

Aesthetic
REJUVENATION

A REGIONAL APPROACH

RANELLA HIRSCH | NEIL SADICK | JOEL L. COHEN

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CHAPTER 1

The Approach to the Cosmetic Patient

Kenneth Beer

INTRODUCTION

When approaching a cosmetic patient, there are many factors to consider. Whether the patient is interested in botulinum toxins, fillers, cosmeceutical advice, lasers, light sources, liposuction, a face-lift, or blepharoplasty, the physician needs to consider the medical condition of the patient, his or her psychologic tolerance for the procedure, the suitability of the procedure for the condition being treated, and patient's financial ability to pay for the procedure.

As the numbers of safe and effective procedures for cosmetic issues continue to increase, so does the demand for these procedures. According to the American Society of Aesthetic Plastic Surgery (ASAPS), approximately 3.2 million people were treated with botulinum toxins in 2006, 1.6 million people received hyaluronic acid fillers, and 2.1 million people were treated with lasers or light sources for resurfacing and hair reduction.¹ These numbers continue to increase and patients who previously had not considered or did not have access to cosmetic procedures find themselves in a physician's office, considering a range of available options.

As more people present for procedures, physicians will encounter more patients with unrealistic expectations, untreated psychopathology, and previous untoward events from prior cosmetic experiences. However, as with any increase in patient visits, there will be a larger number of patients for whom the visit to a physician is a transformative and beneficial experience. Distinguishing between these two types of patients is critical for patient satisfaction and thus a successful cosmetic practice.

The initial interaction with the cosmetic patient should be different from one with a patient coming to the office for a general dermatologic concern. The tone, timing, and appearance of this elective visit must be different from those for the treatment of a medical or surgical condition. Some of these differences will be elaborated in this chapter.

The financial impact of most medical and surgical procedures is mitigated by some type of insurance. This insurance may also dictate the type of treatment provided to the patient depending on guidelines set for the provider. In contrast, for the cosmetic patient there are no

externally mediated guidelines and the financial relationship is a direct one between physician and patient—there is no unseen third party involved. These financial aspects of the relationship must also be considered when treating a cosmetic patient.

INITIAL PATIENT ENCOUNTER

In cosmetic practice, the initial patient encounter occurs long before the physician enters the room. Frequently, the patient makes several decisions regarding his or her treatment prior to meeting the physician. A patient typically first encounters a physician's practice with either a telephone call for an appointment or with a visit to the practice Web site. Unlike a medical dermatology practice where the scarcity of specialists means patients will put up with almost anything to get a visit or a MOHS practice where another dermatologist is deciding where the patient will be treated, the cosmetic patient has a plethora of choices. These range from the sleek-appearing medspa at the local mall (which may be supervised by an inappropriately trained nonspecialist physician or an even less qualified health care professional such as a nurse, PA, aesthetician, or medical assistant).

Typically, patients do not make decisions based on the physician's training, Board Certification, and experience, but rather their subjective experience of the first encounter and the appearance of the office and the physician. This mandates close attention to these aspects of a cosmetic practice.

The phone conversation with a cosmetic patient should begin within three rings. It is important that office staff be educated not to ignore the phone and to limit the patient's time spent on hold. Each of these behaviors projects a sense that the employee is inconvenienced by the patient, and this attitude should not be tolerated by the physician.

Many patients will evaluate the physician and the practice by looking at the office's Web site prior to the visit. For a Web site to be productive, it must clearly state what procedures the practice does and does not offer in its cosmetic repertoire, where it is located (a printable map is helpful to patients), registration paperwork, policies of the office, and any other information that the physician wishes the patient to consider (Figure 1.1).

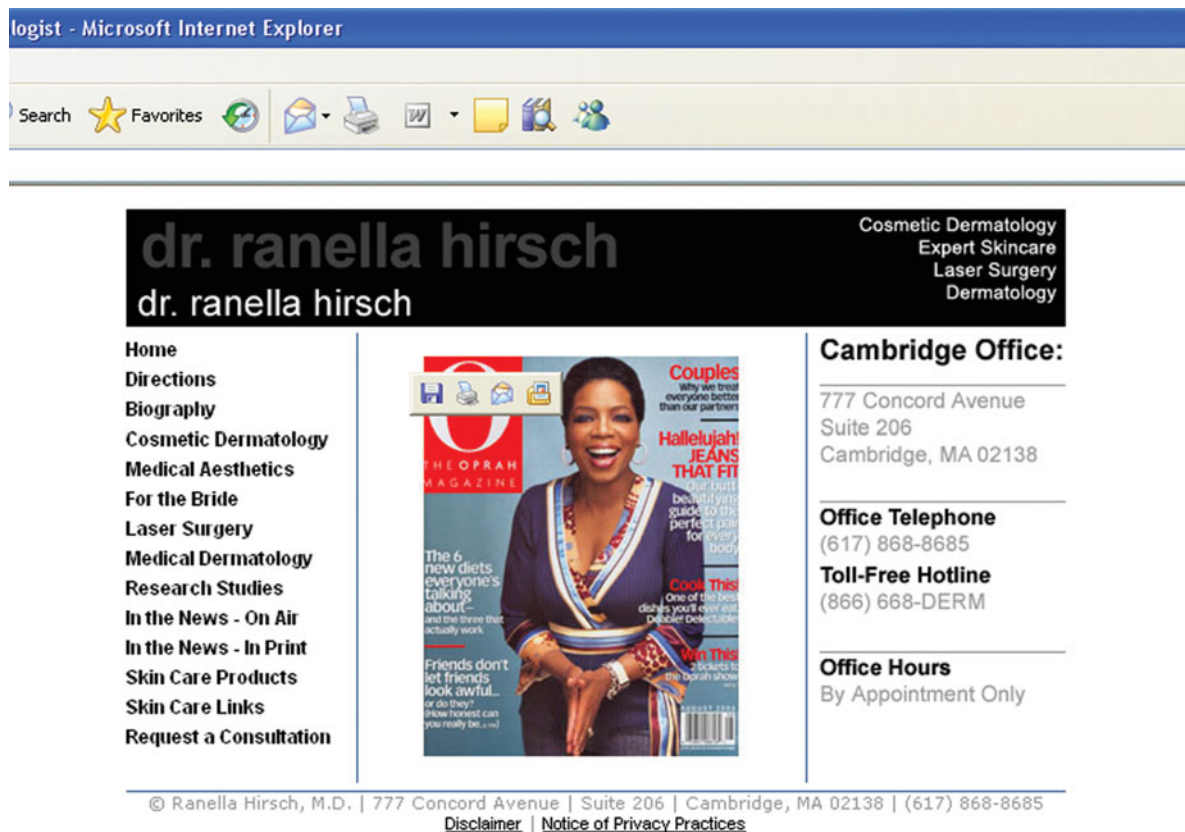


Figure 1.1 A–C A productive Web site for a cosmetic practice includes a menu of services available, a map to the practice, registration paperwork, and office policies

When a patient walks in the door, he or she should be greeted with eye contact and a smile. Nothing conveys a sense of being in the wrong place more than being greeted by the head of a receptionist who is speaking with someone else at the expense of the patient. Although this interaction has nothing to do with the patient's actual treatment, this too has a large impact on a patient's experience and this aspect of the visit should be as controlled as the part that involves the actual procedure.

The patient should be escorted to an examination room in a timely manner (not more than 15 minutes is advised for a cosmetic practice, but in an ideal setting the waiting room time should be limited to 5 minutes). Upon entering the examination room, the patient should be comfortably seated. Once comfortable, a conversation should be initiated with the medical assistant, nurse, or cosmetic coordinator about the patient's goals. During

that time, experienced medical staff will begin to form opinions about the suitability of a particular patient for a specific procedure. Education about the proposed procedure and alternatives to it will also begin during this interval. A good nurse will be able to provide the physician with a great deal of insight about the patient and a good deal of information about the procedure and the physician to the patients. Patients are frequently more candid with staff than they are with the physician and what is discussed during the triage portion of the visit can and should be used to guide the physician's initial interaction.

ACTUAL PHYSICIAN CONSULTATION

When patients select a physician for a cosmetic procedure they are not only selecting the procedure but also the physician. Choosing the physician frequently

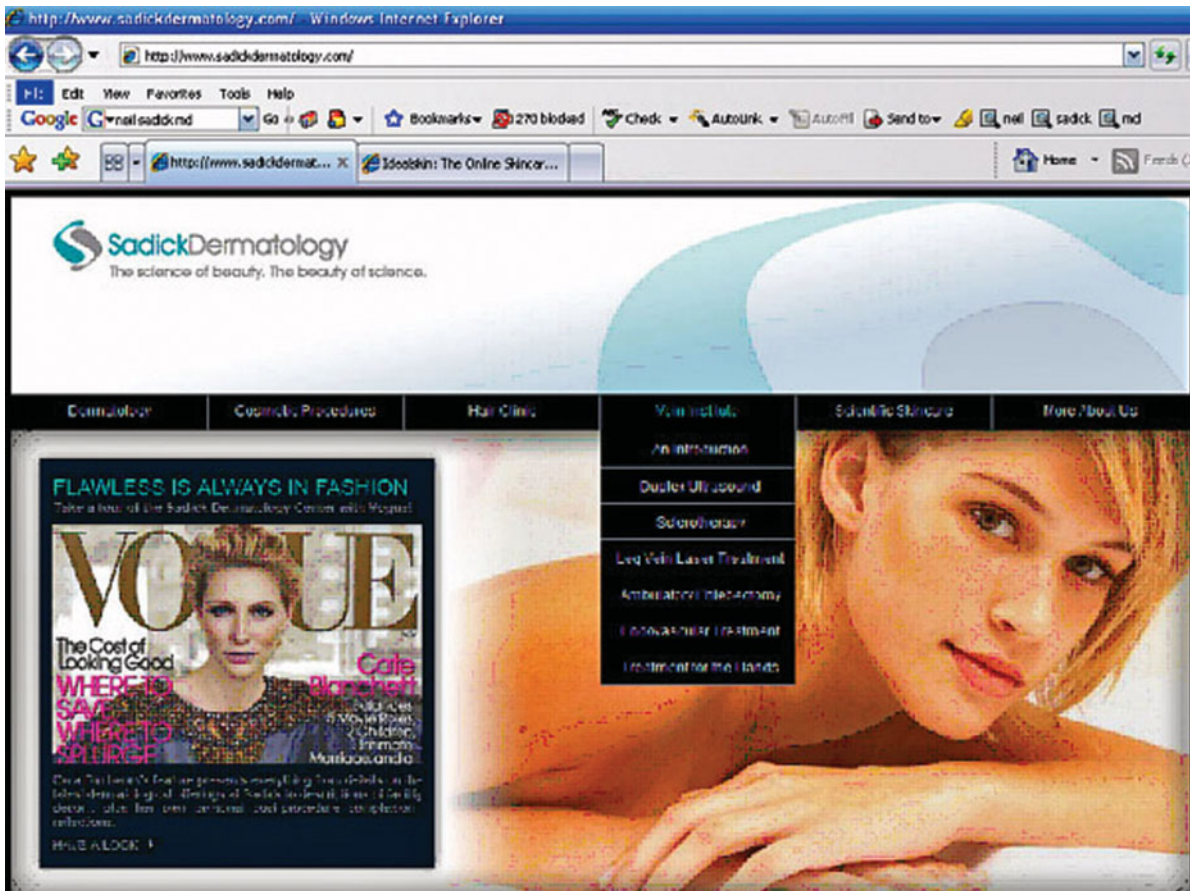


Figure 1.1 A–C (Continued)

depends on factors such as the patient's sense of confidence in the physician and the office staff, the appearance of the office, and the degree to which the patient feels a bond with the physician. This last factor cannot be underestimated and there are several strategies that can help to increase this bond.

The physician should greet the patient by name. There are several methods of beginning a consultation and the physician should try to find the style that fits his or her personality. There is no right or wrong style and patients can usually spot a physician who is trying to act rather than to interact on a personal level. One style that helps to establish a relationship is to use statements such as "Tell me a little about what you would like to change," neutral questions such as "Have you ever had any cosmetic procedures before?" or reviewing the intake form and relating your questions to the areas of interest expressed on the form. Providing the patient with a hand-

held mirror can also evoke a number of previously unmentioned concerns.

A brief review of the patient history form (Figure 1.2) will reveal several salient features about the person's life. Common interests are one potential area for discussion. Many patients presenting for cosmetic procedures will share their educational, social, and economic background to begin a friendly discussion. People typically enjoy discussing how they spend their days, so, if they are employed, it is helpful to discuss what they do for a living. Retired people are happy discussing their activities and other daily events. Most people coming in for cosmetic consultations will have traveled within the past few months and discussing where they went and what they did is a neutral way to begin a dialogue.

Many cosmetic physicians find it helpful to discuss prior cosmetic procedures during the patient consultation. In order to do this, one must integrate this question



Figure 1.1 A-C (Continued)

Dermatology Medical History

Name: _____ Date: _____

Age: _____ Height _____ Weight _____

Are you under a doctor's care? Yes No For what condition? _____

Reason for today's visit _____

Have you ever had dental anesthesia? Yes No Novocaine? Yes No /

Any problem? _____

Have you ever taken a course of Accutane (Isotretinoin)? Yes No

If so, when? _____

Do you have a history of hypertrophic scars/keloids? Yes No

Do you have a history of cold sores? Yes No

If yes, please provide details _____

Pregnancy:

Are you now pregnant? Yes No Are you breast feeding? Yes No

Number of pregnancies? _____ Number of children? _____

Are you sexually active Yes No Are you currently using birth control? Yes No

What type? _____

List any serious or chronic illness:

1. _____ 3. _____ 5. _____

2. _____ 4. _____ 6. _____

List all previous surgery:

1. _____ 3. _____ 5. _____

2. _____ 4. _____ 6. _____

Social History:

Do you drink alcohol? Yes No If YES _____ drinks per day

Do you use IV drugs? Yes No

If YES what? _____ How often? _____

Do you smoke? Yes No Packs per day _____ For how long? _____

Occupation? _____

Skin:

Have you ever had skin cancer? Yes No

Has anyone in your family ever had skin cancer? Yes No

Do you have a history of any specific skin disease? Yes No

If Yes. _____

Do you have problems with healing? Yes No

Do you develop keloids (scars) after surgery? Yes No

Do you bleed easily? Yes No

Figure 1.2 A sample patient history form

Do you have (circle): Dentures	Capped teeth	Bridges	Loose teeth	Chipped teeth	Diseased gums
Contact lenses	Glasses	Hearing aid	Other prosthetic devices: _____		

Important medical conditions: Have you ever had or received treatment for any of the following? (Please circle)

Hepatitis, jaundice, cirrhosis or liver disease?	Yes	No	Blood transfusion?	Yes	No
Asthma, TB, pneumonia	Yes	No	HIV or AIDS?	Yes	No
Bronchitis, emphysema or chest disease?	Yes	No	Anemia or blood disorder?	Yes	No
Heart attack, angina, palpitations, chest pain?	Yes	No	Chronic or recent cough?	Yes	No
Irregular heart beats or heart murmur?	Yes	No	Wheezing?	Yes	No
Shortness of breath or fainting spells?	Yes	No	Alcohol abuse or alcoholism?	Yes	No
Mitral valve prolaps/bacterial endocarditis?	Yes	No	Drug abuse or drug addictions?	Yes	No
Rheumatic fever or congenital heart disease?	Yes	No	Thyroid problems?	Yes	No
High or low blood pressure?	Yes	No	Venereal disease?	Yes	No
Kidney failure, kidney or prostate problems?	Yes	No	Anaphylaxis?	Yes	No
Dialysis?	Yes	No	Stomach ulcers?	Yes	No
Excessive hunger or thirst?	Yes	No	Arthritis/joint deformity?	Yes	No
Amputation?	Yes	No	Arthralgia?	Yes	No
Stomach absorptive disorder?	Yes	No	Limited motion?	Yes	No
Nausea, vomiting, diarrhea or yeast infection when taking antibiotics?	Yes	No	Artificial joint?	Yes	No
Migraines, headaches or chronic head pain?	Yes	No	Epilepsy or seizures?	Yes	No
Lupus, scleroderma or autoimmune disease?	Yes	No	Anorexia or bulimia?	Yes	No
Phlebitis, blood clots or varicose veins?	Yes	No	Nervous breakdown?	Yes	No
Stroke, Bell's palsy or neurological problems?	Yes	No	Personality disorder?	Yes	No
Shingles, cold sores or fever blisters?	Yes	No	Psychological/emotional problems?	Yes	No
Abnormal or excessive bleeding?	Yes	No	Recent weight gain or loss?	Yes	No
Hives, rashes or skin disease?	Yes	No	Anxiety or "panic attacks"?	Yes	No
Diabetes or abnormal "blood sugar"?	Yes	No	X-ray treatments?	Yes	No
Adverse or unusual reaction to anesthesia?	Yes	No	Radiation therapy?	Yes	No
Immune disorder such as vitiligo?	Yes	No	Pacemaker?	Yes	No

Other medical conditions? Explain: _____

Drugs and medicines: Have you, within the last 6 months, taken any of the following? (Please circle)

Accutane?	Yes	No	Anticoagulants or blood thinners?	Yes	No
Homeopathic or herbal medicines?	Yes	No	Pain pills?	Yes	No
Stimulants, appetite suppressants, diet pills?	Yes	No	Birth control pills?	Yes	No
Sedatives, tranquilizers or sleeping pills?	Yes	No	Phen-Phen or Redux?	Yes	No
Antidepressants, antipsychotics or nerve pills?	Yes	No	Recreational or illegal drugs?	Yes	No
Cortisone, prednisone or ACTH?	Yes	No	Asthma meds, inhalers, etc?	Yes	No
Heart medication, Digitalis, Lanoxin?	Yes	No	Diuretics or water pills?	Yes	No
Blood pressure medication?	Yes	No	Nitroglycerine?	Yes	No
Steroids or body building drugs?	Yes	No	Seizure medication?	Yes	No
Headache or migraine medications?	Yes	No	Antibiotics?	Yes	No
Insulin, Orinase or similar drugs?	Yes	No			

Figure 1.2 (Continued)

List medications taken (include prescriptions, over-the-counter meds, vitamins, birth control and herbals):

1. _____ 3. _____ 5. _____
 2. _____ 4. _____ 6. _____

Medications that cause bleeding: Have you taken any of the following in the last 2 weeks? (please circle)

Aspirin or aspirin containing medications?	Yes	No
Vitamin E (excluding multivitamin)?	Yes	No
Anti-inflammatories or muscle relaxants?	Yes	No
Ibuprofen (Motrin, Advil, Nuprin) containing products?	Yes	No
Any products from the health food store?	Yes	No Please specify _____

Allergies and sensitivities: Is there any history of skin reaction or other illnesses following the administration of: (please circle)

Penicillin, sulfa or other antibiotics?	Yes	No	Tetanus toxoid or serum?	Yes	No
Morphine, codeine, Demerol or narcotic?	Yes	No	Tincture of Benzoin?	Yes	No
Novocaine, Lidocaine or other local anesthetic	Yes	No	Adhesive tape?	Yes	No
Iodine, Betadine, chlorhexidine or PhisoHex?	Yes	No	Latex rubber?	Yes	No
Any other drugs/medications or creams?	Yes	No			

If YES, please list and describe the reaction: _____

Is there anything else you would like us to know?

I certify that the above is true and correct. I realize that withholding information about my medical history could result in serious injury to me or harm to those involved in my care. I am aware that providing either false or incomplete information about my medical and surgical history may result in the cancellation of my proposed surgical procedure and also result in forfeiture of my surgical fees.

Patient's Signature

Date

Witness

Figure 1.2 (Continued)

into either the patient intake form or the structured nursing interview. These questions should provide information about what prior procedures were performed and when they were done.

This information can reveal very important data. For instance, patients who have had prior silicone injections may not be candidates for injections of additional fillers if they have had complications with the silicone or if they have had a bad experience with that product. As Dr. David Duffy points out, silicone patients are likely to blame the last person who injected them with silicone if there is a bad outcome and so physicians approaching these patients should discuss and document the patient's impression and physical appearance before injecting them with any substance.² In addition, if a patient has been injected by a less qualified or less reliable source in the past, there is always a need for concern about what precise product was actually injected. Multiple case reports have come forward in recent years of products that were misrepresented to a patient at the time of injection.

Patients who speak poorly of reputable physicians should also be carefully screened during the consultation. In many instances, this type of interaction may be solicited by asking the patient "What did you think of your last treatment?" or "What did you like or dislike about your last experience with X procedure?" This provides numerous insights into the psyche of the potential patient. For instance, one can understand whether the patient is simply shopping for the least expensive provider by listening to a patient, discuss the past providers that they have seen. Experienced cosmetic physicians are attuned to patients who issue scathing reports from a routine side effect such as a bruise and should approach these patients with an increased degree of caution and thoroughly document the review and acceptance of risks with these patients.

Discussions of prior procedures are also an opportunity to manage patient expectations and document the discussion of likely outcomes. Individuals who express dissatisfaction with a static glabellar rhytid following treatment with Botox in this area may be dissatisfied simply because they did not have an adequate understanding of the limitations of toxins without fillers in such a location. Alternatively, they may not be realistic in their expectations or they may be limited in their finances. Each of these situations must be approached differently and the consultation is the time to ferret

out these nuances as well as document these crucial conversations.

DISCUSSION OF FINANCIAL ASPECTS OF A COSMETIC PROCEDURE

A discussion of the financial aspects of a cosmetic consultation is another important aspect of an initial patient interaction. Some patients will expect that payment for an area injected with Botox will encompass ongoing visits throughout the year. Others believe that treating the nasolabial creases with a filler is a fixed-price process, irrespective of the number of syringes involved. Still others have had procedures where they have paid for "a syringe of Botox" that was magically used to treat every rhytid on their face and bring with them the expectation that they can purchase a syringe that will magically perform this miracle from your office as well. Typically, a discussion of the financial aspects of each treatment is taken care of by a member of the office staff rather than the physician. This enables the physician to retain his or her "white coat" and avoid the situation of haggling with the patient over price. The fact that a financial discussion occurred should be documented in as much detail as possible in order to avoid patients who come back saying that she thought the cost of treatment covered them for eternity or for "touch-ups" for glabella covered forehead, crow's feet, and axilla. The written consent form should also clearly and boldly discuss the fact that cosmetic procedures have a fee associated with each additional treatment or procedure so that each person involved in any discussion has the same understanding of what can be expected and whether "touch-ups," if any, are included in initial pricing. Some offices provide a date stamped cost estimate with an expiration, although this is more common with larger surgical procedures such as liposuction. It can be used as a timed incentive to book a procedure.

Although it may at times be uncomfortable to discuss financial aspects, this is also an excellent opportunity for continued education about various products and procedures. For instance, cosmetic physicians will surely encounter patients who have been injected with highly discounted Botox and these patients inevitably want to know "why your Botox costs more." The commoditization of cosmetic procedures is one unfortunate result of the various nonspecialist injectors who presently litter the cosmetic landscape. Fortunately, a discussion of the

substitution of cheaper hyaluronic acids³ or the injection of saline with a few units of Botox or the use of raw toxin by an osteopath in Florida (<http://www.local10.com/news/6426058/detail.html>) helps potential patients to understand the importance of quality rather than price. For those patients unable to grasp this concept, redirection to the Yellow Pages is advisable.

Once the patient's financial limitations have been ascertained, these may present both challenges and opportunities. The challenges that emanate from a disconnect between the creases and the pocketbook are obvious. One should have a candid discussion of these limitations and provide the patient with an honest assessment. In some cases, this may involve telling the patient that one syringe of hyaluronic acid will not make an appreciable difference and that it will not be a good use of their financial resources.

The opportunity presented by financial restrictions stems from your ability to be creative and to understand the patient's focus. For instance, patients needing to have their nasolabial creases filled, their glabella and peri-orbital creases relaxed with botulinum toxins, their perioral lines treated, and their skin resurfaced with lasers, peels, or intense pulsed lights may have a financial limitation of \$1000. In some of these patients, this may mean the use of 25 units of Botox or 75 units of Reloxin to inject their glabella and a small syringe of hyaluronic acid to fill the static component of their glabellar rhytids with any remaining material used to fill the perioral lines. Effacement of the glabellar lines and diminishment of the perioral rhytids can produce a dramatic improvement in the patient's appearance and self-image, and this can be one of the most successful treatments performed. Delivering this type of outcome results in extremely happy patients who can be the best source of referrals for a cosmetic practice.

EVALUATING PATIENTS FOR PERSONALITY DISORDERS IN A MANNER PRACTICAL FOR A COSMETIC CONSULTATION

Body dysmorphic disease is estimated to affect 1% to 2% of the population and is higher in patients interested in cosmetic procedures.⁴ Screening for these patients is frequently recommended and discussed, but the reality of doing so on an ongoing basis is marginal at best. To begin with, few dermatologists or plastic surgeons possess the psychiatric training to assess for this type of

pathology. Those who do may not have the time to perform this assessment and still discuss the proposed procedure if they have a busy practice. Avoiding these patients is a complicated process for most practitioners, but there are a few clues to this type of personality disorder. One is the near constant undertaking of some form of cosmetic enhancement with a lack of long-term satisfaction with any of the procedures performed. Another is the patient who simply does not "feel right" to an experienced member of your staff. This may emanate from an abusive interaction with your receptionist or a discussion with your nurse that raises a red flag. Frequently, patients with dysmorphic disease often reveal themselves during the visit without the need for formal psychological evaluation.

■ Approach to Cosmeceutical Consultations

Perhaps more than any type of consultation, the cosmeceutical consultation is prejudiced by prior experiences that the patient has had. The great majority of patients presenting for cosmeceutical consultation will either be using some type of skin care products or must have used them in the past. Considerations include whether the person will use prescription strength products, whether the physician is dispensing the recommended products in the office, the type of skin the patient has, the monthly budget for products, the number of products that the patient wishes to include in his or her skin care regimen, and the ability and willingness to comply with a regimen. Many of these issues will be addressed in Chapter 3 Cosmeceuticals and it shall suffice for now to state that one should always try to match the patient's tolerance for product use, budget, and goals with the cosmeceuticals either dispensed or recommended. Recommending an expensive, complicated regimen to a patient who has neither the time nor budget for this type of program represents a failure of the physician, and a patient on the receiving end of this will be unlikely to seek additional advice or cosmetic treatments from this practice.

■ Approach to the Cosmetic Patient Receiving Facial Rejuvenation

Once the introduction and initial assessment of the patient is over, a more formal discussion of the proposed

TABLE 1.1 ■ Comparison of Botulinum Type A Toxins Reloxin and Botox

	Reloxin	Botox
Benefits	Quick acting (within days)	Experience of decades
Limitations	Potentially shorter duration	
Mode of action	Inhibits acetylcholine release at neuromuscular junction	Inhibits acetylcholine release at neuromuscular junction
Relative cost		May cost more
Number of treatments typically needed	One	One
Onset of effective results	1–3 d	3–7 d
Expected duration of results	2–4 mo	3–4 mo
Potential complications	Bruising, headaches, ptosis, and asymmetry	Bruising, headaches, ptosis, and asymmetry

treatment should ensue. One of the most productive means of initiating this discussion is to hand the patient a mirror and ask “What would you like to change?” Typically, this opens the door to a discussion about marionette lines, nasolabial creases or the tone and texture of the skin. The patient’s guidance is the most important starting point for a discussion of any proposed treatment. Whether the treatment is with a botulinum toxin, filler, resurfacing modality, skin tightening device, or cosmeceutical, it is important to have some discussion of what the patient can realistically expect.

Botulinum toxin

When discussing treatment with botulinum toxins, it is important to discuss the benefits, limitations, and risks of the products. It is also helpful to discuss how they act, when they begin to act, and the difference between the currently available type A toxins, Reloxin and Botox (Table 1.1). Future additions to the neurotoxin armamentarium must also be discussed along with a timeline for their legal use in the United States. For patients who have not been treated with botulinum toxins, one area that is most easily injected is the glabellar complex and this is a good place to begin discussions, if appropriate. Additional sites such as the periorbital region, platysma, mentalis, perioral region, and frontalis may also be discussed during the initial consultation, and during the consult it is possible to get a sense of the patient’s interest level in each. The consultation is also the appropriate time to discuss potential complications such as bruising and ptosis to learn whether or not a treatment during the visit is practical. For some patients, impor-

tant social or business events preclude the possibility of injections.

Patients who have not been treated in your office should be advised of your office policies with respect to treatment with botulinum toxins. For instance, the need to return for touch-up treatments, if needed, should be discussed as should the frequency of return visits for injections. Many experienced injectors bring first-time patients back to their office for follow-up photography after the first visit. This also enables the physician to solidify the relationship with the patient and to discuss any concerns or questions after the procedure. Touch-up treatments with toxins should be mentioned during the consultation. There is considerable variation in this area. One approach that is useful is to mention the fact that treatments can be “enhanced” (rather than touched up) if there is a need to do so but that since the patient’s time is valuable it will be left for the patient to call the nurse or physician in the event that they feel that such an enhancement is required. This approach has the advantage of letting the patient know that you are proficient in your injection techniques and do not typically require touch-up visits. Whether or not one charges for the enhancement depends on the individual situation. When one is treating several areas for a patient who has an asymmetric brow as a result of an injection, it behooves the physician not to charge for the enhancement. At the other extreme is the patient who pays for one area such as the glabella but then returns complaining that the frontalis (which was not treated) now has wrinkles. This is a key reason why photographs and consent, as well as documentation of

TABLE 1.2 ■ Comparison of Botulinum Toxin Versus Fillers

	Botulinum Toxin	Fillers
Benefits	Reliable result, consistent patient satisfaction, low cost of entry for patients	Longer duration than toxins; significant ability to reshape some of the major cosmetic areas including the nasolabial crease, marionette lines
Limitations	Not good for static rhytids, limited applications in the lower and mid face	Cost may prohibit injection of volume needed for full correction, technically challenging for lips and other areas, lumps and bumps may be unsettling for patients
Relative cost	Low	Depends on the amount used
Number of treatments typically needed	One per quarter	Two per year
Onset of effective results	2–7 d	Immediate
Expected duration of results	3 mo	Months to years, depending on the filler used
Potential complications	Ptosis	Necrosis of skin, blindness
Contraindications	Pregnancy, neuromuscular diseases, aminoglycoside use	Allergy to any of the filler ingredients, pregnancy because of liability concern

discussed treatments, are an essential part of the initial consultation.

Fillers

The approach to a patient considering treatment with a soft tissue augmentation product is significantly different than the approach to a patient getting botulinum toxins. As many fillers are more persistent than presently available toxins the patient needs to understand that he or she will be living with the out come of the procedure for a longer time. In addition, the complication profile for fillers is different and this must also be reviewed during the consultation (Table 1.2).

As with other cosmetic procedures, beginning the approach to patients interested in soft tissue augmentation with an open-ended question regarding what area or areas the patient would like to change is useful as is observing the patient point out areas he or she would like to change using a hand-held mirror. Many patients interested in these procedures are concerned about the nasolabial creases, marionette lines, and lip augmentation. Although these are the most frequent areas requested, the consultation is also an opportunity for the physician to discuss other locations that are appropriate for the

individual. These may include the glabella (usually but not always in conjunction with botulinum toxins), the tear troughs, the temples, the mental crease, the zygomatic arch, and others as appropriate.

After the locations of interest have been discussed, the various materials available for injection should be discussed (Table 1.3). Filler selection is based on the budget of the patient, the areas being treated, tolerance for risk, desire for duration, compliance with needed follow-up care, thickness of the skin, the skill and experience of the physician with the various products under consideration, and several other factors. During consultation, patients will identify the areas they would like to be treated. This imposes some restrictions on the filler that can be injected. For instance, if the glabella is being injected, cross-linked collagen products such as Cosmoplast and Zyplast should not be injected (as per the package insert). Thick-skinned patients desiring long-term correction of the nasolabial creases, marionette lines, or augmentation of the zygomatic arch may be candidates for injections with calcium hydroxylapatite (Radiesse) and the consultation should include discussion of the specifics of this material. For patients interested in volume replacement of the cheeks or temples or other areas that would benefit from collagen stimulation, injections with poly-L-lactic acid

TABLE 1.3 ■ Comparison of Available Fillers

	Restylane	Juvederm	Perlane
Benefits	Reliable molecule with “stiffness” that is great for structure	Smooth, homogenous gel that is great for lips, thin-skinned patients and tear troughs	Large particle size that is good for structural support
Limitations	May be lumpy if placed superficially	May not provide as much structural support	May be too thick for some thin-skinned patients or some areas

(Sculptra) should be discussed. The need for multiple injection sessions, potential for complications, and posttreatment care must be emphasized.

The vast majority of patients in the United States undergoing soft tissue augmentation will receive hyaluronic acid and, as such, many consultations will be focused on this material. During the course of the consultation, the various types of products available should be discussed. Differences between Restylane, Perlane, Juvederm, and some of the newer products should be mentioned because, while patients typically know something about each, this information may or may not be accurate depending on where it originated. Sometimes the information derived from newspapers or Web sites accurately portray the differences between the products, and the consultation may follow a logical progression from the patient's questions to the physician's discussion of its suitability for a specific case and physician's experience with the product. In other instances, the information comes from less reliable sources such as the media, friends, and direct-to-consumer marketing. Newer hyalurons including those containing anesthetic or that are cross-linked differently should be considered during the consultation.

Laser and light treatments

The approach to a patient considering laser or intense pulsed light treatment is markedly different from that for a patient seeking therapy with injectables. While most injections are discrete treatments, most of the laser and intense pulsed light treatments are planned as a series. This means that the approach to the patient needs to be geared toward a conversation about a gradual change in appearance, which necessitates multiple visits. There may also be a larger degree of patient participation for light-based treatments and sun avoidance, the use of bleaching creams (if indicated), and other behavioral

changes should be included during the consultation. As with other procedures, the costs associated should be discussed but many physicians find it helpful to offer a package price for a series of treatments. This not only offers the patient a discount over the price for individual treatments but also commits the patient to seeing the entire course of treatment to completion.

Many more know about radiofrequency treatments that were much hyped, but failed to deliver any appreciable results (for the patient) and this type of baggage must be dealt with during the consultation.

Deep infrared treatments for the purpose of skin tightening are becoming increasingly popular and patients considering this treatment require specific information during their consultation. Many patients will want to undergo infrared treatments for either skin tightening or to attempt treatment of fat or cellulite. The approach to patients interested in these indications must first address the degree to which the procedure will or will not accomplish the desired goals. Complications, costs, time requirements, and other considerations unique to the procedure must also be included (Table 1.4).

■ Approach to the Patient Requiring Eye Rejuvenation

The approach to the patient requiring eye rejuvenation requires the surgeon to have a thorough understanding of both surgical and nonsurgical modalities. For comprehensive eye rejuvenation, blepharoplasty with additional procedures such as laser resurfacing and botulinum toxin injections may be required. During the course of the patient evaluation, it is important to determine what aspects of the eye area are of concern to the patient and to find out whether the patient understands the proposed

TABLE 1.4 ■ Characteristics of Infrared Skin Tightening to Discuss with Potential Patients

	Infrared Skin Tightening Treatments
Benefits	May tighten skin by stimulating and tightening collagen fibers
Limitations	Efficacy, to date, has been suboptimal
Mode of action	Heating of deep dermal tissue
Relative cost	Moderate
Number of treatments needed	4–6
Duration of results	Months to years.
Potential complications	Burns, scarring, hyperpigmentation

procedures. For instance, if the patient needs a blepharoplasty but constantly states that he or she does not want surgery, the consultation must focus on the limitations of a nonsurgical approach. If the patient agrees with these limitations and maintains reluctance to have a surgical procedure, it is essential to document this so that any limitations imposed by these restrictions are understood prior to the procedure.

Surgical approaches to periorbital rejuvenation entail risks that, although not significant, are different from nonsurgical approaches. These too must be discussed during the patient evaluation. Asymmetry, ptosis, visible scarring, and other potential issues must be mentioned during the discussion.

■ Approach to the Patient Requiring Neck Rejuvenation

Patients requiring neck rejuvenation may benefit from several procedures performed alone or in conjunction with each other. These may include surgical plication of the platysmal bands, liposuction, botulinum toxin injections, intense pulsed light, infrared skin tightening, rhytidectomy, and treatment with cosmeceutical and prescription preparations. The approach to a given patient depends on which of these will best accomplish the goals desired.

As with consultations for other areas, it is essential to photograph the area prior to treatment and to document any discussions about proposed procedures. For patients with thin, stringy necks because of platysmal banding, the consultation may focus on treatment with botulinum toxins. Patients with lax skin will most likely require rhytidectomy, whereas those with excessive fat in the area will do well with liposuction. Mild to moderate

poikiloderma of the neck may be treated with combinations of light-based treatments, including intense pulsed light, as well as with retinoids. Discussions with the patient should involve the use of a mirror to point out the various areas that can be treated and enable the patient to visualize what can and cannot be accomplished.

■ Approach to the Patient Requiring Abdominal Rejuvenation

Abdominal rejuvenation may include abdominoplasty, liposuction, and/or the use of an energy device such as infrared treatment. When discussing rejuvenation for this area, it is essential to understand the goals of the patient as well as any limitations that are imposed by your skills, training, and experience. For instance, if the patient requires an abdominoplasty to get the best results but you are capable of performing only liposuction, it is best to advise the patient of this and refer to a colleague who can assist the patient. When discussing abdominal rejuvenation, one needs to consider whether the patient has undergone a weight change recently thus producing an abnormal appearance of the area. If a patient has just had a significant weight change (such as following pregnancy or weight reduction surgery), it is best to defer any treatment for a period of time, which may be as long as a year, in order to ascertain whether the condition that mandated the rejuvenation procedure is stable or transient. It is also a good idea to obtain clearance for the procedure if the condition is the result of a medical or surgical condition. Additionally, the approach requires knowledge of abnormalities in the area such as abdominal or inguinal hernia, which might create significant risks for procedures such as liposuction.

Patients in whom the texture of the abdomen is an issue may do well with infrared, radiofrequency, or ultrasound energy. In these individuals, the consultation should include a realistic discussion of the devices contemplated as well as the risks and time requirements of each. As with other areas and devices, patients will have a great deal of information and misinformation about treatments for the abdomen and the consultation should try to address both.

■ Arm and Hand Rejuvenation

Advances in facial rejuvenation have sparked an interest in other areas of the body. Since the contrast between a rejuvenated face and an old pair of hands is a stark one, many patients are now interested in what can be done to the hands and arms. The approach to patients interested in hand and arm rejuvenation mirrors that taken for other areas and it involves surgery, injectables, use of light-based devices, and cosmeceuticals. Surgical rejuvenation for the arms and hands may involve removal of excessive fat and skin from the posterior upper arm or it may require vein removal from the posterior hands. Discussion of these procedures must include the risks of each as well as the location and extent of any scars. In addition, postoperative potential discomfort must be mentioned (Table 1.5) Nonsurgical rejuvenation of the hands may include the use of poly-L-lactic acid, hyaluronic acid, or other filling agents to replace lost volume from the dorsum of the hands or it may necessitate treatment with intense pulsed lights, lasers, or chemical peels to improve the appearance of dyschromias on the sun-exposed dorsal hands. Consultation regarding nonsurgical renovation should include a realistic assess-

ment of the expected improvement and an educated estimate of the number of visits required to achieve this result.

■ Leg Rejuvenation

Patients seeking cosmetic consultations regarding leg rejuvenation typically are interested in laser treatment sclerotherapy, liposuction, or surgical reduction of the thigh area. As with rejuvenation of the abdomen, it is important to recognize the different indications for each. For patients presenting with unwanted leg veins, it is important to discuss the various modalities available including sclerotherapy, laser, endovenous ablation, and surgical as well as the imaging required to locate vascular abnormalities such as incompetent perforators. Candidates for liposuction of the thighs, knees, ankles, or calves should undergo the same type of consultation as other liposuction patients and it should include the risks of the procedure, expected improvement following the procedure, and the need to wear garments postoperatively. Radiofrequency, ultrasound, or infrared rejuvenation consultations should discuss the degree of success that the device in question has attained (both in peer-reviewed publications and in one's own practice), the risks of the procedure, and the expected number of visits required to accomplish the goals of the rejuvenation program.

■ Breast Rejuvenation

At the present time, breast rejuvenation is primarily surgical and the approach to the patient requiring this type of procedure is one that should involve consultation with

TABLE 1.5 ■ Risks of Laser and Light Treatments Depending on Skin Type, Amount of Energy Used, and Pre- and Postoperative Ultraviolet Exposure			
Location of Proposed Rejuvenation with Lasers or Light Sources	Risk of Hyperpigmentation	Risk of Scarring	Risk of Hypopigmentation
Face	Low; depends on skin type, sun exposure, and settings	Low; depends on skin type, sun exposure, and settings	Low; depends on skin type, sun exposure, and settings
Neck	Moderate	Moderate	Moderate
Chest	Moderate	Moderate	Moderate
Hands	Moderate	Moderate	Moderate

a plastic surgeon. Dermatologic surgeons are beginning to treat the breast area with Botox in an effort to counter the effects of gravity. Breast tissue is also being injected with soft tissue augmentation products in an effort to evert nipples as well as to correct some asymmetries and minimize scars from surgical procedures in this area. Previous efforts involving ablative laser treatments of the breast to tighten collagen have been unimpressive.

CONCLUSION

Perhaps as important as the product or laser used to treat the patient or the technical prowess of the dermatologist or plastic surgeon is the approach one has to the patient. It is critical to listen to the patient's goals and concerns and to assess whether the patient has a realistic chance of being satisfied with your treatment. It is also important to realize that into any cosmetic office on any given day, a patient with unrealistic expectations or body dysmorphic disease will present for treatment. In many instances,

these people are not going to be happy and one should consider not treating them.

Cosmetic surgery can be rewarding and the patients one interacts with are typically fun to work with. The approach that one has with these patients is critical as it sets the stage for all that follows.

REFERENCES

1. American Society for Aesthetic Plastic Surgery. *Statistical Data*. New York, NY: American Society for Aesthetic Plastic Surgery; 2006.
2. Duffy, DM. Liquid silicone for soft tissue augmentation. *Dermatol Surg*. 2005;31(4):1530–1541. doi:10.2310/6350.2005.31238.
3. Beer, K. The use of one soft-tissue augmentation in place of another: repercussions of product substitution in the aesthetic market. *Cosmet Dermatol*. 2007.
4. Mackley, C. Body dysmorphic disorder. *Dermatol Surg*. 2005;31(5):553–558.

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CHAPTER 2

Structural Approach to Aesthetic Rejuvenation

Neil Sadick

INTRODUCTION

As multiple new technologies are introduced into the aesthetic arena on an almost daily basis, the most appropriate use of these technologies remains a challenge to practicing cosmetic physicians. It is important during the initial patient consultation to understand what the major concerns are of the patient so that appropriate modalities may be chosen eventuating in optimal patient outcomes.

The common goals of all aesthetic rejuvenation programs are listed in Table 2.1. These include attention to reducing cutaneous rhytides as part of the aging process and should be included in any structural aesthetic rejuvenation program. Homogenous skin pigmentation, as related to vascularity, as well as increased or decreased melanin content are also of importance. Patients also like smooth and tight skin. Technologies that shrink sebaceous glands and induce both tightening and synthesis of new collagen are able to accomplish these goals.

Finally, volume replacement is increasingly being recognized and addressed, which may be accomplished through multiple modalities, including denervation of hyperkinetic muscle tone and judicious use of fillers.

STRATEGIES FOR REJUVENATION

Strategies for rejuvenation involve common principles in whole body rejuvenation programs (see Table 2.2). These

include cell protection, cell turnover, chromophore targeting, cell stimulation, tissue tightening, and volume depletion.

■ Cell Protection

Protection of the skin against environmental and other stresses associated with photoaging may be ameliorated by using appropriate agents having broad spectrum activity against short-wave UVA and long-wave UVA rays. In addition, the application of a high-dose antioxidant such as idebenone, myebenol, vitamin C, or vitamin E may also provide additional cell protection when applied daily.

■ Cell Turnover

Rejuvenation programs should also strive to increase epidermal cellular turnover. Various cosmeceutical approaches including superficial chemical peeling agents, microdermabrasion devices, and related technologies may help achieve this goal.

■ Chromophore Targeting

Achieving homogeneity of vascular and pigment targets is an integral part of any rejuvenation program. Topical bleaching agents containing 3% to 4% hydroquinone applied at home daily with appropriate photoprotection may play an important role in this setting. In addition, KTP lasers, pulsed dye lasers, and Nd-YAG laser may improve vascular aberrations. Intense pulsed light sources provide the greatest degree of global rejuvenation in terms of pigmentation, vascularity, and skin texture improvement. Q-switched lasers (ruby, alexandrite,

TABLE 2.1 ■ Goals of Aesthetic Rejuvenation Programs

- Skin toning
- Decreased wrinkling
- Homogenous vascularity
- Decreased skin pigmentation
- Improved skin smoothing
- Volume replacement
- Skin tightening

TABLE 2.2 ■ Strategies for Rejuvenation

- Cell protection
- Cell turnover
- Chromophore targeting
- Cell stimulation
- Tissue tightening
- Volume repletion

red Nd-YAG) and fractional photothermolysis technologies have proven the greatest efficacy in terms of pigmentary dyschromia.

■ Cell Stimulation

Increasing fibroblast activity in order to induce dermal remodeling and subsequent new collagen production is important for improvement of rhytides. Ablative and non-ablative lasers, infrared light sources, and fractional photothermolytic devices may help to accomplish these goals. At-home application of a nightly retinoid may also be of benefit in this regard.

■ Tissue Tightening

Tight, firm skin may be achieved by deep dermal and subcutaneous heating, inducing collagen tightening, and deep volumetric remodeling. Broadband near infrared and infrared light sources as well as deeply penetrating radiofrequency technologies are commonly employed in this regard.

■ Volume Depletion

It is well-known that volume loss is a major target of the aging process. Toxins that induce neuromuscular blockage as well as various autologous and synthetic filler agents play an important role in this regard to complete an all-encompassing, noninvasive rejuvenation program.

TABLE 2.3 ■ Aesthetic Approaches

Microdermabrasion
Chemical peels
Light emitting diodes (LED)
Intense pulsed light sources (IPL)
Lasers
Radiofrequency devices
Ultrasound technologies
Toxins
Filler agents

AESTHETIC APPROACHES

The ever-expanding armentarium of technologies available to the practicing noninvasive aesthetic physician is outlined in Table 2.3. These technologies and devices accomplish all of the common aesthetic goals outlined in the previous section.

■ A Novel Classification System of Photoaging

Table 2.4 shows a novel classification system of photoaging based upon the pathophysiology of the aging process as it affects all layers of skin, muscle, and tone. This system allows the practicing aesthetic physician, once he understands particular patients concerns, to develop a rational treatment plan by using appropriate technologies that address these issues.

TABLE 2.4 ■ Structural Classification System of Photoaging (Sadick)

	Clinical Manifestations
Type I—Epidermal, superficial dermal	
Epidermal turnover	Hyper-, hypopigmentation
Chromophore targeting	Telangiectasias, flushing
Hemoglobin	Rough, wide pored skin
Melanin	
Pilosebaceous aberrations	
Type II—Pandermal	Rhytides
Decreased collagen	
Decreased elastin	
Decreased ground substances	
Type III—Subcutaneous, muscle, bone	Skin folds/furrows
Skin laxity	Lipoatrophy
Loss and redistribution of fat, muscle, and bony structures	

■ Type I Rejuvenation

Type 1 photoaging involves epidermal and superficial dermal structures (Table 2.5). The clinical issues associated with these structures include pigmentary and vascular abnormalities as well as pilosebaceous aberrations. The clinical manifestations noted in this setting include, both hyper- and hypopigmentation, telangiectasias, rosacea, and rough, wide-pored skin.

These problems may be addressed in a systematic fashion. Both superficial dermal peels as well as microdermabrasion devices and related technology may induce epidermal turnover. Patients relate this to a feeling of smoother skin. This is accomplished by stripping the basal and granular layers of the skin. Maintenance of this effect requires repeat treatment every 1 to 2 months. Superficial dermal peeling with α -hydroxy and β -hydroxy acid between 40% and 70% or Jessner's solution may accomplish similar goals.

Telangiectasias and flushing syndromes may be addressed by hemoglobin targeting light sources, i.e., KTP lasers (532 nm), pulsed dye lasers (585–600 nm), as well as broadband intense pulsed light sources (IPLs) (500–1200 nm) may accomplish this goal (Figure 2.1).

Pigment dyschromias may include both hyper- and hypopigmentation. Hyperpigmentation may be in the form of discrete lesions, i.e., ephelides, lentigos, or diffuse in nature (melasma or postinflammatory hyperpigmenta-

tion). This may be addressed by any of the Q-switched lasers, i.e., ruby (694 nm), alexandrite (755 nm), or Nd:YAG (1064 nm) technologies. This may be carried out in conjunction with 3% to 4% hydroquinone applied at home daily. Hypopigmentation may be addressed by either excimer laser (308 nm) or targeted phototherapy technologies, which combine both UVB as well as UVA to induce repigmentation of both zones of hypopigmentation and white striae (Figure 2.2).

Rough, wide-pored skin may be associated with sebaceous hyperplasia. This represents the third part of the type I rejuvenation triad. Technologies that temporarily shrink sebaceous glands, such as intense pulsed light technologies, as well as microdermabrasion treatments are helpful in this regard (Figure 2.3). Intense pulsed light sources give the greatest degree of global rejuvenation improvement in Type I photoaging. This is based upon hemoglobin targeting, melanin disruption, and pilosebaceous shrinkage. There is some degree of fibroblast activation as well, which may relate to improvement of fine lines. (Figure 2.4). In addition a photodynamic therapy emphasizing a photodynamic sensitivity, such as D amino 5 levulinic acid (DUSA pharmaceuticals, Worthington, MA), may act to bolster the effects or decrease the number of treatment sessions when IPL treatments are employed for Type I photorejuvenation (Figures 2.5 and 2.6). This is particularly helpful in the more severe photoaged individual who

TABLE 2.5 ■ Type I Photoaging

Goal	Treatment Modalities
Epidermal turnover	Chemical peels Microdermabrasion Aquabrasion Low energy laserabrasion
Chromophore targeting	Intense pulsed light + RF (500–1200 nm) KTP Lasers (523 nm) Pulsed dye lasers (585–600 nm) Q Switched lasers (694, 755, 1064 nm) Fractional photothermolysis devices (1500–1600 nm)
Skin smoothing	Light emitting diodes (LED) Intense pulsed light + RF (500–1200 nm) Infrared/near infrared lasers (1000–1600 nm) Photodynamic therapy (PDT)

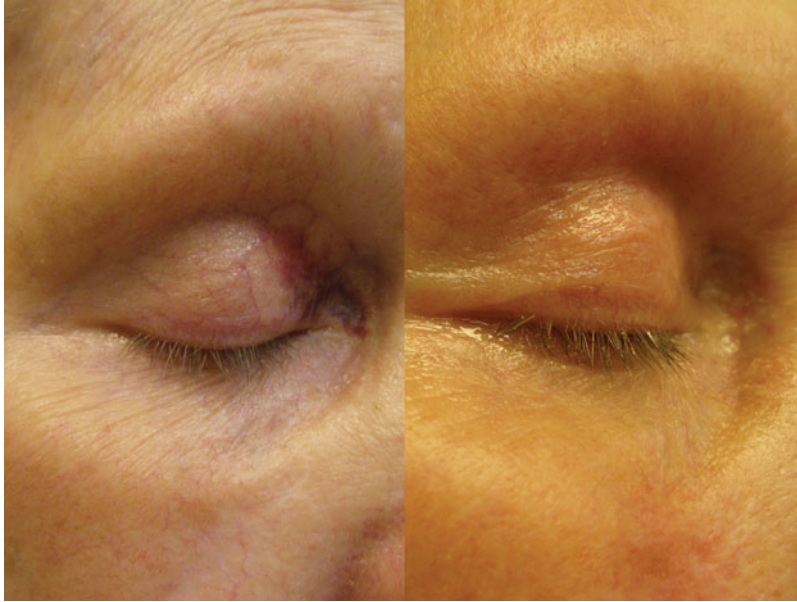


Figure 2.1 Pre- and post-KTP laser (532 nm) treatment of facial telangiectasias (1–5 mm spot 12 J/cm²; pulse duration 20 msec)



Figure 2.2 Pre- and postfractional thermolysis (Fraxel) treatment after diffuse facial hyperpigmentation 8 J/cm² 250 MHz, two passes



Figure 2.3 Combination IPL/RF pre- and post-pilosebaceous complex skin with sebaceous hyperplasia and wide pores. Light 20 J/cm² RF 25J/cm³, three passes

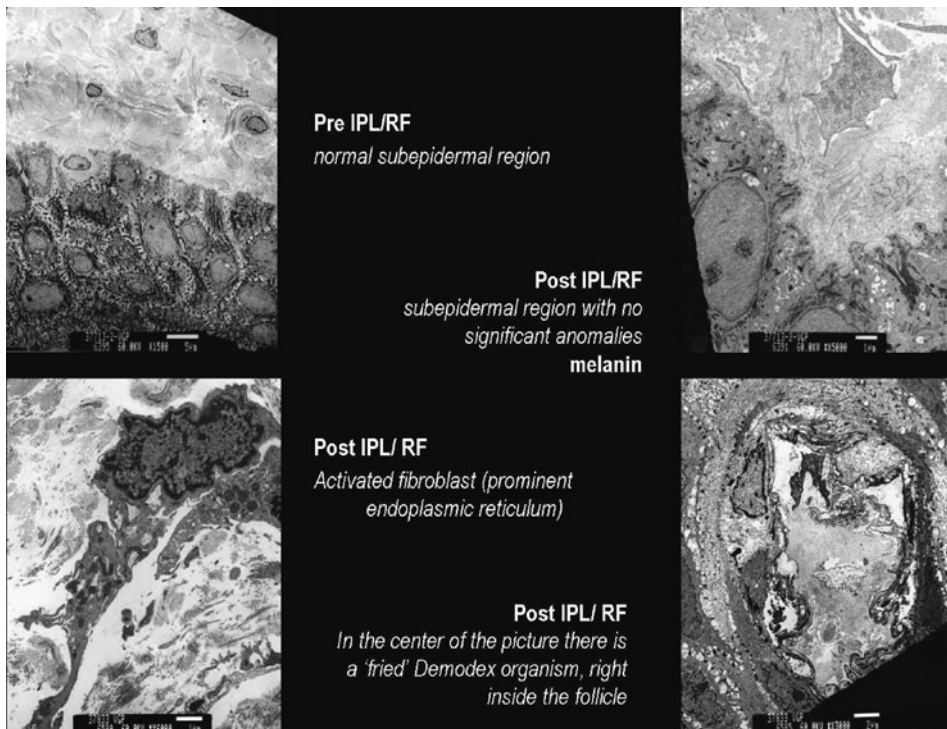


Figure 2.4 Electron micrographs showing changes noted with intense pulsed light and radiofrequency sources for type I photorejuvenation effecting melanin, fibroblast activity, and sebaceous gland shrinkage, which are purported to be responsible for global rejuvenation changes as noted in this setting



Figure 2.5 Application of Levolan for 15 to 30 minutes prior to LED or IPL treatment of type I photoaged skin may boost the effect of these light sources



Figure 2.6 Combined IPL/RF pre- and postsevere Fitzpatrick type IV photoaged skin with multiple active keratoses, light 19 J/cm^2 RF 18 J/cm^2 pulse short, two treatment sessions



Figure 2.7 Pre- and post-Omnilux & ALA treatment combination type I photoaging plus acne with PDT Levulan—1-hour application Red light 633 nm, 10 minutes. Blue light 415 nm, 10 minutes. One treatment session, follow-up 1 month

may also have associated multiple preskin cancers such as actinic keratosis.

Finally, skin toning, which may improve skin texture and surface characteristics, may be achieved by a nonthermal indirect biomodulation effect. These light-emitting diode (LED) light sources are used as maintenance arms of Type I photorejuvenation programs when performed at monthly intervals (Figure 2.7). This technology may employ blue light, which in high doses is reported to diminish propionobacterium acnes on the skin, as well as red and near infrared sources, which may have an anti-inflammatory effect as well. In combination with photodynamic therapy, they can be very useful in providing Type I rejuvenation in conjunction with treatment of mild to moderate acne vulgaris.

■ Type II Rejuvenation

Type II photoaging is involved with pandermal changes in collagen, elastin, and ground substance. The major clinical manifestation noted in this setting is rhytid formation. There are multiple lasers, ablative, minimally ablative,

and nonablative, that are able to accomplish the goal of rhytid reduction. In addition, both medium as well as deep chemical wounding agents can similarly help in this regard.

Wrinkle correction may be achieved in a nonablative manner with dermal heating technologies, such as combination diode lasers/radiofrequency sources. Infrared or near infrared lasers with wavelengths varying from 1064 to 1540 nm. A series of three to five treatment sessions are usually indicated resulting in immediate heating and collagen contraction with wrinkle reduction effects in the longer-term (up to a year) (Figure 2.8).

Fractional photothermolysis technologies, which cause deep microthermal zones of heating separated by normal areas, have been advocated in this setting with mixed results. Newer microablative erbium and CO₂ fractional systems may improve these results. However, these technologies do produce significant efficacy in those patients who suffer from acne scarring (Figure 2.9). These technologies can be used for whole body rejuvenation as well as face and hand (Figure 2.10) rejuvenation.



Figure 2.8 Pre- and post-1320-nm Nd:YAG laser treatment of rhytides; 18 J/cm², pre- and postcooling 30 msec, five sessions



Figure 2.9 Fractional photothermolysis has gained increasing popularity for treatment of acne scars, rough skin, and hyperpigmentation



Figure 2.10 Pre- and postutilization of a 1320-nm Nd:YAG laser for hand rejuvenation. Energy 13 J/cm²; pre- and postcooling 30 msec, six sessions

Ablative laser procedures are also helpful in Type II rejuvenation, particularly in those individuals with more advanced photoaging. The systems include long-pulsed erbium (2940 nm) technologies and CO₂ (10,600 nm). These technologies are associated with downtime, a prolonged wound healing response, and relatively high side-effect profile. For these reasons, more conservative single pass regimens or multiple low-energy treatment protocols have gained increased popularity (Figure 2.11).

A new procedure that keeps the epidermis intact while producing deep heating is plasma cell technology. This novel approach has also been shown to be helpful for the management of moderate deep rhytides. It also improves skin tone, texture, and elasticity and at the same time produces some degree of skin tightening. It, similar to the ablative lasers, may be used in the low energy, multiple treatment paradigms or a high-energy single treatment mode (Figure 2.12).

Finally, medium-depth chemical peels with 25% to 40% TCA or nonoccluded phenol 88% are helpful in the management of moderate to severe rhytides, while deeper Baker's phenol peels and dermabrasion are most often



Figure 2.11 Low-energy multiple treatment, long-pulsed erbium pre- and postresurfacing with the Sciton Profile (Woodsville, CA); 5-mm spot 30, microdepth 7.25 J/cm², three treatment sessions



Figure 2.12 Pre- and post-plasma cell resurfacing. Single treatment, two passes at 2 J/cm²

TABLE 2.6 ■ Type II Photoaging

Goal	Treatment Modalities
Collagen stimulation	Diode laser + RF (815 nm)
Dermal remodeling	Nd:YAG laser (1064 nm)
	Infrared laser (1319, 1320, 1450 nm)
	Erbium glass lasers (1540 nm)
	Fractional photothermolysis (1550 nm)
	Long pulsed erbium laser (2940 nm)
	CO ₂ laser (10,600 nm)
	Plasma cell resurfacing
	Medium/deep chemical peel
	Dermabrasion

used in more severe cases of Type II photoaging. Long wound healing postoperative courses as well as long-term hypopigmentation have limited the popularity of these technologies in clinical practice.

■ Type III Rejuvenation

Type III rejuvenation involves deeper structures (Table 2.7). It has recently been shown that aging involves subcu-

taneous tissue, muscle, and bone. The major clinical manifestations noted in this setting include skin laxity, loss and redistribution of fat, muscle and bone, and contour irregularities.

Skin tightening may be accomplished in a noninvasive fashion by using various broad spectrum flashlamp and near infrared as well as infrared light sources and radiofrequency technologies. Examples of these technologies include the Titan (Cutera, Brisbane, CA), Accent



Figure 2.13 Pre- and post-three treatments with Titan (broad spectrum infrared light sources); 32 J/cm², three passes



Figure 2.14 Pre- and posttreatment with Refirme (infrared light sources plus bipolar radiofrequency device). Three treatments. Fluence 120 J/cm^2

TABLE 2.7 ■ Type III Photoaging

Problem	Treatment Modalities
Skin laxity	Broad spectrum flashlamps (700–2000 nm)
Loss and redistribution of fat, muscle, and bone	Infrared lasers/radiofrequency technologies
Contour irregularities	Ultrasound technologies
	Autologous fat
	Toxins
	Fillers
	Threadlifts

(Alma Lasers, Ft. Lauderdale, FL), ReFirm (Syneron, Richmond Hill, Ontario), and ThermoCool. High-energy radiofrequency technologies such as the Thermage (Hollywood, CA) usually require one treatment session, while light sources or combination light/radiofrequency technologies usually require three to five treatment sessions spaced monthly. The lower third of the face and neck appears to respond best. These technologies are also used for abdominal as well as upper and lower extremity tightening in selected cases (Figures 2.13 to 2.15).



Figure 2.15 Pre- and posttreatment with ThermoCool. Setting 354, three passes, single treatment session



Figure 2.16 Pre- and posttreatment with VelaSmooth for cellulite and body contouring (700–2000 nm) infrared pulse



Figure 2.17 Pre- and post-Botox. 60 units; forehead, interglabellar, and eyes

Newer technologies such as the VelaSmooth (Syneron, Richmond Hill, Ontario Canada) and TriActive (Cynosure, Westford, MA) may also be used for cellulite and body contouring. The technologies incorporate diode laser, infrared light, bipolar radiofrequency, and suction massage (Figure 2.16).

Newer focused ultrasound technologies now are being developed with adipocyte contouring and tissue tightening capabilities. Other ways to improve volume deficits and contour irregularities with associated deep wrinkling are the botulinum toxins and filler products. Toxins by decreasing hyperkinetic muscle activity are excellent for amelioration of deep rhytides at the upper face and eyes but also may be used in the upper face and neck where they produce more subtle results.

Fillers are also used to achieve volume repletion as well as wrinkle reduction, contour improvement, and lip augmentation. The collagens (Zyderm, Zyplast, Cosmederm, Cosmeplast [Allergan, Irvine CA], hyalurons [Allergan, Irvine, CA Medicis, Phoenix, AZ], and Juvederm) lead the list of the wrinkle and lip augmentation products, whereas autologous fat transplantation and volumetrical



Figure 2.18 Pre- and post-L-poly lactic acid Sculptra (four bottles); two treatment sessions; allows pan facial volumetric rejuvenation

fillers such as L-poly-lactic acid (Sculptra Sanofi-aventis, Bridgewater, NJ) and calcium hydroxylapatite (Radiesse, BioForm, San Mateo, CA) may be employed for minimally invasive three-dimensional volume rejuvenation (Figure 2.17). Finally, next generation collagen derivatives such as Evolence (Ortho-Neutrogena, Los Angeles, CA) and collagen/methyl acrylate (Artefill, Artes, San Diego, CA) are new agents recently introduced in this regard (Figure 2.18).

Finally, thread lifts is a newer minimally invasive procedure that utilizes anchoring barbed sutures, allowing for intermediate term correction of facial laxity.

CONCLUSION

By understanding a rational structural approach to whole body, noninvasive rejuvenation based upon pathophysiologic correlates, aesthetic physicians may choose a tailored treatment program with the most advanced technologies that will yield greater patient satisfaction.

REFERENCES

1. Gentile RD. Multimodality aesthetic skin rejuvenation. *Facial Plastic Surg.* 2005;21:120–130.

2. Rokhsar CK, Lee Sa, Fitzpatrick RE. Review of photorejuvenation: devices, cosmeceuticals, or both? *Dermatol Surg.* 2005;1166–1178.
3. Shah GM., Kilmer SL. Combined nonablative rejuvenation techniques. *Dermatol Surg.* 2005;31:1206–1210.
4. Fisher GH, Jacobson LG, Bernstein LJ, Kim KH, Geronemus RG. Nonablative radiofrequency treatment of facial laxity. *Dermatol Surg.* 2005;31: 1127–1241.
5. Weiss RA, Weiss MA, Beasley KL, Munavalli GI. Our approach to nonablative treatment of photoaging. *Lasers Surg Med.* 2005;37:2–8.
6. Carruthers JE, Carruthers AI. The effect of full-face broadband light treatments alone and in combination with bilateral crow's feet botulinum toxin type A chemodenervation. *Dermatol Surg.* 2004;30:355–366.
7. Narukar VA. Lasers, light sources, and radiofrequency devices for skin rejuvenation. *Semin Cutan Med Surg.* 2006;25(3):145–150.
8. Sadick NS, Weiss R, Kilmer S, Bitter P. Photorejuvenation with intense pulsed light: results of a multicenter study. *J Drugs Dermatol.* 2004;3(1) 41–49.
9. Alam M, Hsu TS, Dover JS, Wrono DA. Nonablative laser and light treatments: histology and tissue effects—A review. *Lasers Surg Med.* 2003;33:30–39.
10. Sadick NS. A structural approach to nonablative rejuvenation. *Cos Dermatol.* 2002;12:39–43.
11. Flynn TC. Update on botulinum toxin. *Semin Cutan Med Surg.* 2006;25:115–121.
12. Fernandez EM, Mackley CL. Soft tissue augmentation: A review. *J Drug Dermatol.* 2006;7:630–641.
13. Carruthers JDA, Carruthers A. Facial sculpting and tissue augmentation. *Dermatol Surg.* 2005;31: 1604–1612.
14. Murray CA, Zloty D, Warshawski L. The evolution of soft tissue fillers in clinical practice. *Dermatol Clin.* 2005;23:343–363.

CHAPTER 3

New Generation Cosmeceutical Agents

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INTRODUCTION

The trend in aesthetic rejuvenation has been toward non-surgical treatments that prevent or slow the signs of aging, correct or improve existing signs of aging, and maintain improvement after correction is achieved. Procedures such as nonablative laser therapy and peels, as well as injectables such as neurotoxin and fillers, are part of a comprehensive protocol for treating aging skin. Topical therapies, specifically cosmeceuticals, have grown in popularity as an important component of these nonsurgical antiaging protocols as research and development have improved their efficacy.

Cosmeceuticals are topical products that improve the appearance of the skin beyond cosmetics, which just camouflage or adorn, but have not been tested as drugs that change the structure or function of the skin. It is generally agreed upon by experts in clinical practice that some of these products may indeed have biologic function. They have not, however, made official drug claims so as to not be subjected to Food and Drug Administration (FDA) scrutiny.

Aging of the skin, whether chronologic or photoinduced, has many clinical presentations.¹ It is reasonable to expect that one or more of these signs and symptoms of skin aging should be improved by the cosmeceutical prescribed. It is important, therefore, that the prescribing dermatologist understands the activity of these products in order to be able to make recommendations based on the science as well as specific individual patient needs. This chapter will review popular cosmeceuticals, concentrating on newer trends such as botanicals and peptides as well as describing their purported function and resulting use in antiaging protocols.

VITAMIN A

The vitamin category of cosmeceutical has been one of the oldest and continues to be popular even as new technology develops. Retinoids are natural and synthetic compounds that are derived from and have activity simi-

lar to naturally occurring vitamin A. Beta-carotene is converted to vitamin A, or retinol, after ingestion and is naturally found in yellow and green vegetables. Retinyl esters are found in animal products such as egg yolks, butter, liver, and fish oil and are also converted to vitamin A.² Stramford was the first to use vitamin A for dermatologic purpose for acne vulgaris in 1943.³ In later years, Kligman first noticed that topical retinoids improve the appearance of photoaged skin.⁴

Important cutaneous effects of retinoids include regulating growth and differentiation of epithelial cells, antikeratinization, diminishing malignant cell growth and tumor potential, induction of apoptosis, and immunologic and anti-inflammatory effects.⁵ Retinoids induce uniform distribution of melanin content in the epidermis and decrease epidermal melanin content, thus improving photoinduced pigment irregularities.⁶

During cutaneous metabolism, free retinol is esterified to retinyl palmitate for storage via acylC: retinol acyltransferase. In the cytosol, free retinol is bound by a cytoplasmic retinol binding protein (CRBP).⁷ This complex is used as a substrate for retinol dehydrogenase, which oxidizes retinol to retinaldehyde. Retinaldehyde is then further oxidized by a rate-limiting step to all-trans retinoic acid. The biologically active metabolite of retinol is retinoic acid, or tretinoin.⁵ Figure 3.1 depicts a simplified schematic of retinoid metabolism. The understanding of the biochemistry of retinoids has led to the production of synthetic retinoids such as tazarotene, adapalene, and acetrein, which are available only by prescription and therefore not discussed here.

Nonprescription cosmeceutical creams with vitamin A are very popular in antiaging protocols. Retinol is the most popular vitamin A cosmeceutical, enjoying the benefits of good press that its prescription retinoids have generated because of their scientific studies on improvement of the cutaneous signs of aging. Retinol has been shown to equally penetrate the skin as retinoic acid and cause less irritation.² It is less effective, however, since it must be converted to retinoic acid by the two-step oxidative process. It is also highly unstable. Since retinol degrades into a biologically inactive molecule, packaging must ensure no exposure to light.

Other derivatives, such as retinyl aldehyde, retinal palmitate, and retinyl *N*-formyl aspartamate are other

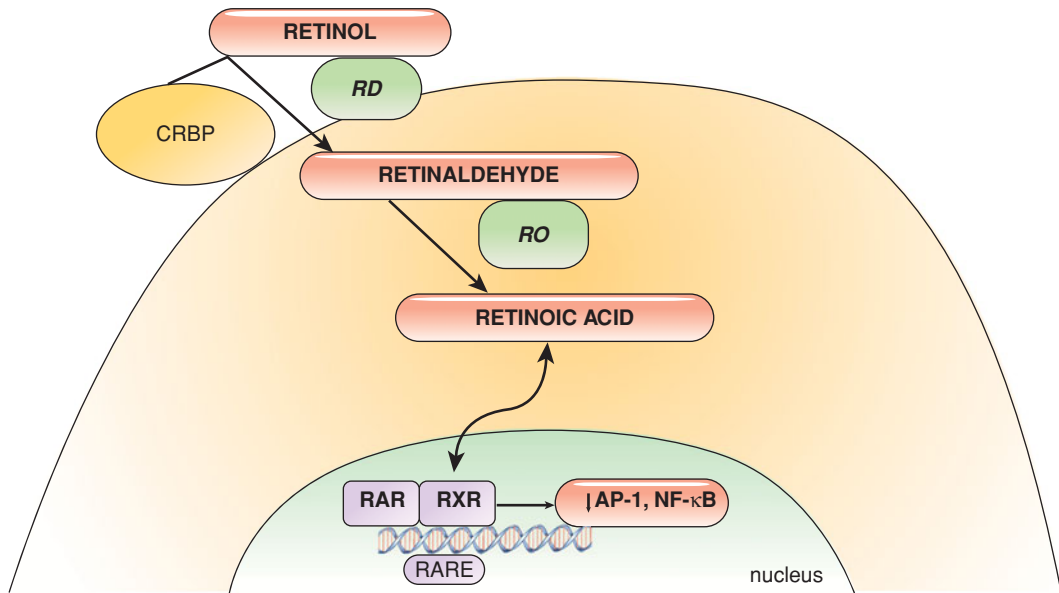


Figure 3.1 *Metabolism of retinoids*

forms of vitamin A often incorporated into cosmeceutical preparations. But these larger molecules, though less irritating, have not been shown to perform as well as stabilized retinol.⁸ Cosmetic benefit from these less active forms of vitamin A is often attributed simply to improved hydration of the epidermis. In general, cosmeceutical vitamin A products are used for the overall improvement of the visible signs of aging when more effective prescription retinoids are not tolerated.

VITAMIN B

Vitamin B has a number of derivatives. Niacin or B3 and its amide derivative, niacinamide, and panthenol; the B5 are the most common in cosmeceuticals. Panthenol is the alcohol form of pantothenic acid. It is a common additive to shampoos to make the hair soft and shiny. The B3 derivatives are by far the most popular B vitamin in cosmeceuticals. Niacinamide, or nicotinamide, is the amide form of niacin. Dermatologic preparations generally are available in this form, as it is chemically stable, formulated easily, and tolerated by the skin in very high concentrations.⁹ Niacinamide has been proposed to have a stabilizing effect on epidermal barrier function by

stimulating effect on ceramide synthesis in the stratum granulosum and speeding up differentiation of keratinocytes.^{10–12} Topical niacinamide has also been shown to prevent immunosuppression and skin cancer induction by UV radiation in mice.¹³ Niacinamide has been studied as a cosmeceutical and found to be effective for acne, rosacea, atopic dermatitis, dyschromia, the general signs of aging skin, and for improving transepidermal water loss (TEWL) via improvement of barrier function.^{10,14–19} It is also found to be effective in improving elasticity; one study has reported improvement in sallowness discoloration also.¹⁰ Patients using topical niacinamide 5% often report an improvement in redness (Figure 3.2).

The use of B3 derivative in antiaging products is the result of its known mechanism of action. Niacin is an important precursor to a family of energy cofactors such as nicotinamide adenine dinucleotide (NAD), nicotinamide adenine dinucleotide phosphate (NADP), and their antioxidant counterpart reduced forms NADH and NADPH. These cofactors are important in numerous metabolic pathways in the body and presumably in the skin.¹⁰ NADPH level has been found to be lower in older skin compared to younger skin.⁹ Niacinamide additives restore NADPH levels to a more youthful level.⁹ There



Figure 3.2 *This patient used a 5% niacinamide treatment on the left side of the face. A marked decrease in redness in comparison to the right side is evident*

was a significant reduction in wrinkle activity after 8 weeks of application of topical niacinamide 5% cream.¹⁰ Niacinamide has been able to increase collagen production in fibroblast culture and has been proposed to reduce excess glycosaminoglycans in the dermis.¹⁰ These two effects are said to be responsible for niacinamide's wrinkle-reducing effect. In the same 8-week topical application study, topical niacinamide was able to improve elasticity in vivo. Yellowness, or sallowness, was also decreased in subjects. Inhibition of glycation of proteins by niacinamide in vivo is hypothesized to be the mechanism responsible for improved elasticity and decreased sallowness. Niacin has been shown in recent studies to decrease melanosome transfer in vitro from melanocytes to keratinocytes. Niacin has also been found to have therapeutic effect in wound healing by stimulating leptin release.²⁰

Most topical niacinamide applications are relatively well tolerated, which is particularly important when used in subjects with sensitive skin as in rosacea patients. Topical preparations are generally of 2% or 5% strength and are used twice daily. At this point, niacinamide is a useful adjunct to treatment for acne, rosacea, melasma, and reversal of photodamage. Further trials and new formulations will allow niacinamide to be used more widely in the future and possibly as monotherapy.

The topical application of dexpanthenol prevents TEWL, accelerates reepithelialization in wound healing, and has shown anti-inflammatory effects.²¹ Topical dex-

panthenol acts as a moisturizer by improving stratum corneum hydration.²² Dexpanthenol has also been shown to induce fibroblast proliferation in vivo and in vitro.²¹ Dexpanthenol can be found in many skin and hair products as a humectant and wound care products to promote wound healing.

In summary, the use of vitamin B derivatives appears to be a good choice for patients with sensitive skin and in need of improved skin hydration. As such, they can be good adjuncts to, or substitute for, retinoids in many patients.

VITAMIN C

Vitamin C is another popular vitamin in cosmeceutical formulas. It is used because of antioxidant and antityrosinase actions. Vitamin C plays a vital physiologic role in humans. Although it goes by several names (ascorbic acid, ascorbate, L-ascorbic acid), each one represents the same water-soluble vitamin that is essential for life. Most animals are able to synthesize ascorbic acid from glucose in the liver. Humans must consume this vitamin because the enzyme necessary for synthesis, L-gulonogamma-lactone oxidase, has become mutated.²³ Vitamin C is also an essential cofactor for lysyl and prolyl hydroxylase, enzymes critical for collagen cross-linking. It is also vital in its role as an antioxidant. It has been found by researchers to be the most abundant antioxidant found in the skin²⁴ and body fluids.²⁵ It works together with another antioxidant, glutathione, to scavenge free radicals in water-soluble areas. Both serve as reducing agents by donating electrons to less stable compounds, which are missing an electron in their outer shell. It serves as an antioxidant by preventing other molecules from being oxidized and losing their electrons. Vitamin C itself is thereby oxidized, becoming a free radical because of its one unpaired electron. Its stability depends on temperature and the ambient pH. Product instability is sometimes responsible for the yellowish discoloration of some topical vitamin C products when they are exposed to oxygen in the air.²⁶ For this reason, topical forms of vitamin C must be delivered in a product with acidic pH, often causing irritation to sensitive-skin patients.

UV radiation is particularly damaging to human skin by virtue of its ability to produce reactive oxygen species (ROS). Studies have demonstrated that ascorbate interferes with the UV generation of superoxide anion,²⁷ hydroxyl radical,²⁸ and singlet oxygen²⁹ in a water-soluble environment. Vitamin C is also able to donate electrons to

oxidized vitamin E regenerating this essential lipophilic antioxidant.

Free radicals in the skin are generated by factors such as sunlight, smoking, and pollution. When light in the UVB spectrum (290–320 nm) is absorbed by DNA, it generates cyclopurimidine and thymine dimers.³⁰ If these errors are not corrected by nucleotide excision repair, there can be resultant C to T and CC to TT mutations, characteristic of actinic damage. The peak spectrum for this is 300 nm. In addition, UV light can be absorbed by trans-urocanic acid to produce singlet oxygen.³¹ The peak spectrum for this is 345 nm. As most dermatologists know, the damaging effects of sun exposure include wrinkling, dryness, telangiectasias, and pigmentation. There can also be upregulation of matrix metalloproteinases 1, 2, 3, and 9, which may breakdown existing collagen.³²

Vitamin C has a number of uses in dermatology, primarily because of its antioxidant action and the skin benefits that would result. Its photoprotective abilities were demonstrated in porcine skin, as a function of reduced erythema and a reduction in the number of sunburn cells.³³ Vitamin C can help decrease inflammation by suppressing activation of the transcription factor, nuclear factor kappa-beta.³⁴ By inhibiting elastin formation, it may also help in photoaged skin where elastin synthesis is increased.³⁵ In addition, Alster and West have reported a reduction in post-laser resurfacing erythema.³⁶

Topical formulations have shown promise for delivering antioxidant benefits directly to the skin. The reasoning for this is that most damage occurs as a result of exogenous radiation or other injury. Research on the percutaneous absorption of L-ascorbic acid demonstrated that it must be formulated at an acidic pH, less than or equal to 3.5, in order to enter the skin.³⁷ This way it does not carry a charge and can permeate the lipid membranes of the stratum corneum. In one study, maximum concentration for optimal absorption was found to be 15% to 20% and levels above that resulted in lower levels of absorption. In the same study, tissue saturation was achieved after three daily applications and bore a half-life of approximately 4 days.

Several double-blinded trials have proven the benefits of topically applied L-ascorbic acid. In one trial, a comparative study of 19 patients comparing 10% ascorbic acid versus vehicle on one half of the face for 3 months optical profilometry image analysis showed a significant improvement in average roughness and mean roughness or depth on the side treated with ascorbic acid.³⁴ Clinical assessments also showed greater improvement in fine wrinkling,

tactile roughness, coarse rhytides, skin laxity or tone, sallowness or yellowing, and overall features. In another study, patients applied 5% vitamin C cream to their neck and forearms for 6 months. Results showed a significant decrease in deep furrows, which was objectively proven with silicon replicas.³⁹ In a third study, punch-biopsies were performed to show increased hydration in areas treated with 10% L-ascorbic acid.⁴⁰

Since topical L-ascorbic acid can be very unstable at neutral pH or in ambient air, yet irritating at acid pH, a number of lipid-soluble ester formulations have been developed for cosmetic usage. Besides L-ascorbic acid, the forms most commonly used in cosmetics are ascorbyl-6-palmitate and magnesium ascorbyl phosphate.⁴¹ Ascorbyl-6-palmitate has been reported to serve as both an antioxidant and an anti-inflammatory formulation, helping to decrease erythema when applied after UV exposure.⁴² In vitro, studies of ascorbyl palmitate demonstrated reduced levels of ROS following UVB radiation, but cautioned that it may increase lipid peroxidation through its lipid component.⁴³ The use of oil-in-water emulsions may be helpful in optimizing its delivery.⁴⁴

Magnesium ascorbyl phosphate has also been studied in vitro and was found to penetrate the epidermis where it is successfully converted to L-ascorbic acid.⁴⁵ There it is able to promote collagen synthesis and wound healing,⁴⁶ lighten dyschromias,⁴⁷ and protect against UVB induced tumor formation in the skin.⁴⁵ Figure 3.3 demonstrates before and after 2 months using a proprietary 6% magnesium ascorbyl phosphate product. Sodium ascorbyl phosphate has been shown to improve acne and acne scarring.^{48,49} Recently a new formulation called tetra-isopalmitoyl ascorbic acid has been introduced and shown to reduce production of interleukin 1-alpha and prostaglandin E2 in UVB-irradiated keratinocytes, while also suppressing pigmentation in areas of topical application.⁵⁰

Many other derivatives of vitamin C exist, including calcium ascorbate, magnesium ascorbate, and sodium ascorbate. Practical application of the science of vitamin C has resulted in its use in cosmeceutical protocols as antioxidant and antipigment agents, usually adjunctive to the use of sun protection in a comprehensive skin care regime to brighten and rejuvenate sun damaged skin.

VITAMIN E

Vitamin E is another important antioxidant found in the body. Like vitamin C, it is not synthesized by humans, so exogenous intake is required. Unlike vitamin C, it is a

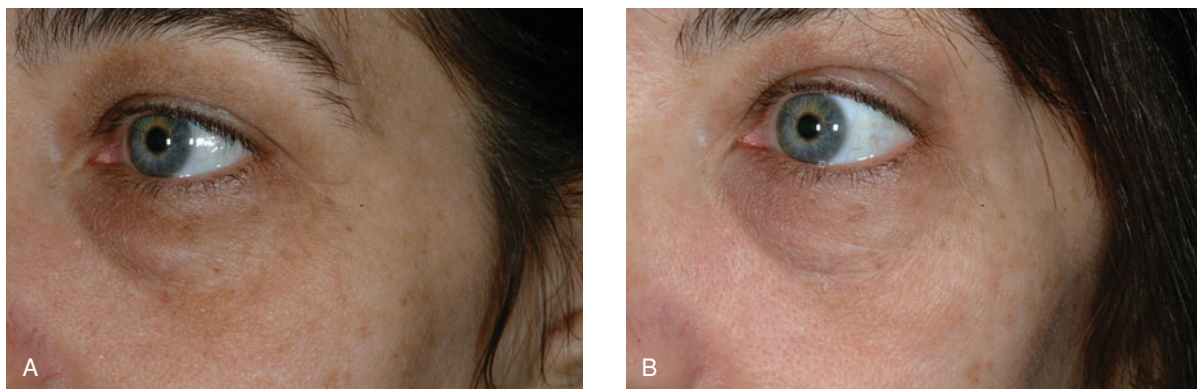


Figure 3.3 (A) Before and (B) after 2 months using a proprietary 6% magnesium ascorbyl phosphate product

lipophilic structure and so helps to protect structures like cell membranes and lipid bilayers. It does not function in the water-soluble compartments. Eight different types of molecules are collectively called vitamin E. Four are tocopherols, which have a phytyl side chain, and four are tocotrienols, which have an isoprenoid side chain. Alpha-tocopherol is the most abundant form of vitamin E in man, and gamma-tocopherol is the second most abundant. The potency of vitamin E is measured in international units (IU).

Vitamin E sequentially donates electrons, acting as a scavenger for lipid peroxyl radicals. The loss of a single electron yields the tocopheroxyl radical. This radical can be reduced back to tocopherol by co-antioxidants such as vitamin C,⁵¹ glutathione, or ubiquinol 10 (coenzyme Q), which may help photoprotection⁵² through similar recycling methods, or, it may react with another free radical to form tocopherol quinone. Like vitamin C, the structure of vitamin E is such that even without two electrons in its outer shell, it is more stable than other free radicals, which may do harm to the body. The molar ratio of vitamin E to polyunsaturated phospholipids is less than 1:1000.⁵³ Therefore, constant regeneration of reduced tocopherol is required in order to maintain its antioxidant capabilities.

Vitamin E has a number of useful applications in dermatology. Its photoprotective effects are well supported in a review paper summarizing 28 studies on vitamin E.⁵³ When used topically prior to sun exposure, it was shown to decrease erythema, sunburn cell formation, lipid peroxidation, DNA adduct and thymine dimer formation, immunosuppression, and UVA-induced binding of photosensitizers. Benefits in terms of scar prevention have

been suggested, but could not be proven in two different studies.^{54,55}

As with vitamin C, the task of identifying optimum topical formulations of vitamin E has been challenging. Tocopherol esters are fat-soluble compounds that are more stable than free tocopherol. They do not have a free aromatic hydroxyl group, which is responsible for the antioxidant properties of vitamin E. Therefore, the ester form must be hydrolyzed during skin absorption in order to be active. This conversion seems to occur only in the nucleated layers below the stratum corneum,⁵⁶ at rates varying from 4% to 50%.^{57,58} Some other cosmetic preparations of vitamin E include tocopherol linoleate, tocopherol nicotinate, and tocopherol succinate. The popularity of vitamin E in cosmeceuticals waxes and wanes. In general, it is seen as an antioxidant and emollient and often used to complement sunscreens in daytime products and in emollient evening moisturizers.

PEPTIDES AND PROTEINS

Recent advances in skin biology have shown us that there may be other ways to impede and partially reverse the cutaneous signs of aging and photodamage. One of the newer emerging therapies involves the use of amino acids and novel peptides, which are short chains of amino acid sequences. These products often have the advantage of inducing less skin irritation when compared to topical retinoids, making them a more tolerable alternative for patients with sensitive skin. This next section covers the science behind cosmeceutical amino acids and peptides and their place in the practicing dermatologist's armamentarium of cutaneous antiaging treatments.

Studies of the extracellular matrix in chronologically aged skin have demonstrated lower procollagen type I mRNA and collagen synthesis, lower protein and greater matrix metalloproteinase-1 (MMP-1) activity inducing the breakdown of collagen, and a slower rate of proliferation of dermal fibroblasts.^{59–62} Natural aging thus results from the decreased turnover of dermal fibroblasts, an increased breakdown of collagen, and a decreased production of new collagen. Photoaged skin also has lower mRNA, collagen production, and lower protein than chronologically aged skin.⁶³ Much of the research demonstrating the role of amino acids and peptides in reversing the cutaneous signs of aging has been a secondary by-product of research on wound healing. In the 1930s, yeast extracts were used in medications for their enhancement of wound healing.⁶⁴ As technology progressed, this allowed a protein fraction to be recovered from the extract of fermented yeast. Through studies of this protein extract, a beneficial effect on wound healing and improved collagen synthesis has been demonstrated.^{65,66} These properties have been attributed to low molecular weight peptides that are enzymatically manufactured by the growing yeast. The peptides are theorized to act by upregulating cellular growth factors leading to skin healing as a result of the stimulation of angiogenesis, increased production of granulation tissue and new collagen synthesis.⁶⁷

As a result of this ongoing research, certain amino acids and peptides have been found that are able to improve the appearance of wrinkles by directly stimulating collagen-producing human dermal skin fibroblasts and downregulating human dermal skin fibroblast collagenase production. Some help by delivering enzymatic cofactors necessary for dermal remodeling and others by blocking the neurotransmitter release necessary for muscle contraction. Finally, there is a separate group of amino acids and peptides that act as antioxidants and anti-inflammatory agents. For these reasons amino acids and peptides retain a position in cosmeceutical marketplace.

AMINO ACIDS

Amino acids are the building blocks for proteins and many other different types of organic molecules including enzymes and DNA. Amino acids are incorporated into cosmeceutical products for several different purposes. Some are purported to have antioxidant and antiwrinkle properties, while others such as capryloyl glycine aid in the emulsification of products and other active ingredients.⁶⁸ Another amino acid, arginine, has been incorporated into

glycolic-acid-containing cosmeceuticals in order to facilitate controlled delivery of the glycolic acid over a period of time and to decrease stinging.⁶⁹ Dipalmitoyl hydroxyproline has been incorporated into cosmeceuticals as an *amplifier of synthesis of collagen III* via enhancement of fibroblast activity.⁷⁰ Valyl-tryptophan is reported to enhance lymphatic circulation by inhibiting angiotensin-converting enzyme and therefore provides a benefit in decreasing the appearance of under-eye circles secondary to periorcular edema as is often seen in allergic patients.⁷¹

Several amino acids are used in cosmeceutical products for their antioxidant effects via iron chelation. Catalytic iron is thought to be released from iron-containing proteins under conditions of oxidative stress, such as exposure to UV light, and catalyzes the generation of free radicals in a process known as the Fenton reaction.⁷² The amino acid histadine is often incorporated into cosmeceutical products as L-carnosine (beta-alanylhistadine) or carcinine (beta-alanylhistamine). Recent studies have shown that it acts as a natural antioxidant with hydroxyl-radical scavenging and lipid-peroxidase activities.⁷³ In a recent study of amino-acid-based iron chelators formed from the condensation of amino acids such as glycine and L-serine with salicylaldehyde, inhibition of the iron-induced hydroxyl radical generation induced by UV light has been demonstrated. These amino acids are proposed to act by mimicking the binding site of iron-sequestering proteins. The compounds also suppressed UV-induced lipid peroxidation and showed protective activity against UV-induced cytotoxicity in murine dermal fibroblasts.⁷² In a further study, *N*-(4-pyridoxylmethylene)-L-serine (PYSer) showed protective effects against skin damage in hairless mice irradiated with UVB radiation. Topical application of PYSer to the skin was shown to significantly delay and/or decrease the visible wrinkle formation induced by UVB irradiation. Results indicated that UVB induced epidermal hypertrophy and lymphocytic infiltration were suppressed by PYSer when a histologic examination was performed. Moreover, PYSer was noted to suppress the UVB induced increase in glycosaminoglycans. The authors of the study suggest that these results indicate that PYSer is a promising antioxidant for the prevention of chronic skin photoaging by its iron-sequestering activity.⁷⁴

Owing to the popularity of the use of botulinum toxin as a neurotransmitter blocking peptide that decreases hyperdynamic facial movements and thus facial wrinkling, other amino acids and peptides with actions that inhibit muscle contraction have been sought. Gamma-aminobutyric acid

(GABA) is a neurotransmitter in the central nervous system that is synthesized in the body from the decarboxylation of the amino acid glutamic acid. The effect of GABA binding results in a negative change in membrane potential leading to hyperpolarization. As a result of this hyperpolarization, an increased stimulus is needed for depolarization and therefore muscle contraction, and therefore it is theorized that topical GABA application may lead to relaxation of facial muscles and thus aid in wrinkle reduction.⁷⁵

OLIGOPEPTIDES

Oligopeptides are short chains of amino acid sequences that make up larger proteins, such as collagen and elastin. There are at least four categories of peptides currently being used in cosmeceutical products. This increase in peptide technology has arisen because of the technology to synthesize fragments that mimic peptide sequences in collagen and elastin with the ability to stimulate production of new collagen and elastin. Currently, there are other peptides available that function primarily as carriers of cofactors for important enzymatic steps in collagen production. Peptide fragments also exist that are able to block neurotransmitter release. Since some wrinkling of the skin is caused by hyperkinetic facial movement, peptides that have actions to inhibit or reverse these actions could have clinical antiaging benefits. Additionally, some peptides have been noted to have antioxidant and anti-inflammatory effects, and others have been discovered that are theorized to reduce hyperpigmentation. The mechanism of action of several of these bioactive peptides is yet to be determined.

SIGNAL PEPTIDES

A peptide with the ability to increase collagen production, decrease collagenase activity, or increase fibroblast turnover should potentially improve the clinical appearance of the fine and coarse wrinkles visible in chronologically and photoaged skin. As a result of wound healing research on the growth and stimulation of human skin fibroblasts, certain bioactive amino acid chains have been discovered that stimulate human skin dermal fibroblast growth in vitro and decrease the depth and length of wrinkles in vivo. A valine–glycine–valine–alanine–proline–glycine (VGVAPG) peptide was discovered in one study of elastin-derived peptides, which significantly stimulated human dermal skin fibroblast

production while simultaneously downregulating elastin expression.^{76,77} Additionally, this novel peptide was shown to be chemotactic for fibroblasts.⁷⁸ Another study examined the tyrosine–tyrosine–arginine–alanine–aspartame–aspartame–alanine peptide sequence. This study found that the peptide inhibited procollagen-C proteinase, which cleaves C-propeptide from type I procollagen, thus leading to decreased collagen breakdown.⁷⁹ A study of the lysine–threonine–threonine–lysine–serine (KTTKS) peptide found on type I procollagen has demonstrated that this peptide stimulates feedback regulation of new collagen synthesis and therefore results in an increased production of extracellular matrix proteins (i.e., collagens I and II and fibronectin).⁸⁰ This pentapeptide is linked to palmitic acid in order to enhance skin stability and improve skin delivery.

A double-blind, placebo-controlled, split face, left–right randomized clinical study of 93 Caucasian women aged 35 to 55 assessed a topical moisturizer versus the same moisturizer containing palmitoyl-KTTKS, over a 12-week period. Pal-KTTKS was well tolerated by the skin and provided significant improvement versus placebo control for reduction in wrinkles or fine lines by both qualitative technical and expert grade image analysis.⁸¹ The tripeptide glycyl-L-histadyl-L-lysine (GHK) is a carrier peptide that has also been shown to have some signal peptide effects as well. It alone has been shown to enhance collagen production by stimulating fibroblasts.⁸² Lipospondin, a tripeptide linked to elaidic acid (elaidyl-KFK or elaidyl-lys-phe-lys) was designed to simultaneously activate latent TGF- β (through its peptide domain) and inhibit MMPs (through its lipophilic moiety, elaidic acid). Studies have demonstrated that it is able to upregulate collagen and TIMP-1 production and downregulate MMP-1 in fibroblast cultures.⁸³ Numerous proprietary peptides with undisclosed sequences have been used in cosmeceutical formulas. Results from studies of these formulas have not been formally published in peer-reviewed articles but in some patients, clinical benefit can be demonstrated (Figure 3.4).

NEUROTRANSMITTER-BLOCKING PEPTIDES

The commercial success of any product often results in products claiming to mimic its benefit. The neurotransmitter-blocking peptides currently incorporated into cosmeceutical products were developed as topical mimics of the

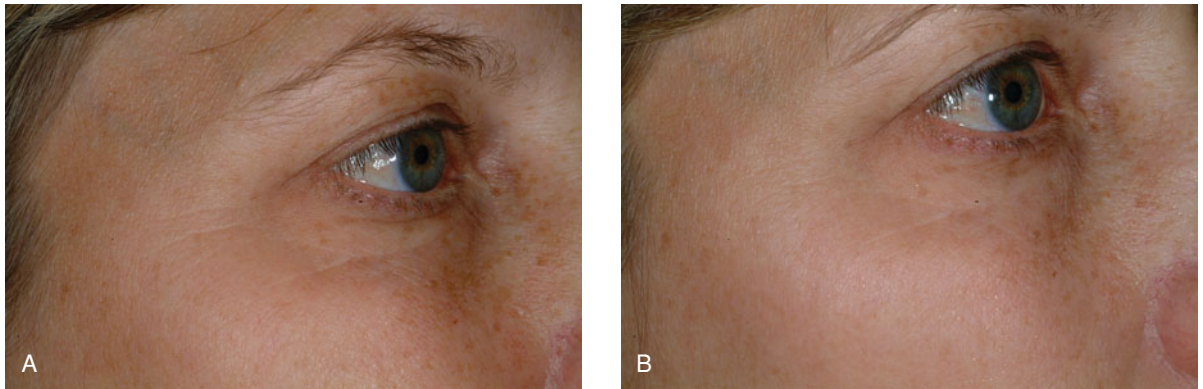


Figure 3.4 (A) Before and (B) after using peptide solutions, a reduction in periorbital wrinkles can be seen

botulinum neurotoxins. Currently, only the botulinum neurotoxin A has been approved by FDA for subcutaneous, intradermal, and intramuscular injection for facial wrinkles.⁸⁴ All botulinum neurotoxin serotypes (A-G) are single-chain polypeptides that inhibit acetylcholine release at the neuromuscular junction in a three-step process. The topical neurotransmitter blocking and releasing peptides that are currently marketed in cosmeceuticals function to decrease facial muscle contraction and thus reduce lines and wrinkles by raising the threshold for minimal muscle activity, requiring more signal to achieve movement and reducing subconscious muscle movement over time.⁸⁵ Most of these peptides act on the SNARE complex, while others target different parts of the neuromuscular junction J or certain neurotransmitters. Although a promising idea, it is yet to be proven whether these topical neurotransmitter blocking and releasing proteins can penetrate to the level of the neuromuscular junction.

Acetyl hexapeptide-3 (AC-gly-glu-met-gln-arg-arg-NH₂) is a synthetic peptide patterned from the N-terminal end of the protein SNAP-25 that inhibits SNARE complex formation and catecholamine release. This peptide was found to inhibit vesicle docking by preventing formation of the ternary SNARE complex, which is involved in synaptic vesicle exocytosis.^{86,87} Theoretically, this peptide may mimic the effects of botulinum toxin injections. Acetyl glutamyl heptapeptide-1, or SNAP-7 and/or SNAP-8, is a molecular mimic of the N-terminal end of SNAP-25, which competes with SNAP-25 for a position in the SNARE complex, thereby modulating its formation. When the SNARE complex is destabilized, the vesicle cannot release neurotransmitters efficiently and therefore muscle contraction is attenuated.⁸⁸

Dimethylaminoethanol bitartrate, also known as DMAE, is a membrane stabilizer that purportedly improves facial muscle tone by releasing acetylcholine. In a multicenter placebo-controlled trial of 156 patients with moderate photodamage applying 3% topical facial gel daily for 16 weeks, a statistically significant mitigation of fine wrinkles was noted in periorbital and cheek skin. Increased skin firmness with possible improvement in underlying facial tone has been noted with topical use. The mechanism of action remains to be elucidated.⁸⁹ The use of these “wrinkle relaxers” remains controversial and marketing claims of benefit to rival FDA approved neurotoxins effects has resulted in skepticism on the part of experienced physicians.

CARRIER PEPTIDES

Carrier peptides function to stabilize and deliver important trace elements necessary for wound healing and enzymatic processes. The most commonly encountered carrier peptide currently is instrumental in stabilizing and delivering copper into cells. Copper is a metal that enhances wound healing and angiogenesis. It is an essential cofactor for collagen and elastin formation, down regulates MMPs, and reduces the activity of collagenase. Copper is a required cofactor for the enzyme superoxide dismutase, (a powerful antioxidant) and lysyl oxidase, an important enzyme in collagen and elastin production. The tripeptide glycyl-L-histadyl-L-lysine is used as a copper carrier.⁹⁰ As a cosmeceutical, copper peptide is thought to improve skin firmness and texture, fine lines, and hyperpigmentation. GHK-copper complex increases levels of MMP/TIMPs and aids in dermal tissue

remodeling.⁹¹ It also stimulates production of collagen I, glycosaminoglycans, cytochrome-c oxidase, and reduces hyperpigmentation via stimulation of tyrosinase.^{92,93}

The increasing number and overall popularity of peptides in cosmeceuticals seems to reflect the desire of consumers to see improvement of visible signs of aging without irritation. Retinoids have long been the antiaging medicine of choice for their various cutaneous effects. Unfortunately, many patients complain of side effects, such as itching, burning, stinging, and peeling. Cosmeceutical amino acids and peptides are less irritating because they do not increase TEWL and thus offer the potential to be a less irritating alternative to retinoids for patients intolerant to retinoids.

GROWTH FACTORS

A recent approach to the treatment of aging skin is the use of growth factors. The likeness of chronic photodamage to an injury such as a chronic wound that has not progressed to complete repair has led investigators to seek using growth factors for skin rejuvenation.^{94,95} Like chronic wounds, ultraviolet radiation (UVR)-induced skin damage requires regeneration of keratinocytes, fibroblasts, dermal extracellular matrix, and the microvasculature.⁹⁶ Growth factors are large peptides that have both anabolic and catabolic functions in cutaneous physiology. Repair and remodeling of epidermal and dermal tissue is a tightly coordinated process that is largely orchestrated by growth factor proteins. Keratinocytes, inflammatory cells, fibroblasts, and endothelial cells are each affected by the presence or absence of these mediators, and changes in cutaneous morphology can often be traced to instability of this communication network. The most well-characterized cutaneous growth factors include epidermal growth factor, fibroblast growth factor, keratinocyte growth factor, platelet-derived growth factor, transforming growth factor (TGF), and vascular endothelial growth factor. These large proteins use autocrine and paracrine signaling to bind cell-surface receptors at low concentrations (10^{-9} molar) and initiate intracellular signal transduction cascades that stimulate inflammatory cell migration, angiogenesis, keratinocyte replication, fibroblast proliferation, and synthesis of dermal extracellular matrix material.⁹⁷ It is the synchronous interaction of multiple growth factors that is significant, with no one growth factor being a more important determinant of outcome in cutaneous repair.⁹⁴

Advances in cutaneous tissue engineering over the past few decades have provided the ability to artificially

construct human skin components that aid in the healing of chronic wounds. These replacement tissue grafts, consisting of cells, extracellular matrix components, or both, provide biologically active dressings that synthesize growth factors regulating tissue repair.⁹⁶ Growth factors are critical to the success of any of any skin substitute in inducing wound repair. Furthermore, biotechnologic advances in tissue culture have permitted the collection of the endogenous growth factors necessary for cutaneous tissue remodeling and repair.⁹⁵ Culture-borne human growth factors can be concentrated and compounded into topical formulations for use in cosmeceutical applications that treat the effects of chronic photodamage, much as they did in their original use for dermal tissue repair in wounds.

TGF- β is perhaps the best known and most widely used growth factor in cosmeceuticals. TGF- β refers to a family of multifunctional proteins capable of regulating proliferation, differentiation, tissue remodeling, and repair among various cell types.^{98,99} In the 1980s, pioneering studies were performed introducing TGF- β by subcutaneous injection in murine models. TGF- β induced a dose- and time-dependent increase in fibroblast proliferation, collagen synthesis, and vasculogenesis. Furthermore, marked reduction in collagen synthesis as measured by a decrease in proline incorporation into collagen, was achieved by administration of antibodies to TGF- β .¹⁰⁰ These pioneering discoveries provided the foundation for understanding cutaneous growth factors as we know them today.

TGF- β is a peptide hormone consisting of two 12-kDa chains linked by disulfide bonds. TGF- β is secreted by activated fibroblasts, lymphocytes, and platelets.^{97,100,101} It acts as a positive growth factor upon fibroblasts and is the most well understood mediator of procollagen, fibronectin, and glycosaminoglycan synthesis.^{99–103} TGF- β binds to the extracellular domain of the TGF- β receptor II (TbRII) transmembrane receptor on fibroblasts and induces a conformational change in the TbRII's intracellular serine-threonine kinase domain. This conformational change triggers a cascade of intracellular events that culminate in the transcription of genes that code for synthesis of dermal extracellular matrix (ECM) material such as collagen, elastin, proteoglycans, and glycosaminoglycans.^{98,99}

UV irradiation alters the dermal matrix both quantitatively and qualitatively. UV irradiation impairs the interaction of TGF- β with the TbRII thereby preventing the cascade of events necessary for fibroblast synthesis of the dermal ECM. The mechanism by which UVA irradiation

interferes with synthesis of dermal components is twofold. UVA irradiation increases the synthesis of a nonfunctional latent TGF- β protein, and UVA wavelengths downregulate the expression of T β RII by fibroblasts within 8 hours of UV exposure.^{99,102,103} The effect of UVA irradiation is a 90% reduction in skin fibroblast T β RII activation, which diminishes procollagen synthesis and directly contributes to the aged appearance of photodamaged skin.¹⁰² T β RII mRNA expression is also diminished after UVA irradiation. Total production reduced by 40% when compared to nonUVA exposed tissue. Skin treated with TGF- β_1 after UVA exposure results in a 20% increase in T β RII expression when compared to controls treated with UVA alone. This suggests that topical TGF- β_1 may act as a “self-protecting” inducer of the T β RII receptor, abrogating UVA-induced reduction in T β RII expression.⁹⁹ UVA irradiation reduces procollagen production by 75%. The addition of TGF- β_1 after UV irradiation blunts this reduction in collagen synthesis.⁹⁹ This data supports the speculation that quantitative reduction of collagen synthesis in photodamaged skin, accounted for by UV-induced changes in TGF- β /T β RII signaling, is a process that can be modified by the addition of TGF- β_1 .

TGF- α is a small growth factor protein with potent keratinocyte mitogenic activity. TGF- α binds to keratinocyte epidermal growth factor receptors (EGFRs) to stimulate keratinocyte proliferation. TGF- α is produced by wounded keratinocytes that are exposed to human serum. Fibrin contained within serum is the physiologic stimulus for keratinocyte synthesis of TGF- α .¹⁰⁴ TGF- α acts in an autocrine fashion to induce keratinocyte proliferation as well as keratinocyte expression of surface EGFR. TGF- α appears to be the strongest mitogen for keratinocytes, surpassing the activity of epidermal growth factor, keratinocyte growth factor, and TGF- β .¹⁰⁵

Epidermal growth factor receptor (EGFR) is a widely expressed cell surface transmembrane receptor with intrinsic protein-tyrosine kinase activity.^{106,107} EGFR ligands include amphiregulin, betacellulin, epiregulin, heparin-binding EGF-like growth factor, and TGF- α .¹⁰⁸ Ligand binding to EGFR induces receptor dimerization and autophosphorylation, two steps necessary for EGFR activity.^{106,107} The EGFR mediates cell-specific anabolic or catabolic effects. EGFR activation promotes keratinocyte proliferation^{106,108} and has been shown to be effective in reducing the healing time in partial-thickness skin wounds.^{108–111} However, activated EGFR also acts as a “negative” growth factor stimulating the degradation of collagen and elastin through induction of ker-

atinocyte synthesis of AP-1 and matrix metalloproteinases (MMPs).^{106,107}

Keratinocyte growth factor (KGF, FGF-7) is one of the 22 members of the fibroblast growth factor family.¹¹² KGF, synthesized by fibroblasts, acts as a paracrine growth factor that induces keratinocyte proliferation.^{113,114} Synthesis of KGF is enhanced by proinflammatory cytokines, interleukin (IL)-1 α , IL-1 β , and TNF- α .¹¹³ KGF has significant effects on epidermal keratinocyte migration during wound repair, but its application in topical preparations has been limited by its short biologic half-life.¹¹² Newer approaches in wound management have used KGF by covalently attaching the growth factor to peptides that are cleaved by products of matrix metabolism during wound repair. Cleavage of the covalent bond releases KGF directly into tissue as a bioactive substrate capable of stimulating keratinocyte proliferation and migration.^{112,114} Bioengineered KGF-peptide complexes applied to wounded cutaneous tissue allow for a gradual and persistent release of KGF and result in significantly reduced epidermal wound closure time.¹¹² Because of its short biologic half-life, KGF applications should employ mechanisms that ensure ongoing secretion of KGF. This can be achieved by application of tissue products capable of synthesizing KGF, or those that contain an altered form of KGF that is slowly released into as a tissue-available substrate.

As with other peptides, research into wound healing resulted in extrapolation of data to justify the use of growth factors in cosmeceuticals. The science of wound healing and the effects of large molecules to speed healing do not necessarily translate into effectiveness of penetration into intact dermis for biologic effect. Clinical benefit has been documented, however, and justifies the use of these ingredients in cosmeceutical formulas for photodamaged skin (Figure 3.5).

BOTANICAL AGENTS

Botanical extracts that support the health, texture, and integrity of skin, hair, and nails are widely used in cosmetic formulations. They form the largest category of cosmeceutical additives found in the marketplace today because of a resurgence of interest and demand for natural products by consumers. Various plant extracts that formed the basis of medical treatments in ancient civilizations and many traditional cultures are still used today in cleansers, moisturizers, astringents, and many other skin care products.

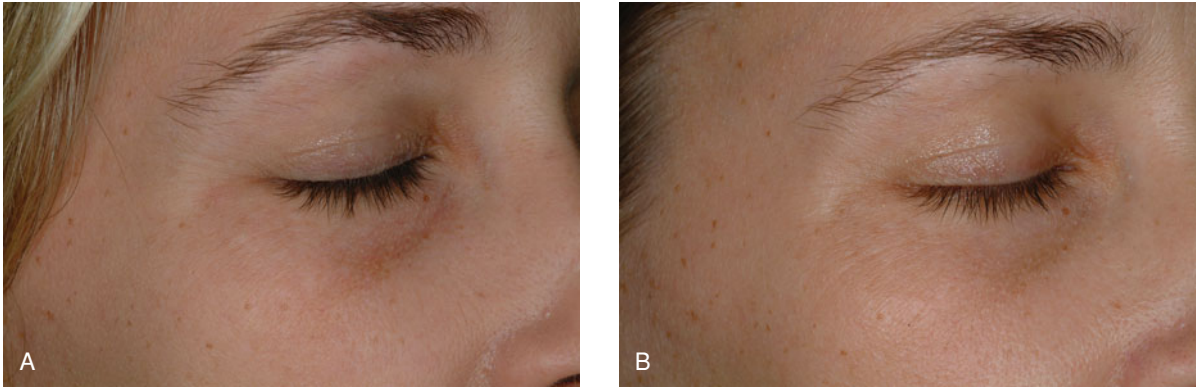


Figure 3.5 (A) Before and (B) after using growth factor treatment

Skincare technologies are constantly changing based on new developments in raw materials and botanical agents, enhanced insight into skin physiology, and evolving consumer demands. New botanical skincare treatments are emerging, presenting dermatologists and their patients the challenge of understanding the science behind these cosmeceuticals. Over 60 different botanicals are integral components of cosmeceutical products. Thus dermatologists must have a working knowledge of these botanicals and keep up with how they can evolve to provide optimal medical care and answer patients' questions (Table 3.1).

Herbal medicine is based upon the principle that a naturally occurring mixture of active plant compounds is more effective and safer than individual molecules and manufactured combinations of synthetic compounds. Many people who use alternative botanical products believe that all "natural" products are safe and somehow provide benefits above and beyond man-made products. However, the incorporation of plant-derived material into contemporary cosmeceutical formulations requires a significant amount of processing, which may greatly affect the biologic activity of the botanical. Multiple factors

TABLE 3.1 ■ Botanical Cosmeceutical Categories

Category	Botanical Additive
Antioxidant	Chamomile, cocoa, curcumin, <i>Echinacea</i> , ferulic acid, ginger, polypodium leucotomos, persimmon, pomegranate, pycnogenol, grape seed, green tea, red clover, resveratrol, rosemary, silymarin, soy, St. John's wort
Anti-inflammatory	Aloe vera, chamomile, curcumin, ginkgo biloba, ginger, green tea, horse chestnut, onion extract, propolis, red clover, silymarin, St. John's wort
Antimicrobial	Aloe vera, chamomile, <i>Echinacea</i> , garlic, ginger, lavender, onion extract, persimmon, pomegranate, resveratrol, St. John's wort, sunflower oil, tea tree oil
Anticarcinogenic	Curcumin, ferulic acid, ginger, green tea, persimmon, <i>Polypodium leucotomos</i> , propolis, resveratrol, rosemary, soy, sunflower oil
Healing/soothing	Allantoin, aloe vera, chamomile, lavender, papaya, prickly pear, sunflower oil, witch hazel

*Many botanical additives are functional in several different categories.

impact the composition of the herb and active ingredients; therefore quality control in the formulation of herbal preparations poses the challenge of balancing the need for consistency in processing the raw materials while maintaining the potency of the bioactivity of the botanical. Standardization of the herbal extract is an important task in producing an effective preparation of herbal cosmeceuticals. Most cosmeceuticals are designed to address the major cause of cutaneous aging, which is the oxidation of skin structures from highly reactive oxygen molecules present in the environment.

Plants have evolved mechanisms for protection against oxidative stresses. These protective mechanisms provide various chemicals that can be extracted and added to topical antioxidant preparations for use in the treatment of benign photodamage. Natural antioxidants that quench free radicals are an essential component of antiaging formulations. They potentially offer protection against tissue damage from the detrimental effects of environmental and other agents. Biochemical reactions that accelerate the progression of skin aging have their roots in inflammatory processes. Natural anti-inflammatory agents are therefore included in antiaging formulations, and serve to soothe, heal, and protect skin tone and integrity. Lipid compounds that provide an occlusive effect to prevent water loss, repair lipid layers, and restore barrier functions are also an integral part of antiaging formulations.

A wide range of natural additives are available for use in skincare formulations. The most popular botanicals incorporated into cosmeceuticals include soy, teas, grape seed, pomegranate, pycnogenol, German chamomile, aloe, and tea tree among many others. While all have been documented to treat dermatologic conditions, many lack scientific support. There are thousands of plants with anecdotal purported skin benefits, but few botanical-based cosmeceuticals have uses that are actually supported by evidence-based science. Only the most popular botanicals commonly incorporated into cosmeceutical formulations will be discussed.

Silymarin is a naturally occurring polyphenolic flavonoid compound, which is an extract of the milk thistle plant, *Silybum marianum*. Milk thistle has been used for thousands of years for multiple medicinal purposes. The extract consists of flavonoids, which are the structural isomers silybin, isosilybin, silidianin, and silychristin.

Silybin is the main constituent and is considered to be the most biologically active in terms of its antioxidant and anti-inflammatory properties.³⁰ Silymarin is a strong

antioxidant shown to inhibit sunburn cells, apoptosis, and lipid peroxidation by scavenging free radical species. Researchers have demonstrated that silymarin, through its antioxidant activity, exhibits preventive and anticancer effects against skin cancer, significantly preventing photocarcinogenesis, and skin tumor promotion in mice.¹¹⁵ Topical application of silybin has been found to impart a strong protective effect against UV-induced damage in the epidermis, to prevent UVB-induced immunosuppression and oxidative stress, and to prevent UVB-induced carcinogenesis.^{116,117}

While silymarin has been introduced in some high-end moisturizers for benign photoaging, it is still mostly available as oral supplementation in the US. Because of its potent antioxidant activity and recognized potential as an antiphotodamage and anticarcinogenic agent, there appears to be compelling evidence for increased inclusion in topical skin care products and sunscreens.

Soy extract has positive research support for its antioxidant, antiproliferative, and anticarcinogenic activities. Topical application of soy has been used for reducing hyperpigmentation, enhancing skin elasticity, controlling oil production, moisturizing the skin, and delaying hair regrowth.¹¹⁸ Soy also shows the potential to decrease photoaging of the skin and prevent skin cancers through the estrogen-like and antioxidant effects of its metabolites.

The major components of soy are phospholipids, such as phosphatidyl choline, and essential fatty acids. The minor components include the most active compounds, such as isoflavones, saponins, essential amino acids, phytosterols, calcium, potassium, iron, as well as the proteases soybean trypsin inhibitor (STI) and Bowman-Birk inhibitor (BBI). The various components of soy have a variety of beneficial effects making them useful additions to skin care products. The most potent isoflavones are the phytoestrogens genistein and daidzein. Genistein is a potent antioxidant, inhibiting lipid peroxidation and chemical and UVB-induced carcinogenesis. Genistein was shown to significantly inhibit chemical carcinogen-induced ROS, oxidative DNA damage, proto-oncogene expression, and the initiation and promotion of skin carcinogenesis in mouse skin.¹¹⁹ Topical estrogens have been shown to promote collagen synthesis and increase skin thickness, which may be beneficial for postmenopausal women who develop a thinner dermis and decreased collagen.¹²⁰ The small proteases STI and BBI appear to promote skin lightening and reduce unwanted facial and body hair in human clinical trials.^{120,121} Beyond the depigmenting activity, STI, BBI, and soy milk

were also found to prevent UV-induced pigmentation both in vitro and in vivo.¹²² In addition, soy lipids, lecithins, and phytosterols are believed to restore barrier function and replenish moisture.

Beyond its moisturizing ability, soy appears to be a safe and effective treatment for postmenopausal women and for hyperpigmentation disorders (other than melasma which is somewhat estrogen-mediated). Although further research is necessary, the antioxidant and anticarcinogenic activities of soy and its isoflavones show a promising role for this botanical in the cosmeceutical industry.

Pycnogenol, or *Pinus pinaster*, is an extract of French marine pine bark. It is a water-soluble antioxidant containing several phenolic constituents, including taxifolin, catechin, and procyanidins. It also contains several phenolic acids, including *p*-hydroxybenzoic, protocatechuic, gallic, canillic, *p*-coumaric, caffeic, and ferulic acids. It is a free radical scavenger that is thought to reduce vitamin C radical activity, returning the vitamin C to its active form. The active vitamin C then regenerates vitamin E and its antioxidant activity. Pycnogenol is an excellent antiaging additive to cosmeceutical products since it currently demonstrates no long-term toxicity, no mutagenicity, no teratogenicity, and no allergenicity.¹²³ It is consumed orally as a preventive for cardiovascular disease and appears to improve benign photoaged skin from both oral and topical use.

Ferulic acid (4-hydroxy-3-methoxycinnamic acid) belongs to the polyphenolic compounds known as hydroxycinnamic acids. It is found in the seeds and leaves of most plants, such as in wheat, rice and oats, as well as in apple, artichoke, spinach, parsley, grapes, and rhubarb.

Ferulic acid may have significant health benefits through its antioxidant, photoprotective, and anticarcinogenic activity. It strongly absorbs UV like its related compounds and is also a potent antioxidant preventing lipid peroxidation and protecting skin from UVB-induced erythema. The vitamin E/ferulic acid compound α -tocopheryl ferulate has the capacity to absorb UV radiation, thereby maintaining tocopherol in a stable state. A recently published study showed that the addition of ferulic acid to a solution of ascorbic acid (vitamin C) and α -tocopherol (vitamin E) stabilized the formulation and doubled photoprotection to skin from fourfold to eightfold as measured by both erythema and sunburn cell formation. This topical preparation may reduce oxidative stress and efficiently reduced the formation of thymine dimers in the skin.¹²⁴ This combination of

antioxidants provides synergistic protection against oxidative stress in skin, providing a useful supplement for protection against photoaging and skin cancer.

Green tea extracts are amongst the fastest-growing herbal products. While there has been enormous growth in green tea consumption as a dietary supplement, the use of tea extracts in cosmeceutical formulations is also on the rise. The complex polyphenolic compounds in tea provide the same protective effect for the skin as for internal organs. They have been shown to modulate biochemical pathways that are important in cell proliferation, inflammatory responses, and responses of tumor promoters.¹²⁵ Green tea has been shown to have anti-inflammatory and antioxidant effects in both human and animal skin.

Since inflammation and oxidative stress appear to play a significant role in the aging process, green tea may also have antiaging effects by decreasing inflammation and scavenging free radicals. Researchers have found that the main active ingredient in green tea, epigallocatechin-3-gallate (EGCG), works well as an anti-inflammatory, antioxidant, and sunscreen. Topical green tea applied to human skin provided a photoprotective effect, reduced the number of sunburn cells, protected epidermal Langerhans cells from UV damage, and reduced the DNA damage that formed after UV radiation.¹²⁶ Green tea polyphenols, when combined with traditional sunscreens may therefore have an additive or synergistic photoprotective effect. Green tea was also found to decrease melanoma cells in tissue culture and squamous cell carcinoma cell formation with topical and oral administration in mice. It improves wound healing by increasing keratinocyte cell differentiation and has been shown to inhibit *Streptococcus* species and *Escherichia coli*.¹²⁷

Natural flavonoids such as green or black tea polyphenols reduced UVB-induced erythema, tumorigenesis, and immunosuppression in mice.^{128,129} White tea appears to be a more potent antioxidant than green tea. Black tea has a much lower content of catechins than green tea, but a higher content of other flavonoids such as quercetin, theaflavin, and kaempferol. Black tea extracts applied before and after ultraviolet light challenge decreased signs of cutaneous photodamage, carcinogenesis, and inflammation in human and mouse skin.^{120,130} Most cosmeceutical products containing tea extracts or phenols have not been tested in controlled clinical trials, but these substances have shown compelling evidence for antioxidant, anti-inflammatory, and anticarcinogenic activities.

GRAPE SEED

Grape seed extract is a powerful antioxidant that has a concentrated source of oligomeric proanthocyanidins. Proanthocyanidins, which belong to the flavonoid family, are found in several plant sources including grape seed, red cabbage, blackberry, cranberry, black currant, almonds, red wine, green, and black tea. Beyond their antioxidant properties, these substances have shown the capacity to stabilize collagen and elastin, thus improving skin elasticity and preventing or reducing wrinkling by protecting against photodamage.

There are countless studies that demonstrate many health benefits of grape seed extract. Some of the cutaneous benefits include its antibacterial, antiviral, and anti-inflammatory properties and its activity as a powerful antioxidant which appears to be more potent than both vitamins C and E. Proanthocyanidins have been shown to protect the skin against UVB damage as well as foster wound healing. Grape seed applied topically improved cutaneous photoprotection to UVB, inhibits histamine synthesis, promotes wound healing, reduces apoptosis induced by chemotherapy, reduces vascular engorgement, is cytotoxic to adenocarcinoma, and inhibits *Streptococcus*. It protects DNA against oxidation more effectively than vitamins C and E and stabilizes collagen and elastin by inhibiting matrix metalloproteinases.^{120,130,131} These properties strongly suggest that grape seed extract should be a helpful tool in improving photoaged skin and preventing further UV-induced damage.

Tea tree oil is the essential oil from an Australian native plant, *Melaleuca alternifolia*. Because of its antimicrobial and anti-inflammatory properties, this oil has become increasingly popular in recent years as a nonprescription remedy for multiple mucocutaneous disorders. It has been used for a wide range of conditions including acne, psoriasis, cold sores, fungal infections, wounds, and sunburn. Tea tree oil contains numerous natural compounds, although the terpinenes, cineole, and terpinen appear to be the key medicinal components.

Tea tree oil's broad-spectrum antimicrobial activity has been well acknowledged and it is also being studied for its potential effectiveness against herpes simplex virus.^{132–135} Multiple double-blinded clinical trials have found tea tree oil to effectively treat acne and fungal or yeast infections. In the treatment of moderate acne, topical application of 5% tea tree oil showed a comparable effect to 5% benzoyl peroxide with significant reduction in inflamed lesions and open and closed comedones.

While studies support the antimicrobial and anti-inflammatory properties of this essential oil, tea tree oil does not appear to have antioxidant activity.

Aloe vera is one of the most widely used herbal products and is well known for its soothing properties. It is a very common ingredient in many over-the-counter products such as moisturizers, soaps, shampoos, deodorants, sunscreens, shaving creams, and other skin care products designed to heal, soothe, and protect the skin. It has been used in the treatment of various skin conditions including burns, bacterial and fungal infections, eczema, psoriasis, ulcers, radiation and stasis dermatitis, seborrhea, acne, and herpes zoster.

The substance released from the plant leaves is a colorless gel and contains mucopolysaccharides, glucomannan, allantoin, flavonoids, amino acids, hydroxyquinine glycosides, carboxypeptidases, and minerals.^{120,136} Aloe is documented to have potent anti-inflammatory and antimicrobial properties, as well as the capability to increase blood flow and enhance wound healing. Research has suggested that the active constituents in aloe, namely emodin, mannose, and lectin, have anticarcinogenic potential and may prevent UVB-induced cutaneous immune suppression by repairing UVB-induced damage to epidermal Langerhans' cells.¹³⁷ However, its significance to the treatment of photoaging has not been established.

Licorice, derived from the roots of *Glycyrrhiza glabra*, has been used medicinally for thousands of years. Glycyrrhizin, one of the main ingredients in licorice, is believed to contribute to the herb's many healing properties. Studies suggest that licorice extract protects the skin from the damaging effects of UV light, reduces inflammation, and soothes irritation.^{138,139}

Licorice extract has also demonstrated efficacy in treating atopic dermatitis by significantly decreasing erythema, edema, and pruritus.¹⁴⁰ Glabridin, one of the primary active constituents in licorice extract, prevented UV-induced redness and inflammation when preapplied to the skin. Licorice extract was also found to reduce melanin synthesis.¹³⁹ Furthermore, licorice extract's antioxidant activity protects skin against damage caused by free radical and ROS.¹³⁸

Licochalcone A, derived from *Glycyrrhiza inflata*, has been shown to inhibit proinflammatory cytokines, eicosanoids, and ROS in human skin cells.¹⁴¹ The potent anti-inflammatory action and high therapeutic index of the compound indicated that lipochalcone-A may be a promising addition to skin care formulations. In one study, a skin

care regimen containing lipochalcone provided significant improvement in the erythema associated with erythematous rosacea or red facial skin not attributable to rosacea.¹⁴² Formulations containing lipochalcone appear to be compatible with other commonly prescribed rosacea medications.

The use of natural and especially botanical products is increasingly sought out by consumers because of a perceived benefit rather than for objective results. The claims of effectiveness are not always convincing. Therefore, in the years to come, the cosmeceutical industry is challenged to provide evidence of the effectiveness of these compounds. In general, this category of cosmeceutical is recommended for those with more sensitive skin or those patients on more aggressive protocols needing soothing hydration. Since this category of cosmeceutical is more likely to appeal to patients with skin easily inflamed and irritated, it is important to remind patients that “natural” is not synonymous with “nonallergenic.”

MISCELLANEOUS

■ Hydroxy Acids

The ancient Egyptians used animal oils, salt, alabaster, and sour milk to aesthetically improve the skin. Lactic acid, an alpha-hydroxy acid, of the sour milk was used to produce smooth skin.¹⁴³ Hydroxy acids are broken into two groups, alpha and beta hydroxy acids. Glycolic acid is the most commonly used alpha hydroxy acid and salicylic acid is the only beta hydroxy acid used in dermatology. The hydroxy acid group penetrates the epidermis down to the dermal–epidermal junction.

■ Alpha Hydroxy Acid

Alpha hydroxy acids are the most frequently used superficial peel agents, and glycolic and lactic acid are the most commonly commercially synthesized.¹⁴⁴ The common structure is a linear carboxylic acid with an attached hydroxyl group at the second (alpha) carbon and a variable length carbon chain.¹⁴⁵ This group is hydrophilic and derived primarily from fruit and food acids. Glycolic acid is derived from sugarcane, lactic acid from sour milk, malic acid from apples, and citric acid from citrus fruits.

At the epidermal level, alpha hydroxy acids act by disrupting corneocyte cohesion at the stratum corneum–stratum granulosum junction.¹⁴⁶ This reduced adhesion

of keratinocytes and increased turnover of the stratum corneum leads to apparently smoother skin.¹⁴⁴ Dermal effects include increased synthesis of collagen and glycosaminoglycans.¹⁴⁵

In one study it was reported that 4 weeks of treatment with 12% lactic acid ammonium lactate lotion resulted in a 19% increase in epidermal thickness and increased amounts of glycosaminoglycans.¹⁴⁶ Additionally, Smith noted increase in epidermal skin firmness and thickness as well as increase in dermal firmness and thickness with application of 12% lactic acid. Clinically, this resulted in reports of improved smoothness.¹⁴⁷ Indications for alpha hydroxy acids include mild photodamage, fine wrinkling, hyperkeratosis (i.e., ichthyosis, xerotic eczema), acne, and actinic keratosis.

■ Beta-Hydroxy Acid (Salicylic Acid)

Salicylic acid is the only beta-hydroxy acid used in cosmeceuticals. Salicylic acid is an organic aromatic carboxylic acid with a hydroxyl group in the beta position. It is hydrophobic and lipophilic; therefore, it is able to enter the sebaceous unit. Salicylic acid acts by solubilizing intercellular corneocyte adhesion, working from the outermost layer downward.¹⁴⁸ Indications for salicylic acid include comedonal acne (OTC 2%); exfoliation of calluses, corns, and warts; removal of undesquamated corneocytes from the scalp of patients with seborrheic dermatitis and psoriasis; facial peels for patients with photodamage and acne; and patients with sensitive skin (less stinging and burning as the primary ingredient does not readily penetrate to the dermis).¹⁴⁹

ANTIGLYCATION END PRODUCTS

The skin, lens, arteries, and tendons are tissues rich in extracellular matrix and long-lived proteins such as collagen and elastin. These tissues have in common age-related increases in stiffness and loss of elasticity.¹⁵⁰ Additionally, these age-related processes are accelerated in patients with insulin-resistant diabetes caused by increased levels of glucose and subsequent increases in glycation. It has been proven that cross-linking of collagen generated by long-term glycation causes increased stiffness in human skin.¹⁵¹

During the aging process, the skin's important structural proteins such as collagen and elastin are constantly undergoing attack by free radicals and nonenzymatic changes

such cross-linking. Abnormal protein cross-linking is mediated by advanced glycation end products (AGEs), which are generated by glycosylation of proteins by reducing sugars such as glucose.¹⁵² This nonenzymatic interaction occurs between the sugar's carbonyl groups and the protein's amino groups to form an unstable Schiff's base which rearranges rapidly to form a more stable Amadori product. Amadori products undergo slow reaction involving rearrangement, oxidation, and dehydration to form a stable, heterogeneous advanced glycosylation end products.¹⁵³ There have been 20 AGEs identified in the human skin. Of these, CML (*N*-(carboxymethyl)lysine) and pentosidine have been shown to increase fivefold in aging skin.^{150,151}

It has recently been demonstrated that the receptor for advanced glycation end products (RAGE) is highly expressed in the skin and upregulated by AGEs and tumor necrosis factor- α .¹⁵⁴ This AGE-RAGE interaction may influence the process of skin aging through mild stimulation of extracellular matrix gene expression, as demonstrated in human foreskin fibroblasts.¹⁵⁴

In addition to the stiffening induced by glycation, it has also been shown that exposure of AGEs to UVA generates ROS.^{155,156} Thus, glycation, by way of generation of ROS, mediate oxidative damage to proteins, DNA, and lipids.

Aminoguanidine (Pimagedine) reacts rapidly with α,β -dicarbonyl compounds to prevent the formation of AGEs.^{157,160} Biomechanical studies of human skin have shown that aminoguanidine decreases the stiffening effect of glycation.¹⁵¹

Research has shown that carnosine serves as an alternative and competitive glycation target.¹⁵⁸ In addition, Szwergold recently demonstrated that carnosine also acts by decomposing the very first intermediates, Schiff bases, of the nonenzymatic glycation cascade. Carnosine also significantly inhibits the inactivation of esterase glycation as well as potentially protecting Cu, Zn superoxide dismutase, and aspartate transferase from glycation inactivation.^{159,160}

Current studies aimed at both preventing glycation as well as breaking previously formed abnormal cross-links offer potential therapeutic tools for the dermatologist's armamentarium.

■ Fructosamine-3-kinase

Fructosamine-3-kinase functions as a deglycating enzyme.¹⁶¹ This enzyme functions by phosphorylating the bound sugar, which becomes unstable and releases 3-deoxyglucosone and restores the original amino group,

thereby reversing the glycation process at an early stage.^{161,160}

■ Alt-711

ALT-711, 4,5-dimethylthiazolium (alagebrium), has been hypothesized to break AGE's protein cross-links.^{152,159} Monnier et al. recently challenged this, arguing that the original target of ALT-711 is not formed during glycation and that the benefit of ALT-711 results from metal chelation. However, this group recognizes the possible application of ALT-711 in cleaving other compounds associated with AGEs.¹⁵⁰

BLEACHING AGENTS

Hyperpigmentation is a cosmetically important and chronic condition with many etiologies. Moreover, it has been recognized that facial pigmentation significantly contributes toward perceived age.¹⁶² Antipigment cosmeceuticals, therefore, are an important component of any cosmeceutical protocol when irregular pigmentation is evident.

Causes of acquired hyperpigmentation include skin diseases, such as melasma and postinflammatory pigmentation; exogenous causes, such as ultraviolet exposure and drugs; and other causes such as pregnancy and systemic diseases.¹⁶³ Hydroquinone is the most commonly used and effective bleaching agent.^{145,164} Hydroquinone blocks the conversion of dopa to melanin through the inhibition of tyrosinase. It is available over the counter in concentrations of 2%, and available in prescription products at concentrations of 3% to 4%.¹⁶⁴

The most common side effects of hydroquinone are skin irritation and rarely contact dermatitis, and both are easily treated with topical steroids. A rare side effect is the development of exogenous ochronosis, however, most reported cases are a result of improper product use. This can be avoided by alternating hydroquinone in 4-month cycles with a natural depigmenting agent and thorough patient education.¹⁶³ Because of this rare side effect, which is generally accepted to be preventable, hydroquinone has come under the scrutiny of the FDA. Of the five to six million users of hydroquinone in the United States, only 15 cases of ochronosis have been reported in the American literature.¹⁶⁵ In one of these cases the patient had been using 2% hydroquinone for 30 years.¹⁶⁶

Kojic acid is a tyrosinase inhibitor produced by *Aspergillus* and *Penicillium* fungi.¹⁴⁵ Kojic acid is used

extensively in Asia at concentrations of 1% to 4%; these products are typically used twice per day for 1 to 2 months.¹⁶³ Erythema and irritant contact dermatitis are commonly reported side effects.^{163,145}

Comparable pigmentation and erythema reductions were demonstrated in patients being treated with kojic acid cream 2% and hydroquinone 4% both with glycolic acid 10% in the treatment of 22 patients with melasma.¹⁶⁷ The study was designed as a randomized, double-blinded, split faced trial. Another study reported superior results in improvement of melasma in a product containing 0.05% hydroquinone, 10% glycolic acid, and 2% kojic acid versus a product containing 2% hydroquinone, 10% glycolic acid, and no kojic acid.¹⁶⁸

SUMMARY

The practice of aesthetic medicine has become more complex in recent decades. Not only are there more surgical procedures being devised, but new, noninvasive treatments are becoming increasingly popular. Often the presenting patient has numerous signs of skin aging, and so multiple problems must be addressed and products combined with procedures for best results. Cosmeceuticals are one aspect of the nonsurgical antiaging treatment protocol. Since drug claims are not made by the manufacturers of these products, the use of these products is not entirely evidence-based. Physicians can responsibly include these treatments for their patients, however, if the treatment rationale can be justified by applying known science and by monitoring the patient for individual benefit as well as any untoward effects.

REFERENCES

1. Lupo MP. Cosmeceutical peptides. *Dermatol Surg.* 2005;31:832.
2. Keller KL, Fenske NA. Uses of vitamin A, C, and E and related compounds in dermatology: a review. *J Am Acad Dermatol.* 1998;39:611.
3. Nguyen EQ, Wolverton SE. Systemic retinoids. In: Wolverton SE, ed. *Comprehensive Dermatologic Drug Therapy*. Philadelphia, PA: W.B. Saunders Company; 2001:269.
4. Kligman LH, Do CH, Kligman AM. Topical retinoic acid enhances the repair of ultraviolet damaged dermal connective tissue. *Connect Tissue Res.* 1984;12:139.
5. Sporn MB, Roberts AB. Retinoids. In: Goodman DS ed. *Biology, Chemistry, and Medicine*. Bethesda, MD;1994:25.
6. Ortonne JP. Retinoid therapy of pigmentary disorders. *Dermatol Therapy.* 2006;19:280.
7. Oblong JE, Bissett DL. Retinoids cosmeceuticals procedures. In: Zoe Diana Draelos ed. *Cosmetic Dermatology: Cosmeceuticals*. China: Elsevier, Inc; 2005:36.
8. Lee MS, Lee KH, Sin HS, et al. A newly synthesized photostable retinol derivative (retinyl *N*-formyl aspartamate) for photodamaged skin: profilometric evaluation of 24-week study. *J Am Acad Dermatol.* 2006;55:221.
9. Bissett D. Topical niacinamide and barrier enhancement. *Cutis.* 2002;60:S8.
10. Bissett DL, Oblong JE, Berge CA. Niacinamide: A B vitamin that improves aging facial skin appearance. *Dermatol Surg.* 2005;31:860.
11. Tanno O, Ota Y, Kitamura N, et al. Nicotinamide increases biosynthesis of ceramides as well as other stratum corneum lipids to improve the epidermal permeability barrier. *Br J Dermatol.* 2000;143:524.
12. Gehring W. Nicotinic acid/niacinamide and the skin. *J Cosm Dermatol.* 2004;3:88.
13. Gensler HL. Prevention of photimmunosuppression and photocarcinogenesis by topical nicotinamide. *Nutr Cancer.* 1997;29:157.
14. Shalita AR, Smith JG, Parish LC, et al. Topical nicotinamide compared with clindamycin gel in the treatment of inflammatory acne vulgaris. *Int J Dermatol.* 1995;34:434.
15. Wozniacka A, Wiecsorkowska M, Gebicki J, et al. Topical application of 1-methylnicotinamide in the treatment of rosacea: a pilot study. *Clin Expir Dermatol.* 2005;30:632.
16. Soma Y, Kashima M, Imaizumi A, et al. Moisturizing effects of topical nicotinamide on atopic dry skin. *Int J Dermatology.* 2005;44:201.
17. Draelos ZD, Ertel K, Berge C. Niacinamide-containing facial moisturizer improves skin barrier and benefits subjects with Rosacea. *Cutis.* 2005;76:135.
18. Imokawa G. Lipid abnormalities in atopic dermatitis. *J Am Acad Dermatol.* 2001;45:S29.

19. Hakozaki T, Minwalla L, Zhuang J, et al. The effect of niacinamide on reducing cutaneous pigmentation and suppression of melanosome transfer. *Br J Dermatol*. 2002;147:20.
20. Kim H, Jacobson M, Kim M, et al. A topical niacin pro-drug enhances wound healing by stimulation of leptin secretion. *J Invest Derm*. 2002;119:347(A840),.
21. Ebner F, Heller A, Rippke F, et al. Topical use of dexpanthenol in skin disorders. *Am J Clin Dermatol*. 2002;3:427.
22. Biro K, Thaci D, Ochsendorf FR, et al. Efficacy of dexpanthenol in skin protection against irritation: a double-blind, placebo-controlled study. *Contact Dermatitis*. 2003;49:20.
23. Nishikimi M, Fukuyama R, Minoshima S, et al. Cloning and chromosomal mapping of the human nonfunctional gene for L-gulonogamma-lactone oxidase, the enzyme for L-ascorbic acid biosynthesis missing in man. *J Biol Chem*. 1994;269:13685.
24. Shindo Y, Witt E, Han D, et al. Enzymatic and nonenzymatic antioxidants in epidermis and dermis of human skin. *J Invest Dermatol*. 1994;102:122.
25. Frei B, England L, Ames BN. Ascorbate is an outstanding antioxidant in human blood plasma. *Proc Natl Acad Sci USA*. 1989;86:6377.
26. Farris, PK. Topical vitamin C: a useful agent for treating photoaging and other dermatologic conditions. *Dermatol Surg*. 2005;31:814.
27. Scarpa M, Stevenato R, Viglino, et al. Superoxide ion as active intermediate in the autoxidation of ascorbate by molecular oxygen: effect of superoxide dismutase. *J Biol Chem*. 1983;258:6695.
28. Cabelli DE, Bielski BHJ. Kinetics and mechanism for the oxidation of ascorbic acid/ascorbate by HO_2/O_2 radicals: a pulse radiolysis and stopped flow photolysis study. *J Phys Chem*. 1983;87:1805.
29. Chou PT, Khan AU. L-ascorbic acid quenching of singlet delta molecular oxygen in aqueous media: generalized antioxidant property of vitamin C. *Biochem Biophys Res Commun*. 1983;115:932.
30. Pinnell SR. Cutaneous photodamage, oxidative stress, and topical antioxidant protection. *J Am Acad Dermatol*. 2003;48:1.
31. Hanson KM, Simon JD. Epidermal transurocanic acid and the UVA-induced photoaging of the skin. *Proc Natl Acad Sci USA*. 1998;95:10576.
32. Rabe JH, Mamelak AJ, McElgunn PJS, et al. Photoaging: mechanisms and repair. *J Am Acad Dermatol*. 2006;55:1.
33. Darr D, Combs S, Dunston S, et al. Topical vitamin C protects porcine skin from ultraviolet radiation-induced damage. *Br J Dermatol*. 1992;27:247.
34. Carcamo JM, Pedraza A, Borquez-Ojeda O, et al. Vitamin C suppresses TNF-alpha induced NF kappa B activation by inhibiting I kappa B alpha phosphorylation. *Biochemistry*. 2002;41:12995.
35. Davidson JM, Luvalle PA, Zoia O, (et al). Ascorbate differentially regulates elastin and collagen biosynthesis in vascular smooth muscle cells and skin fibroblasts by pretranslational mechanisms. *Biol Chem*. 1997;272:345.
36. Alster T, West TB. Effect of topical vitamin C on postoperative carbon dioxide laser resurfacing erythema. *Dermatol Surg*. 1998;24:331.
37. Pinnell S, Yang H, Omar M, et al. Topical L-ascorbic acid: percutaneous absorption studies. *Dermatol Surg*. 2001;27:137.
38. Traikovitch SS. Use of topical ascorbic acid and its effects on photodamaged skin topography. *Arch Otolaryngol Head Neck Surg*. 1999;125:1091.
39. Humbert PG, Haftek M, Creidi P, et al. Topical ascorbic acid on photoaged skin. Clinical, topographical, and ultrastructural evaluation: double-blind study vs. placebo. *Exp Dermatol*. 2003;12:237.
40. Fitzpatrick RE, Rostan EF. Double-blind, half-face study comparing topical vitamin C and vehicle for rejuvenation of photodamage. *Dermatol Surg*. 2002;28:231.
41. Lupo MP. Antioxidants and vitamins in cosmetics. *Clin Dermatol*. 2001;19:467.
42. Perricone NV. The photoprotective and anti-inflammatory effects of topical ascorbyl palmitate. *J Geriatr Dermatol*. 1993;1:5.
43. Meves A, Stock SN, Beyerle A, et al. Vitamin C derivative ascorbyl palmitate promotes ultraviolet-B-induced lipid peroxidation and cytotoxicity in keratinocytes. *J Invest Dermatol*. 2002;119:1103.
44. Jurkovic P, Sentjurc M, Gasperlin M, et al. Skin protection against ultraviolet induced free radicals in microemulsions. *Eur J Pharm Biopharm*. 2003;56:59.

45. Kobayashi S, Takehana M, Itoh S, et al. Protective effect of magnesium-L-ascorbyl-2-phosphate against skin damage induced by UVB irradiation. *Photochem Photobiol.* 1996;64:224.
46. Geesin JC, Gordon JS, Berg RA. Regulation of collagen synthesis in human dermal fibroblasts by the sodium and magnesium salts of ascorbyl-2-phosphate. *Skin Pharmacol.* 1993;6:65.
47. Kameyama K, Saka C, Kondoh S, et al. Inhibitory effect of magnesium L-ascorbyl-2-phosphate (VC-PMG) on melanogenesis in vitro and in vivo. *J Am Acad Dermatol.* 1996;34:29.
48. Ikeno H and Ohmori K. Clinical study of safety and efficacy of 5% sodium L-ascorbyl-2 phosphate lotion to treat acne scars after glycolic acid peels. *Cosmet Dermatol.* 2003;1:29.
49. Ikeno J, Nishikawa T. Clinical evaluation of topical sodium-L-ascorbyl-2-phosphate lotion (5% w/v) in patients with acne. Paper presented at: 3rd Annual Meeting of The Society for Investigative Dermatology; May 15-18, 2002; Los Angeles, CA.
50. Ochiai Y, Kaburagi S, Obayashi K et al. A new lipophilic pro-vitamin C, tetra-isopalmitoyl ascorbic acid (VC-IP), prevents UV-induced skin pigmentation through its antioxidative properties. *J Dermatol Sci.* 2006;44:37.
51. Packer JE, Slater TF, Willson RL: direct observation of a free radical interaction between vitamin E and vitamin C. *Nature.* 1979;278:737.
52. Stoyanovsky DA, Osipov An, Quinn PJ, et al. Ubiquinone-dependent recycle of vitamin E radicals by superoxide. *Arch Biochem Biophys.* 1995; 323:343.
53. Thiele JJ, Hsieh SN, Ekanayake-Mudiyanselage S. Vitamin E: critical review of its current use in cosmetic and clinical dermatology. *Dermatol Surg.* 2005;31:815.
54. Baumann LS, Spencer J. The effects of topical vitamin E on the cosmetic appearance of scars. *Dermatol Surg.* 1999;25:311.
55. Jenkins M, Alexander JW, MacMillan BG, et al. Failure of topical steroids and vitamin E to reduce postoperative scar formation following reconstructive surgery. *J Burn Care Rehabil.* 1986;7:309.
56. Alberts DS, Goldman R, Xu MJ, et al. Disposition and metabolism of topically administered alpha-tocopherol acetate: a common ingredient of commercially available sunscreens and cosmetics. *Nutr Cancer.* 1996;26:193.
57. Trevithick JR, Mitton KP. Topical application and uptake of vitamin E acetate by the skin and conversion to free vitamin E. *Biochem Mol Biol Int.* 1993;31:869.
58. Baschong W, Artmann C, Hueglin D, et al. Direct evidence for bioconversion of vitamin E acetate into vitamin E: an ex vivo study in viable human skin. *J Cosmet Sci.* 2001;52:155.
59. West, MD. The cellular and molecular biology of skin aging. *Arch Dermatol.* 1994;130:87.
60. Furth, JJ. The steady-state levels of type I collagen mRNA are reduced in senescent fibroblasts. *J Gerontol.* 1991;46:B122.
61. Khorramizadeh, MR, Tredget EE, Telasky C, et al. Aging differentially modulates the expression of collagen and collagenase in dermal fibroblasts. *Mol Cell Biochem.* 1999;194:99.
62. Chung, JH, Seo JY, Choi HR, et al. Modulation of skin collagen metabolism in aged and photoaged human skin in vivo. *J Invest Dermatol.* 2001;117:1218.
63. Varoni, J, Spearman D, Perone P, et al. Inhibition of type I procollagen synthesis by damaged collagen in photoaged skin and by collagenase-degraded collagen in vitro. *J Am Pathol.* 2001;158:931.
64. Farrar, GE Jr., Krosnick A. Milestones in clinical pharmacology: wound healing. *Clin Ther.* 1991;13:430.
65. Goodson, W, Hohn D, Hunt TK, et al. Augmentation of some aspects of wound healing by a "skin respiratory factor." *J Surg Res.* 1976;21:125.
66. Bentley, JP, Hunt TK, Weiss JB, et al. Peptides from live yeast cell derivative stimulate wound healing. *Arch Surg.* 1990;125:641.
67. Keller, SJ, Levin RH, Fang, J. Isolation and characterization of a tissue respiratory factor from bakers' yeast. *J Cell Biol.* 1991;304:21.
68. Remedyfind: Capryloyl glycine. <http://www.remedyfind.com/treatments/138/2029/>. Accessed 2006.
69. Morganti, P, Randazzo SD, Fabrizi G, et al. Decreasing the stinging capacity and improving the antiaging activity of AHAs. *J Appl Cosmetol.* 1996; 14:79.

70. Chauduri, RK, Majewski, G, Guitterez, G, et al. A unique liposomal system provides age-defying benefits in skin care by modulating the fibroblast phenotype via keratinocytes to induce synthesis of collagen III. *Cosmetics and Toiletries*. 2000;115:53.
71. Croda: Dipeptide valyl-tryptophan. <http://www.croda.co.kr/sederma/Catalogue/FR/Produitsfr/eyelisfr.pdf>. Accessed 2006.
72. Kitazawa, M, Iwasaki, K. Reduction of ultraviolet light-induced oxidative stress by amino acid-based iron chelators. *Biochemica et Biophysica Acta*. 1999;1473:400.
73. Babizhayev, MA, Seguin MC, Gueyne J, et al. L-carnosine (beta-alanyl-L-histidine) and carbinine (beta-alanylhistamine) act as natural antioxidants with hydroxyl-radical-scavenging and lipid-peroxidase activities. *Biochem J*. 1994;304(Pt 2):509.
74. Kitazawa, M, Ishitsuka, Y, Kobayashi M, et al. Protective effects of an antioxidant derived from serine and vitamin B6 on skin of photo aging in hairless mice. *Photochem Photobiol*. 2005;81:970.
75. Bowery, NG, Enna SJ. Gamma-aminobutyric acid (B) receptors: first of the functional metabotropic heterodimers. *J Pharmacol Exp Ther*. 2000;292:2.
76. Kamoun, A, Landreau JM, Godeau G, et al. Growth stimulation of human skin fibroblasts by elastin-derived peptides. *Cell Adhes Commun*. 1995;3:273.
77. Tajima, S, Wachi H, Uemura Y, et al. Modulation by elastin peptide VGVAPG of cell proliferation and elastin expression in human skin fibroblasts. *Arch Dermatol Res*. 1997;289:489.
78. Senior, RM, Griffen GL, Mecham RP, et al. Val-Gly-Val-Ala-Pro-Gly, a repeating peptide in elastin, is chemotactic for fibroblasts and monocytes. *J Cell Biol*. 1984;99:870.
79. Njieha FK, Morikawa T, Tuderman L, et al. Partial purification of a procollagen C proteinase. Inhibition by synthetic peptides and sequential cleavage of type I procollagen. *Biochemistry*. 1982;21(4):757.
80. Katayama, K, Armendariz-Borunda J, Raghov R, et al. A pentapeptide from type I procollagen promotes extracellular matrix production. *J Biol Chem*. 1993;268:9941.
81. Robinson, LR, Fitzgerald NC, Doughty DG, et al. Topical palmitoyl pentapeptide provides improvement in photoaged human facial skin. *Int J Cosmet Sci*. 2005;27(3):155.
82. Maquart, FX, Pickart L, Laurant M, et al. Stimulation of collagen synthesis in fibroblast cultures by the tripeptide-copper complex glycyl-L-histidyl-L-lysine-Cu²⁺. *FEBS Lett*. 1999;238:343.
83. Cauchard, JH, Berton A, Godeau G, et al. Activation of latent transforming growth factor beta 1 and inhibition of matrix metalloproteinase activity by thrombospondin-like tripeptide linked to elaidic acid. *Biochem Pharmacol*. 2004;67:2013.
84. Vartanian, AJ, Dayan SH. Facial rejuvenation using botulinum toxin A: a review and updates. *Facial Plast Surg*. 2004;20:11.
85. Blanes-Mira, C, Clemente J, Jodas G, et al. A synthetic hexapeptide (argireline) with antiwrinkle activity. *Int J Cosmet Sci*. 2002;24:303.
86. Gutierrez, LM, Cannes JM, Ferrer-Monteil AV, et al. A peptide that mimics the carboxy-terminal domain of SNAP-25 blocks Ca²⁺-dependent exocytosis in chromaffin cells. *FEBS Lett*. 1995;372:39.
87. Gutierrez, LM, Viniegra S, Reuda J, et al. A peptide that mimics the C-terminal sequence of SNAP-25 inhibits secretory vesicle docking in chromaffin cells. *J Biol Chem*. 1997;272:2634.
88. Blanes-Mira C, Merino JM, Valera E, et al. Small peptides patterned after the N-terminus domain of SNAP-25 inhibit SNARE complex assembly and regulated exocytosis. *J Neurochem*. 2004;88:124.
89. Grossman, R. The role of dimethylaminoethanol in cosmetic dermatology. *Am J Clin Dermatol*. 2005; 6(1):39.
90. Pickart, L, Freedman JH, Loher WJ, et al. Growth-modulating plasma tripeptide may function by facilitating copper uptake into cells. *Nature*. 1980;288:715.
91. Simeon A, Emonard H, Hornebeck W, et al. The tripeptide-copper complex glycyl-L-histidyl-L-lysine-Cu²⁺ stimulates matrix metalloproteinase-2 expression by fibroblast cultures. *Life Sci*. 2000;67:2257.
92. Simeon A, Wegrowski Y, Bontemps J, et al. Expression of glycosaminoglycan and small proteoglycans in wounds: modulation by the tripeptide-copper complex glycyl-L-histidyl-L-lysine-Cu²⁺. *J Invest Dermatol*. 2000;115:962.
93. Buffoni F, Pino R, Dal Pozzo A. Effect of tripeptide-copper complexes on the process of skin wound

- healing and on cultured fibroblasts. *Arch Int Pharmacodyn. Ther.* 1995;330(3):345.
94. Fitzpatrick RE. Endogenous growth factors as cosmeceuticals. *Dermatol Surg.* 2005;31(7):827.
 95. Fitzpatrick RE, Rostan EF. Reversal of photodamage with topical growth factors: a pilot study. *Dermatol Surg.* 2003;5(1):25.
 96. Bello YM, Falabella AF, Eaglstein WH. Tissue-engineered skin. Current status in wound healing. *Am J Clin Dermatol.* 2001;2(5):305.
 97. Goldman R. Growth factors and chronic wound healing: past, present, and future. *Adv Skin Wound Care.* 2004;17(1):24.
 98. Wang XJ, Greenhalgh DA, Bickenbach JR, et al. Expression of a dominant-negative type II transforming growth factor beta (TGF-beta) receptor in the epidermis of transgenic mice blocks TGF-beta-mediated growth inhibition. *Proc Natl Acad Sci USA.* 1997;94(6):2386.
 99. Yin L, Morita A, Tsuji T. The crucial role of TGF-beta in the age-related alterations induced by ultraviolet irradiation. *J Invest Dermatol.* 2003;120(4):703.
 100. Roberts AB, Sporn MB, Assoian RK, et al. Transforming growth factor type beta: rapid induction of fibrosis and angiogenesis in vivo and stimulation of collagen formation in vitro. *Proc Natl Acad Sci USA.* 1986;83(12):4167.
 101. Ignatz RA, Endo T, Massague J. Regulation of fibronectin and type I collagen mRNA levels by transforming growth factor-beta. *J Biol Chem.* 1987;15;262(14):6443.
 102. Quan T, He T, Kang S, et al. Solar ultraviolet irradiation reduces collagen in photoaged human skin by blocking transforming growth factor-beta type II receptor/Smad signaling. *Am J Pathol.* 2004;165(3):741.
 103. Fisher GJ, Kang S, Varani J, Bata-Csorgo Z, Wan Y, et al. Mechanisms of photoaging and chronological skin aging. *Arch Derm.* 2002;138(11):1462.
 104. Yamamoto M, Yanaga H, Nishina H, et al. Fibrin stimulates the proliferation of human keratinocytes through the autocrine mechanism of transforming growth factor-alpha and epidermal growth factor receptor. *Tohoku J Exp Med.* 2005;207(1):33.
 105. Li Y, Fan J, Chen M, Li W, et al. Transforming growth factor-alpha: a major human serum factor that promotes human keratinocyte migration. *J Invest Dermatol.* 2006;126(9):2096.
 106. Xu Y, Shao Y, Voorhees JJ, et al. Oxidative inhibition of receptor-type protein-tyrosine phosphatase kappa by ultraviolet irradiation activates epidermal growth factor receptor in human keratinocytes. *J Biol Chem.* 2006;281(37):27389.
 107. Wan YS, Wang ZQ, Voorhees J, et al. EGF receptor crosstalks with cytokine receptors leading to the activation of c-Jun kinase in response to UV irradiation in human keratinocytes. *Cell Signal.* 2001;13(2):139.
 108. Ritte L, Varani J, Kang S, et al. Retinoid-induced epidermal hyperplasia is mediated by epidermal growth factor receptor activation via specific induction of its ligands heparin-binding EGF and amphiregulin in human skin in vivo. *J Invest Dermatol.* 2006;126(4):732.
 109. Brown GL, Curtisinger L 3rd, Brightwell JR, et al. Enhancement of epidermal regeneration by biosynthetic epidermal growth factor. *J Exp Med.* 1986;163(5):1319.
 110. Hong JP, Kim YW, Jung HD, et al. The effect of various concentrations of human recombinant epidermal growth factor on split-thickness skin wounds. *Int Wound.* 2006;3(2):123.
 111. Kwon YB, Kim HW, Roh DH, et al. Topical application of epidermal growth factor accelerates wound healing by myofibroblast proliferation and collagen synthesis in rat. *J Vet Sci.* 2006;7(2):105.
 112. Geer DJ, Swartz DD, Andreadis ST. Biomimetic delivery of keratinocyte growth factor upon cellular demand for accelerated wound healing in vitro and in vivo. *Am J Pathol.* 2005;167(6):1575.
 113. Tang A, Gilchrist BA. Regulation of keratinocyte growth factor gene expression in human skin fibroblasts. *J Dermatol Sci.* 1996;1(1):41.
 114. Tsuboi R, Sato C, Kurita Y, Ron D, Rubin JS, et al. Keratinocyte growth factor (FGF-7) stimulates migration and plasminogen activator activity of normal human keratinocytes. *J Invest Dermatol.* 1993;101(1):49.
 115. Singh RP, Agarwal R. Flavonoid antioxidant silymarin and skin cancer. *Antioxid Redox Signal.* 2002;4:655.
 116. Dhanalakshmi S, Mallikarjuna GU, Singh RP, Agarwal R. Silibinin prevents ultraviolet radiation-caused

- skin damages in SKH-1 hairless mice via a decrease in thymine dimer positive cells and an upregulation of p53-p21/Cip1 in epidermis. *Carcinogenesis*. 2004;25:1459.
117. Katiyar SK, Korman NJ, Mukhtar H, et al. Protective effects of silymarin against photocarcinogenesis in a mouse skin model. *J Natl Cancer Inst*. 1997;89:556.
 118. Baumann LS. Cosmeceutical critique: soy and its isoflavones. *Skin & Allergy News*. 2001;32:17.
 119. Wei H. Photoprotective action of isoflavone genistein: models, mechanisms, and relevance to clinical dermatology. *J Am Acad Dermatol*. 1998;39:271.
 120. Thornfeldt C. Cosmeceuticals containing herbs: fact, fiction, and future. *Dermatol Surg*. 2005;31:873.
 121. Weil H, Spencer JM, Gelfand J, et al. The soy isoflavone genistein: a new agent in dermatology. *Cosmet Dermatol*. 2001;14(2):13.
 122. Paine C, Sharlow E, Liebel F, et al. An alternative approach to depigmentation by soybean extracts via inhibition of the PAR-2 pathway. *J Invest Dermatol*. 2001;116(4):587.
 123. Draelos ZD: Cosmetics. <http://www.emedicine.com.htm>. Accessed April 2005.
 124. Lin FH, Lin JY, Gupta RD, et al. Ferulic acid stabilizes a solution of vitamins C and E and doubles its photoprotection of skin. *J Invest Dermatol*. 2005;125(4):826.
 125. Katiyar SK, Ahmad N, Mukhtar H. Green tea and skin. *Arch Dermatol*. 2000;136(8):989.
 126. Elmets C, Singh D, Tubesing K, et al. Cutaneous photoprotection from ultraviolet injury by green tea polyphenols. *J Am Acad Dermatol*. 2001;44:425.
 127. Spencer JM. Chemoprevention of skin cancer and photoaging. *Cosmet Dermatol*. 2001;14(6):25.
 128. Wang ZY, Huang MT, Ferraro T, et al. Inhibitory effect of green tea in the drinking water on tumorigenesis by ultraviolet light and 12-*o*-tetradecanoylphorbol-13-acetate in the skin of skh-1 mice. *Cancer Res*. 1992;52:1162.
 129. Zhao J, Jin X, Yaping E, et al. Photoprotective effect of black tea extracts against UVB-induced phototoxicity in skin. *Photochem Photobiol*. 1999;70:637.
 130. Jellin JM, Gregory P, Butz F, et al. *Natural Medicines Comprehensive Database*. 3rd ed. Stockton, CA: Therapeutic Research Facility; 2000.
 131. Baumann LS. Cosmeceutical Critique: grapeseed. *Skin & Allergy News*. 2004;35:26.
 132. Walton SF, McKinnson M, Pizzutto S. Acaricidal activity *Melaleuca alternifolia* (tea tree oil). *Arch Dermatol*. 2004;140:563-566.
 133. Baumann LS. Cosmeceutical Critique: tea tree oil. *Skin & Allergy News*. 2002;33:14.
 134. Nenoff P, Haustein UF, Brandt W. Antifungal activity of the essential oil of *Melaleuca alternifolia* (tea tree oil) against pathogenic fungi in vitro. *Skin Pharmacol*. 1996;9(6):388-394.
 135. Hammer K, Carson C, Riley T. In-vitro activity of essential oils, in particular *Melaleuca alternifolia* (tea tree) oil and tea tree oil products, against *Candida* spp. *J Antimicrob Chemother*. 1998;42(5):591-595.
 136. McKeown E. Aloe vera. *Cosmet Toilet*. 1987;102:64-65.
 137. Lee, CK, Han SS, Mo YK, et al. Prevention of ultraviolet radiation-induced suppression of accessory cell function of Langerhans cells by Aloe vera gel components. *Immunopharmacology*. 1997;37(2-3):153-162.
 138. Di Mambro VM, Fonseca MJ. Assays of physical stability and antioxidant activity of a topical formulation added with different plant extracts. *J Pharm Biomed Anal*. 2005;37(2):287-295.
 139. Yokota T, Nishio H, Kubota Y, Mizoguchi M. The inhibitory effect of glabridin from licorice extracts on melanogenesis and inflammation. *Pigment Cell Res*. 1998;11(6):355-361.
 140. Saeedi M, Morteza-Semnani K, Ghoreishi MR. The treatment of atopic dermatitis with licorice gel. *J Dermatolog Treat*. 2003;14(3):153-157.
 141. Dieck K, Ceilley RI, Immeyer J, et al. Anti-inflammatory properties of licochalcone A from *Glycyrrhiza inflata* on various human cells. Paper presented at: Annual Meeting of the American Academy of Dermatology; 2005; New Orleans, LA.
 142. Weber, TM, Scholermann A, Burger A, et al. Tolerance and efficacy of a skin care regimen containing lipochalcone A for adults with erythematic rosacea and facial redness. Paper presented at: Annual Meeting of the American Academy of Dermatology, 2005; New Orleans, LA.
 143. Brody HJ, Monheit GD, Resnik SS, et al. A history of chemical peeling. *Dermatol. Surg*. 2000;26(5):405.

144. Kockaert M, Neumann M. Systemic and topical drugs for aging skin. *J Drugs Dermatol*. 2003; 2(4):435.
145. Clark CP. Office-based skin care and superficial peel: the scientific rationale. *Plast Reconstr Surg*. 1999;104(3):854.
146. Lavker RM, Kaidbey K, Leyden JJ. Effects of topical ammonium lactate on cutaneous atrophy from a potent topical corticosteroid. *J Am Acad Dermatol*. 1992;26:535.
147. Smith WP. Epidermal and dermal effects of topical lactic acid. *J Am Acad Dermatol*. 1996;35:388.
148. Roberts DL, Marshall R, Marks R. Detection of the action of salicylic acid on the normal stratum corneum. *Br J Dermatol*. 1980;103:191.
149. Draelos ZD. Topical agents used in association with cosmetic surgery. *Sem Cutan Med Surg*. 1999; 18(2):112.
150. Monnier VM, Sell DR. Prevention of protein damage by the maillard reaction *in vivo*. *Rejuvenation Research*. 2006;9(2):264.
151. Reihnsner R, Menzel EJ. Two-dimensional stress-relaxation behavior of human skin as influenced by nonenzymatic glycation and the inhibitory agent aminoguanidine. *J Biomechan*. 1998;31:985.
152. Vasan S, Foiles P, Founds H. Therapeutic potential of breakers of advanced glycation end product-protein cross-links. *Arch Biochem Biophys*. 2003; 419:89.
153. Dyer DG, Dunn JA, Thorpe SR, et al. Accumulation of maillard reaction products in skin collagen in diabetes and aging. *J Clin Invest*. 1993;91:2463.
154. Lohwasser C, Neureiter D, Weigle B, et al. The receptor for advanced glycation end products is highly expressed in the skin and upregulated by advanced glycation end products and tumor necrosis factor- α . *J Invest Dermatol*. 2006;126:291.
155. Mazaki S, Okano Y, Sakurai H. Generation of active oxygen species from advanced glycation end-products (AGE) during ultraviolet light (UVA) irradiation and a possible mechanism for cell damage. *Biochim Biophys Acta*. 1999;1428:45.
156. Jeanmaire C, Danoux L, Pauly G. Glycation during human dermal intrinsic and actinic ageing: an *in vivo* and *in vitro* model study. *Br J Dermatol*. 2001; 145:10.
157. Thornalley PJ. Use of aminoguanidine (Pimage-dine) to prevent the formation of advanced glycation end-products. *Arch Biochem Biophys*. 2003; 419:31.
158. Szwergold, BS. Carnosine and anserine act as effective transglycating agents in decomposition of aldose-derived Schiff bases. *Biochem Biophys Res Comm*. 2005;336:36-41.
159. Yan H, Harding JJ. Carnosine protects against the inactivation of esterase induced by glycation and a steroid. *Biochim Biophys Acta*. 2005;1741:120.
160. Harding JJ, Ganea E. Protection against glycation and similar posttranslational modifications of proteins. *Biochim Biophys. Acta*. 2006;1764:1436.
161. Szwergold BS, Howell S, Beisswenger PJ. Human fructosamine-3-kinase; purification, sequencing, substrate specificity, and evidence of activity *in vivo*. *Diabetes*. 2001;50:2139.
162. Bissett D, Matts P, McCclanahan S et al. Reduction of skin hyperpigmentation-cosmetic considerations, edited by Gray, J International Congress and Symposium Series No 264, The Royal Society of Medicine Press Limited; 2006.
163. Rendon MI, Gaviria JI. Review of skin-lightening agents. *Dermatol Surg*. 2005;31:886.
164. Grimes PE. Melasma: etiologic and therapeutic considerations. *Arch Dermatol*. 1995;131:1453.
165. Snider RI, Theirs BH. Exogenous ochronosis. *J Am Acad Dermatol*. 1993;38:662.
166. Kramer KE, Lopez A, Stefanato CM, et al. Exogenous ochronosis. *J Am Acad Dermatol*. 2000;42:869.
167. Hautea S, Verallo-Rowell V. A randomized double-blind clinical trail to compare melanin reduction in melasma by 4% hydroquinone versus 2% kojic acid creams both with 10% glycolic acid. In: Verallo-Rowell VM, eds. *Skin in the Tropics: Sunscreens and Hyperpigmentation*. Pasig City, Philippines: Anvil Publishing, Inc; 2001:272.
168. Lim JT. Treatment of melasma using kojic acid in a gel containing hydroquinone and glycolic acid. *Dermatol Surg*. 1999;25(4):282-284.
169. Padayatty SJ, Katz A, Wang Y, et al. Vitamin C as an antioxidant: evaluation of its role in disease prevention. *J Am Coll Nutr*. 2003;22:18.

CHAPTER 4

Facial Rejuvenation

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INTRODUCTION

Facial aging is a complex process involving a combination of factors. Over time, the skin undergoes many alterations including loss of elasticity and subcutaneous fat as well as fat redistribution caused by gravitational forces. Additionally, skin aging is accelerated by environmental influences such as sun exposure, smoking, and nutritional factors. The net effect of these physiologic and environmental factors is the visible appearance of wrinkles and folds, development of an increasing number of lentigines, and loss of overall skin luster. Photoaged skin creates the appearance of age beyond years, and a tired, dull appearance.

Predictably, the demand for facial rejuvenation cosmetic procedures is growing at a rate commensurate with the aging baby boomer population in the United States. While no single therapeutic modality will be sufficient to address a spectrum of age-related facial changes, many therapies target individual aspects very well. This chapter reviews the many modalities that can be used for facial rejuvenation. While techniques are covered separately, it is essential to realize that they are often used in combination to maximally improve the appearance of aging skin.

KEY POINTS

- Nonablative photorejuvenation lasers and pulsed light devices are directed at improving specific photo-induced color changes in the skin, including lentigines.
- With the addition of photodynamic therapy, results with these nonablative light sources can often be expedited or even amplified.
- Chemical peels of varying depths have long been utilized to address some of these pigment irregularities as well as the textural changes seen in aging skin.
- Neurotoxins are increasingly being employed to prevent dynamic rhytides from becoming “etched-in” lines in various facial regions.
- Traditional ablative resurfacing technologies that have focused on improving prominent textural changes and distinct static rhytides are being supplanted by newer fractional technologies associated with less downtime.
- For volume-depleted facial areas, soft-tissue augmentation has expanded beyond traditional collagen agents to a broader range of agents and devices with increasing safety, efficacy, and duration.
- While surgical lifting techniques remain the most effective procedures for prominent skin laxity, less-invasive procedures such as suture-suspension lifting procedures and radiofrequency tissue tightening technologies have captured much attention for lesser degrees of facial laxity.

Nonablative Techniques for Photorejuvenation of Facial Skin

Girish S. Munavalli and Robert A. Weiss

BACKGROUND

Photorejuvenation is defined as the utilization of visible or infrared light energy sources to reverse the process of sun-induced or environmental damage to the skin.¹ The primary objective of nonablative rejuvenation is to

improve photoaged skin, which includes the appearance of dyspigmentation, static fine wrinkles, coarse texture, prominent pores, telangiectasis, and mild skin laxity. A secondary objective includes the recontouring of surface irregularities, such as scars, via subsequent dermal collagen remodeling.

In order to accomplish photorejuvenation in a nonablative or nondestructive fashion, visible disruption to the

overlying epidermis should not occur. A common pathway for histological and clinical improvement in facially treated skin centers around thermal-damage-induced collagen neogenesis in the dermis. More recent work has proposed reactive oxygen species as another pathway for destruction of old collagen molecules and creation of new ones.² Nonablative devices used for light-energy-based treatment have been categorized into different groups to facilitate their understanding and utilization. A simple scheme involves grouping the devices into coherent and monochromatic laser sources versus broadband light sources.

Melanin, along with water and hemoglobin (oxyhemoglobin), accounts for the major target chromophores for nonablative lasers and light devices in the dermis and epidermis. Further categorization of these devices on the basis of their absorption spectrum aids physicians when evaluating nonablative devices for the treatment of specific photoaging lesions (Table 4.1).

TABLE 4.1 ■ Light and Laser Devices with Specific Chromophores

Visible Laser Sources	Chromophore
Frequency doubled Nd:YAG, KTP (green, 532 nm)	Hemoglobin
Pulsed dye (yellow)	Melanin
Short pulse—585 nm	Hemoglobin
Long Pulse—595nm	
Visible Light Sources	
Pulsed light, 500–1200 nm	Hemoglobin, melanin, water
Light emitting diodes narrowband	NA
Infrared Laser Sources	
1064 nm (Q-switched and millisecond domains)	Melanin, hemoglobin, water
1320 nm	Water
1450 nm	Water
1540 nm	Water
1540-nm nonablative fractional resurfacing	Water
1550-nm nonablative fractional resurfacing	Water
Non-light Sources	
Monopolar radiofrequency	NA

TABLE 4.2 ■ Lesion-Specific Treatment Options with Nonablative Techniques

Photoaging Lesion	Technique/Treatment Options
Telangiectasias	IPL, PDL (extended pulse), large spot size 532 nm
Mottled pigmentation	IPL, large spot size 532 nm, microdermabrasion adjunctive
Q-switched alexandrite or ruby	As adjunctive for resistant areas
Mild rhytides	IPL, 1320 nm, 1450 nm, “nonablative” fractional resurfacing
Moderate rhytides	Nonablative fractional resurfacing, 1320 nm, 1450 nm, monopolar RF, Nd:YAG (1064 nm)
Sagging	Monopolar RF
Surface textural smoothing	All of the above, LED photomodulation

IPL, intense pulsed light; PDL, pulse dye laser; RF, radiofrequency; LED, light-emitting diode.

PATIENT SELECTION AND PREPROCEDURE EVALUATION

The algorithm shown in Table 4.2 is the treatment plan that is often outlined for new patients. Specific problems faced in each component of photoaging are discussed in the following sections. Patients are informed that less downtime will likely require more treatment sessions. More aggressive device settings can be used with fewer total treatments but this will result in possibly more downtime and side effects such as edema, erythema, purpura, and crusting of pigmented regions.³

TECHNIQUE

In our experience, no single nonablative device can address all the issues of photoaging. A multidevice regimen can take advantage of different wavelength–tissue interactions and various delivery systems to treat different properties of recalcitrant lesions. These sentiments have been echoed by other investigators who routinely use multiple devices to treat photoaged skin; they have noted that patient satisfaction is enhanced by this multimodal approach to treatment.⁴ Lesion-specific treatment

options are discussed forthwith. As a rule of thumb, following all treatments, we strongly advise continued use of bland moisturizers and a broad-spectrum sunscreen. Cold packs, low-potency topical steroids, and elevation of the upper torso during sleep can all minimize posttreatment edema.

The components of photoaging can be treated with great success using the algorithm discussed in Table 4.2. All patients do not respond to the treatment, and results vary; however, most patients with realistic expectations do well.

■ Telangiectasias

Many lighter-skinned patients have telangiectasias as the primary component of photoaging (Figure 4.1). If these are accompanied by other manifestations such as mottled pigmentation, as is typically the case, we recommend a technique that is most likely to treat all the components of the problem. In our practice, we initially suggest intense pulsed light (IPL) for Fitzpatrick skin types I to III. Extensive experience with a single device is preferable to limited use of multiple devices as this allows the physician to develop expertise with a tool and its particular

settings. For example, with the Lumenis Quantum SR (Lumenis, Santa Clara, CA), we use a double pulse with a 560-nm filter having a very thin layer of gel to allow the chilled crystal to reduce surface temperature. The pulse durations we use are 2.4 and 6 milliseconds with a 10-millisecond delay between the pulses for a total pulse time of 18.4 milliseconds and a total fluence of 25 to 28 J/cm². Following these parameters, excellent results can be achieved (Figure 4.2). We recently have also added the Palomar Starlux (Palomar, Burlington, MA) with the “G” handpiece at a 20-millisecond pulse duration with energy of 36 to 40 J/cm² as an alternative.

There are some patients who are poorly responsive because of the curved topography along the nasal alae, which can be difficult to treat with large crystals of IPL. Some patients may find IPL too painful or there may be multiple fine red telangiectasias less than 0.3 mm in diameter that may not be responsive to IPL. For these patients, we use the extended pulse dye laser (PDL) as an alternative. We typically use the Cynosure Cynergy (Cynosure, Chelmsford, MA) with the Zimmer air cooler set at 1. Typically, the starting settings are 10-millisecond duration, 10-mm spot, and 7.5 J/cm². Treatment



Figure 4.1 Presence of telangiectasis on the untreated cheek on the left and its absence on the right following two pulsed light treatments



Figure 4.2 Resolution of telangiectasias after two treatments with a pulsed light device

consists of a 50% overlap or double pulsing (as necessary) to observe a visible photodarkening with diminished capillary refill. We rarely reduce the pulse duration to 6 milliseconds as this would increase the risk of purpura; however, some patients may, on rare occasions, require shorter pulse durations with accompanying purpura to induce clearing of vessels that are less than 0.2 to 0.3 mm in diameter.

Recently we have begun to use the Gemini (Laserscope, San Jose, CA) for initial treatment of the telangiectatic component of photoaging. This device features a large (10-mm) spot size of 532-nm KTP. This is now recommended for fast results in patients with the most severe photodamage. As 532 nm is absorbed most intensely by hemoglobin and melanin, we believe that fewer treatments are necessary for these components of photoaging (Figure 4.3). The patients are informed that there is a higher risk for edema lasting up to 3 days and erythema lasting for 24 hours, but the trade-off for fewer treatments is one that some patients are willing to accept.

Care should be taken to maintain slight overlap between pulses to ensure confluent treatment of the target area and avoid “skip” (untreated) areas as these skip areas may leave a footprint of dyspigmentation.⁵ Complications from these devices can include ulceration, scarring, and textural



Figure 4.3 Improvement in lentigines and telangiectasias following two treatments with 532 nm KTP

changes. Fortunately, when proper patient selection and appropriate treatment parameters are utilized, these complications are rare. Careful observation of the immediate tissue reaction for vessel coagulation and epidermal changes is essential. Striking erythema or graying of the epidermis is a sign of excessive thermal damage and possible tissue necrosis. Adequate cooling is essential to minimize damage to surrounding tissue and potential for scar formation.⁵

■ Lentigines

Our approach to the patient with dyschromias is very similar to that for telangiectasias as these two components of photoaging often coexist. However, in patients for whom mottled pigmentation is the predominant component, we are more likely to use microdermabrasion as an adjunctive procedure. This is mainly to enhance the penetration of topical hydroquinones and retinoids, which is also essential for long-term success, especially in regions of the world where the sun is always intense. We may also change the settings of the various devices utilized to address differences in skin type and pigmentation, such as changing to a shorter pulse-duration cycle of 2.4 and 4 milliseconds with a 10-millisecond delay for a total duration of 16.4 milliseconds typically at a fluence of 25 to 28 J/cm².

Our clinical experience has been that shorter pulse durations as well as lower cutoff filters with IPL improve the response to pigmentation. The lower cutoff filters of the V handpiece of the Starlux device may be used for residual pigmentation. We prefer not to use extended PDL lasers as the efficacy can be less compared to devices with lower wavelengths that are more highly absorbed by melanin. We are more likely to suggest large spot size 532 nm for patients with considerable mottled pigmentation photoaging component, as melanin would effectively absorb 532 nm more effectively (Figure 4.3).

If persistent pigmented spots remain following full-face IPL or 532-nm treatment, options include follow-up with more intense treatment using higher fluences, lower cutoff filters, and less skin-surface cooling. Alternatively, Q-switched alexandrite (755 nm) or Q-switched ruby (694 nm) lasers can be used for spot treatment of resistant lesions. Complications from device treatment of lentigines are similar to that of telangiectasias. Because these patients are more apt to have sun-tanned skin, it is critical to assess for this prior to treatment. To prevent tissue damage, it is important that Q-switched lasers are not used in a pulse stacking or overlapping fashion.

■ Mild Rhytides

In addition to the above treatments, it is often advisable to employ a longer wavelength water-only absorbing laser to treat fine rhytides. This type of laser heats up water molecules around collagen causing a controlled wound-healing response.¹⁻³ The devices used for this include the 1320-nm CoolTouch 3 (CoolTouch3, CoolTouch Corp, Roseville, CA) with fluence of 17 to 19 J/cm², with the latest iteration allowing for pre-, mid-, and post-laser pulse cooling. Pulse duration is fixed at 50 milliseconds, so cooling and fluence are the primary treatment parameters modified. Our preferred settings for collagen contraction are 10 milliseconds precooling, 5 milliseconds mid-cooling, and 10 milliseconds post-cooling for a total of 25 milliseconds of cooling. We no longer monitor skin temperature at the conclusion of a pulse but allow the device to cut off when skin temperature has reached 40°C.

A typical treatment now involves two to three passes. As skin temperature rises so does the pain of treatment. The vast majority of patients elect to have a topical anesthetic applied 45 minutes prior to the procedure. Topical anesthetic is rarely needed for IPL, extended PDL, or large spot (532 nm). For patients with thinner skin, we find that the use of a less-penetrating wavelength (more highly absorbed by water) may yield better results. For those patients, we use the 1450-nm Smoothbeam (Candela, Wayland, MA) with typical settings of 11 to 12 J/cm² energy, a fixed pulse duration, 35 to 40 milliseconds of cryogen spray, and a 6-mm spot size. Two to three passes are the norm. As with any infrared laser, double pulsing or pulse stacking should be avoided, as this can cause blistering and could result in depressed scars.

■ Moderate Rhytides

The treatment of moderate rhytides is probably the most controversial and leads to the most passionate discussions. Many devices are now available for this application. The problem of moderate rhytides may also be accompanied by significant sagging, which can make both treatment and response more difficult. Our approach at this time is to treat with a 1320 nm wavelength using three passes as described earlier with 1450 nm utilized as an additional pass. We have the least success with this indication; when used for upper lip moderate rhytides, which often require

ablative resurfacing, even lower success rates are achieved.

■ Textural Smoothing

Textural roughness is a problem that seems to be aided by almost every device listed. This, in our opinion, arises as a consequence of the thermal energy applied and the papillary dermal capillary leakage and subsequent inflammatory and cytokine pathways activated. This effect is seen most dramatically in photographic images in which the skin takes on a more flash-reflective appearance. One of the newest concepts in acquiring this smoother appearance of the epidermis that is related to stimulating very superficial collagen production in a nonthermal way is light-emitting diode (LED) photomodulation (Gentlewaves, LightBioScience, Virginia Beach, VA). The initial clinical trial with this device has been previously described.⁶ We also use LED photomodulation to enhance the results from all other techniques. Our clinical experience is that photomodulation can also help to reduce erythema from IPL, PDL, 532 nm-, 1320 nm-, 1450 nm-devices, and fractional resurfacing. We routinely treat all patients with the nonthermal LED device, immediately following these treatment modalities.

REFERENCES

1. Weiss RA, McDaniel DH, Geronemus RG. Review of nonablative photorejuvenation: Reversal of the aging effects of the sun and environmental damage using laser and light sources. *Semin Cutan Med Surg.* 2003;22(2):93-106.
2. Lubart R, Friedmann H, Lavie R, et al. A reasonable mechanism for visible light-induced skin rejuvenation. *Lasers Med Sci.* 2007;22(1):1-3.
3. Weiss RA, Weiss MA, Beasley KL, Munavalli G. Our approach to non-ablative treatment of photoaging. *Lasers Surg Med.* 2005;37(1):2-8.
4. Trelles MA, Mordon S, Calderhead RG. Facial rejuvenation and light: Our personal experience. *Lasers Med Sci.* 2007;22(2):93-99.
5. Dawson E, Willey A, Lee K. Adverse events associated with nonablative cutaneous laser, radiofrequency, and light-based devices. *Semin Cutan Med Surg.* 2007;26(1):15-21.
6. Weiss RA, McDaniel DH, Geronemus RG, Weiss MA. Clinical trial of a novel non-thermal LED array for reversal of photoaging: Clinical, histologic, and surface profilometric results. *Lasers Surg Med.* 2005;36(2):85-91.

Fractional Laser Resurfacing

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BACKGROUND

For years, the industry standard for treating signs of severe photoaging has been ablative laser resurfacing with carbon dioxide and erbium lasers. Despite major advances in technology over the past decade, this continues to be the case. The overall trend in the current patient population, however, is for devices that deliver more benefits with less downtime and risk. The most significant advance in this direction over the past 20 years has been the arrival of fractional photothermolysis. Fractional resurfacing provides significant and measurable improvement for photoaging, mild to moderate rhytides, acne scars, and skin pigmentation with a minimal downtime recovery period.

■ Theory and Technology

The ability to deliver effective outcomes with minimal downtime results from the fractional nature of these treatments. Instead of a continuous beam that scans 100% of the skin surface ablating tissue as it goes, the collimated beam is fractionated into hundreds of micrometer-sized beams that are separated by islands of untreated skin. Furthermore, the infrared laser does not vaporize the skin cells; rather, they form columns of desiccated tissue known as microthermal treatment zones.¹

The advantages of treating only focal zones of tissue are twofold. First, by leaving zones of untreated skin, reservoirs of healthy skin remain that enable rapid migration and tissue proliferation to repair the damaged areas.^{2,3} Second, because there is a readily available

source for tissue repair, it is possible to generate fluences capable of penetrating to depths of 500 to 1000 microns with a significantly reduced risk of scarring. This represents a distinct advantage over ablative devices such as CO₂ or erbium lasers where ablation of 100% of the skin surface at such depths would result in permanent disfiguring scars.

The shortcoming of this fractional technology is that it falls short of matching the degree of skin tightening seen in ablative laser modalities. The dramatic type of wound healing and tissue repair that is created through full thickness and total ablation of the epidermis is not replicated. Future studies comparing the differences between cell signaling pathways following ablative versus fractional technologies may hold the key to future advances in laser resurfacing for photorejuvenation.

PATIENT SELECTION AND COUNSELING

The factors that must be considered when deciding upon the appropriate laser therapy include healing time or patient downtime, the level of desired improvement, patient expectations, and safety and side effect profile of the device. In sum, the physician and the patient must

weigh the adverse effects versus the potential outcome in deciding which device to use. In general, severe conditions and an advanced age require more aggressive therapies to affect a substantial result.

Conditions with proven benefit from fractional resurfacing include dyschromias, photoaging, mild to moderate rhytides, acne scars (Figures 4.4 and 4.5), and melasma.^{4,5} Various case reports have described the use of fractional resurfacing for many other conditions including poikiloderma of Civatte⁶ and colloid milium⁷ as well as other dermatologic issues. The recovery time usually consists of several days of erythema, with occasional episodes of light peeling after more aggressive treatments. In most cases, patients are able to continue the use of cosmetics and sunscreens. Exercise and daily activities are not limited by these treatments, in contrast to ablative resurfacing.

A final consideration is the safety profile offered by fractional resurfacing. Fractional treatment of the skin provides skip areas for rapid healing with minimal risk for long-term adverse effects making it safe and effective for most skin types. Furthermore, the safety of this technology permits safe and effective applications for treating “off-face” skin areas such as neck, hands, and



Figure 4.4 Improvement in periorbital rhytides following fractional resurfacing. This patient is shown before (left panel) and after (right panel) two treatments with a combination 1440- and 1320-nm fractional resurfacing device



Figure 4.5 Improvement in acneiform scars following multiple treatments with a fractional resurfacing device. This patient is shown before (left panel) and after (right panel) five treatments with a 1550-nm fractional resurfacing laser. Fractional resurfacing provides an effective and safe option for treating patients with darker skin phototypes, as shown here, with only a rare occurrence of posttreatment hyperpigmentation

chest.⁸ This is a significant advantage over ablative technologies that carry a high risk of scarring when used on off-face areas.

Potential side effects of fractional photothermolysis devices include a low incidence of superficial blistering leading to several days of peeling, a low incidence of transient hyperpigmentation in darker phototype patients, and rare transient edema of 1 to 2 days' duration. One series examined posttreatment hyperpigmentation in Asian patients and reported an incidence of 7% to 12% depending on the energy and density used.⁹ The chance of permanent scarring is minimal and hypopigmentation has not been reported.

TECHNIQUE

■ Devices

Since the first fractional device (Fraxel SR®, Reliant Palo Alto, CA) was approved for use in 2004, interest in this technology by both patients and physicians has continued to soar. This popularity has led to an increasing number of fractional devices available in the market along with a new category of ablative fractional devices being offered today. The majority of these devices are similar in their basic characteristics. They are near-infrared lasers that use water as the target chromophore, and have the capability of penetrating to depths of approximately 400 to 1000 microns. Differences mainly consist of variations in proprietary technology that fractionates the beam.

The original Fraxel® laser and subsequent models use an optical tracking system of mirrors that creates the fractionated beams and focuses them on the skin. This mechanism is able to provide constant output and adjustment corrections for changes in velocity. This means that the user delivers the pulses while constantly moving the handpiece across the skin surface. Other manufacturers use special lenses that fractionate the beam. Cynosure's Affirm® and Affirm Multiplex® lasers use such lenses to fractionate the beam into 1000 pixels in a 10-mm spot size. These are then delivered as single pulses. The addition of forced air cooling adds improved patient comfort and safety to these systems. Palomar has a 1540-nm laser (Lux1540™) that uses a fractionating lens to evenly disperse the 10-mm laser beam and deliver energy penetrating to depths of over 1000 microns.

Newer technologies include the aforementioned Affirm Multiplex® and fractional CO₂ and erbium lasers. The Affirm Multiplex® represents a unique type of treatment in that it offers the combination of two wavelengths in one treatment session. Each treatment delivers sequential pulses of both 1440-nm and 1320-nm lasers. The 1440-nm wavelength is predominantly absorbed in the epidermis and upper dermis penetrating to depths of 400 to 500 microns, and the 1320-nm laser penetrates more deeply to provide bulk heating of deeper dermal elements (2 mm) for additional skin tightening (Figure 4.6).

Fractionated CO₂ (Active FX®, Lumenis) and erbium lasers (Harmony Pixel™, Alma Lasers™; PROFractional, Sciton Inc.) offer a different type of fractional resurfacing



Figure 4.6 Photographs demonstrating tissue tightening following treatment with a combined 1440- and 1320-nm fractional resurfacing laser. This patient is shown before (left panel) and after (right panel) two treatments with the combined laser. Notice the improved skin tightening with overall reduction in the volume of the jowl area and the concomitant reduction in the “marionette lines”

for skin rejuvenation. Longer wavelengths of CO₂ (10600 nm) and erbium (2640 nm) lasers do not penetrate as deeply as the near-infrared systems mentioned earlier. As a result, the energy is absorbed more superficially in the epidermis. The fractionated beams still permit more rapid healing than total surface ablation. Multiple passes may be used to increase the density of the fractionated spots in order to achieve a more targeted or more complete treatment. These devices produce moderate erythema for 4 to 6 days along with some fine epidermal peeling. Early results show efficacy for mild to moderate rhytides, dyschromias, and photoaging after a series of treatments.

The future offers further advances in fractional technology. Currently, a fractionated CO₂ device that is able to deliver ablative pulses that penetrate several hundred microns is under investigation.^{10,11} The combination of deep tissue penetration and the thermal diffusion characteristics of the CO₂ laser reportedly provide immediate tissue contraction approaching that of full-surface tissue ablation.

■ Step-by-Step Technique

Preparation for a fractional laser treatment is very basic. Any history of recurrent perioral herpes infection is pre-treated to prevent outbreaks. Prior to the treatment, all patients are asked to wash their face to remove any cosmetics, sunscreens, or excess moisturizers. Application of a topical anesthetic is then performed. Although topical anesthetics are not necessary, their use can be very helpful for the comfort of a patient and also enable the operator to use higher energy settings during the treatment.

Generally, it is recommended that the passes be performed after a short period of cooldown time but before any significant edema begins. Any excess fluid disperses the laser energy and lessens the effect on the target tissues. In our practice, we routinely treat a regional unit such as the cheek or one side of the neck and then perform a second pass before moving on to the next region. This allows ample cooldown time without permitting excess time for edema.

Repeat treatments are generally performed every 3 to 4 weeks. Shorter intervals can be performed; however,

the skin is often more sensitive and easily irritated by closely spaced treatments. There are no published studies, to date, evaluating the ideal treatment interval for fractional resurfacing. Typically, four to five treatments are performed; however, these must be individualized to the patient's needs and desires. Many patients with melasma or acne scars receive more than five treatments as they continue to see improvement with each additional session.

■ Complications to Avoid

Caution must be exercised when using high potency or combination topical anesthetic preparations on large surface areas. A case of a single patient with early signs of lidocaine toxicity and elevated serum lidocaine levels following fractional resurfacing has been reported.¹² This patient received treatment on the face and neck following pretreatment with a 30% lidocaine ointment. A follow-up study revealed that patients do experience an increased uptake of topical anesthetic with a concomitant rise in their serum lidocaine levels immediately after fractional laser treatment.

As with any laser treatment, determining the appropriate energy settings and number of passes is essential for achieving the desired result. Each subsequent pass creates a higher density of microthermal treatment zones, correlating to increased resurfacing and enhanced thermal activation. An excessive number of passes or a very high density results in blistering of the skin.

POSTPROCEDURE CONSIDERATIONS

After completion of a treatment, patients will experience varying degrees of erythema and mild edema, with most patients experiencing only minimal symptoms. Posttreatment skin care consists of using moisturizers, gentle cleansers, and sunscreen. Patients are also asked to avoid any retinoids, glycolic acid preparations, or harsh cleansers for 48 to 72 hours after treatment. The skin becomes more sensitive following these treatments, and the use of any of these products can create additional erythema and irritation; several cases of allergic contact dermatitis have occurred in the early posttreatment period. Excessive scrubbing or harsh cleansing can result in focal epidermal sloughing or microblisters. If any areas of blistering are seen, the application of petrolatum ointment for 2 to 3 days can encourage wound repair.

REFERENCES

1. Manstein D, Herron GS, Sink RK, Tanner H, Anderson RR. Fractional photothermolysis: A new concept for cutaneous remodeling using microscopic patterns of thermal injury. *Lasers Surg Med.* 2004; 34(5):426-438.
2. Laubach H, Chan HH, Rius F, Anderson RR, Manstein D. Effects of skin temperature on lesion size in fractional photothermolysis. *Lasers Surg Med.* 2007;39(1):14-18.
3. Laubach HJ, Tannous Z, Anderson RR, Manstein D. Skin responses to fractional photothermolysis. *Lasers Surg Med.* 2006;38(2):142-149.
4. Alster TS, Tanzi EL, Lazarus M. The use of fractional laser photothermolysis for the treatment of atrophic scars. *Dermatol Surg.* 2007;33(3):295-299.
5. Rokhsar CK, Fitzpatrick RE. The treatment of melasma with fractional photothermolysis: A pilot study. *Dermatol Surg.* 2005;31(12):1645-1650.
6. Behroozan DS, Goldberg LH, Glaich AS, Dai T, Friedman PM. Fractional photothermolysis for treatment of poikiloderma of Civatte. *Dermatol Surg.* 2006;32(2):298-301.
7. Marra DE, Pourabbani S, Fincher EF, Moy RL. Fractional photothermolysis for the treatment of adult colloid milium. *Arch Dermatol.* 2007;143(5): 572-574.
8. Wanner M, Tanzi EL, Alster TS. Fractional photothermolysis: Treatment of facial and nonfacial cutaneous photodamage with a 1550-nm erbium-doped fiber laser. *Dermatol Surg.* 2007;33(1):23-28.
9. Chan HH, Manstein D, Yu CS, Shek S, Kono T, Wei WI. The prevalence and risk factors of post-inflammatory hyperpigmentation after fractional resurfacing in Asians. *Lasers Surg Med.* 2007;39(5): 381-385.
10. Hantash BM, Bedi VP, Chan KF, Zachary CB. Ex vivo histological characterization of a novel ablative fractional resurfacing device. *Lasers Surg Med.* 2007; 39(2):87-95.
11. Hantash BM, Bedi VP, Kapadia B, et al. In vivo histological evaluation of a novel ablative fractional resurfacing device. *Lasers Surg Med.* 2007;39(2): 96-107.
12. Marra DE, Yip D, Fincher EF, Moy RL. Systemic toxicity from topically applied lidocaine in conjunction with fractional photothermolysis. *Arch Dermatol.* 2006;142(8):1024-1026.

Botulinum Toxin

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BACKGROUND

Botulinum toxin (BTX) is the most popular cosmetic procedure performed in the United States, with 3.2 million treatments performed in 2006.¹ In 2002, the Food and Drug Administration (FDA) approved BTX for cosmetic therapy of dynamic glabellar frown lines. Although BTX is most commonly employed for relaxation of dynamic rhytides in the upper third of the face, advanced treatment techniques for additional anatomic sites have been developed and effectively used off-label.² This chapter reviews the cosmetic indications, relevant anatomy, preoperative evaluation, injection technique, and safety profile for effective use of BTX.

Seven distinct serotypes of BTX are produced by the bacterium *Clostridium botulinum*: A, B, C1, D, E, F, and G. Irrespective of the subtype, all BTXs inhibit the release of acetylcholine from the presynaptic motor neuron, leading to chemodenervation and paralysis of muscle activity. Given the long duration of experience, proven safety profile, and FDA approval for glabellar cosmetic indication of Botox®, this is the most common BTX used by physicians in the United States. All discussions in the chapter regarding BTX-A will therefore refer to Botox®.

ANATOMIC LANDMARKS

A comprehensive understanding of superficial facial anatomy is crucial for achieving optimal aesthetic outcome and avoiding adverse effects from injection of BTX-A. Dynamic rhytides form perpendicular to underlying muscle fibers and result from prolonged, repeated muscle activity combined with the decrease in skin laxity that occurs with aging. By weakening the activity of these specific muscles and muscle groups with BTX, dynamic lines can be softened and smoothed. Treating physicians should critically evaluate patients for individual variation in muscle position and activity. Accurate toxin placement into targeted muscles is achieved by asking patients to animate. A rote “recipe” or standardized approach for the treatment of all patients is discouraged.

PATIENT SELECTION/PREPROCEDURE EVALUATION

During pretreatment evaluation, patients should be instructed that the effects of BTX-A are temporary. Onset of action is not immediate, and relaxation of target muscles typically begins 3 to 7 days following injection and does not peak for several weeks. Patients should also be informed that, although dynamic rhytides fade with onset of muscle relaxation, improvement of static “etched-in” lines may only be evident after a prolonged period of muscle inactivity with repeated treatments over time. During the consultation, deep imprinted lines as well as any preexisting baseline anatomic asymmetry should be documented and pointed out to the patient. Preprocedure photographs allow for an objective reference for comparison of posttreatment results.

Contraindications

BTX-A is contraindicated in patients with neuromuscular disorders, such as myasthenia gravis or amyotrophic lateral sclerosis, as these conditions may exaggerate the paralytic effect of the drug. In addition, care should be exercised when administering BTX-A to patients who are taking medications that interfere with neuromuscular transmission, as these drugs may theoretically increase the paralytic effect of the toxin. BTX-A should not be administered in any anatomic area with an active infection. While BTX-A is classified as pregnancy category C and should be avoided in pregnant or lactating women, there have been no reports of complications in pregnant women who have incidental exposure to BTX-A prior to becoming aware of pregnancy.³ Finally, patients with demonstrated allergy to any components of BTX-A should not be treated.

TECHNIQUE

Equipment and Instrumentation

Botox® is sold in crystalline form, with vials containing 100 units of vacuum dried powder. The package insert recommends reconstitution with 0.9% sterile, nonpreserved saline. However, using preserved saline can significantly reduce the pain and burning associated with injection

because of the presence of benzyl alcohol, which acts as an anesthetic.⁴

For cosmetic purposes, a vial can be reconstituted with a range of dilutions, often 1 to 5 mL of saline. There are no agreed upon ideal concentrations. Many physicians feel that the dilution of BTX does not make a significant difference in patient outcomes and recent studies seem to confirm this conviction.^{5,6} However, some authors have argued that higher concentrations administered via smaller injection volumes result in more precise placement of BTX-A, with a decreased risk of unintended diffusion.⁷

The package insert recommends use of Botox® within 4 hours of reconstitution. However, experience has demonstrated that BTX-A can maintain its efficacy for a much longer period of time and BTX-A reconstituted with preserved saline can be stored for up to several weeks without loss of activity or risk of bacterial contamination.^{8,9} BTX-A can be drawn into 1 mL syringes and injected via a 30 to 32 gauge needle. Some physicians prefer to use insulin syringes, which have an integrated 30-gauge syringe and a hubless system to reduce waste of volume. While the discomfort associated with injections is minimal, topical application of ice or lidocaine cream can further reduce any pain.^{10,11}

■ Patient Positioning

A patient should be positioned comfortably on an examination table, with his or her head elevated. This allows for

evaluation of how an individual's unique muscle patterns affect rhytid formation, helping to ensure accurate toxin placement.

Complete documentation of individualized treatment sites should always be performed. In this manner, consistent results can be expected with each subsequent follow-up treatment. For patients who are undergoing the treatment for the first time, a follow up at 2 weeks can be useful to assess and reassure the patient or to perform an additional touch-up treatment if some adjustment is required.

■ Step-by-Step Technique

Glabellar frown lines

Glabellar frown lines result primarily from contraction of the corrugator supercilia, which pull the brows medially, and the procerus, which pulls the brow inferiorly. Treatment of this area is the only FDA-approved cosmetic indication for BTX-A (Figure 4.7). In addition to smoothing the vertical glabellar rhytides, inactivation of the brow depressors in this region results in unopposed action of brow elevator action of the frontalis. The end result of these muscular interactions is a slight, but noticeable brow lift—a particularly desired aesthetic effect in women.¹²

Approximately 20 to 30 units of BTX-A are typically placed in this region using a five-point injection method.¹³ Male patients, and those with larger muscle mass, may require a higher number of units (40–60 units) in order to achieve the desired effect.¹⁴



Figure 4.7 (A) Glabellar complex before treatment. The corrugator muscles are easily noted with furrowing of the brow (B) 2 weeks after botulinum toxin type A treatment of glabellar complex



Figure 4.8 Injection of botulinum toxin into corrugator muscle. Note how the muscle is isolated by grasping it between the thumb and forefinger

There is normal anatomic variation in the orientation and length of the corrugator supercilii, and the origin and insertion should be identified with the patient seated in an upright position. When the patient is instructed to frown, there is often dimpling of the skin marking the lateral insertion site of this brow depressor. With the muscle contracted, the physician can grasp the corrugator between the thumb and index finger to help isolate the muscle and ensure accurate toxin placement (Figure 4.8). A general rule to avoid ptosis is to avoid injection within 1 cm of the bony superior orbital rim.

Horizontal forehead lines

Horizontal forehead lines are caused by contraction of the frontalis muscle. The frontalis is a vertically oriented muscle that originates superiorly at the galea aponeurotica and inserts inferiorly into the dermal eyebrow skin, orbicularis oculi, corrugator supercilii, and procerus. Contraction of this muscle produces movement and elevation of the eyebrows, structures often regarded as the aesthetic center of the upper face. Careful consideration of the variations in anatomy is therefore required to diminish forehead lines without inducing brow ptosis or the complete lack of expressivity. The treatment endpoint is simply “weakening” of the frontalis, as total paralysis can result in brow ptosis and heaviness. Extra caution should be exercised in older patients who use the frontalis to raise their eyebrows in order to see.

Since the lower region of the frontalis is primarily responsible for eyebrow elevation, injections are generally limited to the upper half to two-thirds of this muscle



Figure 4.9 (A) Horizontal forehead rhytides before treatment. Lines are accentuated when the patient raises the brow **(B)** 2 weeks after botulinum toxin type A treatment of frontalis

(2–3 cm above the orbital rim) in order to help minimize brow ptosis (Figure 4.9). About 10 to 20 units of BTX-A are placed in series of 5 to 10 injections across the upper half of the forehead.^{15,16}

Crow's-feet

The crow's-feet rhytides extend concentrically from the lateral canthus and are caused by the contraction of the lateral portion of the orbicularis oculi. Much like treating the frontalis, the goal is to relax, rather than immobilize the muscle. Despite successful treatment with BTX-A and achieving a significant reduction of lines, rhytides may still persist when a patient smiles because of the upward motion of the cheek. A brief explanation of superficial anatomy, along with a demonstration of the successful

reduction of rhytides with squinting as compared to smiling, can help minimize misunderstandings with patients. Approximately 8 to 12 units of BTX-A are then placed at each lateral orbit.¹⁷ A superficial bleb is usually raised in an average of three injections per side approximately 1 cm lateral to the orbital rim. Different patterns of crow's-feet prominence have been described and the location and number of injection sites will vary based on the individual.¹⁸ For a more detailed review of this region, see Chapter 5 on "Periocular Rejuvenation."

"Bunny" lines

Contraction of the nasalis muscle creates dynamic rhytides on the sides of the nose ("bunny" lines). Approximately 2 to 6 units of BTX-A can be placed just medial to the midpoint of this muscle¹⁹ (Figure 4.10).

Perioral lines

Contraction of the orbicularis oris muscle around the mouth can lead to vertically oriented muscle columns as well as "etched-in" lines. They are most commonly seen in smokers as well as photodamaged and aging women. Small amounts of BTX-A can be placed in each lip quadrant to weaken the muscles and reduce the appearance of evolving rhytides.²⁰ Generally, a total of 4 to 8 units are used when treating upper and lower lips. Injections are placed into the muscle in the visible crease on the cutaneous aspect of the vermilion border. Care must be taken to avoid weakening the muscle to such a degree that speech and mouth function are impaired.

Mouth frown

Contraction of the depressor anguli oris muscle causes a depression of the lateral corners of the mouth. By weakening this muscle with BTX-A, unopposed lip elevators (zygomaticus major and minor) can create a slight upturn of the corners of the mouth. Though subtle, this effect may result in a significant aesthetic improvement in select patients and is best used in conjunction with fillers placed in the commissures.²¹ The muscle can be identified by asking the patient to clench their jaw. Approximately 3 units of BTX-A are placed into the inferior aspect of each depressor anguli oris muscle.

Mentalis

Excessive contraction of the mentalis muscle can lead to a "peau d'orange" or pebbly appearance of the chin. The mentalis muscle originates in the lower incisive fossa and inserts in the skin of the chin. Small amounts of BTX-A (2–10 units) can be injected into the center of the mentalis muscle at the apex of the mentum in order to smoothen the chin.²²

Platysmal bands

Platysma is a large muscular sheet that arises from the pectoralis and deltoid fascia, crosses the clavicle, and extends onto the sides of the neck. Platysmal bands occur with age as the cervical skin loses its elasticity, submental fat descends, and the platysma separates to form two distinct vertical bands. Preoperative evaluation is critical for treating these patients, as BTX-A does not correct skin laxity or fat deposition. Instead, BTX-A is best used

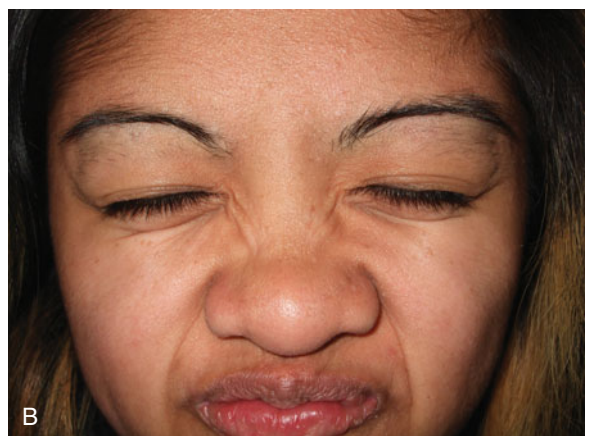
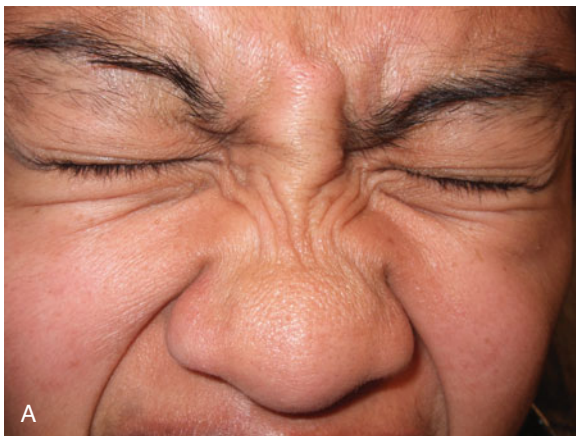


Figure 4.10 (A) "Bunny lines" before treatment. Rhytides are created from contraction of the transverse portion of the nasalis muscle (B) 2 weeks after botulinum toxin type A treatment of "bunny" lines

in those with good skin elasticity and obvious resting vertical banding.²³

Patients are asked to contract the platysma, which allows for accentuation and visualization of these vertically oriented neckbands. The physician can then grasp these bands between the thumb and index finger, and injections placed along the length of the band in 1-cm intervals. A total dose of toxin placed usually ranges from 10 to 40 units.

■ Complications to Avoid

General effects

Adverse events with the cosmetic use of BTX-A are usually temporary in nature and not of serious consequence.²⁴ BTX-A purified protein has been used safely for medical treatment for two decades. The therapeutic dose for cosmetic use of BTX-A is well below the therapeutic index LD₅₀ of 2500 to 3000 units.²⁵ Complications of treatment include transient pain at the injection site, erythema, edema, and bruising. Less common adverse effects are headache, nausea, and flu-like symptoms.²⁶ Weakness of nontargeted muscles can occur with improper injection technique. Physicians should only use FDA-approved BTX-A product, as unlicensed BTX-A has resulted in severe, life-threatening botulism.²⁷

Because of its molecular structure, BTX-A is a protein capable of producing an immune response and neutralizing antibodies in patients who receive repeated treatments and large amounts of toxin. Loss of efficacy of BTX-A caused by formation of antibodies is an unusually rare event when used for cosmetic indications, with only a single case report in the literature.²⁸

Glabellar frown lines

The most common and concerning adverse events when treating glabellar rhytides with BTX-A are upper eyelid ptosis and diplopia. If the toxin diffuses through the orbital septum into the orbit, the levator palpebra can be weakened, resulting in upper eyelid ptosis.²⁹ Care should be taken to palpate the superior bony orbital rim and inject at a point more than 1 cm above this landmark margin. In the event of eyelid ptosis, use of α -adrenergic-agonist eyedrops, such as apraclonidine hypochloride 0.5% eyedrops (Iopidine, Alcon, Fort Worth, TX), can provide some improvement.³⁰ The mechanism of action is adrenergic stimulation of the adjacent Müller's muscle (which can take over some of the function of the para-

lyzed levator palpebrae superioris), resulting in a 1 to 2 mm lift in the lids. A rare complication is diffusion of BTX-A into the orbital septum resulting in paralysis of the extraocular muscles and diplopia. Dry eyes and superficial punctate keratitis have been reported as potentially rare complications of BTX-A treatment of glabellar lines.³¹

Horizontal frown lines

Total paralysis of the frontalis muscle can cause brow ptosis and/or mask-like expressionless facies.³² Physicians will often inject the glabellar brow depressors in conjunction with the forehead to help decrease the occurrence of brow ptosis. Patients with an existing brow ptosis, or those who actively contract their frontalis to compensate for low-set eyebrows are best not treated in the forehead region.

Another adverse event unique to treating the frontalis is an exaggerated raise of the lateral eyebrow, creating a quizzical or "Mr. Spock" brow (Figure 4.11). In general, if the upper lateral fibers of the frontalis remain totally untreated, the increased resting muscle tone will raise the lateral edge of the eyebrow. The exaggerated lateral eyebrow raise can be avoided or corrected by injecting a small amount of BTX-A to relax the lateral fibers of the superior forehead that create the upward pull.

Crow's-feet

Potential adverse events in this area include bruising, ectropion, upper lid ptosis, asymmetrical smile, horizontal diplopia, and dry eyes. The increased risk of bruising in



Figure 4.11 "Quizzical" brow complication caused by unopposed muscle contraction of the upper lateral fibers of the frontalis

this anatomic location results from the presence of numerous periocular superficial veins. Use of proper lighting and stretching the skin can help the physician visualize and avoid these small veins. Ice and/or pressure immediately after the injections can also reduce the risk of bruising.

Diffusion of the toxin into the zygomaticus major and minor can occur because of overzealous chasing of the inferior muscle prominences in the crow's-feet region. Weakening of these muscles causes ipsilateral lip ptosis and an asymmetric smile.³³ Diplopia is caused by the diffusion of toxin into the extraocular muscles such as the lateral rectus. Finally, tear formation can be affected (excessive tears or dry eyes) if BTX-A directly diffuses into the lacrimal gland.^{34,35} Avoiding these complications can be maximized by palpating the bony lateral orbital rim and placing injections greater than 1 cm lateral to this site. In addition, injections should be limited to at least 1 cm above the zygoma. For a more detailed review of this region, see Chapter 5 on "Periocular Rejuvenation."

Perioral lines

Functional adverse events related to overtreatment in this area include decreased ability to purse the lips. This makes it difficult to whistle, use a straw, or play a wind musical instrument. Speech difficulties include inability to make "b" or "p" sounds. These adverse effects are more likely to occur with excessive treatment of the lower lip. Drooling and oral asymmetry can also occur in cases of severe lip weakening. Treatment of the midline upper lip should be avoided as this may result in a flattening of the Cupid's bow.

Mouth frown

Care must be taken to avoid inadvertent asymmetrical weakening of the depressor labii inferioris muscle, which can lead to an asymmetric smile. Other adverse effects include drooling or mouth incompetence and difficulty with speech.

Mentalis

Excessive toxin use or improper placement too close to the orbicularis oris or the depressor labii inferioris muscle can lead to impairment of oral motor function. Adverse effects such as difficulty in eating, drooling, and abnormal speech can be avoided with proper treatment technique.

Platysmal bands

Dysphagia, dysphonia, and neck weakness can be caused by excessive or misplaced BTX-A, which results in involvement of the strap muscles of the neck. A report of profound dysphagia resulting in nasogastric tube placement for 6 weeks indicates the potential severity of

complications in this location.³⁶ In our opinion, knowledge of the relevant anatomy, toxin dosage of no more than 40 units, and careful placement can dramatically reduce this risk.

POSTOPERATIVE CONSIDERATIONS

Immediately after the injections, cold compress or ice packs can be applied to the skin in order to reduce swelling and bruising. Some physicians recommend that patients actively use the treated muscle for a period of time following treatment to expedite toxin uptake into target muscles. There is a theoretical risk of unwanted toxin diffusion with vigorous physical activity immediately following treatment.

REFERENCES

1. The American Society for Aesthetic Plastic Surgery. *11.5 Million Cosmetic Procedures in 2004*. Available at <http://surgery.org/press/news-release.php?iid/465>. Accessed April 22, 2007.
2. Carruthers J, Fagien Matarasso S, and the Botox Consensus Group. Consensus recommendation on the use of botulinum toxin type A in facial aesthetics. *Plast Reconstruct Surg*. 2004;114:1S-22S.
3. de Oliveira Monteiro E. Botulinum toxin and pregnancy. *Skinmed*. 2006;5:308.
4. Alam M, Dover JS, Arndt KA. Pain associated with injection of botulinum A exotoxin reconstituted using isotonic sodium chloride with and without preservative: A double-blind, randomized controlled trial. *Arch Dermatol*. 2002;138:510-514.
5. Carruthers A, Carruthers J, Cohen J. Dilution volume of botulinum toxin type A for the treatment of glabellar rhytides: Does it matter? *Dermatol Surg*. 2007;33: S97-104.
6. Carruthers A, Bogle M, Carruthers JD, et al. A randomized, evaluator-blinded, two-center study of the safety and effect of volume on the diffusion and efficacy of botulinum toxin type A in the treatment of lateral orbital rhytides. *Dermatol Surg*. 2007;33:567-571.
7. Hsu TS, Dover JS, Arndt KA. Effect of volume and concentration on the diffusion of botulinum exotoxin A. *Arch Dermatol*. 2004;140:1351-1354.
8. Hexsel DM, De Almeida AT, Rutowitsch M, et al. Multicenter, double-blind study of the efficacy of injections

- with botulinum toxin type A reconstituted up to six consecutive weeks before application. *Dermatol Surg.* 2003;29:523-529.
9. Alam M, Yoo SS, Wrone DA, White LE, Kim JY. Sterility assessment of multiple use botulinum A exotoxin vials: A prospective simulation. *J Am Acad Dermatol.* 2006;55:272-275.
 10. Sarifakioglu N, Sarifakioglu E. Evaluating the effects of ice application on the pain felt during botulinum toxin type-A injections: A prospective, randomized, single-blind controlled trial. *Ann Plast Surg.* 2004; 53:543-546.
 11. Carruthers A, Carruthers J. Single-center, double-blind, randomized study to evaluate the efficacy of 4% lidocaine cream versus vehicle cream during botulinum toxin type A treatments. *Dermatol Surg.* 2005;31:1655-1659.
 12. Huilgol SC, Carruthers A, Carruthers JD. Raising eyebrows with botulinum toxin. *Dermatol Surg.* 1999;25:373-375.
 13. Carruthers A, Carruthers J, Said S. Dose-ranging study of botulinum toxin type A in the treatment of glabellar rhytides in females. *Dermatol Surg.* 2005;31:414-422.
 14. Carruthers A, Carruthers J. Prospective, double-blind, randomized, parallel-group, dose-ranging study of botulinum toxin type A in men with glabellar rhytides. *Dermatol Surg.* 2005;31:1297-1303.
 15. Carruthers A, Carruthers J, Cohen J. A prospective, double-blind, randomized, parallel-group, dose-ranging study of botulinum toxin type a in female subjects with horizontal forehead rhytides. *Dermatol Surg.* 2003;29:461-467.
 16. Levy JL, Pons F, Jouve E. Management of the ageing eyebrow and forehead: An objective dose-response study with botulinum toxin. *J Eur Acad Dermatol Venerol.* 2006;20:711-716.
 17. Lowe NJ, Ascher B, Heckmann M, Kumar C, Fraczek S, Eadie N, Botox Facial Aesthetics Study Team. Double-blind, randomized, placebo-controlled, dose-response study of the safety and efficacy of botulinum toxin type A in subjects with crow's feet. *Dermatol Surg.* 2005;31:257-262.
 18. Kane MAC. Classification of crow's feet patterns among Caucasian women: The key to individualizing treatment. *Plast Reconstr Surg.* 2003;112(5 Suppl): 33S-40S.
 19. Tamura BM, Odo MY, Chang B, Cuce LC, Flynn TC. Treatment of nasal wrinkles with botulinum toxin. *Dermatol Surg.* 2005;31:271-275.
 20. Semchyshyn N, Sengelmann RD. Botulinum toxin A treatment of perioral rhytides. *Dermatol Surg.* 2003; 29:490-495.
 21. Carruthers J, Carruthers A. Botulinum toxin A in the mid and lower face and neck. *Dermatol Clin.* 2004;22:151-158.
 22. Beer K, Yohn M, Closter J. A double-blinded, placebo-controlled study of Botox for the treatment of subjects with chin rhytides. *J Drugs Dermatol.* 2005;4: 417-422.
 23. Brandt FS, Boker A. Botulinum toxin for the treatment of neck lines and neck bands. *Dermatol Clin.* 2004;22:159-166.
 24. Cote TR, Mohan AK, Polder JA, Walton MK, Braun MM. Botulinum toxin type A injections: Adverse events reported to the US Food and Drug Administration in therapeutic and cosmetic cases. *J Am Acad Dermatol.* 2005;53:407-415.
 25. Aoki KR. A comparison of the safety margins of botulinum neurotoxin serotypes A, B, and F in mice. *Toxicon.* 2001;39:1815-1820.
 26. Alam M, Arndt KA, Dover JS. Severe, intractable headache after injection with botulinum A exotoxin: Report of 5 cases. *J Am Acad Dermatol.* 2002;46: 62-65.
 27. Chertow DS, Tan ET, Maslanka SE, et al. Botulism in 4 adults following cosmetic injections with an unlabeled, highly concentrated botulinum preparation. *JAMA.* 2006;296:2476-2479.
 28. Lee SK. Antibody-induced failure of botulinum toxin type A therapy in a patient with masseteric hypertrophy. *Dermatol Surg.* 2007;33:S105-S110.
 29. Rzany B, Dill-Muller D, Grablowitz D, Heckmann M, Caird D; German-Austrian Retrospective Study Group. Repeated botulinum toxin A injections for the treatment of lines in the upper face: A retrospective study of 4,103 treatments in 945 patients. *Dermatol Surg.* 2007;33:S18-S25.
 30. Omoigui S, Irene S. Treatment of ptosis as a complication of botulinum toxin injection. *Pain Med.* 2005; 6:149-151.
 31. Northington ME, Huang CC. Dry eyes and superficial punctuate keratitis: A complication of treatment of

- glabellar dynamic rhytides with botulinum exotoxin A. *Dermatol Surg.* 2004;30:1515-1517.
32. Redaelli A, Forte R. How to avoid brow ptosis after forehead treatment with botulinum toxin. *J Cosmet Laser Ther.* 2003;5:220-222.
 33. Matarasso SL, Matarasso A. Treatment guidelines for botulinum toxin type A for the periocular region and a report on partial upper lip ptosis following injections to the lateral canthal rhytides. *Plast Reconstr Surg.* 2001;108:208-214.
 34. Arat YO, Yen MT. Effect of botulinum toxin type A on tear production after treatment of lateral canthal rhytides. *Ophthal Plast Reconstr Surg.* 2007;23: 22-24.
 35. Matarasso SL. Decreased tear expression with an abnormal Schirmer's test following botulinum toxin type A for the treatment of lateral canthal rhytides. *Dermatol Surg.* 2002;28:149-152.
 36. Carruthers J, Carruthers A. Practical cosmetic Botox techniques. *J Cutan Med Surg.* 1999;3(suppl 4): S49-S52.

Fillers for Facial Rejuvenation

Joel L. Cohen and Anna Bar

BACKGROUND

Assessment of the aging face by the cosmetic surgeon includes not only the evaluation of photoaging and rhytides, but also the volume status of the face. Specifically, aging changes seen in the midface include loss of subcutaneous fat and gravitational changes such as descent of the malar fat pads, jowling, and laxity. To address these volumetric changes of aging, soft-tissue augmentation procedures have become part of our therapeutic armamentarium. The filler choice must be precisely tailored to the cosmetic concerns of the patient as well as the longevity of the product. Currently used filling agents have different modes of action and differing injection techniques, so the dermatologic surgeon must be knowledgeable about the product, its advantages and disadvantages, and its optimal uses.

PATIENT SELECTION/PREPROCEDURE EVALUATION

The choice of appropriate filling agents for each patient begins at the consultation visit. Often the physician finds that the areas of concern to the patient are not necessarily what would be noticed as primary factors in facial aging. In our practice, fillers are discussed in relation to their best cosmetic uses, longevity, cost per volume, and potential adverse effects. This enables the patient to

decide on which filler would best suit their cosmetic needs and budget.

In the consultation, a review of the patient's medications and past medical history is performed. Patients on nontherapeutic aspirin, nonsteroidal anti-inflammatory drugs, and some potentially blood-thinning vitamins and supplements are often advised to stop the medication 7 to 10 days prior to their planned treatment. In circumstances where agents cannot be stopped (including coumadin and therapeutic aspirin), the patient should be warned of the risk of significant bruising and swelling. There are scattered reports and many anecdotal discussions suggesting the utility of topical vitamin K products and arnica gel after treatment in reducing the amount of bruising and swelling.

Notable points in the medical history include confirmed or possible pregnancy, lactation, history of anaphylactic reactions, beef allergy (for bovine collagen fillers), lidocaine allergy (for collagen fillers and other agents) and personal or family history of collagen vascular disease (for all collagen fillers). Filling agents have not been studied in pregnant or lactating women, or in patients younger than 18 years.

Informed consent should be obtained prior to the procedure with all applicable risks outlined. The risks include but are not limited to infection, bleeding, bruising, asymmetry, nodule formation, allergy, and unsatisfactory cosmetic results. There is a risk of cutaneous necrosis as well. Before injecting fillers, photographs of the patient should be taken to document the improvement as well as to identify any areas of baseline asymmetry.

TECHNIQUE

■ Equipment and Instrumentation

Biodegradable fillers

Bovine collagen The first filling agent that became widely used was bovine-derived collagen, which was developed by a group of investigators at Stanford University in the late 1970s.¹ Zyderm I (Allergan) was approved by FDA in 1981 and was harvested from an isolated US herd. Zyderm I, Zyderm II and Zyplast were subsequently developed. These three bovine collagen products differ in their collagen concentration (35 mg/mL for Zyderm I and 65 mg/mL for Zyderm II and Zyplast). In addition, Zyplast is cross-linked with glutaraldehyde for greater stability and longevity in tissues. Zyderm is recommended for more superficial lines, while Zyplast is used for deeper rhytides of the face.

One disadvantage of these bovine-derived collagen products is their potential for immunogenicity; thus, prior to injection with either Zyderm or Zyplast, skin testing should be performed. Documentation of two negative skin tests is recommended prior to initiating treatment with these products.² Zyderm I is used for skin testing screenings, as it is thought to be more immunogenic because of lack of cross-linking. At the first visit, an intradermal injection of Zyderm I is placed just below the antecubital fossa (similar to a PPD test). Ninety percent of positive reactions (edema, induration, erythema) are seen within the first 72 hours, and the remaining 10% of positive reactions will be seen by 4 weeks. A follow-up visit is scheduled for 4 weeks, at which time the test site can be assessed. In patients who have a negative initial skin test, another skin test is placed, often at the anterior scalp line. Overall, the incidence of allergy to bovine collagens has been estimated at 1% to 5%. After two negative skin tests, the incidence of allergic reaction with treatment is 1% or less.²

Advantages of these bovine-collagen-derived fillers include their ease of use and a relatively long track record of efficacy and safety. Zyderm and Zyplast are formulated with 0.3% lidocaine, which can decrease pain on injection.

The disadvantages of these products include the aforementioned need for skin testing prior to injection, shorter duration of correction compared to some newer fillers, and requirement for refrigeration. Current recommendations involve avoiding the treatment of patients with a history of lidocaine allergy, anaphylactoid event, beef allergy, and sensitivity to collagen products in the past. Initial fears about the possibility of bovine collagen leading to

autoantibody production to human collagen have not materialized; no causal link has been established to date. In a study by Hanke and colleagues, the rate of dermatomyositis or polymyositis occurrence in patients who have had bovine collagen injections was no higher than in age-matched controls.³

Human bioengineered collagen Harnessing the advantages of collagen without a need for skin testing was a key breakthrough in the spectrum of filling agents. Cosmoderm and Cosmoplast (Allergan) are manufactured from a single cell line of human fibrocytes, which has been tested for viruses, tumorigenicity, genetic disease, and retroviruses. They are the only FDA-approved dermal fillers comprised of human collagen from a noncadaveric source.

Cosmoderm and Cosmoplast are similar in properties to Zyderm and Zyplast, without the need for skin testing. Cosmoderm I is formulated with a collagen concentration of 35 mg/mL and consists of non-cross-linked collagen fibers. This product is designed for injection into the superficial dermis. Cosmoplast is composed of cross-linked collagen fibers with a concentration of 35 mg/mL, designed for injection into the mid to deep dermis for correction of deeper rhytides. Cosmoderm II has a concentration of 65 mg/mL and is also used for superficial correction.

The advantage of the Cosmoderm products over their bovine predecessors is the ability to treat the patient at the first visit, without the need for skin testing. The disadvantages are similar to those of the bovine products including need for refrigerated storage, and the limited duration of effect compared to newer products such as hyaluronic acid derivatives.

Hyaluronic acid fillers Hyaluronic acid is a naturally occurring linear polysaccharide and is a component of the extracellular matrix of connective tissues. It is found in all vertebrate animals, yet exhibits no organ or species specificity, minimizing risk of allergic reaction.⁴

The advent of hyaluronic acid fillers represented a significant advance over collagen fillers in terms of longevity. The average hyaluronic acid filler usually lasts 2 to 4 months longer than collagen fillers. The actual hyaluronic acid macromolecules swell in the tissue by absorbing water.⁴ No refrigeration is required with these products. Their ease of use and safety profile has greatly expanded the use of filling agents in the United States.

Restylane (Medicis) was the first hyaluronic acid filler to be approved by FDA (in 2003) and is the most popular filler in the United States today. Prior to US approval, it had been used in Europe for many years. Restylane is

formulated from nonanimal sources and is partially cross-linked. This formulation has been trademarked as NASHA (nonanimal stabilized hyaluronic acid). The concentration of the hyaluronic acid is 20 mg/mL. The Restylane line of products includes Restylane Fine Lines and Perlane. Perlane was approved by the FDA in the spring of 2007 for correction of moderate to severe facial wrinkles and folds, and proves useful for facial contouring. Restylane Fine Line is now being evaluated in US studies, but is currently available in Europe and Canada for treatment of very superficial rhytides.

Hylaform gel (Allergan) was developed in the 1980s and has been used worldwide since 1998,⁴ but became available in the United States only in early 2004. It is a purified, cross-linked hyaluronic acid derived from the dermis of rooster combs. Currently it is available in two forms for the treatment of mild to moderate rhytides: Hylaform and Hylaform Plus. While both products have a concentration of 5.5 mg/mL, Hylaform Plus has a larger particle size and is intended for deeper injection.

Captique (Allergan) is another nonanimal source hyaluronic acid filling agent that has many similarities to Hylaform with a concentration of 5.5 mg/mL and particle size of 500 μm . It is also injected with a 30-gauge needle. Captique was approved by FDA in late 2004. It has a cross-linking process through divinyl sulfone. Many physicians have anecdotally felt that Captique and Hylaform inject more easily than Restylane and thus may result in less pain, swelling, and bruising. Despite these reported properties, however, they do not seem to offer the longevity of Restylane with often only 4 months of correction compared to 6 to 8 months offered by Restylane products.⁵

Juvederm (Allergan) was approved by FDA in the United States in 2006, and seems to offer more comparable longevity to Restylane than the Hylaform and Captique profiles of Allergan products. This nonanimal derived hyaluronic acid formulation is composed of a homogenous gel, in contrast to other hyaluronic acid products with particles of different sizes composing the mixture. Juvederm is available in two different formulations in the United States: Juvederm Ultra and Juvederm Ultra Plus for deeper folds and rhytides. Both products have a concentration of 24 mg/mL, although Juvederm Ultra Plus has a higher degree of cross-linking, yielding a thicker product. Juvederm Ultra is packaged with a 30-gauge needle, while Juvederm Ultra Plus is supplied with a 27-gauge needle.

Calcium hydroxylapatite Radiesse (Bioform Inc) is composed of calcium hydroxylapatite. It was initially approved

by FDA for treatment of vocal cord insufficiency, oral and maxillofacial defects, and radiographic tissue marking before being approved for correction of moderate to severe facial rhytides in 2006. Calcium hydroxylapatite is a mineral found in bones and teeth. The particle sizes of the synthetic calcium hydroxylapatite found in Radiesse range from 25 to 45 μm in diameter, in an aqueous gel (glycerin and sodium carboxymethylcellulose).⁶ Radiesse does not require refrigeration or a skin test.

Radiesse is used primarily to treat nasolabial folds and marionette lines. It can also be used in nose and chin augmentation, cheek augmentation, and diffuse facial contouring such as for HIV-associated lipoatrophy.^{7,8} It is injected through a 27-gauge needle into the deep dermis. After injection, the material can be massaged and molded to fit the contours of the face. Patients generally require nerve blocks or local anesthesia injection for placement of this filler.

Poly-L-lactic acid Sculptra (Sanofi Aventis/Dermik, USA) is composed of poly-L-lactic acid (PLA) derived from a vegetable source. A form of PLA has been used in synthetic suture material for over 40 years. Sculptra has been used in Europe under the name New Fill for several years, and in 2004 received FDA approval specifically for the treatment of HIV lipoatrophy in the United States. Sculptra has not received FDA approval for uses other than HIV lipoatrophy; any uses outside of this indication are all considered to be off-label.

Sculptra is designed for correction of diffuse facial volume loss rather than the correction of a specific rhytid. After injection, the final correction is not immediately apparent as with other fillers. The correction occurs as the PLA particles stimulate collagen production in the tissue; this may not be evident for months. This type of neocollagenesis is thought to occur through a macrophage and fibroblast response to the PLA particles, which are degraded over time. This foreign body reaction and subsequent degradation is accompanied by increased collagen production in the tissue.⁹ Several sessions are typically required although some small degree of improvement can be seen after the first session of two vials of product. More severe facial volume loss may require four or more sessions to achieve correction.

Sculptra is packaged as a sterile freeze-dried preparation in a clear glass vial. One disadvantage is that this product must be reconstituted 2 to 72 hours prior to injection with at least 4 to 5 mL sterile water, and 1 mL of 1% or 2% lidocaine is usually added prior to injection. Most physicians, however, prefer to reconstitute the

product at least 8 hours before use to decrease the amount of particulate matter in the solution, so same-day procedures are not performed unless product has already been mixed. The product is injected through a 25- to 26-gauge needle into the subcutis. This larger gauge of needle is required to facilitate passage of the particulate material, which can clog needles of smaller diameter.

Nonbiodegradable fillers

Silicone Silicone is one of the most controversial and polarizing filling agents in use today. Liquid silicone has been used as a filling agent for decades. Silicone fluid, or polydimethylsiloxane, comes in two forms that have been approved for use by the US FDA specifically for retinal detachment: AdatoSil 5000 (Bausch & Lomb) and Silikon 1000 (Alcon Labs). The numeral at the end of the brand name of the product refers to the viscosity in centistokes, with 100 cS defined as the viscosity of water. In facial augmentation, Silikon 1000 is used by some aesthetic physicians (AdatoSil being too viscous to inject through a small needle), although this use is considered off-label.

Dr. Orentreich was the pioneer of the microdroplet injection technique, in which 0.005 to 0.01 mL of product is delivered into the subdermal plane in a serial puncture technique at intervals of 2 to 10 mm, with multiple treatments being necessary.¹⁰ The treatments are repeated typically at monthly intervals until desired results are achieved. Improvement can usually be seen after several treatments as the body produces new collagen around the silicone droplets, thus waiting in between treatments is crucial to avoid overcorrection.

Polymethylmethacrylate ArteFill is a combination filler composed of polymethylmethacrylate (PMMA) spheres and purified bovine collagen from an isolated closed herd in the United States. ArteFill was approved by FDA in the United States in late 2006. The European and Canadian predecessor of this filler is known as Artecoll. PMMA, commonly known as Plexiglas or Lucite, is widely used in medical devices. The PMMA spheres, which compose 20% of the mixture, are permanent, while the bovine-collagen component composing the remaining 80% is biodegradable. The collagen component provides instantaneous correction while the PMMA spheres are, in theory, chemically inert and stable for many years.¹¹ The microsphere size ranges from 30 to 50 μm in diameter in a gel carrier packaged with 0.3% lidocaine. Because of the bovine-collagen component, a skin test is recom-

mended with an evaluation period of 28 days. After injection, the collagen carrier is degraded by the body, while the PMMA microspheres become surrounded by the patient's own collagen.¹² Because the collagen used in ArteFill is partially denatured, it is thought to be less likely to produce an allergic reaction than Zyderm.¹³

The Artecoll formulation was used in Europe since 1994 on an estimated 200,000 patients. The reported incidence of granuloma formation because of this product was at rate of 0.01% to 0.024%.¹¹ Patients who developed granulomas with Artecoll, many of whom showed manifestations several years after treatment, respond fairly well to injection with intralesional corticosteroids.¹⁴ In the ArteFill trial of 251 patients there were no reported granulomas cases.¹⁵ Some expert physicians, however, have advocated a need for histologic evaluation of the effects of this product after cutaneous implantation prior to widespread cosmetic use.

■ Patient Positioning

Injectors have various preferences as to positioning of patients for soft-tissue augmentation. We prefer to inject patients in an upright position so that the natural appearance of redundancy and volume loss can be appreciated. If an injector prefers to have the patient tilted back for some areas, we strongly encourage that the areas intended for filler placement be marked carefully on the patient's skin prior to adjusting their position. A power table is recommended so the injector can be comfortable and alter the height and degree of tilt of the patient depending on the region of injection.

■ Step-by-Step Technique

Anesthesia

Anesthetic need varies depending on the filler that is chosen for augmentation and on the location of placement. Many fillers can be comfortably injected with only topical anesthesia. One exception to topical anesthesia is lip augmentation. For lip injections, we prefer a gingival sulcus block that can be quickly administered with 1% lidocaine (with or without epinephrine) into the upper and lower gingival sulcus.

Fillers that are injected with larger needles (25–27 gauge) can sometimes be uncomfortable without local anesthesia or nerve blocks. Nerve blocks can be performed with 1% lidocaine, with or without epinephrine. The infra-orbital and mental nerves are quite amenable to nerve

blocks; nerve blocks are helpful for some patients prior to injecting the nasolabial folds and oral commissures as well as often necessary for many patients prior to mid-cheek augmentation.

Injection techniques

Techniques for injection vary by location of treatment and by specific filling agent. Although often there is no one “right” or “wrong” way, each injector has individual preferences for different fillers.

There are four main types of injection techniques: serial puncture, linear threading, fanning, and cross hatching (Figure 4.12). Many injectors prefer a retro-grade linear threading injection technique for most loca-

tions and filler agents. This entails inserting the needle to the furthest injection point and injecting small even aliquots during withdrawal of the needle. Care is taken not to inject at the final point of withdrawal as this can lead to superficial placement.

Depth of injection is very important. When using fillers that are meant to improve the appearance of superficial facial rhytides (Cosmoderm, Zyderm), injection into the superficial to mid dermis is crucial to provide correction. Many fillers are injected into the deep dermis or superficial subcutis (including Restylane, Perlane, Juvederm, Radiesse, Cosmoplast, as well as the longtime filler Zyplast) to provide correction of moderate to severe facial rhytides rather than very superficial “etched” lines.

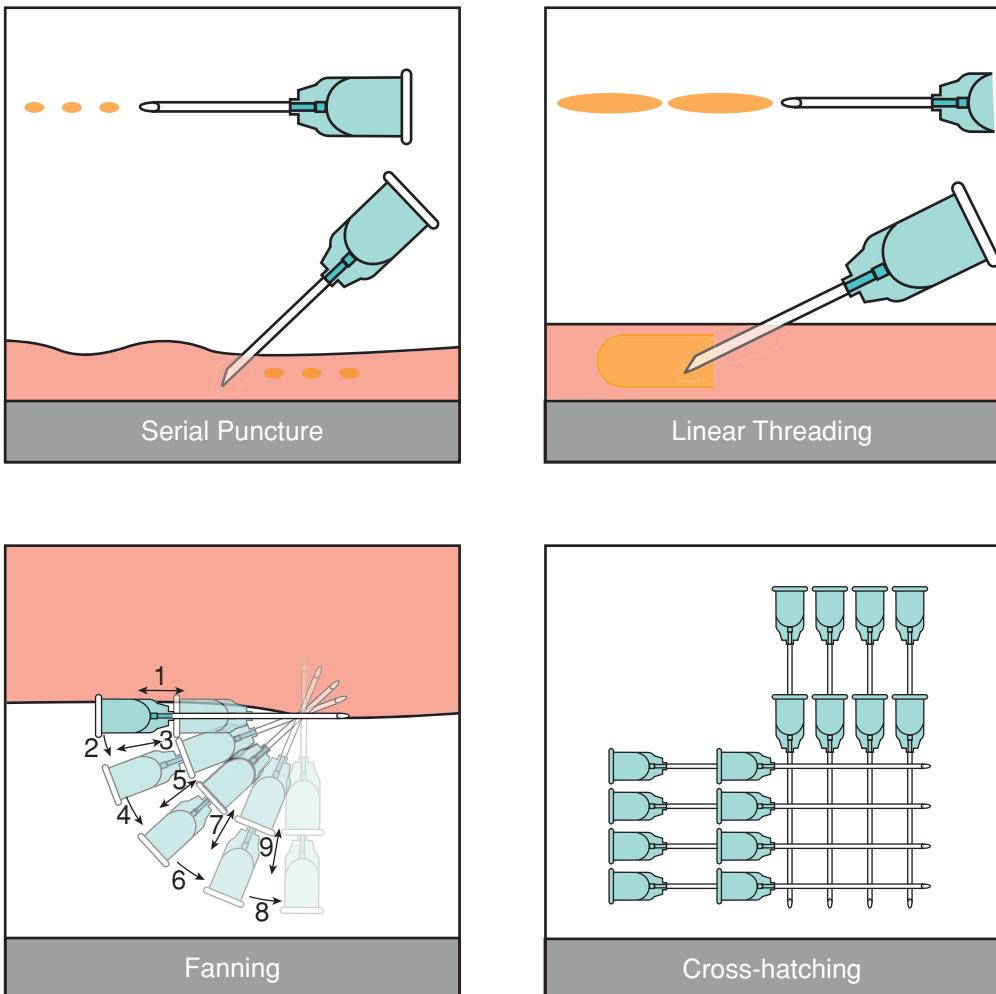


Figure 4.12 *The four most common techniques for filler injections*

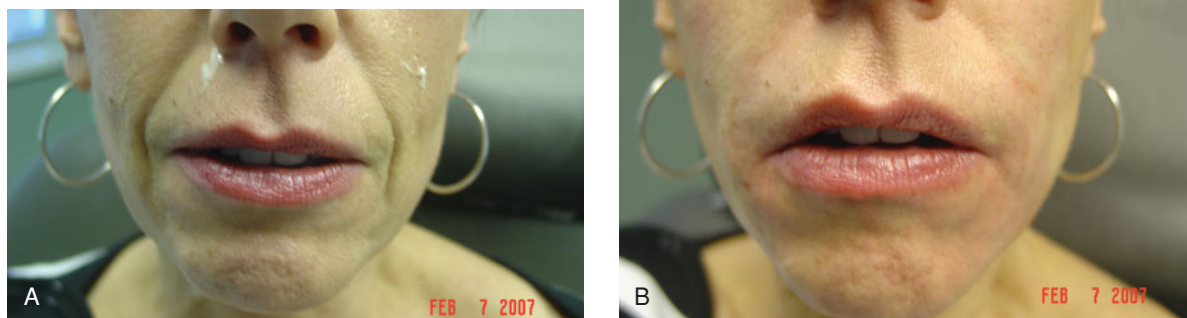


Figure 4.13 (A) Pre- and **(B)** immediately post two syringes (total 1.6 mL) of Juvederm Ultra into nasolabial folds. Topical anesthesia was used

Sculptra is an example of a filler that requires even deeper injection, specifically into the subcutis.

The nasolabial folds are the most “forgiving” areas for a novice to learn injection techniques. This area is amenable to all four of the injection techniques, and often physicians use a combination of the four techniques in this area. Figure 4.13 shows Juvederm Ultra placement into the nasolabial folds.

Marionette lines are similar to nasolabial folds in that they are quite forgiving and tolerate a variety of injection techniques, but correction often requires a substantial amount of filler. To provide a good cosmetic result for patients with deep nasolabial folds and marionette lines, one can easily use several milliliters or syringes of a given product. While injecting these areas, adding some filler to the lateral commissures can improve downturning at the corners of the mouth, which occurs with facial aging. Figure 4.14 shows Restylane placement in nasolabial folds and oral commissures, along with 6 months, follow-up.

Lip augmentation is extremely technique dependent. Poor injection technique can lead to blurring of the vermillion-skin junction, blunting of Cupid’s bow, asymmetry, and lumpiness. Overinjection of the upper lip without maintaining the normal 1/3 to 2/3 ratio of upper to lower lip can lead to “duck lips” where the upper lip appears large and protuberant. Figure 4.15 shows an “unnatural” lip appearance after an overzealous lip augmentation by a nonphysician. Figure 4.16 shows a more “natural”, small volume augmentation of the lips achieved by injecting the vermillion border as well as the lip body (off-label), and paying careful attention to relative proportions and accepted aesthetic principles.

Tear trough injections of fillers are usually placed below the orbicularis oculi muscle and just above the periosteum. Superficial injection of filler can be visible in this area where the eyelid skin is thin. Figure 4.17 shows delayed nodules of PLA after a nonphysician likely injected this patient in a too superficial plane along the lower eyelid.

When fillers are used in the glabellar area, extra caution must be exercised. There are reports of necrosis with various fillers injected into this area, presumably with use of larger volumes of product compressing adjacent vessels or frank intravascular injection.¹⁶ Physicians treating the glabellar area should consider injecting superficially, aspirating before injecting to confirm placement, and using low volumes (often spaced over two sessions) of small-particle fillers (such as Zyderm and CosmoDerm) to avoid compression of adjacent vessels.¹⁷

■ Complications to Avoid

Complications seen with fillers include sensitivity, infection, necrosis, and superficial placement. Reactions that can occur independent of injection technique include allergic reaction and infection. Allergic reaction has been well documented with bovine collagens, necessitating the need for skin testing. These reactions typically resolve within 4 to 24 months.² Treatment of sensitivity reactions from these bovine products includes topical, intralesional, and oral corticosteroids, topical calcineurin inhibitors, PDL, nonsteroidal anti-inflammatory medications, and in one case report, cyclosporine.¹⁸

Although reactions against hyaluronic acids (HA) are rare, they have been reported. Data from 1999 involving

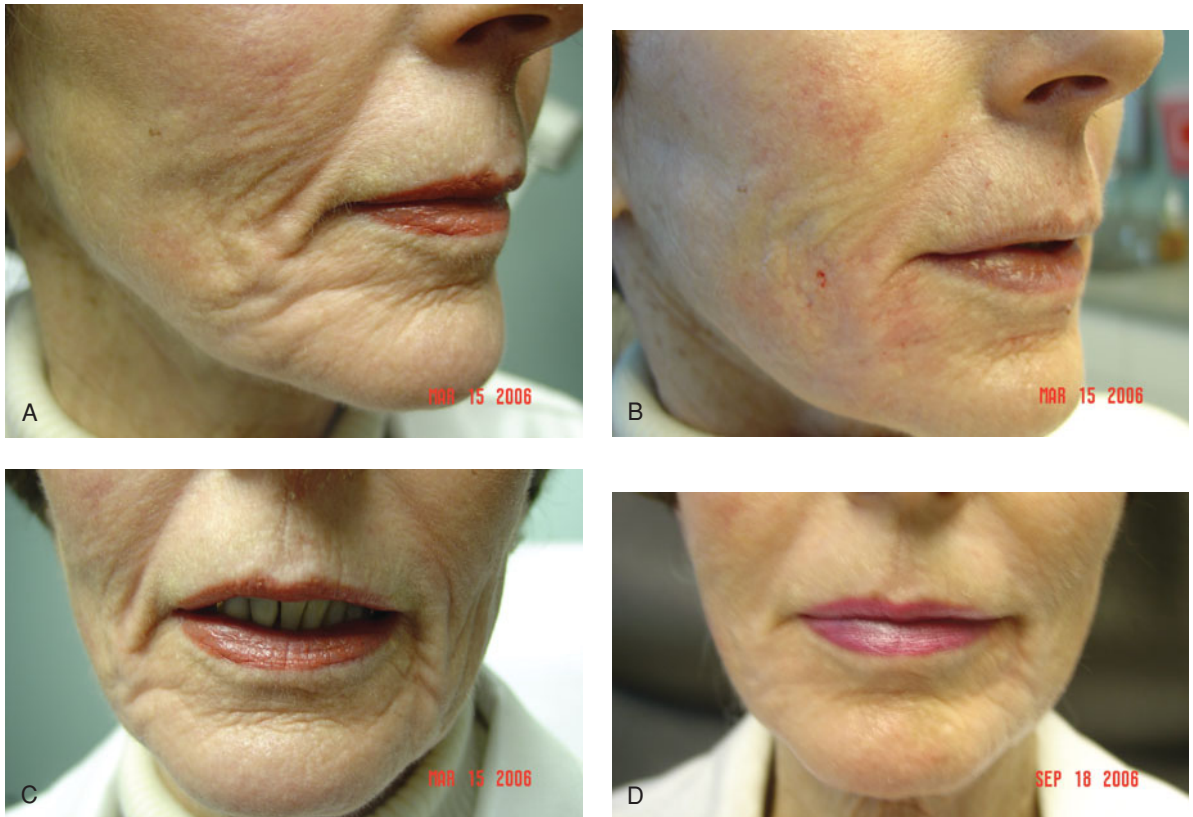


Figure 4.14 (A&B) Pre and post 3 mL of Restylane into bilateral nasolabial folds and oral commissures **(C&D)** Pre-Restylane photo and follow-up photo at 6 months posttreatment. In addition, the patient was also treated with three full-face fractional resurfacing (Palomar 1540) procedures subsequent to the filler placement



Figure 4.15 Patient treated by nonphysician with hyaluronic acid in the lips and nasolabial folds. The appearance of these lips is an unnatural “plunger-look” caused by overfilling and little regard for aesthetic principles of volume and proportions. Poor placement of filler is also seen at the patient’s right nasolabial fold, where a “sausage-lump” of product is seen “ridging” at the angle of the mouth

an estimated 144000 patients treated showed adverse events in 0.15%, including redness, swelling, acneiform lesions, bacterial infection, and localized granuloma formation.^{19,20} With the reformulation of Restylane in 1999, the protein content of the product was decreased sixfold, resulting in a concomitant drop of reported hypersensitivity reactions. In 2000, there were fewer adverse events and hypersensitivity reactions reported despite more patients receiving treatment.²¹ Acute hypersensitivity reactions to HA have been infrequently described, including an acute angioedema-type reaction occurring hours after injection in the lip.²²

Delayed erythema with papules or nodules, or so-called angry red bumps have been reported with hyaluronic acid fillers. Although the etiology is debated, several have been proposed: allergic, foreign body granuloma, infection or sterile abscess. Narins and colleagues provided an algorithm for management of the

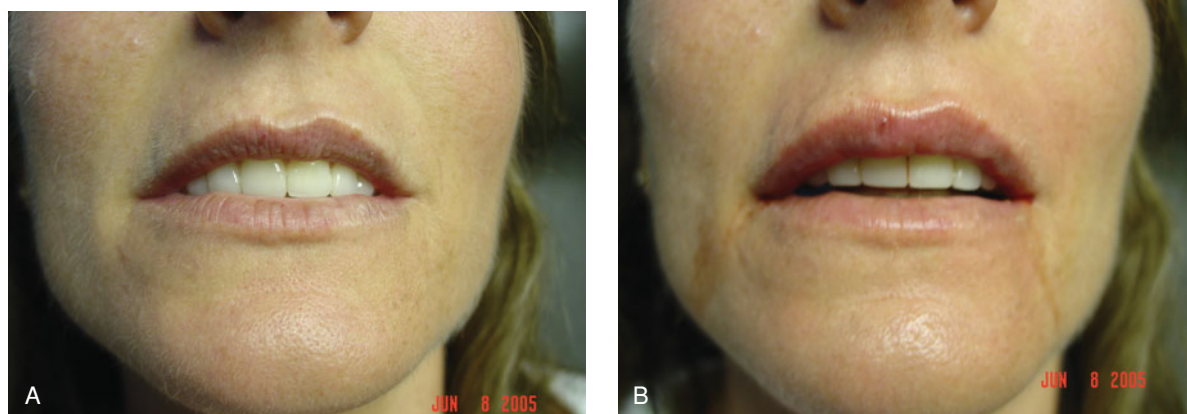


Figure 4.16 (A&B) Patient received a total of 0.7 mL of Restylane into lips. She desired only a small augmentation, and requested topical anesthetic only. The product was placed both along the vermillion border as well as at the wet-dry junction of the upper and lower lips. Careful attention was paid to anatomic proportions, as well as pre-injection asymmetry (left-sided Cupid's bow peaking)

angry red bumps with needle aspiration or incision and drainage of fluctuant lesions with culture for bacteria and mycobacteria, followed by a course of Biaxin pending culture results. For nonfluctuant lesions, a consensus panel recommended Biaxin for coverage of mycobacteria and possible use of intralesional steroids or topical calcineurin inhibitors.²³

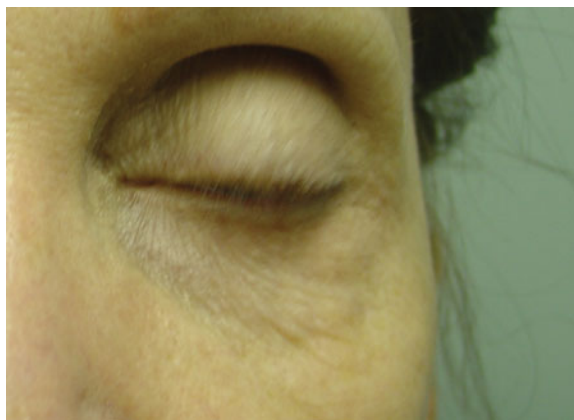


Figure 4.17 Sculptra nodules at lower lid. Patient was injected by a nonphysician, and nodules have persisted over a year

Viral and bacterial infections can also rarely occur after filler injections. If bacterial infection is suspected, cultures should be obtained and antibiotic therapy should be started immediately. If a herpetic process is suspected, antiviral therapy is initiated. Filler injections in the perioral area can potentially cause reactivation of herpes simplex in patients with a history of multiple recurrent episodes of cold sores. In our practices, these patients usually receive oral antiherpetic prophylaxis before treatment.

Regarding the use of silicone, the controversy stems from reports of side effects, which can be long-lasting or permanent. Silicone fillers have been linked to granuloma formation and nodules for many years. Excessive volumes have also resulted in filler migration.²⁴ When using silicone, it is recommended to correct a specific area over several sessions using small volumes and microdroplet technique. Disastrous complications from injection of adulterated, nonmedical grade silicone by inexperienced or unlicensed injectors have been widely publicized.²⁴ However, when a pure, medical grade silicone is injected by an experienced, aesthetic physician who is familiar with the microdroplet technique, the adverse effects are minimized. Potential complications from silicone still include granuloma and nodule formation, ulceration, migration, connective tissue disease, and infection.²⁴

Necrosis is a feared complication of any filler that can result either from injection of too much material causing compression of vascular structures, or direct injection into a vessel. Injection necrosis is a well-described phenomenon (especially in the glabella) that can present initially with painless blanching, bruising, or reticular erythema of the treated area. Over days, the skin may appear dusky and then black. Necrosis and ulceration may follow.¹⁶ Treatment for this complication should start immediately; if impending necrosis is suspected at the time of injection, immediately stop injection and massage the treated area. Application of heat along with a topical nitroglycerine paste may encourage vasodilation of the area. If the culprit product was a hyaluronic acid filler, hyaluronidase may help avert a dusky, reticulated impending necrosis from progressing to full necrosis.^{25,26} If necrosis occurs, low molecular weight heparin has been used along with daily wound care to minimize scarring.¹⁶

Superficial placement can occur with any of the filling agents. When injected too superficially, the most common complication is again a visible nodule. In many instances, these filler nodules can be nicked with an 11 blade and expressed. With hyaluronic acid fillers, superficial placement can lead to the Tyndall effect, where a blue tinge is seen on the skin. Hyaluronidase has also been used in these types of cases to dissolve the undesired hyaluronic acid product.^{27,28}

Delayed nodules have been reported with injection of Radiesse as well, particularly with injection in the lips. Histologically, these are not granulomas but rather nodules of calcium hydroxyapatite that seem to have been “pushed” superficially over time because of the mechanical pumping action of the underlying orbicularis oris muscle. These small nodules occur on the mucosal surface of this lip, and will often respond to massage; however, incision and drainage, surgical extrusion, or even excision may be required in refractory cases. Many injectors do not recommend using Radiesse in the lips because of frequent occurrence of these nodules.⁶

There are various reports of subcutaneous papule formation related to Sculptra injection. These papules are typically palpable, but only sometimes visible on the surface (mostly in thin-skinned areas). This reaction is thought to be related to superficial injection technique, unequal distribution, and/or lower volume dilution resulting in more particulate matter per injection site.²⁹ The incidence of this complication has been estimated at 6% to 44%.^{30,31} There are, however, reports of patients who

have developed actual granulomatous skin reactions over 1 year after injection with Sculptra.³⁰ Sculptra granulomatous nodules have been managed with intralesional steroids and surgical excision in refractory cases.²⁹ Dermal injection of PLA, as was originally prevalent in Europe, has been linked to significant nodule formation as well as reports of disfiguring atrophic hypopigmented scars³⁰; thus, subcutaneous injection of a dilute PLA mixture reconstituted several hours before injection is now the favored technique.

POSTPROCEDURE CONSIDERATIONS

Following the procedure, we recommend patients stay in the office and ice the areas of treatment for 15 to 20 minutes. Patients are instructed not to apply makeup until at least 6 hours after procedure because of the theoretic risk of occluding and possibly implanting a foreign material into the actual injection penetration sites. Patients are often instructed to avoid exercise until the following day as there is a potential for accentuated blood flow eventually leading to an exacerbation of swelling or bruising. Potentially blood-thinning agents stopped prior to the filler appointment are not recommended to be resumed until at least 3 days after the procedure.

Finally, patients are usually told to avoid manipulation of the product and injection sites for at least 3 days in order to allow the swelling to go down. For PLA, however, patients are usually instructed to massage the treated area (usually with a gentle lotion) several times a day for about 5 minutes for 1 to 2 weeks after injection in order to ensure more even dispersion of the material and to avoid clumping.

REFERENCES

1. Klein AW, Elson ML. The history of substances for soft tissue augmentation. *Dermatol Surg.* 2000;26: 1096-1105.
2. Klein AW. Skin filling. Collagen and other injectables of the skin. *Dermatol Clin* 2001;19:491-508.
3. Hanke CW, Thomas JA, Lee WT, Jolivet DM, Rosenberg MJ. Risk assessment of polymyositis/dermatomyositis after treatment with injectable bovine collagen implants. *J Am Acad Dermatol.* 1996;34: 450-454.
4. Monheit GD. Hyaluronic acid fillers: Hylaform and Captique. *Facial Plast Surg Clin North Am.* 2007; 15(1):77-84.

5. Rao J, Genevieve C, Goldman MP, et al. Clinical comparison between two hyaluronic acid-derived fillers in the treatment of nasolabial folds: Hylaform versus Restylane. *Dermatol Surg.* 2005; 31(s4): 1587-1590.
6. Berlin A, Cohen JL, Goldberg DJ. Calcium hydroxylapatite for facial rejuvenation. *Semin Cutan Med Surg.* 2006;25:132-137.
7. Alam M, Yoo SS. Technique for calcium hydroxylapatite injection for correction of nasolabial fold depressions. *JAAD.* 2007;56(2):285-289.
8. Tzikas TL. Evaluation of the Radiance FN soft tissue filler for facial soft tissue augmentation. *Arch Facial Plast Surg.* 2004;6:234-239.
9. Vleggaar D. Facial volumetric correction with injectable poly-L-lactic acid. *Dermatol Surg.* 2005; 31(s4):1511-1518.
10. Orentreich DS. Liquid injectable silicone: Techniques for soft tissue augmentation. *Clin Plast Surg.* 2000; 27:595-612.
11. Lemperle G, Romano JJ, Busso M. Soft tissue augmentation with Artecoll: 10-year history, indications, techniques, and complications. *Dermatol Surg.* 2003;29:573-587; discussion 587.
12. Cohen S, Holmes R. Artecoll: A long-lasting injectable wrinkle filler material: Report of a controlled, randomized, multicenter clinical trial of 251 subjects. *Plast Reconstr Surg.* 2004;114: 964-976.
13. Gelfer A, Carruthers A, Carruthers J, Jang F, Bernstein S. The natural history of polymethylmethacrylate microspheres granulomas. *Dermatol Surg.* 2007;33:614-620.
14. Conejo-Mir J, Guirado S, Munoz M. Adverse granulomatous reaction to Artecoll treated by intralesional 5-fluorouracil and triamcinolone injections. *Dermatol Surg* 2006;32:1079-1081.
15. Cohen SR, Berner CF, Busso M, et al. ArteFill: A long-lasting injectable wrinkle filler material—Summary of the U.S. Food and Drug Administration trials and a progress report on 4- to 5-year outcomes. *Plast Reconstr Surg.* 2006;118(3)(suppl): 64S-76S.
16. Schanz S, Schippert W, Ulmer A, et al. Arterial embolization caused by injection of hyaluronic acid (Restylane®). *Br J Dermatol.* 2002;146:928-929.
17. Glaich AS, Cohen JL, Goldberg LH. Injection necrosis of the glabella: Protocol for prevention and treatment after use of dermal fillers. *Dermatol Surg.* 2006;32(2): 276-281.
18. Baumann L, Kaufman J, Saghari S. Collagen fillers. *Dermatol Ther.* 2006;19:134-140.
19. Micheels P. Human anti-hyaluronic acid antibodies: Is it possible? *Dermatol Surg.* 2001;27:185-191.
20. Friedman PM, Mafong EA, Kauvar A, Geronemus RG. Safety data of injectable nonanimal stabilized hyaluronic acid gel for soft tissue augmentation. *Dermatol Surg.* 2002;28(6):491-494.
21. Leonhardt JM, Lawrence N, Narins RS. Angioedema acute hypersensitivity reaction to injectable hyaluronic acid. *Dermatol Surg.* 2005;31:577-579.
22. Narins RS, Jewell M, Rubin M, Cohen J, et al. Clinical conference: Management of rare events following dermal fillers—Focal necrosis and angry red bumps. *Dermatol Surg.* 2006;32:426-434.
24. Narins RS, Beer K. Liquid injectable silicone: A review of its history, immunology, technical considerations, complications, and potential. *Plast Reconstr Surg.* 2006;118(3)(suppl):77-84.
25. Hirsch RJ, Cohen JL, Carruthers JDA. Successful management of an unusual presentation of impending necrosis following a hyaluronic acid injection embolus and a proposed algorithm for management with hyaluronidase. *Dermatol Surg.* 2007;33(3): 357-360.
26. Hirsch RJ, Lupo M, Cohen JL, Duffy D. Delayed presentation of impending necrosis following soft tissue augmentation with hyaluronic acid and successful management with hyaluronidase. *J Drugs Dermatol.* 2007;6(3):325-328.
27. Brody HJ. Use of hyaluronidase in the treatment of granulomatous hyaluronic acid reactions or unwanted hyaluronic acid misplacement. *Dermatol Surg.* 2005; 31(8, pt 1):893-897.
28. Hirsch RJ, Cohen JL. Surgical insights: Challenge: Correcting superficially placed hyaluronic acid. *Skin & Aging.* 2007;15:36-38.
30. Beljaards RC, de Roos K-P, Bruins FG. NewFill for skin augmentation: a new filler or failure? *Dermatol Surg.* 2005;31(7 pt 1):772-776; discussion 776.
31. Mest D, Humble G. Safety and efficiency of poly-L-lactic acid (Sculptra) injections for patients with HIV-associated facial lipoatrophy. *Antiviral Ther.* 2004; 9:L36.

Chemical Peels

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BACKGROUND

The use of chemical peels is a versatile tool for any physician interested in aesthetic facial rejuvenation. Chemical peels are most often classified by the depth of injury they induce, as shown in Table 4.3. Clinical uses, associated

downtime, and side effect profiles vary among different types of peels.

PATIENT SELECTION AND PREOPERATIVE COUNSELING

As with any aesthetic procedure, proper patient selection and establishment of realistic expectations are vital to

TABLE 4.3 ■ Comparison of Available Chemical Peels

	Depth of Penetration	Specific Agents	Clinical Indications
Superficial, very light	Stratum corneum +/- superficial stratum spinosum	10%–20% TCA Low potency α -hydroxy acid (i.e., glycolic acid 10%–50%, 92% lactic acid, tartaric acid, citric acid, malic acid) β -Hydroxy acid (i.e., salicylic acid) Tretinoin 1% solution	Actinic keratoses and cheilitis, acne, dyschromia (solar lentigines, melasma, postinflammatory hyperpigmentation, ephelides), seborrheic keratosis, very fine lines, keratosis pilaris (generally produces only partial improvement of these preceding conditions). Enhances penetration of topical medications (i.e., 5-fluorouracil)
Superficial, light	Full thickness of epidermis	25%–30% TCA Jessner's solution 70% Glycolic acid	
Mid-level	Upper reticular dermis	35%–50% TCA Solid carbon dioxide plus 35% TCA 70% Glycolic acid plus 35% TCA Jessner's plus 35% TCA	Acne scars (only minimal effect), fine lines and rhytides, dyschromia (solar lentigines, melasma, postinflammatory hyperpigmentation, ephelides), epidermal and superficial dermal growths (actinic keratoses, seborrheic keratoses, sebaceous hyperplasia, syringomas, trichodiscomas, xanthelasma). Blends areas treated with resurfacing laser
Deep	Mid-reticular dermis	Baker–Gordon phenol peel	Moderate and deep rhytides, actinic keratoses, acne scars, dyschromia (solar lentigines, melasma, postinflammatory hyperpigmentation)

TCA, trichloroacetic acid.

success. The chosen peel should match the patient's degree of photoaging and tolerance for postprocedure downtime. It is also necessary to elicit a history of factors that could cause poor wound healing and discuss potential adverse effects. (Table 4.4) Nicotine use or prior surgery involving wide undermining such as rhytidectomy within 3 to 6 months can disrupt cutaneous blood supply and may adversely affect healing. Laser resurfacing has been performed concomitant with lifting procedures without adverse consequence, suggesting that superficial or mid-level chemical peels are likely safe in this scenario as well.¹

Use of oral isotretinoin within 6 to 12 months, prior superficial irradiation, or previous ablative laser resurfacing or dermabrasion can affect pilosebaceous units and their associated epidermal stem cells.^{2,3} Although superficial chemical peels are often performed in these patients, medium and deep peels should be considered with caution because of an increased risk for scarring. Active dermatitis or prepeel use of laser, radiofrequency, or IPL devices can disrupt the epidermal barrier and lead to inadvertent deep penetration of a peel. Peels should be avoided in patients with active herpes simplex virus (HSV), molluscum contagiosum, or human papilloma virus infections. We routinely prescribe oral antiviral prophylaxis for patients with a history of recurrent orolabial HSV infection who are receiving medium or deep facial chemical peels. Although uncommonly used with superficial peels, antiviral prophylaxis can be considered with a history of frequent HSV outbreaks.

TECHNIQUE

■ Patient Preparation

Preprocedure skin preparation can improve clinical outcomes and minimize adverse effects. Photoprotection and application of hydroquinone-containing bleaching agents may be implemented 4 to 6 weeks before a peel to decrease the possibility of postinflammatory hyperpigmentation. Use of topical retinoids daily for 2 or more weeks preoperatively has been shown to result in a more even and rapid frost with mid-level peels, and more rapid wound healing.⁴ Retinoids should, however, be discontinued 4 to 7 days before the procedure to ensure the presence of an intact stratum corneum.

Immediately prior to administering a peel, the skin should be cleaned and degreased. Our approach is to clean the skin thoroughly with Ingasam (Septisol, Vestal

Laboratories, St Louis, MO), followed by application of 70% isopropyl alcohol and a final degreasing with acetone. We utilize this skin preparation for all of our peels. Preoperative anesthesia is not required for superficial chemical peels, but we often recommend local nerve blocks during mid-level peels. Oral or intramuscular sedatives are occasionally beneficial for mid-level chemical peels, and deep chemical peels may require sedation or even general anesthesia. In addition, we have a low threshold to use sublingual triazolam or oral diazepam for anxious patients undergoing mid-level or deeper chemical peels.

■ Specific Peeling Agents

Superficial chemical peels

Individual superficial chemical peels rarely produce significant results, and a series of 4 to 10 peels is usually required to achieve optimal outcomes. Conditions amenable to superficial chemical peels are listed in Table 4.3. Superficial chemical peels are further classified as very light peels (which penetrate the stratum corneum with possible involvement of the superficial stratum spinosum), and light peels (which affect the entire epidermis). Superficial peels have the advantage of minimal downtime and a favorable side effect profile. Very light superficial peels are safe for all skin tones because of the lack of significant associated inflammation, but care must be taken with light superficial chemical peels in darker skin types, as they can induce hyperpigmentation. Superficial peels can be a component of a skin maintenance regimen in combination with daily sunscreen, topical retinoids, and topical bleaching agents if appropriate. Used in isolation, superficial peels can result in more supple and reflective skin, but have only mild effects on epidermal growths.

Trichloroacetic acid (10%–25%) At different concentrations, trichloroacetic acid (TCA) can be used as a superficial or medium-depth peeling agent. The agent 10% to 25% TCA produces an injury superficial to the papillary dermis. Penetration of TCA is self-limited since it is rapidly neutralized and induces coagulation of proteins as it progresses through the layers of the skin, manifesting as a white frost. The desired endpoint of a superficial TCA peel is a level I to II frost (Table 4.5).⁶ We most frequently utilize superficial TCA peels for mild dyspigmentation including solar lentigines and epidermal melasma, and as an adjunctive treatment for actinic keratoses and photoaging.

TABLE 4.4 ■ Side Effects of Chemical Peels

Adverse Event	Management	Comment
Postinflammatory hyperpigmentation	Often resolves spontaneously in several months. Topical hydroquinone bleaching creams. Mid-level chemical peel if persistent.	More common after superficial peels in darker phototypes (Fitzpatrick III–VI).
Hypopigmentation	Often permanent.	More common after deep peels.
Milia	Extraction via 19-gauge needle and comedone extractor. Topical retinoids following re-epithelialization.	Related to occlusive ointments. Avoid aggressive extraction to prevent scarring.
Acne and folliculitis	Usually resolves spontaneously in 1–2 wks. May use oral antibiotics, but topical acne therapy should be deferred until re-epithelialization is complete.	Related to occlusive ointments.
Prolonged erythema	Rule out infection and contact dermatitis. Erythema and induration may indicate impending scarring and should be treated as below.	Erythema is prolonged if greater than 3–5 d for a superficial peel, 15–30 d for a mid-level peel, and 90 d for a deep peel.
Scarring	Topical, intralesional, or oral corticosteroids; pulsed dye laser; intralesional 5-fluorouracil, occlusive dressings, massage.	More common with deeper peels. Increased on the neck and bony prominences of the zygoma, jawline, and chin.
Bacterial infection	Obtain cultures and start empiric antibiotics.	Often related to poor wound care. Can include staphylococcus, streptococcus, and pseudomonas. Early management mitigates risk of scarring.
Fungal infection	Usually responds well to topical or oral antifungals.	Candidal infections are usually related to occlusive dressings. Early management mitigates risk of scarring.
HSV reactivation	Oral antivirals.	Can result in widespread infection of the peeled area (herpes varicelliform eruption). Early management mitigates risk of scarring.
Contact dermatitis	Eliminate irritants and allergens, topical and/or oral corticosteroids, bland emollients.	May have increased because of impaired skin barrier function. Resorcinol in Jessner's solution may be a cause.
Systemic toxicity (cardiotoxicity, hepatotoxicity, and nephrotoxicity)	Supportive measures.	Phenol peels only. Decrease risk by treating one segment risk by treating one segment intervals and hydrating before and during procedure. Cardiac monitoring is mandatory.

TABLE 4.5 ■ Endpoints of TCA Peels

Level of Frosting	Clinical Findings
0	Pink or erythematous skin
I	Pink or erythematous skin with speckled white frost
II	White frost with erythema showing through
III	Opaque white frost

Jessner's solution Jessner's solution consists of 14 g each of resorcinol, salicylic acid, and lactic acid in 200-proof ethanol to make 100 mL. Because of occasional allergic contact dermatitis to resorcinol, some advocate use of a modified solution consisting of 17% lactic acid, 17% salicylic acid, and 8% citric acid in 200-proof ethanol.⁵ Similar to superficial TCA peels, the desired endpoint is a level I to II frost (Table 4.5), sometimes requiring two to four applications at intervals of 5 to 6 minutes to achieve. Although we most often use Jessner's solution peels in conjunction with other agents to produce a mid-level peel (discussed later), we occasionally use them in isolation for indications similar to those of superficial TCA peels.

Salicylic acid Salicylic acid is a lipophilic β -hydroxy acid that occurs naturally in multiple plants and fruits and has keratolytic, antimicrobial, and anti-inflammatory effects. Commonly used preparations include 20% or 30% salicylic acid solutions. Treatment typically consists of one to three coats of salicylic acid, each left in place for 3 to 5 minutes prior to rinsing with water. We have found salicylic acid peels most useful in the treatment of acne.

Alpha hydroxy acids Glycolic acid and lactic acid are both α -hydroxy acids. They do not self-neutralize via protein coagulation, but rather must be neutralized with water or sodium bicarbonate. The depth of a glycolic acid peel is a function of the concentration, volume, and duration of application. Glycolic acid is one of the most frequently used superficial peeling agents and we find it useful for epidermal melasma, solar lentigines, acne, and mild photoaging. A solution containing 92% lactic acid has also been used as a peeling agent and found to have effects comparable to Jessner's solution in the treatment of melasma.⁷

Mid-level chemical peels

Mid-level peels induce coagulative necrosis to the level of the papillary dermis, with inflammation extending to the reticular dermis. Conditions treated by mid-level peels are listed in Table 4.3. In our experience, a single mid-level chemical peel usually produces results superior to those from a series of superficial peels.

Trichloroacetic acid (50%) This traditional mid-level peeling agent has largely been replaced by combination peels utilizing lower concentrations of TCA because of less frequent adverse effects. At concentrations of 50% and greater, TCA is characterized by irregular and non-homogenous depth of penetration.

Combination 35% TCA peels Use of 35% TCA can be preceded by application of solid carbon dioxide (Brody peel), Jessner's solution (Monheit peel), or 70% glycolic acid (Coleman peel) to produce a mid-level chemical peel.^{8–10} Pretreatment with these substances disrupts the epidermis, allowing deeper and more uniform penetration of 35% TCA, giving more predictable results and an improved safety margin compared to 50% TCA. The clinical endpoint is a level II to III frost (Figure 4.18). The Jessner's solution or 35% TCA peel is our most frequently used mid-level peel and we find it effective in the treatment of solar lentigines, melasma, actinic keratoses, fine lines, photoaging, and in conjunction with laser resurfacing to blend lased and nonlased areas.

Deep chemical peels

Only phenol-based solutions are commonly used for deep peeling with extension to the reticular dermis. Indications for their use are found in Table 4.3.

Phenol peels The Baker–Gordon formula has been the most popular method for deep chemical peeling. It consists of 3 mL of 88% phenol, 8 drops of Septisol liquid soap, 3 drops of croton oil, and 2 mL of distilled water. Croton oil enhances penetration of phenol. The entire face cannot be treated at once, but rather should be broken down into subunits, allowing 10 to 15 minutes between subunits to avoid toxic blood levels of phenol. The eyelids should be treated with extreme caution, and the pretarsal lids should never be treated because of the increased risk of scarring. Postoperative wound care can consist of an open technique with application of petrolatum ointment, or a closed technique with occlusion by tape, resulting in deeper peeling. Results are comparable to those achieved by laser resurfacing.



Figure 4.18 (A&B) Jessner's solution and 35% trichloroacetic acid peel. At baseline, this patient has multiple solar lentigines and mild actinic damage **(C)** A level II to III frost is achieved immediately following application of 35% TCA **(D)** and rapidly fades over several minutes (continued)



Figure 4.18 (Continued) (E) Desquamation is appreciated 4 days following the peel (F&G) A durable response is achieved, with decreased solar lentigines and improved skin texture and tone persisting at 3 months postpeel

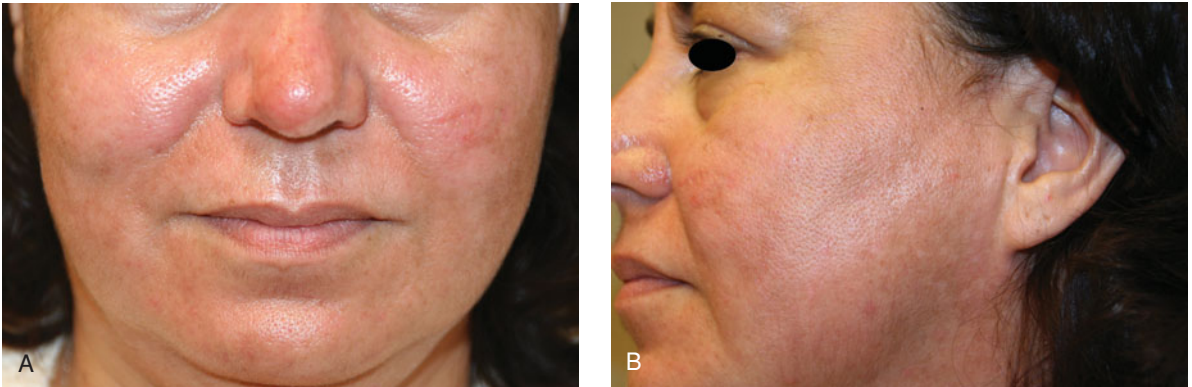


Figure 4.19 (A&B) Post-inflammatory hyperpigmentation. Hyperpigmentation in this patient appeared approximately 4 weeks after a mid-level chemical peel with Jessner's solution and 35% TCA. This complication is more common in patients with darker skin tones. Treatment can include topical hydroquinone, topical steroids, topical retinoids, sun protection, or repeating a mid-level peel. Pigmentation often resolves spontaneously over several months

■ Complications to Avoid

With proper patient selection and technique, complications from chemical peels are uncommon. Adverse events increase with depth of peel penetration and are very uncommon following appropriate use of superficial and mid-level peels. Table 4.4 lists some potential adverse events and the approaches adopted for their management (Figure 4.19).

REFERENCES

1. Alster TS, Doshi SN, Hopping SB. Combination surgical lifting with ablative laser skin resurfacing of facial skin: A retrospective analysis. *Dermatol Surg.* 2004;30:1191-1195.
2. Rubenstein R, Roenigk HH, Stegman SJ, et al. Atypical keloids after dermabrasion of patients taking isotretinoin. *J Am Acad Dermatol.* 1986;15: 280-285.
3. Wolfe SA. Chemical face peeling following therapeutic irradiation. *Plast Reconstr Surg.* 1982;69(5): 859-862.
4. Hevia O, Nemeth AJ, Taylor JR. Tretinoin accelerates healing after trichloroacetic acid chemical peel. *Arch Dermatol.* 1991;127(5):678-682.
5. Fulton JE, Jr. Jessner's peel. In: Rubin MG, ed. *Chemical Peels.* Philadelphia, PA: Elsevier Saunders; 2006:57-71.
6. Rubin MG. *Manual of Chemical Peels: Superficial and Medium Depth.* Philadelphia, PA: Lippincott-Raven; 1995.
7. Sharquie KE, Al-Tikreety MM, Al-Mashhadani SA. Lactic acid chemical peels as a new therapeutic modality in melasma in comparison to Jessner's solution chemical peels. *Dermatol Surg.* 2006;32: 1429-1436.
8. Brody HJ. Variations and comparisons in medium-depth chemical peeling. *J Dermatol Surg Oncol.* 1989;15(9):953-963.
9. Monheit GD. The Jessner's + TCA peel: A medium depth chemical peel. *J Dermatol Surg Oncol.* 1989; 15:945-950.
10. Coleman WP III, Futrell JM. The glycolic acid trichloroacetic acid peel. *J Dermatol Surg Oncol.* 1984;20(1):76-80.

Photodynamic Therapy in Facial Rejuvenation

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BACKGROUND

Photodynamic therapy (PDT) is a useful tool in the armamentarium of the cosmetic physician for treating photoaging. In topical PDT, a prodrug is applied to the skin, and is naturally converted in vivo into a photosensitizing molecule (in this case, protoporphyrin IX [PpIX]) in the cells. Various light sources can be used to activate the photosensitizing agent. The choice of light source is determined depending on the patient's skin condition as well as practical considerations.

PDT is defined as a photochemical, oxygen-dependent reaction involving light and a photosensitizer, resulting in the generation of singlet oxygen to cause oxidative damage to target cells. In addition, indirect damage to these areas occurs through inflammatory cascades, which may also play a role in the effects of PDT in photoaging.¹ The exact mechanism of this effect has not been fully elucidated, though the cosmetic benefits are thought to be mediated through multiple pathways.² Some of the benefits may simply be from clearing of scaly and/or erythematous lesions such as actinic keratomas or thin seborrheic keratoses. Additionally, a generalized inflammatory reaction elicited by PDT may contribute to generalized skin texture and tone improvement through a peeling process such as those seen with chemical peels or superficial laser peels.

The inherent properties of the light source used may also contribute to the effectiveness of PDT for rejuvenation. Filtered blue light (417 ± 5 nm) has traditionally been used for activation of PpIX because of the peak in the porphyrin absorption spectrum referred to as the Soret band (400–410 nm).³ The effects of blue light on *Propionibacterium acnes* bacteria are well known, since they endogenously produce protoporphyrins with a similar absorption spectrum in the blue-light range. The therapeutic effects of commonly used light sources such as IPL and PDL therapy are summarized in Table 4.6. Using such alternative light sources for activation takes advantage of the inherent beneficial effects of each light source as it pertains to cosmetic improvement of the skin.

PATIENT SELECTION/ PREPROCEDURE EVALUATION

PDT rejuvenation can certainly be considered in any patient with AKs and photodamage of the skin, since the treatment of AKs is its primary indication in the United States. Physicians as well as patients have noted the improvement of facial skin texture and tone following PDT treatments for AKs. One such early report was in a study by Touma and colleagues looking at the effects of topical 5-delta-aminolevulinic acid (5-ALA) therapy combined with a blue-light source for the treatment of AKs.⁴ This study demonstrated an improvement in overall photodamage as well as improvement in sallowness and fine wrinkling.

PDT facial rejuvenation can also be considered in almost any patient who would benefit from IPL treatments for signs of photoaging. These treatments can target photodamage (including sallowness), mottled pigmentation, telangiectasia or poikiloderma of Civatte, fine lines, and rough skin texture. Using PDT with IPL can also lead to fewer treatments, overall, to achieve the same results compared to IPL alone.⁵

TECHNIQUE

Equipment

Light sources

Even though its highest absorption peak lies in the visible blue light spectrum, PpIX has several other peaks in its absorption spectrum; therefore, light sources with wavelengths in any of these other peaks can be used as activators (Table 4.6).

After the findings of Touma and colleagues using blue light were published, studies using IPL-activated PDT emerged. In one split-face study, 20 subjects demonstrated the significantly increased efficacy of IPL-activated PDT versus IPL alone in the improvement of global photodamage, mottled pigmentation, and fine lines.⁵ Subsequent publications of similar design demonstrated comparable results.^{6,7}

Patient Positioning

The best position for the patient during both the application of the topical agent and the treatment processes is

TABLE 4.6 ■ Light Sources Used in Photodynamic Therapy

Source	Type	Common Uses Besides PDT
Blue light	Continuous wave	Acne
Red light	Continuous wave	Acne
Pulsed dye laser	585–595 nm laser	Telangiectasias and various vascular lesions, acne
Intense pulsed light	400–1200 nm	Telangiectasias and lentigines, acne

generally a sitting reclined position or a supine position on a comfortable examination table. Appropriate eye protection for the patient and staff in the room should be used in accordance with the recommendations of specific light source.

■ Step-by-Step Technique

Topical PDT treatments involve a five-stage process. These stages are outlined in Table 4.7. First, the area to be treated is identified and prepared. The preparation varies tremendously from physician to physician. At the very least, a gentle cleanser is recommended for the treatment area in order to remove surface debris, makeup, and surface lipids. Some advocate further preparation of the skin with acetone scrubs or microdermabrasion immediately before the treatments. One study of ALA PDT with a 595-nm PDL demonstrated a greater erythematous response with two passes of microdermabrasion followed by a 10-minute incubation of ALA versus a

TABLE 4.7 ■ Steps in Performing Topical Photodynamic Therapy

Preparation (to assure even application of the topical 5-ALA)
Application (of the 5-ALA in a uniform pattern)
Incubation (the time allowed for the topical 5-ALA to have contact with the skin before washing the face)
Activation (exposure of the skin to the activating light source)
Protection (of the skin from ambient light)

5-ALA, 5-delta-aminolevulinic acid.

1-hour incubation of ALA without microdermabrasion.⁸ Retinoids are often used for several weeks prior to the procedure, but should be discontinued 5–7 days before the treatment session, and may be restarted once all post-treatment erythema and irritation has subsided. As with any cosmetic procedure, a good clinical baseline photograph prior to the procedure is recommended to document the clinical effects of the treatment.

Once the skin has been prepared, the prodrug is applied. In the United States, a 20% 5-ALA solution delivered with a sponge-tip applicator (Levulan Kerastick, DUSA Pharmaceuticals, Inc., Wilmington, MA) is available through its FDA approval for the treatment of actinic keratoses. This solution is prepared by crushing the two ampules within the applicator stick and shaking the applicator vigorously but carefully for 2–3 minutes while holding the stick with the applicator tip pointed upwards. By using two layers and applying the strokes oriented in different directions (horizontal in one pass, and vertical in another), the chance of missing any areas in the field of treatment is minimized. The prodrug is absorbed and converted to the active compound, PpIX, within the skin structures in a time-dependent fashion.

Several protocols have been described ranging from standard incubation (>3 hours) to short incubation (1–3 hours) to ultrashort incubation (30 minutes to 1 hour). From a practical standpoint, 1-hour incubation is often used for cosmetic indications; however, this may be adjusted depending on whether a more vigorous or less vigorous response is desired. This must be weighed against the increased chance of phototoxic reactions and more significant pain with longer incubation times versus less discomfort and likely decreased efficacy with shorter incubation times. When in doubt, utilize a shorter incubation time for the first treatment session and increase incubation time as necessary to achieve the desired response in the subsequent treatment sessions. This caveat applies more to darker skin types, which can have significant pigmentary alteration (hypopigmentation or hyperpigmentation) if the inflammatory response is too vigorous. At the end of the incubation period, the face is cleansed again.

The face is then treated with the desired light source to activate the PpIX. After activation, sunscreen is applied and strict photoprotection is reviewed with the patient. Education regarding postprocedural sun protection or avoidance is a critical portion of the treatment process and is covered in greater detail later.

TABLE 4.8 ■ Potential Complications with Topical PDT Treatments

Phototoxic reaction
Persistent erythema
Blistering
All complications associated with light source (burns, retinal damage, hypopigmentation, hyperpigmentation)
Herpes simplex outbreak
Secondary infection of skin

COMPLICATIONS TO AVOID

Although adverse events with ALA PDT for photorejuvenation are not common, caution is to be advised when initially gaining experience with the technique. Some of the complications that have been reported are listed in Table 4.8. The risk of complications goes up when treating darker skin types and with patients who are on photosensitizing agents. Reduced incubation times and a more conservative light treatment can help minimize these risks.

POSTTREATMENT INSTRUCTIONS AND CARE

Thorough posttreatment care instructions and education for the patient are essential to help prevent phototoxic reactions. Patients should be educated both prior to and after the procedure regarding the importance and proper use of sunscreens, sun protective clothing, and sun/light avoidance. Specifically, patients need to be instructed to avoid any sunlight, daytime outdoor exposure, windows,

skylights, and even bright indoor lights for at least 2 days following the treatment.

REFERENCES

1. Goldman MP, Atkin D, Kincaid S. PDT/ALA in the treatment of actinic damage: Real world experience. *Lasers Surg Med.* 2002;14S:79.
2. Korbek M. PDT-associated host response and its role in the therapy outcome. *Lasers Surg Med.* 2006;38(5):500-508.
3. MacCormack MA. Photodynamic therapy. *Adv Dermatol.* 2006;22:219-258.
4. Touma D, Yaar M, Whitehead S, Konnikov N, Gilchrist BA. A trial of short incubation, broad-area photodynamic therapy for facial actinic keratoses and diffuse photodamage. *Arch Dermatol.* 2004;140(1):33-40.
5. Dover JS, Bhatia AC, Stewart B, Arndt KA. Topical 5-aminolevulinic acid combined with intense pulsed light in the treatment of photoaging. *Arch Dermatol.* 2005;141(10):1247-1252.
6. Alster TS, Tanzi EL, Welsh EC. Photorejuvenation of facial skin with topical 20% 5-aminolevulinic acid and intense pulsed light treatment: A split-face comparison study. *J Drugs Dermatol.* 2005;4(1):35-38.
7. Gold MH, Bradshaw VL, Boring MM, Bridges TM, Biron JA. Split-face comparison of photodynamic therapy with 5-aminolevulinic acid and intense pulsed light versus intense pulsed light alone for photodamage. *Dermatol Surg.* 2006;32(6):795-801; discussion 801-803.
8. Katz BE, Truong S, Maiwald DC, Frew KE, George D. Efficacy of microdermabrasion preceding ALA application in reducing the incubation time of ALA in laser PDT. *J Drugs Dermatol.* 2007;6(2):140-142.

Threads and Mini-lifts

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Hayes B. Gladstone**

BACKGROUND

The cornerstone of maximizing facial rejuvenation is still a surgical lifting procedure. This procedure comes in many varieties. Traditionally, lifting procedures can be divided into neck, lower face, mid face, and upper face.

Combined with liposuction and platysmal placcation, a formal neck lift will greatly improve the neck by removing redundant skin, creating a more optimal cervico-mental angle, and even decreasing rhytides to a degree.

For the lower face, facelifts can be divided into anterior and posterior. The ancestor to the S-lift—an anterior lift—was first described by Suzanne Noel, a Parisian cosmetic dermatologist in the 1920s. These anterior lifts have limitations given its short flap and lack of posterior undermining.



Figure 4.20 An example of the incision for the “G” lift

Rhytidectomies that are more complete include the posterior incision, and vary from a modest “G” flap to the conventional rhytidectomy (Figure 4.20). Dermatologic surgeons have made a major contribution in the arena of traditional supra-superficial musculoaponeurotic system (SMAS) rhytidectomies by demonstrating that they can be performed effectively without general anesthesia by using a tumescent solution.

Given the public demand for less-invasive procedures with decreased downtime, thread lifts have become a potential alternative. While Contour thread lifts are currently off the market, the concept of using sutures was first introduced over a decade ago, and is a reasonable option. Endotine, which uses a bioabsorbable fixation device, has also been demonstrated to be effective for minimally invasive brow and mid facelifts.

■ Mini-Lifts

The role of traditional rhytidectomy in achieving dramatic results has long been recognized. Nonetheless, over the

past few decades there has been a growing appreciation for our ability to achieve significant outcomes, albeit more subtle, among those seeking less-invasive surgeries with shorter recovery time.¹ While facelifts have been performed mostly by plastic and facial plastic surgeons, over the past decade dermatologic surgeons have increasingly performed this procedure. Because many facial repairs such as a cervicofacial rotation flap require a similar knowledge of the anatomy and surgical skill set as the rhytidectomy, it is not surprising that dermatologic surgeons have now become innovators for this procedure. The term “mini-lifts” has come to define these simpler, more conservative procedures.

The Webster Lift, described in the late 1970s and early 1980s, featured a smaller skin flap with shorter anterior and posterior incisions, and simple plication of the SMAS-platysma unit.² In Germany, in 1983, Ansari coined the term S-lift to describe a procedure with a smaller S-shaped incision, limited primarily to the preauricular region, starting behind the ear lobule and ending in the temporal hair-bearing area.³ The central advantage of this approach was the elimination of a retroauricular incision and scar therefore helping to avoid potential complications such as hairline distortion. Fulton and others have more recently helped to popularize this procedure, proposing variations in extent of skin, SMAS and platysma dissection, and site of fixation.⁴ Although the S-lift technique usually involved simple plication of the SMAS-platysma unit, the “short scar” method proposed by Baker combined the S-lift approach with lateral SMA-Sectomy, claiming that the vectors created on closure could aid in correction of both the nasolabial fold and lower face, including the jowl.⁵

Adjunct procedures such as liposuction, fat transfer, chemical peels, or laser resurfacing can enhance results when used in combination with the above techniques.⁴ Perhaps the most important factors to consider for achieving optimal results and patient satisfaction with any mini-lift are patient selection and appropriate expectations, especially given the limitation in these procedures to address more extensive cervical laxity often seen in older adults.⁶

■ Suspension Lifts

Various modified sutures have been introduced for the purpose of elevating ptotic tissues of the neck and face, including the brow, jowls, and mid face. Their use was popularized in Russia in 2002 by Sulamanidze with the



Figure 4.21 (A) Pre mini-lift (B) post mini-lift at 6 months

introduction of blue APTOS (anti-ptosis) threads.⁷ These polypropylene self-anchoring sutures studded with numerous bidirectional (converging) barbs are inserted into the subdermis where the hook-like projections support and fix local tissue. This procedure, marketed as the Feather-Lift, has not yet received FDA approval. In comparison, clearance was granted in late 2004 for the use of Contour Threads™, a clear suture with unidirectional barbs.⁸ It has a long, straight inserting needle on the distal end to aid in placement in the mid-subcutaneous tissue, and a curved needle on the proximal aspect to secure the thread to fascia.

While these two “thread lifts” utilize tiny barbs, a third technique introduced by Eremia uses an anchor suspension suture created by the placement of 7 to 9 mm pieces of suture secured by basic square knots along a slowly absorbable 2-0 monofilament suture.⁶ The 5 to 9 cross suture bits, spaced about 1 cm apart, act as anchors allowing tissue elevation once the proximal ends are secured to fascia.

The multiple supporting structures in these three suspension lift techniques allow tension to be distributed over a larger area of the adjacent subcutaneous tissue. Additionally, secondary fibrosis along the tract of the inserted suture should also help maintain support of lax tissue. Limited studies to date indeed show a relatively good safety profile and initial cosmetic results have thus far often been impressive (Figure 4.21) when used either in conjunction with open facelifts or alone as a pure suspension lift.^{9,10} Nevertheless, questions remain regarding whether sustained results are feasible, and recent data with anchor suspension sutures suggest their benefit is mostly faded within 1 year when used alone.¹⁰ Ultimately, combinations of mini incisional lifts and threads may provide medium term solutions for moderate facial aging.

ANATOMICAL LANDMARKS

Mastery of pertinent cervicofacial anatomy is central to avoiding complications and achieving optimal results with

all facial rejuvenation, but an in-depth discussion is beyond the scope of this review. A good understanding of the SMAS and the fibromuscular superficial fascial layer of the face and neck, is especially important in mini-lifts. The SMAS is continuous with the platysma muscle inferiorly, and extends superiorly near the zygomatic arch until transitioning to galea and superficial temporalis fascia. Its lateral border inserts into parotid fascia, overlying the parotid gland, and approaches the nasolabial fold medially.

The SMAS encloses the facial muscles and is connected to the overlying skin by ligamentous extensions. During mini-lift procedures, limited undermining allows the preservation of these attachments so that exertion applied with SMAS-platysma tightening, by either plication or partial excision, is transmitted to the overlying skin with visible repositioning of sagging tissues.³ While the risk of serious injury to vessels and nerves is reduced, given the lack of more extensive undermining indicated in traditional facelifts, caution should still be taken. In particular, thinning of the SMAS medially and superiorly indicates that special care must be taken to avoid the more vulnerable superficial components of the facial nerve in these areas,⁶ such as the frontal branch along the mid-zygoma.

Suspension lifts require similar care, as sharp inserting devices pose a risk, and a thorough understanding of the subdermal plane facilitates their appropriate use. For example, although Contour and APTOS threads were designed for placement in the mid to superficial subcutaneous tissues, anchor suspension sutures require deeper placement, just above the SMAS, as superficial deployment of this larger suture may result in a palpable component.⁸

PATIENT SELECTION AND PREOPERATIVE CONSIDERATIONS

The S-lift is indicated for patients who have mild jowling and lower anterior facial skin laxity with redundant skin. More extensive mini-lifts and ultimately the conventional rhytidectomy incorporate posterior incisions that are appropriate for patients who have greater jowling and neck skin laxity. Contour Threads™ are indicated for elevation and fixation of the brow, mid-face, and neck. Silhouette Mid-Face Suture™ is indicated for elevation and fixation of the mid-face.

The S-lift is suited best for young patients with early laxity of the lower face. Generally, this includes patients in their late fourth to fifth decades. The patient should be in good health with appropriate expectations. A patient who under-

went a previous full rhytidectomy, but now has sagging may also be a candidate for this less-invasive procedure.

The thread lift is suited for a patient with mild laxity of the brow, mid-face, and neck who desires less postoperative downtime, a more subtle lift, and who understands the shorter durability compared with an S-lift or full face lift.⁸

Absolute contraindications to these procedures include active infection, blood clotting disorder, and pregnancy. Relative contraindications include a history of keloids, hypertrophic scarring, or any condition with wound healing abnormalities.

At the initial consult, the goals of the S-lift or thread lift should be discussed with the patient. These goals include providing a moderate lift to the lower face, reducing marionette lines, and creating a smoother jaw line. The postoperative course should be discussed, including initial bruising and swelling, which, for the S-lift procedure, will generally resolve at 1 week. Risks, including postoperative bleeding and nerve injury, should also be discussed. These issues should be thoroughly reviewed at the preoperative visit when the informed consent is signed.

As with other invasive procedures, preoperative instructions usually include avoiding aspirin for 2 weeks, and nonsteroidal anti-inflammatory drugs for 1 week prior to the procedure. This practice may be modified if the patient takes aspirin because of a personal history of a stroke, blood clot, or myocardial infarction. It is best to consult the patient's internist or cardiologist in these circumstances. If a patient cannot stop these medications, then it may not be in the best interests of the patient to undergo this type of elective procedure since safety is paramount in cosmetic surgery. Alcohol consumption should stop at least 2 days before the procedure, since it can also alter clotting factors. If the patient uses tobacco, then it is strongly advised that the patient stops its consumption 2 weeks before the procedure, since smoking may compromise even a short flap facelift.

TECHNIQUE

■ Mini-Lift: Equipment, Positioning, and Step-by-Step Technique

Following informed consent and preoperative photos, the patient receives oral sedation and analgesia. The patient is then marked. The extent of the incision depends on which lift is chosen. A tumescent solution of 0.1% lidocaine and 1/500,000 epinephrine is then infiltrated into the neck and



Figure 4.22 *Tumescing the face*

the lower face (Figure 4.22). Using smaller gauge cannulas (sizes 14 and 16), neck liposuction is performed from stab incisions performed in the submentum and the infra-auricular regions. The liposuction is carefully performed above the mandible to create a more optimal contour.

After contouring with liposuction, the incision is then performed. Prior to raising the flap, tunnels are made with the liposuction cannula with the suction switched off. Using either a “baby” Metzenbaum or Mayo scissors, the flap is undermined to approximately 6 cm anteriorly. The neck is undermined to midline, hermostasis achieved. Plication with 3-0 absorbable or nonabsorbable of the SMAS is then performed (Figure 4.23). The vector of

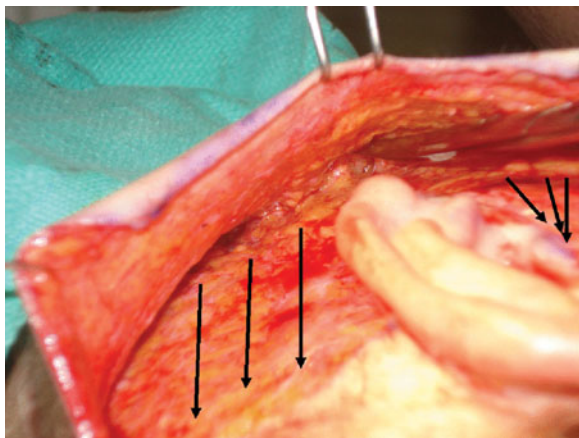


Figure 4.23 *Plication of the SMAS and platysma should be in the range 60 to 90 degrees in order to avoid the “wind tunnel” look*

these sutures ranges from 60 to 90 degrees. Generally, six to eight plication sutures are performed anteriorly, and four to six are performed posteriorly depending on the length of the incision. The skin is anchored superiorly and the excess is then trimmed. There should be no tension. The incision is closed in two layers with 4-0 monocryl along with 6-0 fast absorbing gut for the anterior aspect and 5-0 fast absorbing gut for the posterior aspect of the incision. A compression bandage is placed.

■ Contour Thread Lifts: Equipment, Positioning, and Step-by-Step Technique

Following informed consent and preoperative photos, the patient is marked. The vectors should be as vertical as possible. Depending on the site, the apex/entry point of the thread should be within the hairline. For neck thread lifts, the entry point is infra-auricular. Tumescant anesthesia is infiltrated. Using a 2.5-mm punch, a small incision is made for the entry point. This area is undermined with tenotomy scissors so that a small pocket is formed. Using a cannula, the marked pathway of the suture as well as adjacent areas are bluntly undermined. This procedure essentially creates a small sliding flap.

A CT400 barbed thread on a keith needle is inserted through the entry point, and then guided in the subcutaneous layer in a sinusoidal fashion following the markings. The needle exits just superior to the eyebrows for the browlift, just lateral to the nasolabial folds for the midface lift, and just lateral to midline for the neck lift. The thread should be pulled through until there are no barbs at the entry point. The thread is pulled gently retracted locking the barbs. The suture is cut leaving a “whisker.” Then the other end of the CT400 is inserted through the same entry point, and the procedure is repeated. Before inserting this needle, a Gore-Tex pledget may be placed onto the thread and positioned on the smooth portion, which will act as an anchor. Depending on the site, several sets of the sutures may need to be placed.

The patient is placed in a sitting position, and contouring begins by holding the “whiskers” and manually pushing up on the lax skin in a superior direction. This is continued until there is a visible lift and bunching of skin at the entry point. The excess sutures are then cut flush with the skin at the exit points. Some practitioners do not cut the suture, but have the patient return on the second and

third day to repeat the contouring process before trimming the sutures. A protective dressing is placed that makes it difficult for the patient to manipulate the lifted regions.

■ Complications to Avoid

Minor complications of surgical as well as thread-lifting procedures include ecchymosis and swelling. Other complications include hematoma, infection, widened scars, asymmetry, and premature sagging. Nerve complications include injury to branches of the facial nerve.^{11,12}

For the S-lift and more extensive facelifts, complications can be avoided by detailed knowledge of the facial anatomy and meticulous technique. For mini-lifts, the plane of dissection is above the SMAS. If this plane is maintained, then the risk of injury to the facial nerve will be minimized. When performing liposuction of the neck, it is important to remain superficial when along the jawline in order to minimize the risk of injury to the marginal mandibular nerve.

If a postoperative hematoma occurs, evacuation is important to decrease the risk of skin slough and infection. Depending on the size of the hematoma, it can be aspirated, or the flap may need to be taken down and a drain may be placed. If a nerve injury occurs and it is recognized, it should be reattached via microsurgery—particularly if it is a branch of the facial nerve.

While the above complications can occur with thread lifts, the most common adverse effects include thread breakage, thread migration, asymmetry, dimpling of skin, and thread extrusion.¹³ In case these complications occur, the thread may need to be removed, and the procedure repeated.

POSTOPERATIVE CONSIDERATIONS

It should be emphasized to the patient that bleeding may occur during the first 48 to 72 hours. The patient should not do any heavy lifting or vigorous activity for 1 week. The patient should sleep with her/his head on multiple pillows for the first three nights. A compression dressing is placed following surgery. The dressing is removed in the office after the first 24 hours, and is then replaced. For the first week, the patient should perform dressing changes on a daily basis, and clean the incision sites with a small amount of hydrogen peroxide on a cotton-tipped applicator and then apply a topical antibiotic.

While the thread lift is minimally invasive, in many ways, the postoperative care is more complicated and

tenuous than that for the incisional facelift. The thread-lift patient must be very gentle when cleaning the area where the procedure has been performed. Palpating the region or too vigorous wound care may “pop” the suture’s barbs from its grip, and release the lift. Therefore, the patient must also sleep on her/his back for the first 3 weeks. It is believed that after this period, there will be sufficient fibrosis to maintain the lift in case the barbs release. In reality, the patient is instructed to be extremely careful when manipulating the regions for the first 6 weeks. Additionally, the thread-lift patient may have some mild bruising and swelling that usually resolves in several days. Strenuous exercise should be avoided for 3 weeks.

REFERENCES

1. McCarty ML, Brackup AB. Minimal incision facelift surgery. *Ophthalmol Clin North Am*. 2005;18(2):305-310.
2. Webster GV, Davidson TM, White MF, et al. Conservative face lift surgery. *Arch Otolaryngol*. 1976;102:657-662.
3. Ansari P. Elimination of the retroauricular incision in face lifts. *Aesthetic Surg J*. 2003;23:14-19.
4. Fulton JE, Saylan Z, Helton P, et al. The S-lift featuring the U-suture and O-suture combined with skin resurfacing. *Dermatol Surg*. 2001;27(1):18-22.
5. Baker DC. Minimal incision rhytidectomy (short scar face lift) with lateral SMASectomy: Evolution and application. *Aesthetic Surg J*. 2001;21:14-26.
6. Eremia S. Rhytidectomy. *Dermatol Clin*. 2005;23(3):415-430.
7. Sulamanidze MA, Fournier PF, Paikidze TG, et al. Removal of facial soft tissue ptosis with special threads. *Dermatol Surg*. 2002;28(5):367-371.
8. ASDS Technology Report: Suspension Sutures.
9. Sulamanidze MA, Paikidze TG, Sulamanidze GM, et al. Facial lifting with “APTOS” threads: Featherlift. *Otolaryngol Clin N Am*. 2005;38(5):1109-1117.
10. Eremia S, Willoughby MA. Novel face-lift suspension suture and inserting instrument: Use of large anchors knotted into a suture with attached needle and inserting device allowing for single entry point placement of suspension suture. Preliminary report of 20 cases with 6 to 12-month follow-up. *Dermatol Surg*. 2006;32(3):335-345.
11. Saylan Z. The S-lift: Less is more. *Aesthetic Surg J*. 1999;19:406-409.

12. Choong JL, Jun Ho P, Sun Hye Y, et al. Dysesthesia and fasciculation: Unusual complications following face-lift with cog threads. *Dermatol Surg.* 2007; 33(2): 253-255.
13. Silva-Siwady José G, Díaz-Garza Celina, Ocampo-Candiani Jorge. A case of APTOS thread migration and partial expulsion. *Dermatol Surg.* 2005;31(3): 356-358.

Tightening Devices

Murad Alam

BACKGROUND

Historically, facial tightening has been the province of surgical excision procedures. The gold standard remains full-face rhytidectomy (face-lift), which offers the promise of dramatic improvement, with long-term reduction of mid-face lines, jowls, and sagging facial architecture. In general, lasers and other external energy devices have been less successful in inducing facial tightening than in improving other manifestations of skin aging, like dyspigmentation.

However, within the past 10 years, energy-based tightening devices have become available that offer some of the benefits of face-lifts with reduced risk and downtime. Chronologically first among these was carbon dioxide laser, which causes skin tightening via thermal skin contraction.¹ Particularly useful for effacing fine perioral and periorbital lines and skin redundancy, CO₂ laser can be used concurrently with face-lift to simultaneously affect both peripheral and central skin tightening.²⁻⁵

PATIENT SELECTION AND PREPROCEDURE CONSIDERATIONS

More recently, nonablative energy devices have been designed to diffusely tighten the entire facial and neck area.^{6,7} These devices are potentially attractive to both patients and physicians. Patients may find nonsurgical tightening less frightening, and they may view tightening devices as safer than face-lifts. From an anatomic standpoint, devices also offer the promise of results without visible scars, or the risk of focal neurovascular injury.

Physicians may also find tightening devices an attractive option. To the extent that tightening devices are unlikely to cause infection, dehiscence, injury, or asymmetry, they are safer. Postprocedure complications are rare, and additional corrective surgery or follow-up visits

are unlikely to be necessary. Additionally, tightening by device is less time and resource intensive than surgical tightening. For these reasons, more patients are good candidates for nonablative tightening than surgical lifting.

Device-based tightening is most appropriate for well-selected patients, such as patients who (a) show early skin sagging and have a good overall contour and skin consistency, (b) are not candidates for a face-lift or do not want one, and (c) have reasonable expectations and are likely to be satisfied with a modest benefit (Figure 4.24). For maximal combined effect, nonablative tightening should be delivered in association with other minimally invasive cosmetic agents, such as neuromuscular relaxants, injectable fillers, and lasers and light devices for improvement of skin surface pigment and vascularity.

■ Indications and Contraindications

For optimal results, it is important for physicians and patients to understand the indications for noninvasive skin tightening. Such tightening is most likely to be successful for young to middle-aged patients with mild to moderate skin laxity. Anatomic areas that may be responsive include the jowls, brows, nasolabial folds and mid-face, neck, and abdomen. Reasonable patient expectations are important since tightening devices offer the possibility of modest improvement but seldom result in the dramatic improvement of a “home-run.” Energy-based tightening is best for patients who definitely do not want a face-lift but would be satisfied with modest, safe tightening if such were attainable.

There are few absolute contraindications to noninvasive tightening, but some exclusion criteria are generally observed. Patients who expect the same degree of improvement from noninvasive skin tightening as is possible with a face-lift will likely be disappointed by even the maximal result obtained; hence, such patients should be excluded. Similarly, patients who are not prepared to be



Figure 4.24 (A) Prior to treatment skin laxity is observed in the jowl region; (B) Six months after treatment appearance of the jowl and neck is improved slightly.

in the minority that has no discernible objective improvement after treatment should also be excluded. If patients are more concerned about crepe-like skin or pigmentary abnormalities than about wrinkles and loose skin, vascular or pigment lasers may help them more than a tightening device. Extremely sagging skin and collapse of facial substructure require a face-lift, not energy-based tightening. Finally, patients who are not worried about having a face-lift and would like maximal results regardless of the degree of downtime and potential risk should be encouraged to consider rhytidectomy.

TECHNIQUE

■ Equipment and Devices

Monopolar radiofrequency: Emergence and adaptation

The first device specifically designed for noninvasive facial skin tightening was a monopolar radiofrequency machine introduced in 2002 (Thermage Thermacool).^{6,7}

The method of action was shown to include collagen remodeling, as with other nonablative devices. Additionally, Ross⁸ demonstrated contraction of the fibrous septae in the subcutis; this suggested that deeper thermal injury and contraction, and potentially greater tightening efficacy, were possible with this device than with earlier nonablative devices. It was also postulated but not proven that SMAS may undergo thermal contraction. The monopolar radiofrequency device was approved for brow elevation, and was also used for reduction of mid-face wrinkles, jowls, and redundant skin of the neck.

Bipolar radiofrequency, infrared light, hybrid devices, and ultrasound

A number of successor tightening devices have been developed^{9–15} (Table 4.9). Many of these claim to refine the underlying technology to optimize the tightening pioneered by nonablative monopolar radiofrequency. Specifically, newer tightening devices have employed some or all of the following components: (a) bipolar radiofrequency; (b) intense pulsed light, often

TABLE 4.9 ■ Technologies for Noninvasive Energy-Based Skin Tightening

Monopolar radiofrequency
Bipolar radiofrequency
Intense infrared light
Combined radiofrequency and light
Ultrasound

in the infrared range; (c) hybrid radiofrequency and light systems; (d) ultrasound; and (e) suction or vacuum for cooling.

■ **Step-by-Step Technique**

In general, with all these devices, clinically significant skin tightening requires many treatments several weeks apart. Overall, clinical benefit is usually mild to moderate, and interpatient variation remains common, with some noting a significant increased effect.

■ **Side Effects and Complications to Avoid**

Adverse effects are generally mild (Table 4.10), with transient erythema and edema, but rare burns and blisters may occur with some devices at high treatment levels. Intraoperative pain is usually tolerable and patients can return immediately to work or social engagements. Because of the time required for nonablative skin tightening, these treatments are frequently

TABLE 4.11 ■ Benefits and Limitations of Noninvasive Skin Tightening

Benefits	Limitations
Safe	At best, mild to moderate efficacy
Minimal discomfort	Significant variability of effect across patients
Minimal downtime	Questionable persistence of effect
Often efficacious	Difficult to quantify improvement
Suitable for most skin types	Not comparable in effect of face-lift and invasive modalities

delegated to nurses or other nonphysician medical providers.

CONCLUSION

Nonablative skin tightening of the face is a promising clinical technique that is still limited in its efficacy (Table 4.11). On the one hand, tightening by device is minimally invasive, well tolerated, and associated with little or no downtime. Moreover, the newest data does reveal significant efficacy under good conditions for selected patients. Limitations include the possibility of insufficient clinical benefit, the substantial cost and time involved to complete a treatment course, and the risk of rare but troublesome complications.

TABLE 4.10 ■ Adverse Events Associated with Noninvasive Skin Tightening

Frequency	Type	Method of Correction
Common	Erythema	Resolves within 1–3 d
	Edema	Resolves within 1–3 d
	Mild pain	Ceases after procedure
Uncommon	Erosions	Resolve within 1 wk (can use emollients and topical steroids)
	Hyperpigmentation	Resolves within several weeks to months (can use peels, bleaching agents)
Rare	Depressions/indentations	May require fillers or autologous fat grafting
	Burns	Require high potency topical steroids
	Textural abnormalities	May consider subcision

REFERENCES

1. Fitzpatrick RE, Rostan EF, Marchell N. Collagen tightening induced by carbon dioxide laser versus erbium:YAG laser. *Lasers Surg Med.* 2000;27:395-403.
2. Fulton JE, Saylan Z, Helton P, et al. The S-lift face-lift featuring the U-suture and O-suture combined with skin resurfacing. *Dermatol Surg.* 2001;27:18-22.
3. Weinstein C, Pozner J, Schefflan M, et al. Combined erbium:YAG laser resurfacing and face lifting. *Plast Reconstr Surg.* 2001;107:593-594.
4. Weinstein C, Pozner J, Schlefman M. Combined erbium:YAG laser resurfacing and face lifting. *Plast Reconstr Surg.* 2001;107:586-592.
5. Achauer BM, Adair SR, VanderKam VM. Combined rhytidectomy and full-face laser resurfacing. *Plast Reconstr Surg.* 2000;106:1608-1613.
6. Hsu RS, Kaminer MS. The use of nonablative radiofrequency to tighten the lower face and neck. *Semin Cutan Med Surg.* 2003;22:115-123.
7. Ruiz-Esparza J, Gomez JB. The medical face-lift: A noninvasive, nonsurgical approach to tissue tightening in facial skin using nonablative radiofrequency. *Dermatol Surg.* 2003;29:325-332.
8. Ross EV, Zelickson BD. Biophysics of nonablative dermal remodeling. *Semin Cutan Med Surg.* 2002;21:251-265.
9. Busciani A, Curinga G, Menichini G, et al. Nonsurgical tightening of skin laxity: A new radiofrequency approach. *J Drugs Dermatol.* 2007;6:381-386.
10. Bunin LS, Carniol PJ. Cervical facial skin tightening with an infrared device. *Facial Plast Surg North Am.* 2007;15:179-184.
11. Goldberg DJ, Hussain M, Fazeli A, et al. Treatment of skin laxity of the lower face and neck in older individuals with a broad-spectrum infrared light device. *J Cosmet Laser Ther.* 2007;9:35-40.
12. Mayoral FA. Skin tightening with a combined unipolar and bipolar radiofrequency device. *J Drugs Dermatol.* 2007;6:212-215.
13. Taub AF, Battle EF Jr., Nikolaidis G. Multicenter clinical perspectives on a broadband infrared device for skin tightening. *J Drugs Dermatol.* 2006;5:771-778.
14. Sadick N, Sorhaindo L. The radiofrequency frontier: A review of radiofrequency pulsed-light technology in aesthetic medicine. *Facial Plast Surg.* 2005;21:131-138.
15. Kulick M. Evaluation of a combined laser-radiofrequency device (Polaris WR) for the nonablative treatment of facial wrinkles. *J Cosmet Laser Ther.* 2005;7:87-92.

CHAPTER 5

Periorbital Rejuvenation

Brian S. Biesman

BACKGROUND

The periorbital region is often perceived as the first to reflect signs of aging. It is thus no surprise that requests for periorbital rejuvenation are among the most commonly received in an aesthetic surgery practice. In order to prescribe an appropriate treatment plan, it is essential to have a thorough understanding of both periorbital anatomy and perceived signs of aging or fatigue. In general, changes in skin texture, tone, laxity, or pigmentation are treated with chemical peels, lasers, or technology-based solutions. Orbicularis oculi muscle function can be modified via botulinum toxin (BTX) injections or surgery. Periorbital volume changes are best treated with surgical removal or redistribution of fat, or via volume replacement. This chapter will explore each of these approaches to periorbital rejuvenation.

ANATOMIC LANDMARKS

■ Skin

Aging changes in the skin are characterized by development of rhytides, pigmentary or textural abnormalities, and loss of elasticity.¹ Because skin quality is not altered by blepharoplasty surgery, adjunctive therapies such as dermabrasion, chemical peeling, or laser skin resurfacing may need to be incorporated into rejuvenation strategies.

■ Forehead and Eyebrows

The forehead extends from the hairline to the eyebrow and is higher in men than women.^{2,3} Dynamic horizontal forehead rhytides may indicate compensatory eyebrow or eyelid elevation, which, when present simultaneously, are indicative of underlying ptosis. The eyebrows normally rest at or above the superior orbital rim. In the upper third of the face (from the hairline to the upper lid), soft tissue aging changes manifest as forehead rhytides, glabellar furrows, or eyebrow descent.⁴ Since the eyebrow is contiguous with the upper eyelid skin, brow ptosis will affect the eyelid folds (Figure 5.1).

Eyebrow configuration can be a powerful communicator of moods. Raising the eyebrows produces a surprised look, while lowering of the medial eyebrow may give the impression of anger or sternness, particularly when accompanied by visible glabellar frown lines. Generalized drooping of the eyebrows may signal sadness or fatigue, especially when the downward displacement aggravates hooding of upper eyelid skin.

There is no single “correct” resting position or shape of the eyebrow and concepts of “normal” vary with gender and race. In general, male eyebrows rest at the level of the orbital rim and female eyebrows lie superior to the orbital rim. The eyebrows tend to be more arched in women, peaking between the lateral limbus and lateral canthus meridians. Eyebrows positioned below the superior rim are considered to be ptotic. If eyebrow ptosis is present, the upper eyelid skin becomes compressed downward, producing the impression of dermatochalasis. For such patients, eyebrow lifting alone may correct the apparent skin excess or may need to be combined with upper lid blepharoplasty. However, treating these patients with blepharoplasty alone will only drag the eyebrow further downward, worsening the upper lid crowding and resulting in a cosmetically unacceptable outcome.



Figure 5.1 Left upper eyelid ptosis (Note elevation of brows in an effort to lift the ptotic upper eyelid. Also note how a change in the eyelid position affects the relative amount of dermatochalasis present.)

■ Upper Eyelid and Palpebral Dimensions

The upper eyelid edge normally rests 2 mm below the superior corneal limbus. The maximal distance between the upper and lower eyelids (vertical palpebral dimension) with the eyes in primary (straight ahead) position is 9 to 12 mm. Abnormalities in the upper eyelid position should be recognized since underlying etiologies may need to be evaluated or the conditions be corrected. In individuals with *eyelid retraction*, the lid margin rests higher than normal and simulates “staring” or signals “surprise” (Figure 5.1). Those individuals with *ptosis* have a narrowing of the vertical palpebral dimension, imparting a “sleepy” appearance.

The upper *eyelid crease* is usually 9 to 12 mm above the central lid margin in adult Caucasian females and 8 to 10 mm above the lid margin in adult Caucasian males (Figure 5.1).⁵ The upper lid crease in Asian individuals is lower, usually in the range of 2 to 5 mm from the eyelid margin. There are additional anatomic differences between Asian and Occidental eyelids.^{6,7} The lid crease is created by the anterior insertion of the levator aponeurosis into the orbicularis and dermis. The distance from the eyelid crease to the inferior border of the eyebrow should be approximately twice the distance from the eyelid margin to the eyelid crease.

The *lid fold* represents the skin and orbicularis muscle that together drape over and obscure the lid crease when the eye is in primary position. Excessive folding or hooding may result from dermatochalasis, brow ptosis, or both. Affected individuals may perceive a sensation of lid “heaviness,” early fatigue with reading or watching television, and even loss of peripheral vision, a finding easily demonstrated on visual field testing.

The *superior sulcus* is the region between the superior orbital rim and the eye. In young or thin individuals, the sulcus may be flat or concave. Sulcus fullness may be caused by orbital fat herniation or generalized lid edema. In patients with both concave and convex sulcus areas, liposculpting or fat redistribution can be performed to even the sulcus definition and avoid exacerbating a peaked appearance.

The horizontal width of the palpebral fissure from medial to lateral canthi is approximately 28 to 30 mm.⁸ The lateral canthal angle is situated approximately 2 mm higher than the horizontal meridian of the medial canthal angle. Involutional lowering of the lateral canthal angle can produce a “sad” look.



Figure 5.2 “Pseudoherniation” of lower-eyelid fat

■ Lower Eyelid and Midface

Normally, the lower lid margin covers the inferior corneal limbus. With increasing age, there is a tendency for the lid to bow downward and rest at a lower position, sometimes resulting in scleral show.⁹ In youth, the lower lid contour is generally flat. Over time, the lower lid fatty tissue may protrude anteriorly, resulting in steatoblepharon (prominence of the orbital fat pads) and creating the impression of “bags under the eyes,” which may signal “fatigue” (Figure 5.2). The lower eyelid fatty tissue appears clinically to protrude in three zones, conceptualized as medial, central, and temporal compartments.¹⁰ However, anatomic studies indicate that there is actually no division and there exists a single large lower orbital fat compartment.^{11,12}

Additional signs of aging include progressive skin laxity and subcutaneous fat atrophy leading to descent of the midfacial structures (middle third of the face). This downward migration, in conjunction with laxity of the orbitomalar ligament, creates a nasojugal tear trough, malar festoon, or a decrease in cheek projection (Figure 5.3).

Dark circles in the nasojugal areas of the lower lids and medial canthus (periorbital hyperpigmentation) are an extremely common cause of aesthetic concern. They may represent epidermal or dermal melanin deposition, or can be created by a “shadowing” effect produced by pseudoherniation of orbital fat.¹³ Most frequently, there are numerous coexistent causes for dark circles necessitating multiple treatment modalities.



Figure 5.3 *Involuntional changes in the midface have led to flattening of the midface and development of a deep “tear trough” defect*

NONINVASIVE EYELID SKIN TIGHTENING

■ Patient Selection and Preoperative Evaluation

The only noninvasive skin tightening device that has been tested for safety on the eyelids is the Thermage ThermaCool (Thermage Inc., Hayward, CA)—use of this technology for facial rejuvenation is reviewed in Chapter 4 (see “Tightening Devices” by Alam). I have previously described both the safety and efficacy studies performed with this device.^{14,15} I strongly advise against the use of any noninvasive skin tightening or resurfacing device on the eyelid skin unless it is well designed and clinical safety trials have been performed. This is particularly true of 1064-nm Nd:YAG lasers and broadband infrared devices because of associated vision risks.

Noninvasive skin tightening treatment of the eyelids is best indicated for patients with mild to moderate dermatochalasis, reasonably good skin tone, and minimal eyebrow ptosis, eyelid ptosis, or herniated orbital fat. Ideal candidates for this noninvasive treatment either do not want or do not need blepharoplasty surgery. In general, younger patients who do not want or need to accomplish a very marked change in their eyelid appearance, or patients who have previously undergone blepharoplasty who are noticing a gradual recurrence of skin laxity are the best candidates for monopolar radiofrequency (RF) skin tightening.

Although efforts to conclusively implicate several factors such as tobacco use, advanced age, and sun exposure in negatively affecting skin tightening treatment outcomes have been unsuccessful, these factors should be carefully considered when selecting patients for noninvasive eyelid tightening treatment. Patients should be counseled to anticipate only modest improvement from this treatment technology as the amount of tightening achieved can vary dramatically from patient to patient. The factors responsible for this variability remain largely unknown (Figure 5.4).

The preoperative evaluation for patients undergoing noninvasive eyelid skin tightening or any other eyelid procedure should include an ocular history specifically including contact lens wear, dry eye syndrome, ocular infections or other ocular disease, and any ocular medications (prescription or over-the-counter). Objective assessment of visual acuity on either a standard Snellen eye chart or a handheld card must be obtained and documented in the chart. Visual acuity must be assessed



Figure 5.4 (A) A 42-year-old female before monopolar radiofrequency (RF) treatment of the eyelids (B) 6 months after monopolar RF treatment of the eyelids

one eye at a time as the contralateral eye is occluded, and should be assessed in the same manner at the patient's first posttreatment visit as well.

■ Technique

When used on the lids, the Thermage device should be used with the eyelid treatment tips developed specifically for this indication. Plastic corneoscleral protective shields (Oculo-Plastik, Montreal, Canada) must be used, but may be placed in one eye at a time so as to avoid unnecessary discomfort and a claustrophobic feeling that may be associated with bilateral simultaneous visual obstruction. Metal shields must not be used under any circumstances when using RF energy. The lenses should only be placed after a drop of propacaine hydrochloride 0.5% (Alcon Laboratories, Fort Worth, TX) anesthetic has been placed in each eye. If the patient develops ocular irritation or discomfort while the shield is in place, artificial tears should be applied. If these drops fail to provide symptomatic relief, propacaine drops may be used intermittently. The use of propacaine should be minimized to avoid inadvertent corneal injury. The internal aspect of the shield should be lightly coated with an ophthalmic lubricant such as Lacrilube (Allergan Inc., Irvine, CA) to further protect against disruption of the ocular surface. Because of concerns about the use of alcohol around the eyes, the removable grid usually applied prior to Thermage treatment is not used when the eyelids are treated.

Thermage treatment of the eyelids may be performed with the patient in either the supine or semi-upright position. A sitting position demonstrates the gravitational effects on the eyelids and brows more clearly. Treatment is typically begun on the upper eyelid pretarsal skin. Treatment energies are adjusted to patient comfort but fine-tuning of energies throughout the session will be required. The eyelids are kept on stretch to avoid “bunching” of the treated skin, which would result in uneven distribution of energy to the eyelid. Treatment is continued until a visible end point of tissue tightening is achieved. Treatment is performed up to the level of the eyebrows and down to the level of the eyelashes in the upper eyelid and from the inferior orbital rim up to the eyelashes in the lower eyelid. The lateral canthal or “crow’s-feet” region is also included in the treatment zone. Energy levels typically need to be reduced when treating the thin skin overlying the lateral orbital rim. Copious application of coupling fluid to the treatment



Figure 5.5 Plastic corneoscleral protective lenses must be used when performing any RF treatment (monopolar or bipolar) on the eyelids

zone throughout the session will maximize patient comfort and minimize the risk of eyelid skin burns. As the treatment of each eye is completed, the corneoscleral protective lens is removed using an appropriate suction cup (Figure 5.5). Treatment is then performed on the opposite eyelids. At the end of the treatment session, the coupling fluid is gently washed away with water.

■ Postoperative Instructions and Care

Patients are instructed to resume their normal eyelid skin care immediately. As there are no open wounds, specific wound care is not required. Patients may resume normal activities immediately after treatment. Gradual improvement can be expected for up to 6 months after treatment. Topical treatments such as TNS Recovery Complex (Skin Medica, Carlsbad, CA) or ascorbic acid may enhance outcomes, but experience with these products after skin tightening is anecdotal at best.

LASER SKIN RESURFACING OF THE EYELIDS

■ Background

Carbon dioxide laser skin resurfacing still remains the most efficacious method for treatment of advanced facial wrinkling and photoaging, including the periocular skin. The long posttreatment recovery period and the

substantial side effect profile associated with CO₂ resurfacing make it appropriate for use only in a limited number of patients who are unlikely to achieve favorable outcomes with any other treatment (except phenol peeling, an equally undesirable option for most). In an effort to improve fine wrinkling and dyspigmentation of the eyelid skin without the long recovery time or risks associated with ablative techniques, I have recently developed protocols for fractional and plasma resurfacing techniques.

Fractional resurfacing for facial rejuvenation may be accomplished with a number of different devices (as discussed in the section “Fractional Laser Resurfacing,” Chapter 4). The Fraxel laser (Reliant Inc., Mountainview, CA) is a 1550-nm diode-pumped erbium fiber laser that is approved by the Food and Drug Administration (FDA) for skin resurfacing, treatment of melasma, periorbital wrinkles, and scars created by acne or surgery. This device coagulates tissue in multiple 75- to 150-micron microthermal treatment zones (MTZs) with depth of injury extending up to 750 microns. Reepithelialization typically occurs within 24 hours.¹⁶ Previously, technical limitations prevented use of the Fraxel device on the eyelids. Development of a special treatment tip of the same diameter as the existing small treatment tip, but extending several millimeters from the housing, now permits manipulation on the eyelids and other small areas.

Safety and efficacy of the Fraxel laser treatment on the eyelids has been evaluated with a two-phase study. In the first phase, *ex vivo* testing was performed on human eyelid skin removed during routine blepharoplasty to determine acceptable treatment parameters. In the second phase, 20 patients with Fitzpatrick Type I to III skin received four treatment sessions at approximately 2-week intervals. All treatments occurred under topical anesthesia with follow up at 1 and 3 months following the final treatment. There were no serious complications after any of the treatments. Both the physician and the patient assessed the textural improvement, wrinkle reduction, and overall improvement in eyelid appearance using a quartile score. Patients returned to everyday activities within 2 to 3 days, by which time temporary posttreatment edema, erythema, and swelling had resolved. Preliminary evaluation of the data suggests that these techniques result in an average improvement in eyelid texture of 2.8, wrinkle improvement of 2.6, and overall eyelid appearance of 2.5.¹⁷

■ Patient Selection and Preoperative Evaluation

Ideal patients for fractional resurfacing of the eyelids have mild to moderate wrinkling and/or dyspigmentation of the eyelid skin without marked laxity or dermatochalasis. Fraxel treatments may be performed on patients who have undergone recent transconjunctival blepharoplasty, and, in fact, may be performed at the conclusion of the procedure. If a skin flap alone (as opposed to a skin-muscle flap) is elevated during the course of surgery, any ablative resurfacing treatments should probably be delayed for 6 months to permit full revascularization. Fractional resurfacing can probably be safely performed earlier in the postoperative period, but this has not been formally addressed so far.

A baseline ophthalmic history and evaluation must be performed prior to performing fractional resurfacing of the eyelids. Patients should be counseled to expect a few days of mild eyelid swelling and erythema, depending on the device and parameters used for treatment. Makeup can generally be restarted within a day or two after treatment. The potential adverse events associated with fractional resurfacing techniques are similar to those associated with ablative techniques (pigmentary change, scarring, textural change, etc.) but the relative risk of these events is dramatically lower than that anticipated after ablative resurfacing. Ectropion formation and marked hypopigmentation, both well known to be associated with CO₂ resurfacing of the eyelids, should be extraordinarily unlikely after fractional resurfacing because of the more limited injury associated with fractional techniques.

■ Technique

In our study of Fraxel treatments, topical anesthetic (compounded betacaine, lidocaine, or tetracaine) was carefully applied approximately 1 hour before treatment, ensuring that the topical anesthetic did not get into the eyes. After removal of the anesthetic, an optical tracer dye was applied and the patients were treated with the SR 750 device at fluences ranging from 8 to 20 mJ. Either 125 or 250 MTZ/cm² and a total of 1000 to 2000 MTZs were administered in each area in alternating passes that varied by 90 degrees in orientation until all passes were completed. In general, the higher the energy provided, the lower the density and the total number of MTZs. There was no observable association between



Figure 5.6 (A) Before fractional skin resurfacing of the eyelids using the Fraxel SR 750 **(B)** 1 month after four Fraxel SR 750 treatments (Note smoothing of fine wrinkles, improvement of skin texture, and even modest skin tightening)

total energy delivered and amount of postoperative edema and erythema. Superimposing passes and skip areas were carefully avoided. Metal corneoscleral protective lenses were used (Oculo-Plastik, Montreal, Canada) for all treatments, and the entire upper eyelid, lower eyelid, and lateral canthal region was treated. Systemic sedation or analgesics were not required (Figure 5.6).

Another fractional resurfacing device known as the Affirm (Cynosure Inc., Chelmsford, MA) has also been used successfully on the eyelids. This device delivers a 1440-nm beam that is separated via a diffraction grating into individual “microspots.” The depth of injury produced by this device is approximately 300 microns, corresponding to the observed depth of solar elastosis observed histologically, following treatment. Topical anesthesia is not required with this device and epidermal protection is provided via forced cold air. When used to treat the eyelids, energy settings of 3 to 5 J/cm² are typically employed. Two to three passes are usually applied, keeping the thin lid skin on stretch so as to ensure even distribution of energy. Spots are overlapped by 20% to 30%

and two to three passes are typically made, ensuring adequate time for skin cooling between passes. Pulse stacking must be avoided. Metal corneoscleral protective lenses should be placed behind the eyelids during this procedure. As with any fractional resurfacing procedure, four to five treatments are required to achieve best outcomes. Treatments are usually spaced 2 to 3 weeks apart but this interval may be varied to accommodate scheduling needs.

Postoperative Instructions and Care

After the operation, patients used cold compresses and kept their eyelids moist with a bland ointment such as petrolatum jelly until the wounds healed and makeup could be reapplied. Aggressive use of sunblock is encouraged on all treated areas. Products such as retinoic acid and its derivatives, glycolic and other fruit acids, and other agents commonly associated with irritation and dermatitis are usually avoided until the eyelids return to normal.

PLASMA SKIN RESURFACING OF THE EYELIDS

Background

For patients with moderate to severe degeneration or photoaging of the eyelids whose goal is to achieve skin tightening, smoothing of eyelid and periorbital rhytides, and improvement of dyspigmentation, a more aggressive treatment approach is required. While ablative techniques may be appropriate for some of these patients, many would accept a more modest improvement in return for a greater safety profile than traditional CO₂. In an effort to achieve this objective, a recently completed study evaluated *plasma skin resurfacing* of the eyelids.

Plasma skin resurfacing involves creation of high-energy gas via the delivery of a pulse of ultra high frequency RF, which excites a tuned resonator, imparting energy to a flow of inert nitrogen gas. When the plasma is delivered to the skin, rapid molecular energy transfer occurs with transmission to dermal layers. The depth of effect is determined by the amount of energy delivered per pulse. Differences between plasma skin resurfacing and laser skin resurfacing techniques include lack of chromophore dependency and maintenance of the structural integrity of treated tissue after delivery of plasma

energy. After the delivery of plasma energy to the skin, there are resulting zones of thermal damage and thermal modification (similar to ablative skin resurfacing). The zone of thermal damage is ultimately sloughed, while the zone of thermal modification recovers. Maintenance of an intact epidermis overlying the area of injured tissue appears to support shorter healing times relative to ablative skin resurfacing techniques. The greater the energy, the deeper the plasma is delivered to the dermis.¹⁸

The only plasma skin resurfacing device commercially available is produced by Rhytec Inc., Waltham, MA. This device is cleared by the FDA for cosmetic improvement for treatment of facial and nonfacial areas for rhytides, superficial skin lesions, actinic keratosis, seborrheic keratosis, and viral papillomata. Until recently, this device had not been formally evaluated for its safety and efficacy in treating the eyelids. In a recent study this device was used to treat the eyelids of 20 patients. Patients included in this study had more substantial photoaging and lid laxity than those included in the fractional resurfacing eyelid study. After performing a series of *ex vivo* eyelid treatments, parameters were selected to treat the remaining patients. Some treatments were performed at low-energy settings, while others were performed at high energy. Most treatments were accomplished with topical anesthesia only. Recovery typically ranged from 4 to 10 days. Patients indicated that the average amount of time required for their skin to return to a level where they were comfortable applying makeup was 11 to 13 days. Final data analysis has not been completed but preliminary evaluation suggests that most patients achieved marked skin tightening and wrinkle reduction (approximately 35%) and that the overall improvement in eyelid skin appearance ranged from 60% to 90%. Ideal parameters for plasma resurfacing of the eyelids are still under development.¹⁹

■ Patient Selection and Preoperative Evaluation

Patients selected to undergo plasma skin resurfacing of the eyelids should have more significant laxity, wrinkling, and dyspigmentation. A comprehensive ophthalmic history and examination should be performed prior to initiating treatment. Patients should be counseled to prepare for approximately 2 weeks of recovery time during which they may not be able to apply makeup. As these patients will have more extensive changes in their facial skin, treatment of the full face in conjunction with the perior-

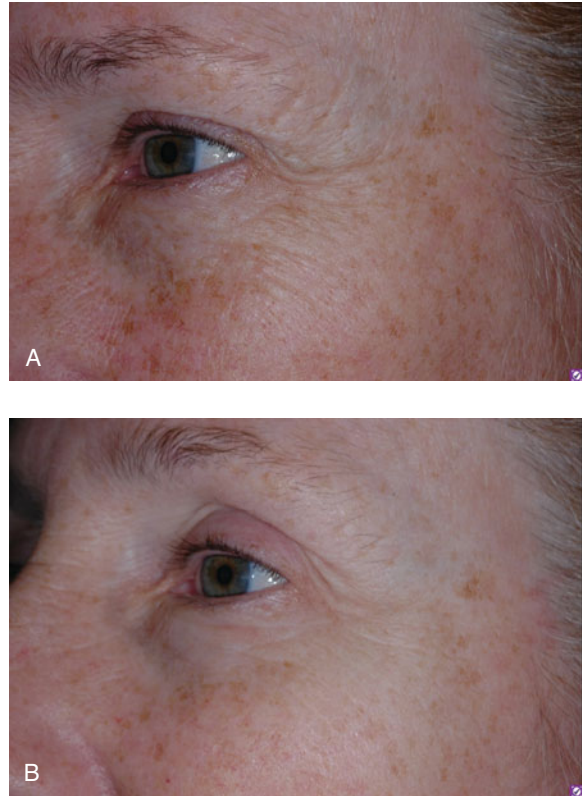


Figure 5.7 (A) Before plasma skin resurfacing of the eyelids (B) 3 months after a single treatment session (Note marked improvement in skin wrinkling, pigmentation, and tightening.)

bital region is often considered. Potential adverse events are similar to those associated with ablative resurfacing and may include infection, postinflammatory hyperpigmentation, persistent erythema, hypopigmentation (never reported), scarring or textural change, demarcation lines, and others. Again, the relative risk of these untoward outcomes is believed to be lower than is associated with CO₂ resurfacing. Pretreatment of the skin or the use of systemic antibiotics is not required for treatment limited to the periorbital region (Figure 5.7).

■ Technique

Topical anesthesia (such as compounded benzocaine 20%, lidocaine 8%, or tetracaine 4%) is applied to the eyelids approximately 1 hour prior to the procedure. The anesthetic agent is carefully and completely removed

with gauze and a drop of 0.5% proparacaine solution is placed in each eye. Plastic corneoscleral protective lenses must be used as the plasma is produced via an RF generator. Petrolatum ointment is placed on the eyebrows and eyelashes to prevent injury. The petrolatum does not ignite with exposure to plasma energy. The patient is placed in either a supine or a partially reclining position and the plasma device is brought into the field (Rhytec Inc., Waltham, MA). One to two passes are made at an energy setting of 1 to 3 J, depending on the amount of change present and the treatment goal. Occasionally, when treating at 3 J, patients will be unable to tolerate the treatment because of discomfort, and infiltration of lidocaine 1% or 2% solution into the treated areas may be required. Lidocaine injections should be avoided if possible as the temporary orbicularis oculi muscle paresis can lead to exposure and drying of the ocular surface.

When performing treatment with the plasma skin resurfacing device, it is imperative that the handpiece be kept perpendicular to the patient at a distance of approximately 5 mm from the point at which the aiming beam is focused. Overlap of spots by up to 20% is acceptable but pulse stacking should be avoided. The upper eyelids are treated from the lashes to the eyebrows, the lower eyelids are treated from the lashes to the inferior orbital rim, and the entire crow's-feet region is treated as well. Eyelid treatments should err on the side of under- as opposed to overtreatment because persistent erythema and textural change can result. At the conclusion of the procedure, the corneoscleral lenses are removed and bland petrolatum jelly is applied to all treated areas.

■ Postoperative Instructions and Care

Postoperative care following plasma skin resurfacing is similar to that following ablative skin resurfacing techniques. Petrolatum ointment is kept on the treated areas at all times until the devitalized tissue has been fully replaced by intact new epithelium. Antibiotic-containing ointments should not be applied because of the increased risk of topical hypersensitivity inducing contact dermatitis. Reepithelialization typically takes 3 to 10 days, depending on the relative aggressiveness of the treatment. Patients are advised to use dilute vinegar compresses (one teaspoon of white vinegar or one cup of water) several times per day until the skin is healed. If full-face treatment is performed, antiviral prophylaxis is recommended. Antibiotic prophylaxis after full-face treatment is optional but frequently administered. Antibiotic

and antiviral prophylaxis is generally not required if only periorbital treatment has been performed. Makeup may be applied once the wounds have reepithelialized. Agents such as retinoids, hydroquinones, azelaic acid, and others recognized to induce dermatitis should be avoided until the wounds have been completely healed for at least a few weeks.

PERIORBITAL USE OF BTX

■ Patient Selection and Pretreatment Evaluation

It is critically important to understand exactly what feature of the periocular region is of the most concern to the patient before making therapeutic recommendations. Some patients complain of “looking tired” when in fact they are concerned about loss of skin tone, excessive eyelid skin (dermatochalasis), eyebrow ptosis, dark circles under the eyes, hollowness in the tear trough area (medial lower eyelid below the orbital rim), bulging orbital fat pads, “hypertrophic” pretarsal orbicularis oculi muscle, and/or deep lateral canthal rhytides. Periocular rejuvenation is a complex topic and addressing only one of many needs may or may not provide a satisfactory result.

The best candidates for periocular BTX injections are those with mild to moderately deep lateral canthal rhytides and/or those who develop a “roll” of pretarsal orbicularis muscle as they smile. There are very few absolute contraindications to periocular BTX injection. It should be used with great caution in patients with true dry eye syndrome or systemic diseases that may produce dry eyes such as Sjogren syndrome and severe rheumatoid arthritis, and also in patients with ocular myasthenia gravis or other conditions that may affect extraocular muscle function. It should be used with great caution in patients whose eyes do not close well (a condition known as lagophthalmos) due to previous seventh nerve palsy, thyroid eye disease, or previous blepharoplasty.

Examination of the patient begins with evaluation of the patient literally from across the room. The patient's facial features are assessed for typical facial stigmata of aging including lentigines, erythema, fine wrinkling, loss of skin laxity, eyebrow and/or eyelid ptosis (or chronic eyebrow elevation to correct latent brow or lid ptosis), midfacial ptosis, jowling, loss of facial volume, and deep dynamic rhytides in the glabellar, perioral, and periocular regions. It is particularly important to view the patient

as a whole before concentrating on the periocular (or any other individual) region, as the goal of any treatment is to create a harmonious facial appearance. An observer's eye is actually drawn to asymmetry more so than rhytides or lines. That is, creation of a "smooth island in a sea of rhytides" produces a less natural appearance than if the patient were not treated at all.^{20,21}

The patient is then evaluated from a frontal perspective. Particular attention is paid to the presence of rhytides at rest, eyebrow contour and position, horizontal forehead rhytides that may be indicative of chronic brow elevation, extent of photoaging, presence of dermatochalasis in the upper eyelid, position of the upper eyelid margin relative to the pupil, and lower eyelid position. If white sclera is visible above the superior limbus or below the inferior corneoscleral limbus, additional ophthalmic evaluation is warranted. The presence of horizontal forehead rhytides may either indicate overactive use of the muscles or facial expression or a compensatory response to eyebrow ptosis. One must make this distinction, as weakening the frontalis muscle in the latter setting will uncover a previously latent eyebrow ptosis. That is, patients who have been elevating their eyebrows continuously in order to prevent impairment of their superior visual field will no longer be able to do so if their frontalis muscle is weakened.

The examiner can identify the patient with latent brow ptosis by studying the patient when the frontalis muscle is completely relaxed (instruct the patient to relax their forehead). If the eyebrows assume a lower position when the frontalis muscle relaxes, BTX should not be injected into the forehead. These patients need eyebrow and/or eyelid surgery to correct their underlying problem. Next, the patient is asked to gently (not forcibly) close the eyes to ensure complete apposition of the upper and lower lids. Some patients who have had prior surgery, trauma, or thyroid disease may be able to forcibly close their eyes but have an incomplete blink that leaves them highly vulnerable to symptomatic dry eyes if the orbicularis oculi muscle is weakened. The patient is then instructed to forcibly close the eyes. This permits evaluation of the pretarsal orbicularis oculi muscle in its dynamic state. The appearance of a prominent "bulge" in the pretarsal region is suggestive of "hypertrophic" orbicularis oculi muscle. Importantly, this condition must be differentiated from a "bunched" lower eyelid occurring as a result of cheek tissue recruitment with contraction of the zygomaticus major and minor muscles.

Horizontal rhytides in the lateral canthal region should be evaluated in both frontal and lateral views. The patient is viewed from each side first at rest, then with gentle eyelid closure, and finally during forced closure. This series of maneuvers permits the examiner to differentiate between fine skin wrinkles due to loss of elasticity and rhytides caused by action of the orbicularis oculi muscle. Next, a "map" of the orbicularis oculi muscle should be constructed by asking the patient to repeatedly squeeze their eyes tightly while the examiner palpates the lateral canthal region with the tip of the index finger. The orbicularis oculi muscle may be relatively small and confined to the region overlying the lateral orbital rim or may extend almost to the tragus laterally, into the lateral extent of the temporal region superiorly, and into the upper region of the midface inferiorly. It is important to map the orbicularis oculi muscle on both sides as its distribution may be asymmetric.

Finally, the position of the eye is assessed relative to the orbital rim. Patients with prominent eyes caused by high myopia (a condition in which the globe is actually longer than normal), thyroid eye disease, or shallow orbits should be treated with greater care as they are more prone to lagophthalmos and change in lower eyelid position after BTX injection.

■ Treatment Goals

BTX may be used in the periorbital region to diminish dynamic rhytides in the lateral canthal region, to weaken eyebrow depressors and thereby elevate or contour the brows, and to treat "hypertrophic" orbicularis oculi muscle in the lower eyelid.²⁰ In any given patient, it may be desirable to accomplish one or more of these goals. They are not mutually exclusive and should be considered independently.

When treating lateral canthal rhytides, the injector must distinguish between the lines caused by the action of the orbicularis oculi muscle, those caused by contraction of zygomaticus major and minor muscles (causing upward movement of the cheek with smiling), and those due to photoaging of the skin. Only those lines clearly caused by contraction of the orbicularis oculi muscle should be treated with BTX (Figure 5.8).²² Attempts to treat too far inferiorly may produce an unnatural appearance when smiling, facial asymmetry, or, in the most extreme circumstance, facial drooping. It is usually best to use the least amount of toxin necessary to produce the desired clinical effect while still providing adequate efficacy.



Figure 5.8 (A) Before injection of crow's-feet rhytides with botulinum toxin type A, active (B) 3 weeks after injection of crow's-feet rhytides with botulinum toxin type A, active—orbicularis oculi activity is reduced but not eliminated (C) Before injection of crow's-feet rhytides with botulinum toxin type A, passive (D) 3 weeks after injection of crow's-feet rhytides with botulinum toxin type A, passive (Note improvement in rhytides even when in repose.)

Use of too much toxin will minimize the action of the orbicularis oculi muscle to the point where the lateral canthus does not wrinkle at all with smiling and other facial expressions. This can signal the appearance of insincerity in some patients, and thus overaggressive injection in the crow's-feet region should be avoided.

A careful injection record should be kept and first-time patients are usually followed up 2 weeks after their injection. At the follow-up visit, the treatment goals should be reviewed and compared to the clinical results. If additional injections are needed to achieve the desired goals, these should be administered at the follow-up visit. If the patient has not achieved the best possible outcome,

careful notes should be made about adjustments that need to be made in dosage, placement, or both.

BTX can also be used to weaken the pretarsal orbicularis oculi muscle. This can reduce the prominent “roll” that appears with smiling in patients with “hypertrophic” orbicularis muscle and can also increase the size of the palpebral fissure (the distance between the upper and lower eyelids). While these goals are separate, they are indistinct in that it is difficult to accomplish one objective without the other. That is, treatment of hypertrophic orbicularis oculi muscle will not only reduce the muscle bulk upon smiling but will also make the eyes appear to be open wider. Flynn reported this change to be well

accepted by the patients, but lowering of the eyelid after blepharoplasty surgery is generally considered a complication to be avoided.²³ Further, some patients who complain of “bunching” of their lower eyelid with smiling or animation actually have either excess eyelid skin or a normal amount of eyelid skin that is compressed into a small area by the upward movement of the cheek associated with strong contraction of the zygomaticus muscles. These patients will not benefit significantly from BTX injection into the lower eyelid. Thus, a careful examination and discussion about realistic goals is needed prior to treatment.

The dosage of BTX-A injected into the periorbital region should be determined by the treatment goal, the size and strength of the orbicularis oculi muscle, and the position of the globe relative to the orbit. As detailed earlier, the size and distribution of the orbicularis oculi muscle may vary dramatically from patient to patient and even within individuals from one side of the face to the other. Individuals with the muscle distributed over a large area or with relatively hypertrophic muscle will require larger doses of toxin than may be considered “standard.” Similarly, patients with smaller muscles that do not contract as forcefully should receive relatively lower doses of toxin. The dosage should also be reduced when injecting the lower eyelid and inferior lateral canthal region of patients with prominent globes caused by shallow orbits, thyroid eye disease, or myopia (extreme near-sightedness due to a longer than normal eye) as these patients are at higher risk for ectropion or incomplete eyelid closure.²⁴

It is more helpful to think in terms of number of units per injection site than the total dose required to treat the entire region. As a general rule, 2.5 to 5 U per site is appropriate in the lateral canthal area. When treating the inferior pretarsal orbicularis oculi muscle only 1 to 2 U should be used per site.

■ Injection Technique

It is impossible to construct an injection site map or scheme that can be applied to patients in a generic manner. While some general guidelines can be applied, it is not feasible or appropriate to recommend a fixed number of injection sites, total dose of toxin, or the exact location of injection sites; treatments must be individualized to each patient. Asymmetry in orbicularis oculi muscle distribution often necessitates varying the dose of toxin between the two sides of the face. Figure 5.8 demon-

strates some typical injection sites on patients with normal and large sized orbicularis oculi muscles. When injecting in the lateral canthal region, the risk of toxin diffusion into the orbit increases with proximity to the canthal angle. While there a study is yet to be performed to address this issue directly, it seems that injecting at a distance 1 to 1.5 cm from the lateral canthal angle minimizes the risk of clinical effects of toxin on the extraocular muscles.

Pain management techniques for periorbital BTX injections range from no anesthesia, to the use of ice packs immediately prior to injection, to the use of topical anesthetic agents. Extremely anxious patients may very rarely wish to take a benzodiazepine prior to injection; but in this scenario, transportation arrangements must be made as driving while under sedation cannot be permitted. If a topical anesthetic agent is applied, it should be thoroughly removed prior to injection as the small 30 to 32 gauge needles may otherwise become occluded.

Although brow contouring has been briefly covered in Chapter 4 (see “Botulinum Toxin” by Neuhaus), it is appropriate to make at least a few comments on this topic within the context of periorbital rejuvenation since the brows exert such a strong influence on eyelid aesthetics. The rationale for using BTX-A to change eyebrow position is based on the selective relaxation of brow depressors. Centrally, the corrugator, depressor supercilii, and procerus muscles are all important brow depressors. Temporal to the papillary midline, the orbicularis oculi muscle is the sole brow depressor. The orbicularis oculi muscle is opposed by the frontalis muscle medial to the temporal line of fusion (conjoined tendon) and is unopposed temporal to this landmark (in the region overlying the temporalis muscle). Therefore, relaxation of the temporal brow depressor leads to brow elevation.

With regard to the placement of these injections, anecdotal reports suggest a variety of options including directly into the tail of the brow, above the tail, and below the lateral brow. As long as the orbital orbicularis oculi muscle is affected by the toxin, the exact placement of the injection probably is not critical, provided the diffusion into the inferior aspect of the frontalis muscle is avoided. Those injectors using a higher dilution (e.g., 4 mL of saline) and thus a greater volume may be well advised to inject relatively more superiorly so as to avoid unexpected diffusion of toxin into the levator muscle.

As the injections given to contour the brows are administered in the periorbital region, a few general comments in this chapter are warranted. First, injections given along the

superior orbital rim should be performed with care. This does not imply that injections cannot be safely given in this region. I routinely treat patients with intractable blepharospasm by injecting 5 U of BTX-A in each of the three positions along the superior orbital rim (a total of 15 U) along with an additional 2.5 to 5 U in the medial and lateral portions of the upper eyelid itself (a total of 5–10 U), without adverse sequelae. When injecting in this area, it is important to always direct the needle away from the orbit as directing the toxin toward the eye may result in diffusion of toxin into extraocular muscles or even serious injury if the patient were to move suddenly during the injection.

While difficult to study or prove definitively, the risk of toxin diffusion into the orbit may be reduced if the thumb of the nondominant (or noninjecting) hand is placed firmly along the orbital rim. A novice injector may wish to either stabilize his or her injecting hand against the patient, or the opposite, nondominant hand as it rests on the patient. This will prevent inadvertent perforation of veins or the eye in the event of an unexpected movement. When administering injections in the periorbital region, the injector must take great care to always point the needle away from the eye. To minimize the risk of ecchymosis, injections in this region are best administered intradermally or subcutaneously. When possible, injecting immediately over vessels should be avoided. Finally, injections should be administered from the side, and never from the front of the patient, to avoid directing the needle towards the orbit.

Although it is generally true that absolute rules about dosage and number of injection sites should not be made, great care should be taken when injecting the pretarsal orbicularis oculi muscle. This muscle can be injected in its central portion (immediately below the pupil) or, alternatively, may be injected in two sites, roughly corresponding to the medial and lateral corneoscleral limbus. If a single central injection is planned, a dose of no more than 2 U should be injected initially. Additional injections can be administered as needed at the 2 weeks follow-up visit. If two separate sites are used, no more than 1 to 1.5 U should be delivered at each site. Excessive delivery of toxin in this region can impair eyelid closure and may lead to tearing, ectropion, and even inward turning of the eyelid against the eye (entropion). Entropion can occur when the preseptal orbicularis oculi muscle contracts forcefully in the setting of an immobile pretarsal muscle. In this situation, the preseptal muscle moves upward overriding the pretarsal muscle and pushing the pretarsal muscle and eyelid margin inward toward the eye. This usually produces severe ocular pain

and irritation. When administering lower eyelid injections, it is advisable to deliver the toxin subcutaneously to avoid diffusion into the inferior oblique muscle, which occupies a relatively anterior position in the orbit. If the inferior oblique muscle is affected, vertical and/or torsional diplopia can result.

■ Postoperative Instructions and Care

Postinjection instructions after the administration of BTX injections vary widely as there are no solid data upon which recommendations can be based. Some physicians feel that repeated contraction of the treated muscles for several hours may be of benefit. Others instruct patients not to exercise, lie down, or assume other unusual positions that may result in migration of the toxin away from its intended destination before it can be taken up into the motor nerve termini. Each injector must develop strategies that work best in his or her practice. Ice packs are not typically required after BTX injection, unless localized bruising is suspected.

PERIORBITAL SOFT TISSUE AUGMENTATION

■ Background

Volume augmentation in the periorbital region can be extraordinarily useful for the treatment of deep nasojugal lines (often referred to as “tear trough” defects), elevation of the temporal brow, and even smoothing of fine eyelid wrinkling. The choice of product for each application varies according to personal preference.

■ Patient Selection and Pretreatment Evaluation

Patients with deep tear trough defects, ptosis of the mid-face, and descent of the tail of the brow may all be excellent candidates for periorbital soft tissue volume augmentation. Beware of patients who do not have a deep tear trough defect but complain of “dark circles” under the eyes as these patients are most likely have dyspigmentation of the skin, and thus will not benefit from volume augmentation. Patients must be counseled that bruising and swelling may be more common after periorbital soft tissue volume injection than after injections elsewhere. Bruising is quite common and can, occasionally, be severe. When medically acceptable, patients are

often recommended to avoid the use of anticoagulants for 10 to 14 days prior to injection.

Ptosis of the temporal “tail” of the brow often contributes to an aging or “tired look” in the periorbital region. Surgery has been the mainstay of treatment for brow ptosis, but now BTX and fillers may be used alone or in concert to elevate the tail of the brow. While BTX produces passive elevation by changing the dynamic balance of the brow elevators and depressors, fillers exert a direct effect by physically supporting the brow. Injections may be administered into the underlying retroorbicularis oculi fat (ROOF) pad, which lies above the orbital rim posterior to the brow cilia. The ROOF is invested by the inferior extension of the galea aponeurotica. Hyaluronic acid products are the current agents of choice in this region.

Etched-in lines at the lateral canthus that do not respond adequately to BTX and laser resurfacing treatment may be treated with fillers as well. Great care must be taken while administering the products. In the United States, Cosmoderm may be the best option for these fine etched-in lines as smaller particle size hyaluronic acid fillers have not been approved for use here by the FDA. Hyaluronic acid products may be delivered through a 32-gauge needle to minimize the particle size of the injected product in an effort to produce a smoother, more even result. Fillers should be used as the last resort in crow's-feet area as the skin is so thin that the product may be easily visible thus producing surface contour irregularities and/or discoloration because of Tyndall effect (Figure 5.9).^{25–27}

■ Technique

Soft tissue volume augmentation of the tear trough region may be performed with injections administered deep into the orbicularis oculi muscle (between orbicularis oculi muscle and periosteum) or with superficial injections between skin and orbicularis muscle. Hyaluronic acid products, such as Juvederm™ and Restylane®, are preferred for use in the tear trough region. With proper patient selection and product placement, spectacular results may be achieved. Most patients require treatment of the region from the nasal-third to half of the lower eyelid, although some require administration of the filler all the way to the lateral canthal region.

The aesthetic improvements seen with the use of a filler in the tear trough region are realized at a somewhat greater risk than are encountered in other areas. This anatomic region is tricky and unforgiving; administration of product too quickly will result in excessive swelling. Bruising is common and can, occasionally, be severe, as already stated. Injection of large “globules” of product too superficially will result in an uneven, swollen appearance and may produce Tyndall effect. Postinjection modification via massage can help manage lumpiness. Blindness is always at least a remote risk when injecting fillers in the periorbital region. Although this devastating complication has not been reported following hyaluronic acid injection, to believe that it cannot occur is naïve. I strongly feel that products such as calcium hydroxylapatite and poly-L-lactic acid should be used with great caution by experienced



Figure 5.9 (A) A 42-year-old female prior to hyaluronic acid (Restylane®) injection into tear troughs **(B)** 2 weeks after hyaluronic acid (Restylane®) injection into tear troughs

injectors in the tear trough region due to the risk of palpable or visible lumps that cannot be modified without surgery.

Profound anesthesia of the skin overlying the tear trough region may be easily achieved using topical agents alone, and injection of a local anesthetic is not required in almost all circumstances. Topical anesthetic agents are applied approximately 30 minutes prior to injection and may even be applied by the patients before presenting to the office. If a nerve block is desired, the eyelid branch of the inferior orbital nerve should be injected with a small volume of sodium lidocaine solution.

Tear trough injections may be given subcutaneously (between the skin and orbicularis oculi muscle), deep (between the muscle and underlying periosteum), or into the muscle itself. The decision regarding the level to which the product should be placed must be made on a case-by-case basis depending on the needs of the patient. It is recommended by some that 32 gauge needles be used to deliver product in this region in order to minimize the risk of delivery of large particles intravascularly, as well as to slow the actual speed of injection.

When injecting the tail of the brow, products with a larger particle size, such as Perlane® or Juvederm™ UltraPlus, may be of greatest benefit. These products are injected into the ROOF pad from a temporal to nasal direction. Care should be taken when injecting in the region of the junction of the temporal and central third of the brow as a large supraorbital vessel is consistently present in this location, and will produce a large hematoma if traumatized by the needle. It may be advisable to inject the product as the needle is advanced so as to “push” the vessels out of the way. However, this is not a fail-safe method to avoid bleeding. Injections of the brow may also be administered more superficially within the dermis to help enhance the skin quality and “push” the brow superiorly. Care must be taken while injecting thin-skinned patients using this technique, as the product can be highly visible if placed too superficially.

Injection into etched-in, “crow’s-feet” lines may also be tedious and challenging. If the lines are of sufficient depth and the skin is thick enough, favorable results may be achieved. However, when treating thin, delicate skin and/or fine lines, overcorrection may be difficult to avoid. If injections are to be administered in the crow’s-feet region, product must be placed superficially and with emphasis on slow and meticulous placement.

BLEPHAROPLASTY

■ Background

Blepharoplasty surgery is indicated when the eyelids require recontouring. Specifically, blepharoplasty is most useful when excess skin needs to be removed, the orbicularis oculi muscle is hypertrophic or requires tightening, or orbital fat needs to be removed or redistributed.

■ Patient Selection and Preoperative Evaluation

As is true prior to performing any eyelid or periorbital procedure, patient assessment begins with measurement of visual acuity using a standard eye chart or a near card. The visual acuity is clearly recorded in the chart. The eyebrow position is assessed by placing the patient in an upright position with the eyes gazing straight ahead. In this manner, the eyebrows and forehead are assessed, noting height above the orbital rim, the presence of horizontal furrows, and photoaging of the skin. The eyebrows are examined for position, contour, and fullness of the ROOF pad as manifested by prominence of the superior lateral orbital rim below the eyebrow. The position of the globe is noted relative to the orbital rim. In the setting of a flattened malar eminence, a shallow orbit, or axial myopia (seen in a near-sighted patient with a large globe), the eye will appear prominent and the upper and lower eyelids may appear retracted. In such cases, removal of tissue from the eyelids must be performed with great care as the proptotic appearance of the globe may be emphasized, creating an aesthetically unacceptable “staring” look and possibly even limiting the ability of the eyelids to close completely. True proptosis may be detected by exophthalmometry measurements.

Attention should then be turned to the upper eyelid itself. The eyelid position is observed and retraction or ptosis noted. The skin is inspected for evidence of medial canthal webs or epicanthal folds, wrinkling, old surgical scars, pigmentary change, and tumors or other defects, which may need to be addressed. The superior sulcus is examined for fullness or concavity. Gentle pressure on the globe through a closed eyelid will demonstrate the size and position of the central and nasal fat pads. If the superior sulcus is relatively concave preoperatively, care should be taken to avoid aggressive resection of orbital fat as this may produce excessive indentation of the superior fornix.

Next, the lateral portion of the eyelid is examined for the presence of fullness, which may represent brow ptosis, descent of the ROOF into the eyelid, prolapse of the lacrimal gland, or a combination of these factors. The position and definition of the upper eyelid crease is noted. If the crease is poorly defined, or if there is an asymmetry between the eyelids, the mirror should be used to demonstrate this difference. The “normal” position of the upper eyelid crease is slightly higher in females than males, but again will vary from one individual to the next. There are significant differences in eyelid crease position and contour between Asian and Occidental eyelids.¹⁰ Because of the social significance placed on this feature by most Asian societies, these differences must be recognized and respected. Patients usually are much more likely to notice a difference between their eyelids after surgery due to the careful attention typically given to the lids during the postoperative recovery period. Finally, a comprehensive ophthalmic examination is completed.

■ Technique: Upper Eyelid

Marking the incision

While there are many ways to mark an upper eyelid incision, serious problems may be avoided if some basic rules are followed. It is helpful to mark the eyelids with a fine point surgical marking pen. All makeup should be removed and the lids gently cleansed so that oil on the skin's surface does not disrupt the marking ink. The inferior border of the incision should be placed in the eyelid crease assuming it has not been distorted by previous surgery, disinsertion of the levator aponeurosis, or other factors. In Caucasian patients, the central portion of the crease is highest while the temporal and nasal ends are lower. In the Asian lid, the crease is more level than arched. If the creases are asymmetric, this should be discussed with the patient prior to the day of surgery. Nasally, the incision should extend no closer than 5 mm from the superior punctum and, in general, should not extend medial to a line drawn vertically through this structure. Carrying the incision too far medially can result in cicatricial band formation also known as “webbing.” The superior border of the incision should pass no closer than 1 cm from the inferior border of the eyebrow, the approximate point of transition between the thicker skin of the eyebrow and the thinner skin of the eyelid, so as to avoid induction of an eyebrow ptosis.

To estimate the amount of eyelid skin to be removed, the eyes are gently closed and one arm of a smooth forceps is placed in the eyelid crease, while the other pinches the redundant skin until the eyelashes just begin to rotate. This marks the maximum amount of skin that may be safely removed. The temporal extent of the incision should not extend lateral to a line drawn vertically through the lateral end of the eyebrow (Figure 5.10). Once the eyelids have been marked with the patient in a supine position, the patient may be placed in a sitting position and the markings are carefully compared for symmetry. Eyelid marking should be done prior to administration of sedative agents.

Anesthesia and patient positioning

Upper blepharoplasty may be performed with local anesthesia alone or in conjunction with systemic oral or intravenous sedation. Occasionally patients will even request general anesthesia. Local anesthesia, usually lidocaine with or without bupivacaine in a solution containing 10% hyaluronidase, is injected subcutaneously in the central upper lid using a 30-gauge needle. The orbicularis muscle should not be injured. A “bleb” containing 1.5 to 2.0 mL of anesthetic is placed between the skin and orbicularis muscle in the central region of the upper lid. Digital massage is then used to facilitate distribution medially and laterally throughout the upper eyelid, thus achieving adequate anesthesia from a single injection. This technique of delivering anesthesia avoids multiple punctures in the eyelid skin, thus reducing trauma and the likelihood of hematoma formation.

The patient is then prepped and draped in the usual sterile fashion. If a CO₂ laser is to be used, all laser-safe precautions are observed.

The procedure is begun with the placement of corneoscleral eye protection (Oculo-Plastik, Montreal, Canada). If a laser is to be used to make the incision, the ocular protection must be laser-safe. The incision is made with either a no. 15 Bard Parker blade or a CO₂ laser of the surgeon's choice; the laser is positioned in such a fashion that there is no tension on the handpiece as the surgeon holds it lightly. This may require proper positioning of the laser or adjustment of counterweights or springs.

It is difficult to execute the fine maneuvers required to perform blepharoplasty surgery when fighting against an ergonomically unfavorable device. The laser used for incisional periorbital surgery should produce the smallest possible beam diameter, ideally no more than 0.2 mm.

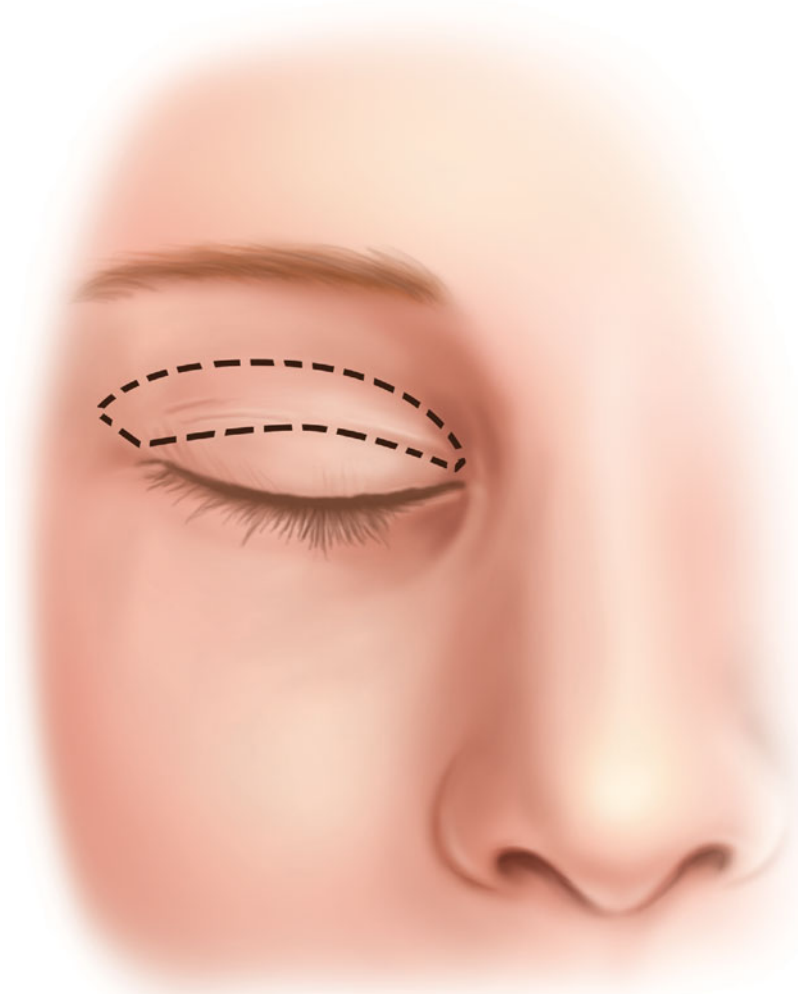


Figure 5.10 *Borders of a typical upper eyelid blepharoplasty incision*

The laser may be operated in continuous wave or high frequency pulse mode.

Step-by-step technique

Regardless of which instrument is used, the temporal portion of the incision should extend only through skin so as to avoid dividing the terminal branch of the lacrimal artery, which passes between skin and orbicularis muscle at the level of the lateral orbital rim.⁸ Once the initial incision is complete, a toothed forceps is used to elevate the temporal end of the ellipse so that a skin flap may be dissected with the laser. This is performed easily if traction is kept on the

skin flap to help separate it from the underlying orbicularis muscle. Once the orbital rim has been crossed, the incision is deepened to include orbicularis muscle. This dissection should stay anterior to the orbital septum at all times.

Once the skin muscle flap has been removed, the orbital septum will be clearly visible. The septum is divided just below the superior orbital rim to expose the preaponeurotic fat pad. The fat pad is grasped with forceps and carefully separated from the underlying levator aponeurosis to which it is loosely adhered. If excessive fat is present, a small amount may be conservatively resected. This should be performed with great care as

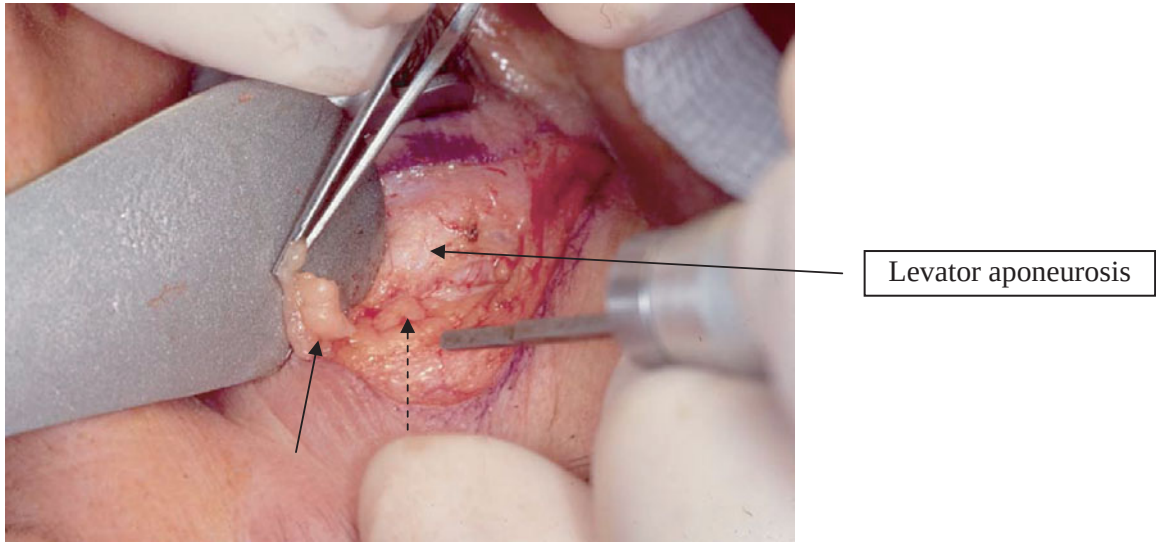


Figure 5.11 Intraoperative photograph demonstrating orbital fat resection with a carbon dioxide laser during upper eyelid blepharoplasty. The solid line indicates the light colored medial orbital fat while the dashed line demonstrates the yellow central or preaponeurotic fat

over-resection of fat produces a “hollow” look in the eyelid. The nasal fat pocket is explored and may be very conservatively contoured. Gentle pressure may be applied to the eyelid to help prolapse orbital fat into the surgical field. The nasal fat is white in color, while the central fat is yellow (Figure 5.11). The inferior edge of the orbital septum may be reflected inferiorly and transected just above its fusion with the levator aponeurosis.

Attention is then turned to the lateral brow where fullness and contour irregularity must be addressed. A pronounced appearance to the lateral brow may be caused by prominence of the underlying frontal bone or by relative enlargement or descent of the brow fat pad. This fat pad, usually referred to as ROOF, tends to migrate inferiorly due to loss of support and natural attenuation. While many patients are reluctant to undergo reduction of the bony orbital rim, contouring of the ROOF complex may be readily performed with the laser to achieve an optimal aesthetic result. The details of this procedure are beyond the scope of this chapter.

Hemostasis must be maintained in a meticulous fashion throughout the entire procedure to avoid orbital hemorrhage and the possibility of blindness. Hemostasis may be achieved with defocused laser, bipolar cautery, or even monopolar cautery.

Wound closure may be performed in many ways. In cases where the lid crease is not to be formed, inter-

rupted or running 6-0 or 7-0 permanent monofilament suture such as nylon or polypropylene may be used. A small cutting needle (P-1 or PC-1 Ethicon, Somerville, NJ) works very well although some surgeons prefer a taper-cut needle, which may cause less trauma to the tissue but which also becomes dull more quickly than the cutting needles. Sutures should be passed from skin edge to skin edge without engaging the underlying orbicularis muscle; it may bleed and cause a hematoma if cut by the needle. Care should be taken to ensure that the wound is properly aligned across its entire extent. Because of the curvilinear shape of the incision, the vectors of force vary across the entire width of the wound. If interrupted sutures are used, the wound should be aligned by placing cardinal sutures centrally, nasally, and temporally prior to filling in the areas in between. Care should be taken to ensure wound edge eversion. Running sutures may also be used.

If a lid crease is to be created, an interrupted 6-0 undyed polygalactin suture is placed through skin and orbicularis muscle on the inferior border of the wound, which engages the levator aponeurosis at the superior tarsal border, and then is passed through orbicularis muscle and skin on the superior border of the wound. Three such sutures are placed—one in the nasal portion of the lid, one in the central portion, and one in the temporal portion. The wound in between these sutures is

closed, skin edge to skin edge, with permanent suture material. At the end of the procedure, an ophthalmic ointment preparation that does not contain neomycin is placed on the lids, followed by sponges soaked in iced saline.

■ Technique: Lower Eyelid

Selecting the approach to lower lid blepharoplasty depends upon whether there is excess skin, excess orbicularis muscle, excess herniated fat, or malar descent. When there is fat herniation in the absence of excessive lower lid skin, then a *transconjunctival approach* is favored. When there is moderately excessive lower lid skin, herniated orbital fat, or hypertrophy of the orbicularis muscle, a skin-muscle flap approach is used.

■ Technique: Transconjunctival Blepharoplasty of Upper Eyelid

Anesthesia and patient positioning

Areas of prominent fat protrusion should be marked with the patient sitting upright; when in a supine position, the fat falls posteriorly into the orbit and is difficult to assess. Topical anesthetic is administered and scleral shields lubricated with an ophthalmic ointment are inserted to protect the eye. Local anesthetic is injected subconjunctivally, into the fatty tissue compartments (posterior to the rim where the septum originates) and along the inferior orbital rim, near the infraorbital nerve. If the anesthetic is injected into the inferior oblique muscle, temporary pupillary dilation and diplopia may result. The lower lid is retracted inferiorly using a Desmarres retractor, which should rest against the maxilla anterior to the inferior orbital rim. A lid plate or malleable retractor is placed in the fornix and over the protected globe to ballot the eye so as to induce protrusion of orbital fat.

Step-by-step technique

The incision is created horizontally through conjunctiva and lid retractors overlying the area of fat herniation, usually halfway between the inferior fornix and inferior tarsal border. Since the lower lid retractors may fuse with the orbital septum as low as 2 to 3 mm below the inferior tarsal border, the orbital fat does not extend superiorly beyond this point and the incision should be made below this level. A battery-operated disposable hot cautery, Colorado needle, RF device, or CO₂ laser may be used to achieve hemostatic cutting. Once the conjunctiva and

lower eyelid retractors have been divided, the anterior orbital fat becomes visible within its fibrous sheath. While the wound edges are retracted with scleral hooks, dissection is extended through the capsulopalpebral fascia until the central fat pad is visualized. The incision is extended laterally to the lateral canthal angle and medially to a point just lateral to the plica semilunaris.

The amount of anticipated fat removal is determined in the preoperative evaluation, the goal being to eliminate the fatty tissue prominence while avoiding a “hollow” appearance. A Desmarres retractor can be placed within the wound to retract and expose the orbital fat. If the fat is not transected with a hemostatic device, it should be carefully clamped with a hemostat prior to division. If a CO₂ laser is used to transect the fat, clamping is not necessary. Fat excision should proceed in a systematic fashion and only the fat herniating anterior to the level of the orbital rim is removed. Care must be taken to avoid injury to the inferior oblique muscle when working in the medial and central portions of the lower eyelid. The lateral fat pad is usually divided into two compartments—one adjacent to the central fat pad and another located more posteriorly and laterally. To adequately expose these lateral fat lobules, the arcuate expanse of the inferior oblique may be divided. The inferior oblique muscle divides the central and nasal fat compartments and should be recognized by its horizontal orientation in the anterior-inferior orbit. Injury to this muscle or its sheath may result in double vision. The presence of prominent vessels associated with the medial fat compartment should be anticipated. Medial fat repositioning can be performed with the transconjunctival approach. The orbital septum remains intact and the periosteum can be incised at the arcus marginalis in order to access the subperiosteal space.

■ Technique: Laser Transconjunctival Blepharoplasty of Lower Eyelid

Lower eyelid transconjunctival blepharoplasty is readily accomplished using the CO₂ laser or standard incisional devices. If the laser is used, standard laser safety precautions including metal ocular protection are observed at all times. The laser is set at 6 to 8 W continuous wave or 15 to 25 mJ per pulse (5 to 8 W pulsed mode) with a 0.2-mm focused handpiece and used to make an incision through the conjunctiva and lower lid retractors to expose the orbital fat. This approach maintains a plane of dissection posterior to the orbital septum. If a laser is

used, the handpiece is held in a focused position and directed anteriorly and inferiorly, aiming toward the inferior orbital rim, as the conjunctiva and lower eyelid retractors are divided. After two to three passes in the same incision, the orbital fat should present itself into the wound. The assistant holds the retractor in one hand and the Jaeger plate in the other, taking care to maintain the plate's position while applying gentle posterior pressure on the globe. The surgeon holds the laser in one hand and a toothed forceps in the other while the fat pads are exposed. The central fat pocket with its surrounding fascial investments is exposed in the midportion of the wound. The fibrous septae surrounding the fat should be carefully stripped away from the fat pad itself using blunt dissection with the laser handpiece or a cotton-tipped applicator. Once the entire pocket has been isolated, it may be resected with the CO₂ laser or transposed inferiorly once the arcus marginalis has been released along the inferior orbital rim (Figure 5.12). Inadvertent laser burns are avoided if the laser is directed against the metal retractor and held stationary while the fat pad is

grasped with a forceps and moved slowly from side to side. If large vessels are apparent within the fat, these should be closed with bipolar electrocautery prior to division of the pad with the laser. If using a laser, every effort should be made to minimize the total amount of laser energy applied to the orbit. The laser should never be directed posteriorly as the globe or extraocular muscles may be inadvertently divided or damaged.

■ Technique: Transcutaneous Lower Lid Blepharoplasty

Skin marking

Preoperative markings should be drawn with the patient sitting upright. It is helpful to delineate the nasojugal tear trough, specific location and severity of fatty prolapse, and the extent of orbicularis muscle hypertrophy when applicable. Forceps should be used to gently pinch the lower lid, to determine the maximum amount of skin that can be removed from the subciliary area without inducing downward bowing of the lower lid margin. This mark

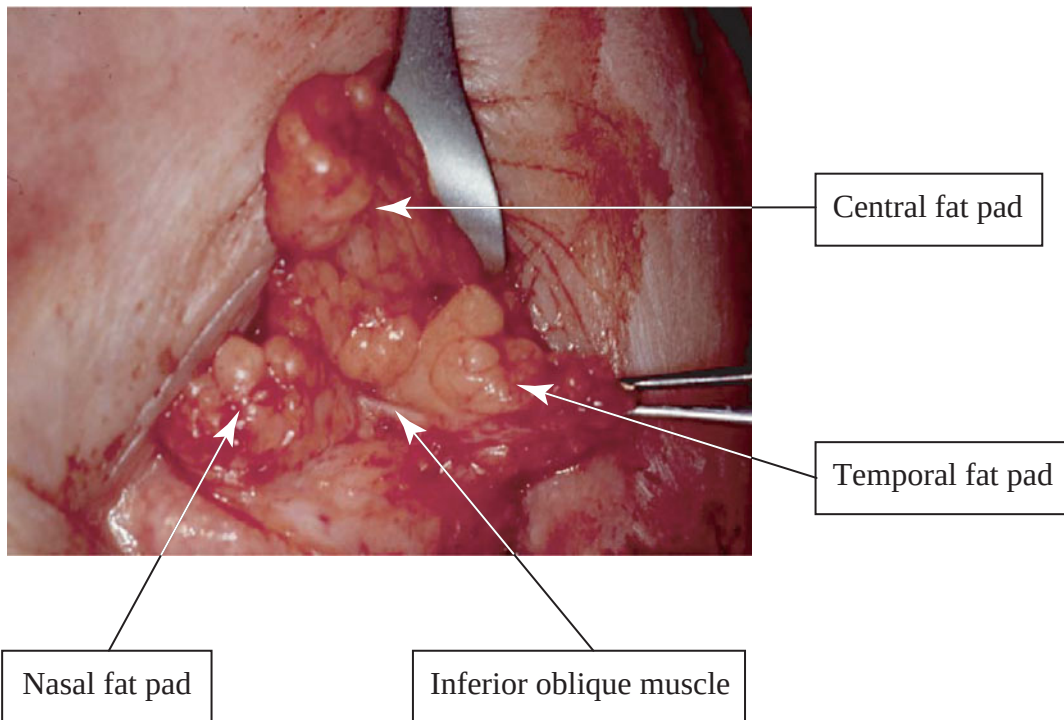


Figure 5.12 Anatomy of transconjunctival lower blepharoplasty, surgeon's view of the right eye. The metal retractor is pulling the lower eyelid inferiorly. Once exposed the fat pads can be managed appropriately

will remind the surgeon of the upper limit of vertical shortening when skin is actually removed in subsequent steps. Preoperative photographs are useful for intraoperative reference. Topical anesthetic is instilled in both eyes and protective scleral shields lubricated with artificial tear ointment are placed behind the lids. Local anesthetic for subcutaneous injection, consisting of 2% lidocaine with 1:100,000 epinephrine in a 1:1 mixture with 0.75% bupivacaine with 1:200,000 epinephrine, provides analgesia and hemostasis.

Incision and dissection

A standard subciliary incision placed 1.5 mm below the cilia can be initiated laterally with a no. 15 Bard-Parker blade. The incision is extended medially to within 2 mm of the lacrimal punctum using sharp Westcott scissors or with a no. 15 Bard-Parker blade. Lateral to the lateral canthal angle, the incision is directed inferotemporally along a natural laugh line. This lateral portion of the lower lid incision is extended if more skin needs to be removed, but initially, the length need only facilitate skin and muscle dissection inferiorly. A 6-0 polypropylene or silk suture placed at partial thickness through the tarsus centrally and just at the subciliary incision (avoiding skin puncture) is useful for providing countertraction.

Generally, a combined skin and muscle flap method is used. However, it may be advantageous to elevate skin only in the pretarsal orbicularis area, since keeping the muscle layer undisturbed may decrease the chance of developing a depressed subciliary scar. Once the orbicularis muscle has been exposed, a pair of blunt-tipped scissors is used to buttonhole the preseptal muscle. The muscle is then opened across the full extent of the wound and dissection is performed inferiorly in the sub-orbicularis oculi plane until the inferior orbital rim is exposed. Meticulous hemostasis is maintained during this dissection.

Removal of hypertrophic orbicularis muscle

When orbicularis muscle hypertrophy is present, a skin flap can be dissected to expose the muscular bulge. The superior edge of prominent orbicularis mounding often begins 3 to 5 mm below the lash line and is accentuated with smiling. The muscle is reduced by partially and superficially excising muscle tissue layers along the orientation of the fibers, decreasing the anterior to posterior prominence. Bleeding is anticipated but is easily controlled in this region. An alternative method uses electrosurgical modification and ablation of the orbicularis muscle to reduce the muscle prominence.

Removal of orbital fat

In contrast to the transconjunctival approach, the orbital fat cannot be exposed without opening the orbital septum when the transcutaneous approach is utilized. The entire width of the septum may be incised at a point just above the arcus marginalis in order to expose the medial, central, and lateral fat compartments. Alternatively, three small buttonholes can be made in the septum to allow access to the fat. Gentle pressure applied to the orbit through the lids will accentuate fat herniation. When the first lateral fat pad is removed, balloting the globe can expose a second more posterior fat pad. This fat may also need to be sculpted to avoid residual temporal lower eyelid fullness. Next, the central and nasal fat pads are addressed. Fat that protrudes anterior to the rim can safely be removed using techniques described earlier. Sitting a patient upright during surgery will allow better judgment of the adequacy of liposculpting.

Fat repositioning

Traditional blepharoplasty techniques emphasizing fat removal alone are now recognized to be too limited a strategy in rejuvenative surgery. In the presence of significant cheek fat descent, rejuvenation may additionally require a midface lift, discussion of which is beyond the scope of this chapter.

Tear trough deformities without significant malar descent can be corrected by fat preservation and repositioning. In this setting, nasal fat removal would worsen a sunken lid appearance. The inferomedial orbital rim is palpated and the septum is incised at the arcus marginalis, near the origin of the levator labii superioris alaeque nasi muscle. A periosteal elevator is used to create a subperiosteal pocket 10 mm distal to the incision spanning the length of the trough to be filled. Damage to the infra-orbital foramen located 7 to 10 mm inferior to the rim at the junction of the medial third and lateral third of the eyelid is avoided.

The medial fat capsule is incised to liberate the fat lobules and allow fat to be advanced anteriorly without creating traction. The fat is fashioned into a mallet-shaped fat flap with a pedicle of 5 to 7 mm diameter and positioned into the subperiosteal pocket. If properly performed, there should be no tendency for the fat to recoil posteriorly, and extraocular motility should not be restricted. The fat can be secured using a 6-0 polygalactin or nylon horizontal mattress stitch that loosely engages the anterior edge of periosteum and part of the fat pedicle.

Skin excision

The skin-muscle flap is draped without traction over the subciliary incision. The amount of skin to be removed is determined with the patient gazing upward and opening his or her mouth. Excess skin extending above the subciliary incision is excised with sharp Westcott scissors. The primary lid skin tightening effect is achieved by horizontal shortening at the laugh line, rather than by shortening vertically. Therefore, conservative amounts of skin are removed from the subciliary region. Aggressive *vertical* shortening is almost guaranteed to cause lower lid malposition. The excised subciliary skin is a horizontally oriented triangle with apex pointing medially with maximal widths usually ranging from 1 to 4 mm.

As mentioned, skin tightening should be accomplished in the horizontal vector. The edge of the skin-muscle flap is draped laterally along the curvature of the lower lid with a gentle amount of traction and the overlap is excised to reduce skin redundancy. The extra “dog ear” of tissue is eliminated by a combination of extending the original laugh line incision (usually not more than 15 mm) and excising a triangle of the skin-muscle flap with apex at the inferior extent of the lateral incision.

Skin closure

The subciliary portion of the wound should not be under tension and can be closed using 7-0 chromic gut, silk, or nylon sutures in a running fashion from medially to laterally. The lateral wound extent is closed in an interrupted fashion to maximally align the closure and distribute tension. Sutures are removed in 5 days, although in laser-assisted incisions, the sutures should remain for 7 to 10 days.

Patients are instructed to maintain ice compresses as often as possible for the first 48 hours, to apply antibiotic ointment to the sutures three to four times per day, to keep their head elevated during the day and even at night by sleeping on extra pillows, and to avoid heavy bending, lifting, exercise, or other activities that may require a Valsalva maneuver for at least 1 week. Airplane travel should be discouraged to avoid potential exposure to decreased atmospheric pressure, which may allow bleeding to occur. Satisfactory postoperative analgesia may usually be achieved with acetaminophen, although some patients may require narcotics. Nonsteroidal anti-inflammatory agents (including ketolorac [Toradol, Roche Pharmaceuticals, Nutley, NJ]) must be avoided to prevent orbital hemorrhage. Sutures are removed 6 to 10 days after surgery (Figure 5.13).



Figure 5.13 (A) Preoperative view of a 45-year-old female **(B)** 1 month after upper and lower blepharoplasty with fat repositioning and periorbital carbon dioxide laser skin resurfacing

Wearing contact lens may be resumed 1 to 2 weeks after surgery. Patients who wear contact lenses should be carefully advised of this preoperatively as many do not have current glasses prescriptions. Makeup may be applied to the upper eyelids 1 to 2 days after the sutures have been removed and concealer may be applied to the lower eyelids to cover ecchymosis several days after surgery. Erythromycin ophthalmic ointment is applied until makeup use is resumed. Patients are told to expect 80% of the swelling to resolve over 2 weeks, while the remaining 20% (which is not usually aesthetically significant) may take months to completely resolve. Consumption of salty foods or a systemic state of fluid retention may produce increased swelling in the eyelids. Low-level exercise

may be resumed after 1 week, and more vigorous activities after 2 weeks.

■ Postoperative Clinical Considerations

Written postoperative instructions should include information on activity restrictions, wound care, and expected wound appearance. It is difficult to list every specific restriction or contraindication; therefore, an explanation of why the limitations are recommended will allow the patient to extrapolate and employ good judgment. The patient should be informed of signs or symptoms that warrant medical attention and then urged to maintain a low threshold for seeking your medical advice. It is important to indicate whom to contact in the event of an ocular emergency.

Wound care and appearance

Patients are instructed to apply ice compresses as often as possible for the first 48 to 72 hours in order to reduce eyelid swelling and improve comfort level. Crushed ice or frozen peas can be placed in plastic resealable plastic bags. Direct application of compresses to the skin has been known to cause frostbite and can be avoided by placing a moistened gauze or towel on the lids to buffer the intense cold of the ice pack. On the day of surgery, ice applications initially of 30 minutes duration each hour is suggested, with decreasing intensity over the subsequent 2 days after surgery. Beyond this, ice has limited efficacy and warm compresses are sometimes recommended for improved subjective comfort. The patient is instructed to keep his/her head and neck elevated by approximately 30 to 45 degrees with two to three stacked pillows during sleep to further reduce periocular accumulation of dependent edema for at least the first 3 days. A combination antibiotic–steroid ointment is applied to the suture line three to four times per day to reduce tissue reaction to sutures.

Patients should be prepared to expect eyelid swelling and bruising beginning on the first postoperative day. The edema is expected to diminish 2 to 3 weeks after surgery. Consumption of salty foods or a systemic state of fluid retention may aggravate eyelid swelling. Darkening of the skin in the early postoperative period may represent deposition of hemosiderin and resolves spontaneously. Persistent pigmentation may occur as a result of sun exposure while lids are ecchymotic.¹¹¹ Patients are thus instructed to avoid such exposure by wearing sunglasses as well as a hat.

Activity restrictions

To avoid bleeding induced by positive pressure to the orbital or eyelid vasculature during the first postoperative week, patients should refrain from heavy lifting or exertion, lowering the head beneath the heart, or other activities that result in a Valsalva maneuver. If patients live a significant distance from a medical facility, they should arrange to stay nearby for the first night after surgery. Patients should be contacted by telephone on the evening of surgery and the following day. If a patient reports persistent bleeding, swelling, significant pain, decreased vision, erythema, or discharge from the wound, prompt examination is required. If there are no unanticipated concerns, the patients are then seen again 5 to 10 days postoperation for suture removal.

Analgesia

Satisfactory postoperative analgesia is usually achieved with acetaminophen alone, although some patients required narcotics. Nonsteroidal anti-inflammatory agents and aspirin must be avoided for at least 3 to 4 days after surgery to diminish bleeding risks. Application of ice compresses often provides additional comfort.

Anticipated recovery

Low-exertion exercise may be resumed after 1 week, and more vigorous activities after 2 weeks. Wearing contact lens may be resumed 1 to 2 weeks after surgery, but the patient must be reminded to limit lid manipulation. This anticipated postoperative abstinence from contact lens wear should be discussed with patients preoperatively so they may be prepared to wear glasses during the early postoperative period. Patients are told to expect 80% of the swelling to resolve over 2 weeks, while the remaining 20% may take 3 months or longer to resolve completely. Makeup may be applied to the upper eyelids 1 to 2 days after the sutures have been removed. Concealer may be applied to the eyelids remote from incision lines to cover ecchymosis soon after surgery; however, the patients must exercise caution in removing the cosmetics to avoid excessive periocular manipulation.

■ Complications of Blepharoplasty

Complications of blepharoplasty include blindness, scarring, diplopia, asymmetry, dissatisfaction with surgical outcome, over- or underresection of fat, eyelid malposition, ptosis, and others. Careful attention to detail will help minimize the risk of these problems.

REFERENCES

1. Wiess JS, Swanson NA, Baker S. Anatomy and physiology of aging skin. In: Krause CJ, Pastorek N, Mangat DS, eds. *Aesthetic Facial Surgery*. Philadelphia, PA: Lippincott; 1991:461-467.
2. Tolleth H. Concepts for the plastic surgeon from art and sculpture. *Clin Plast Surg*. 1987;14:585-586.
3. Farkas LG, Kilar JC. Anthropometric guidelines in cranio-orbital surgery. *Clin Plast Surg*. 1987;14:1.
4. Gonzalez-Ulloa M, Flores ES. Senility of the face: basic study to understand its causes and effects. *Plast Reconstr Surg*. 1965;36:239-246.
5. Callahan M, Beard C, eds. *Beard's Ptosis*. 4th ed. Birmingham, AL: Aesculapius Publishing; 1990:1-50.
6. MT Doxanas, RL Anderson. Oriental eyelids: An anatomic study. *Arch Ophthalmol*. 1984;102:1232-1235.
7. Chen WPD. Comparative anatomy of the eyelids. In: Chen WPD, ed. *Asian Blepharoplasty: A Surgical Atlas*. Boston, MA: Butterworth-Heinemann; 1995: 1-19.
8. Whitnall SE. *The Anatomy of the Human Orbit and Accessory Organs of Vision*. 2nd ed. London, UK: Oxford Medical Publishers; 1932:57-65.
9. Shore JW. Changes in lower eyelid resting position, movement, and tone with age. *Am J Ophthalmol*. 1985;99:415-423.
10. Lemke BN, Lucarelli MJ. Anatomy of the ocular adnexa, orbit, and related facial structures. In: Nesi FA, Lisman RD, Levine MR, eds. *Smith's Ophthalmic Plastic and Reconstructive Surgery*. 2nd ed. St. Louis, MO: Mosby; 1997:1-75.
11. Barker DE. Dye injection studies of intraorbital fat components. *Plast Reconstr Surg*. 1977;59: 82-85.
12. Yousif NJ, Sonderman P, Dzwierzynski. Anatomic considerations in transconjunctival blepharoplasty. *Plast Reconstr Surg*. 1995;96:1271-1278.
13. Lowe NJ, Wieder JM, Shorr N, Boxrud C, Saucer D, Chalet M. Infraorbital pigmented skin. Preliminary observations of laser therapy. *Derm Surg*. 1995;21: 767-770.
14. Biesman BS, Carruthers J, Baker S, Holloman E, Leal H. Monopolar radiofrequency treatment of human eyelids: A prospective, multicenter, efficacy trial. *Lasers Surg Med*. 2006;38(10):890-898.
15. Biesman BS, Pope K. Monopolar radiofrequency treatment of the eyelids: A safety evaluation. *Derm Surg*. 2007;31:794-801.
16. Mannstein D, Herron GS, Sink RK, Tanner H, Anderson RR. A new concept for cutaneous remodeling using microscopic patterns of thermal injury. *Lasers Surg Med*. 2004;34:426-438.
17. Biesman BS. Fractional resurfacing of the eyelids: Initial clinical experience. *Lasers Surg Med*. 2007; (suppl 19):55.
18. Bogle MA, Arndt KA, Dover JS. Evaluation of plasma skin regeneration technology in low-energy full-facial rejuvenation. *Arch Dermatol*. 2007;143(2):168-174.
19. Biesman BS. Plasmakinetic resurfacing of the eyelids. *Lasers Surg Med*. 2007;(suppl 19):17.
20. Fagien S. Botox for the treatment of dynamic and hyperkinetic facial lines and furrows: Adjunctive use in facial aesthetic surgery. *Plast Reconstr Surg*. 1999;103:701-708.
21. Biesman BS, Arndt KA. Periocular treatment. In: Carruthers A and Carruthers J, eds. *Botulinum Toxin. Procedures in Cosmetic Dermatology*. Elsevier Inc.; 2005:45-57.
22. Kane MAC. Classification of crow's feet patterns among Caucasian women: The key to individualizing treatment. *Plast Reconstr Surg*. 112(5):33S-39S.
23. Flynn TC, Carruthers JA, Carruthers JA. Botulinum-A toxin treatment of the lower eyelid improves infra-orbital rhytids and widens the eye. *Dermatol Surg*. 2001;27(8):703-708.
24. Flynn TC, Carruthers JA, Carruthers JA, Clark RE II. Botulinum A toxin in the lower eyelid: Dose-finding study. *Dermatol Surg*. 2003;29(9):943-950; discussion 950-951.
25. Kane MA. Treatment of tear trough deformity and lower lid bowing with injectable hyaluronic acid. *Aesthet Plast Surg*. 2005;29(5):363-367.
26. de Maio M. The minimal approach: An innovation in facial cosmetic procedures. *Aesthet Plast Surg*. 2004;28(5):295-300.
27. Biesman BS. Soft tissue augmentation using Restylane. *Facial Plast Surg*. 2004;20(2):171-177; discussion 178-179.

CHAPTER 6

Neck Rejuvenation

Steven H. Dayan, Benjamin Bassichis, Ryan M. Greene, and Amit B. Patel

KEY POINTS

- Frequent aesthetic concerns focus on the sagging neck or “turkey gobbler” appearance, which can make patients appear heavy, full faced, and aged.
- Loss of the cervicomenal angle is a common sign of aging, inciting patients to seek facial rejuvenation.
- Neck contouring involves management of the subcutaneous fat, platysma muscle, and cervical skin.
- The youthful neck that most patients seek is characterized by thin, soft tissue with
 - visible underlying structures,
 - an acute cervicomenal angle, and
 - the absence of fat, sagging, dyspigmentation, and wrinkles.
- Traditionally, neck-lift procedures have been recommended to achieve maximum tightening of the neck and overcome skin laxity.
- Current trends of smaller incisions and reduced morbidity and recovery times mean that successful neck rejuvenation depends upon
 - effective facial analysis,
 - understanding of the facial aging process,
 - careful patient selection, and
 - skillful execution of combined surgical and nonsurgical techniques.

ANATOMIC LANDMARKS

A thorough understanding of superficial neck anatomy is critical in diagnostic planning and achieving a superior result in neck rejuvenation. Evaluation should involve an assessment of bony structures, musculature, subcutaneous tissues, and skin of the neck. Cervical anatomy has many variations, and appropriate preoperative evaluation is paramount in tailoring the ultimate treatment plan.

The anatomic limits of the neck are the mandible superiorly, the supraclavicular area inferiorly, and the anterior borders of the trapezius laterally. The platysma

muscle is a quadrangular sheet of muscle that originates from fascia that overlies the pectoralis and deltoid muscles. The superficial temporal fascia divides to envelop the platysma and is anchored to the overlying dermis by multiple dense connective tissue bands. The anterior platysmal fibers tend to decussate in the midline with the fibers of the opposite side before inserting into the mentum. Variations have been noted with regards to this decussation. Partial decussation has been noted to occur in approximately 61% to 75% of the population¹ and in 10% there is no decussation at all.²

The external jugular vein perforates the superficial layer of the deep cervical fascia at the middle portion of the clavicle. The communicating vein descends to connect with the anterior jugular vein. This communicating vein and external jugular vein are at risk during transection of the platysma and may be avoided during surgery by adhering to fascial planes that separate the platysma from its underlying structures.

Cutaneous sensation in the anterior neck is derived from the second, third, and fourth cervical nerves termed as the cervical plexus. A transverse branch of the plexus passes beneath the platysma before dividing into fibers that pierce the muscle to supply the anterior neck skin. The primary innervation of the platysma is by the cervical branch of the facial nerve. Another nerve of interest is the marginal mandibular branch of the facial nerve. This nerve is responsible for innervating the depressor labii angularis, depressor anguli oris, mentalis, and portions of the platysma.

The cervicomenal angle is the landmark that is most influential in creating a slender neckline. It is formed by the intersection of two lines: the first is from the gnathion to the hyoid bone, and the second is from the hyoid to the sternal notch. The ideal cervicomenal angle is 90 to 105 degrees (Figure 6.1). The level of the hyoid bone is particularly influential in this angle and neck contour. It is typically located at the level of the third or fourth cervical vertebrae (C3 or C4). While a slightly higher hyoid denotes a well-defined jaw line, a low hyoid leads to the appearance of a sloping neck and poorly defined jaw line.

A blunted cervicomenal angle may occur because of the ptosis of adipose tissue in the anterior neck. There are two planes of fat defined by their relation to the investing fascia of the platysma: the subcutaneous, or supraplatysmal, and subplatysmal planes.

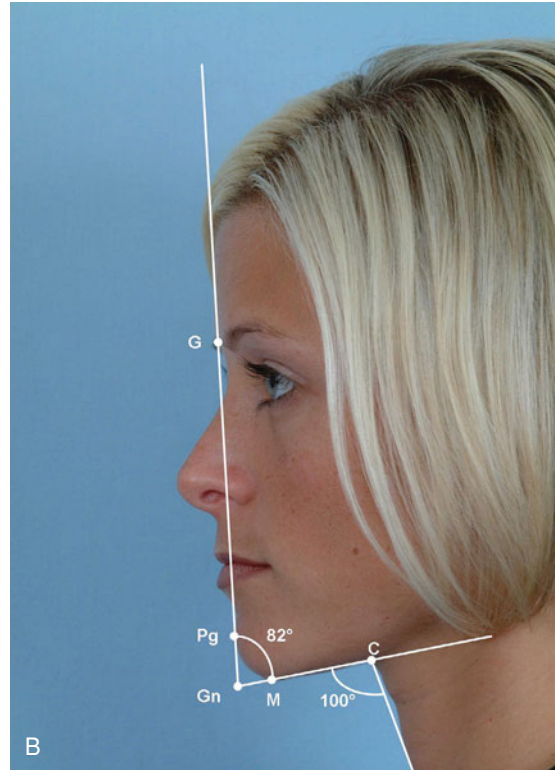
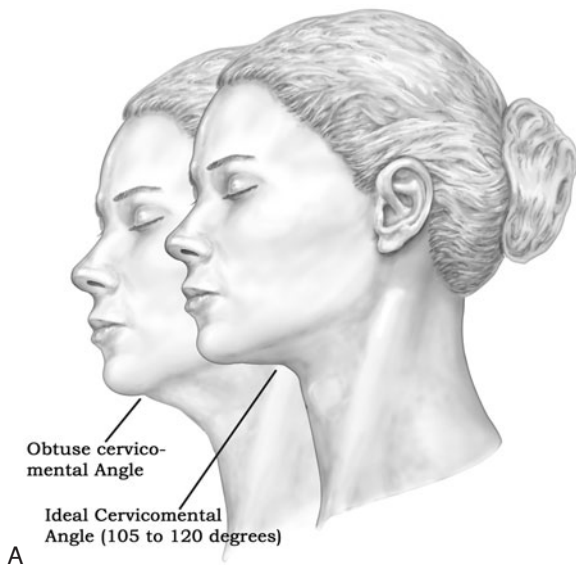


Figure 6.1 A&B Cervicomenal angle is ideally between 105 and 120 degrees. Diagram of a favorable cervicomenal angle

In addition to the hyoid, chin position also influences the appearance of the neck. A short jaw line and unfavorable chin position can accentuate an obtuse cervicomenal angle (Figure 6.2). A more desirable relationship exists when the angle defined by a vertical tangent drawn from the glabella to the pogonion intersects with a second horizontal tangent drawn from the cervical point through the menton, known as the gnathion. A 75- to 90-degree angle between these two tangents is desirable (Figure 6.3).

PATIENT SELECTION AND PREOPERATIVE EVALUATION

■ Indications and Contraindications

Obtaining superior aesthetic results in neck rejuvenation requires accurately diagnosing the underlying anatomic abnormality in context of the aging process.³ The aging neck results from the triad of accumulated submental fat,

platysmal banding, and redundant dyspigmented skin. These physical attributes are the result of the intrinsic aging process compounded by the extrinsic environmental factors affecting all tissue and structures in the submentum and neck area.

A successful surgical outcome of neck rejuvenation surgery initiates with careful patient selection and realistic patient expectations. Any medical problems contraindicating elective surgery will also contraindicate neck contouring. A review of the general health status, past medical and surgical history, medications, allergies, and social habits is also important to reveal any contraindications or underlying illnesses that may affect elective surgery and anesthesia. A history of keloid or hypertrophic scar formation, autoimmune and inflammatory diseases, and allergic dermatitis of the face and neck skin should be well controlled prior to surgery to avoid poor healing.

Of extreme importance is any recent history of tobacco use. The deleterious and dangerous effects of smoking

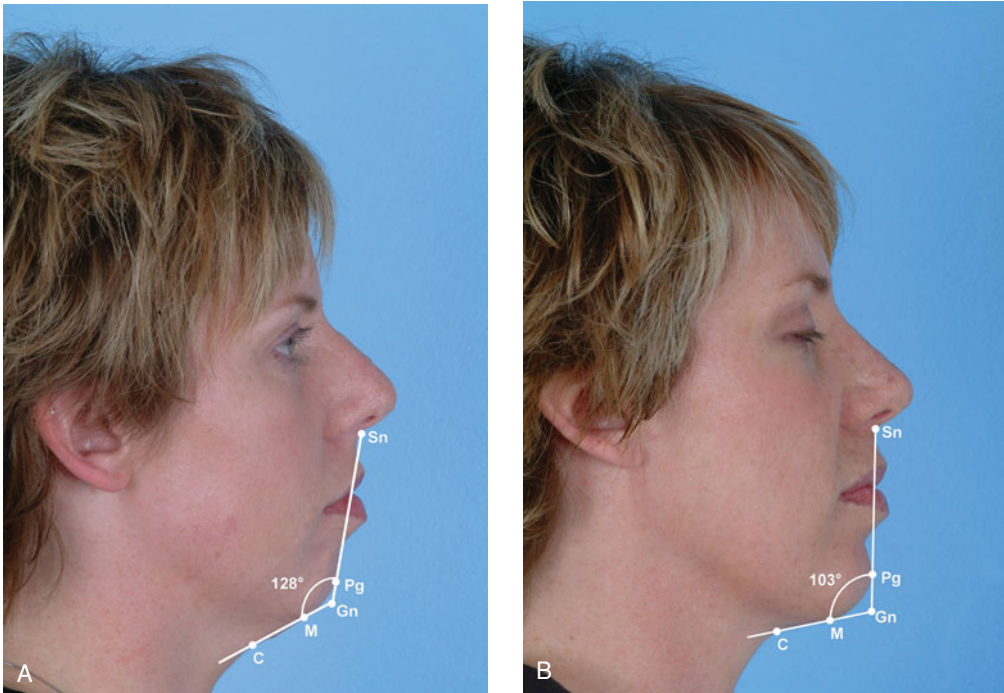


Figure 6.2 A&B An underprojected chin position leads to an unfavorable obtuse cervicomental angle. A chin implant increasing chin projection can improve the cervicomental angle

on postsurgical healing are well documented in the literature and should be explicitly explained to patients currently using tobacco products. Patients should discontinue the use of all tobacco and nicotine at least 1 month before and after cosmetic surgery.⁴

All medications or supplements that increase the risk for bleeding, including anticoagulant medications such as aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), warfarin sodium (Coumadin), steroid medications, as well as some vitamins and herbal supplements, should be reviewed by the surgeon and discontinued prior to surgery. In addition, a history of easy bruising, bleeding gums, frequent epistaxis, a family history of bleeding dyscrasias, heavy alcohol use, or liver disease can increase the risk for postoperative bleeding and should be evaluated before surgery.

Preprocedure assessment

As with all cosmetic procedures, it is important for cosmetic surgeons to consider the psychologic status of the cosmetic patient. Before agreeing to perform any elective procedure, it is crucial to understand the patient's motivation and expectations of the surgical outcome. The key

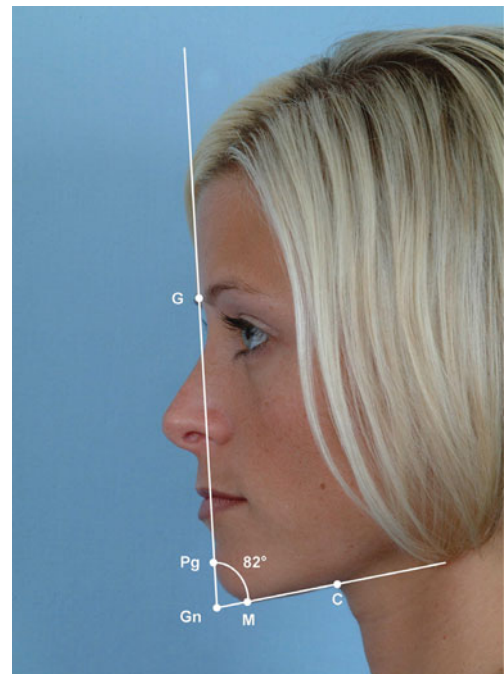


Figure 6.3 A mental angle between 75 and 90 degrees is desirable



Figure 6.4 *Standard five pretreatment photos for facial rejuvenation procedures*

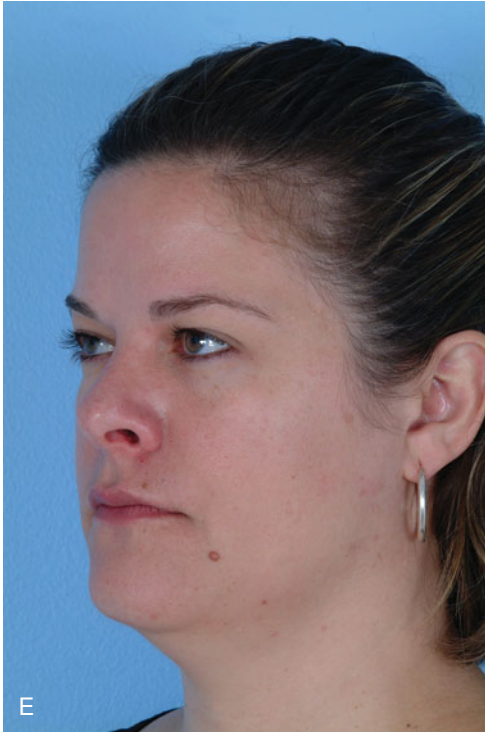


Figure 6.4 (Continued)

to a satisfied patient is one with clearly defined achievable goals. Once the history is obtained, a thorough physical examination of the full face and neck is performed. Any asymmetry of the head and neck should be documented and discussed with the patient. If these are not recognized preoperatively, they may become a focus of concern and dissatisfaction postoperatively.

Patient assessment begins with standard photographic representation and a complete physical examination. Standard face-lift pictures are taken in the Frankfort horizontal plane. These include full-face frontal, left and right oblique, and right and left lateral views. In addition, close-up views of the anterior neck and each ear with particular attention to the pre- and postauricular hairlines are helpful. A full set of multiview photographs (Figure 6.4) allows the surgeon and patient to (1) discuss and review the patient's expectations, (2) counsel the patient on any preexisting asymmetries, (3) relay a realistic outcome to the patient, (4) use as an intraoperative reference, and (5) evaluate postoperative outcome.

Digital modification of the photographs can present realistic possibilities for outcomes of neck-rejuvenation

procedures. Modification can be helpful not only in terms of preoperative planning but also to ensure that the patient's expectations and surgeon's proposed outcome are in agreement. In addition, the patients should sign a disclaimer explaining that photoimaging is not a guarantee of results, but merely a tool to visualize the possible outcomes.

The physical examination includes a complete assessment of each anatomic component of the neck in both static and dynamic positions in order to accentuate specific structures and their contribution to the existing deformity. Attention is focused on the following elements³:

1. Excess skin—skin quality.
2. Excess fat—extraplatysmal and subplatysmal.
3. Platysmal muscle abnormalities—flaccid and decussations/banding.
4. Malpositioned tissues/unfavorable anatomy—ptotic submandibular glands, overdeveloped suprahyoid musculature, and abnormal hyoid position.
5. Chin projection.

In an ideal scenario, a bony cartilaginous framework serves as a scaffold for the overlying soft tissue envelope (comprised of muscle, fat, glandular elements, and skin) to drape across it and cast an elegant, sweeping contour that transitions smoothly from the facial architecture. The presence of an unfavorable cervicomenal contour may reflect the effects of aging on one or multiple anatomic elements. For example, blunting of the cervicomenal angle may be the result of excess skin laxity, fat hypertrophy, soft tissue ptosis, and/or an unfavorable hyoid position. Failure to properly diagnose the underlying cause(s) may compromise patient satisfaction as a result of persistence of the preoperative deformity following surgical rejuvenation attempts. Therefore, each anatomic element must be considered closely as relates to the overall shape of the neck in efforts to formulate a sound treatment plan.

Assessment of skin The intrinsic effects of aging on skin over time lead to thinning of the epidermis and subcutaneous fat layers with effacement of the dermal–epidermal junction. When combined with a progressive disorganization of the elastic fibers and collagen, the external characteristics of skin wrinkling and laxity become visible.⁵ Excess skin may range from mild redundancy to severe laxity often described as the turkey gobbler neck. Male patients often present with a greater degree of skin



Figure 6.5 *Submental liposuction in isolation in a patient with ptotic platysmal muscles and inelastic skin can lead to an unfavorable sequelae of loose submental skin*

redundancy.⁶ Management of skin elasticity will vary depending on the degree of excess.

During evaluation, the surgeon should consider both the degree redundancy by gently mobilizing the skin from the underlying tissues and how well the skin rebounds to subtle retraction. If the skin possesses a favorable rebound quality, liposculpture techniques should be considered in the treatment plan. Patients with excess skin laxity, on the other hand, may benefit from a lifting procedure. Isolated liposuction to such a patient would worsen the skin laxity because of the poor intrinsic elasticity (Figure 6.5).⁷

Assessment of fat Fat deposits may exist in the subcutaneous or subplatysmal plane. Excess accumulation has the tendency to project a “doughy” appearance on the submental region and is often seen with aging, weight gain, and congenital lipodystrophies. This area is particularly prone to sagging because of congenital or age-related diastasis of the platysmal sling.^{1,5,6} On examination, asking the patient to alternate between

tightening and relaxing their neck muscles will help to target the additional fat deposits. Distinguishing the location of fat accumulation aids surgical planning in that subplatysmal fat is less amenable to liposuction because of its more fibrous nature and, instead, may require direct excision.

Assessment of platysma muscle The thin, sheetlike nature of the platysma muscle provides an anatomic sling for the deeper neck structures such as subplatysmal fat, cervical musculature, and the submaxillary glands. In a majority of patients, a congenital absence of midline decussation of the muscle fibers renders a central strip where the muscular sling’s support is deficient. As the effects of aging ensue, platysmal tone gradually diminishes and diastasis of the medial borders of the platysma progressively increases and manifests as platysmal bands.⁸

Assessment of hyoid position On lateral view the superior and inferior limbs of the cervicomental angle converge to a vertex at the level of the hyoid bone.⁸ The height of the hyoid bone, as well as its anteroposterior projection, should be noted after careful palpation with multiview photographs. The structure contributes to the cervical framework that provides a scaffold for the overlying skin-soft tissue envelope, and thus it can directly influence cervical contour. A hyoid complex that rests in a low and forward position will unfavorably direct the vertex of the cervicomental angle anterior and inferior, and thereby, blunt the transition of the superior and inferior limbs.

Assessment of submaxillary gland The submaxillary glands, also referred to as submandibular glands, may be visible in a thin neck and generally become more ptotic with age. For a right-handed examiner, bimanual palpation with the first two fingers of the right hand placed intraorally under the tongue and the first two fingers of the left hand placed extraorally under the mandibular body will help distinguish the gland’s borders from surrounding structures.

Assessment of chin position Proper chin position and projection is elemental in an attractive, youthful jaw line. Position may change with age secondary to ptosis of the chin pad and resorption of bone. Failure to diagnose and treat a deficient chin can compromise an otherwise successful rejuvenation procedure.

Mandibular length is assessed with the patient in profile view. An imaginary line is dropped vertically from the

vermillion border of the lower lip in order to assess its positional relationship with the pogonion. In a favorable scenario, this line will be tangent to the pogonion (Figure 6.3).⁹ Patients seeking rejuvenation surgery often do not recognize the influence of a poorly positioned chin on their cervical deformity. Photographs, three-way mirrors, and/or computer video-imaging are recommended to help with preoperative counseling.

The goals for cosmetic neck surgery are to achieve a youthful neckline by re-establishing a well-defined mandibular line and a sculptured cervicomental angle by effective management of the subcutaneous fat, platysma muscle, and cervical skin. The successful use of a chosen procedure is dependent upon the surgeon's ability to select patients whose anatomic attributes and psychologic profile indicate that they are appropriate candidates. Safe and effective aesthetic surgery is possible when the anatomic changes associated with the aging face are appreciated in combination with proper patient selection.

TECHNIQUES

■ Submental Liposuction

Patient selection

Ideal candidates for submental liposuction as a primary procedure are those who demonstrate fat deposition, an obtuse cervicomental angle, and early signs of aging in the neck structures.¹⁰ Good candidates have good skin elasticity and tone and are of average weight for their height. Those who demonstrate localized submental adipose deposition that is out of proportion to the remainder of their body habitus will benefit most from localized fat removal. Young to middle-aged patients with slight skin redundancy typically do not require skin removal after liposuction. Once the fat is removed, the skin contracts over the lost volume permitting improved skin redraping across the anterior neck (Figure 6.6). Redundant and inelastic skin types as well as the thicker skin of a man tend not to redrape as favorably after neck liposuction. In



Figure 6.6 A&B *In those with thick elastic skin, submental liposuction in isolation can be performed with favorable results*

general, the greater the fullness or laxity of the neck, the longer it will be before the final result is realized. Patients need to be well informed of this period of maturation. Middle-aged and older patients may primarily present with evidence of fat in the submentum; however, for these patients to benefit from an isolated submental liposuction, they should not have severe skin redundancy. Inappropriate candidates for cervicofacial liposuction are those with moderate-to-severe skin laxity, substantial cervical rhytides, marked ptosis of the muscular sling, prominent platysmal bands, and distinct lateral jowling. Neck liposuction can be used as a primary aesthetic sculpting mechanism or in combination with other regional rejuvenation procedures.

Today facial liposuction is used as a primary rejuvenation procedure or in combination with other regional aesthetic operations. Submental liposuction offers important advantages of minimum scarring, reduced tissue trauma, shorter recuperative period, and the ability to hide submental incisions. The advent of smaller liposuction cannulas has advanced the science of fat removal permitting more customized results.

Patient positioning and anesthesia

With the patient upright, the angle of the mandible, medial edge of each sternocleidomastoid muscle, submental crease, and the thyroid notch are marked. Preoperative marking is imperative because when the patient lies supine, the fatty accumulations tend to shift and occasionally disappear. Submental liposuction can be performed under local, intravenous sedation, or general anesthesia as subject to the surgeons' preference and patients' needs. After administration of adequate sedation and preparation and draping of the patient, the marked region is infiltrated with local anesthetic.

Step-by-step technique

A 5- to 8-mm incision in submental crease is made and carried down with a No. 15C blade through skin into subcutaneous tissue. The Adson forceps is used to lift the inferior aspect of the incision, while Kaye dissecting scissors gently create a pocket between skin and underlying subcutaneous tissue. A 4-mm spatula cannula is introduced into the incision to complete the undermining of the planned operative field (Figure 6.7). It is advantageous to use smaller diameter cannulas (3 to 4 mm) to perform fat reduction in the anterior neck. The liposuction tubing is attached to the cannula and while the cannula is in the pocket, the liposuction machine is turned on. The cannula is then moved quickly back and forth



Figure 6.7 Submental liposuction is performed with a spatula cannula through a submental skin incision in a supraplatysmal plane

through the fatty tissue. The holes in the cannula tip should always be directed toward the neck tissue and away from the dermis to minimize the likelihood of damage to the undersurface of the dermis, with possible resultant superficial scarring.

The nondominant hand guides the tip of the cannula, directing fat into the lumen, while the dominant hand actively advances the cannula. Care should be taken to maintain the cannula at all times above the platysma. Failure to maintain strict adherence to this principle can result in serious damage to structures deep in the platysma. Peripheral feathering of the lateral edges of the liposuction field will permit a smooth transition with surrounding tissues. The surgeon should frequently inspect the progress by palpating the skin and the underlying fat layer. A sufficient amount of fat is removed when the surgeon can feel a thin layer of adipose tissue remaining between two opposing skin layers.¹⁰

The process of lipolysis induced from surgical manipulation continues over several weeks following liposuction and contraction of the overlying skin continues over the ensuing months. The goal of submental liposuction is to

rejuvenate and recontour areas of adiposity by precise reduction in submental volume while minimizing external irregularities or scars. A conservative approach to fat reduction is prudent as overaggressive fat resection can create unnatural contours. If too much fat is removed, unmasking of platysmal bands can occur as can creation of a masculinized appearance secondary to skeletonization of the thyroid notch. Leaving a thin layer of subcutaneous fat is required to achieve a supple skin contour.^{11–13} The submental crease incision is closed using 6-0 Prolene suture.

Complications to avoid

Attention should be given to the removal of the appropriate amounts of fat during liposuction and flap procedures. “Cobra neck” deformity or prominent jowls are a direct result of aggressive fat removal in the central submental area without peripheral feathering of the fat dissection. Prominent jowling may be best managed with a combined face and neck-lift. Overreduction of the subcutaneous fat from the skin flap during a neck-lift can give rise to dermal scarring that appears as skin dimpling or abnormal folds and creases during platysmal and facial muscle contraction. To avoid a complication, a minimum of 3 mm of subdermal fat needs to be maintained on the skin flap. Cicatricial banding can occur when only dermis is left on the fat. Skeletonization of the submandibular gland can leave a visible, ptotic submandibular gland, which can be a difficult aesthetic issue. Moderation is the key and conservative fat removal is superior to excessive fat removal.

Improvement of residual cervical skin wrinkling or laxity after neck liposuction should occur over the following 3 to 6 months. During this time, skin contraction and remodeling of subcutaneous fibrosis will occur. Unfortunately, thicker skin usually does not contract as well as thinner skin does; therefore, a longer period of time may be required before the final result is seen in skin that has greater laxity preoperatively.¹⁰ If there is residual skin wrinkling and laxity after this period, it is likely the result of poor patient selection for an isolated submental liposuction procedure (Figure 6.5). In this case, more benefit would have been derived from a combined face and neck-lift instead. Residual neck laxity after a formal neck-lift procedure is likely because of poor technique and may require a tuck-up procedure for amendment.

Postoperative complications

In addition to 1 week of restricted activities and supportive care, the patients should wear compressive chin/neck

strap dressings for 2 to 3 weeks postoperatively to promote healing and recontouring in the submental region. Pain is minimal and there are no dietary restrictions. Sleeping with the head of the bed elevated will promote lymphatic drainage and help with postoperative edema. Most patients return to work within 1 week.

■ Platysmal Plication

Patient selection

Platysmal plication is indicated for those patients demonstrating pronounced platysmal banding. The neck skin quality should retain adequate capacity for redraping and contraction. Recreation of a more acute cervicomental angle and obliteration of the platysmal bands will achieve more youthful contours (Figure 6.8). Platysmal plication can be incorporated with submental liposuction and/or neck-lift, but it is not an alternative for a neck-lift if the cervical skin is significantly redundant or if jowling is excessive.¹⁴ Patients affected by the full spectrum of cervical aging involving skin, muscle, and fat usually also present with jowls; therefore, an isolated platysmaplasty or fat removal can actually highlight the jowls if they too are not simultaneously addressed with a needed face- or neck-lift. While it is preferred to perform minimum surgery to obtain a maximum result, an aesthetically unbalanced surgical outcome can appear less pleasing than a face and neck that is balanced and aging naturally.

Patient positioning and anesthesia

While the patient is in the seated position the submental crease, medial edges of platysmal bands, and a horizontal line representing the location of the new cervicomental angle are marked with a marking pen. After anesthesia with intravenous sedation, the submental crease incision is infiltrated with local anesthetic.

Step-by-step technique

Using a No. 15C blade, a 1- to 2-cm incision in the submental crease is carried through skin into subcutaneous tissue. If submental liposuction is planned concomitantly to platysmal plication, it is performed prior to the plication procedure.

The incision is then completed and face-lift scissors are used to undermine the skin from underlying soft tissue and platysma, extending from anterior sternocleidomastoid muscle to the other sternocleidomastoid muscle (approximately 5–6 cm lateral to midline bilaterally). This is helpful if performed under direct visualization with



Figure 6.8 *Platysmal plication and liposuction can be performed through a submental skin incision in those with mildly ptotic platysmal muscles and mild looseness in submental skin*

face-lift scissors tenting the skin upward as the dissection proceeds. Bipolar cautery is used to control bleeding in the visible connecting vessels extending from muscle to skin. The laxity and redundancy of the platysma is assessed with accommodation for the significant variation in the platysma muscle and aging pathology that may occur. To provide definition and contouring of the new cervicomental angle, the medial edges of the platysma are identified and bipolar cautery is used to cauterize approximately 2 cm of muscle, which is then transected using the face-lift scissors^{15,16} (Figure 6.9). The medial edges are plicated using a permanent suture such as 3-0 Prolene or Mersilene (Ethicon) extending from the new cervicomental angle to the submental crease to create a smooth, flat seam, leaving no free muscle edges to return as visible bands. If there is redundant platysma muscle evident, it can be addressed with conservative resection prior to plication. Before closing the skin incisions, the surgeon should verify that the cervical skin drapes

smoothly over the newly plicated platysma. If undermining was adequate, the skin will easily conform to the new concavity of the neck. If skin dimpling is identified, additional undermining over the platysma should be undertaken. When closing the submental crease incision, achieving wound edge eversion is beneficial for optimal scar camouflage. The submental crease incision is then closed using 5-0 monocril deep to appose dermal edges and 6-0 Prolene suture to reapproximate the skin edges.

Complications to avoid

If prominent platysmal bands are evident after neck-contouring procedures, they may have been undiagnosed preoperatively and not corrected during surgery. They may also have been unmasked by removal of fat and skin, or the plication sutures may have failed. By having patients contract their platysma muscle, hidden bands can be exposed and this complication can be avoided.¹³ Residual prominent banding may necessitate a revision procedure.



Figure 6.9 To accentuate the cervical mental angle, the platysmal fibers are incised horizontally at the level of the hyoid

Postoperative instructions

In addition to 1 week of restricted activities and supportive care, the patients should wear compressive chin/neck strap dressings for 2 to 3 weeks postoperatively to promote healing and recontouring in the submental region. Pain is minimal and there are no dietary restrictions. Sleeping with the head of the bed elevated will promote lymphatic drainage and help with postoperative edema. Most patients return to work after 1 week.

Neck-Lift

Patient selection

The goals of the neck-lift procedure are to rejuvenate the neck to achieve balanced, youthful contours and to restore a more youthful cervicomental angle. Patients with significant excess neck skin and tissue do not have a clear separation between the end of the jaw line end and the beginning of the neck. Often patients with a heavy or “turkey gobbler” neck have significant laxity of submental skin in addition to evidence of platysmal banding, submental, and submandibular adipose deposits. Older patients in whom the skin has lost its elasticity are often candidates (Figure 6.10). The focus of the operation is creating a more defined neck angle. Distinct lateral jowling is a contraindication for neck-lift, as these patients would be better served by a traditional facial rhytidectomy (Figure 6.11).



Figure 6.10 A&B Neck-lift with platysmal plication can be performed in isolation in those with a thin neck and minimal jowling



Figure 6.11 A–D Heavy neck and jowls is best treated with aggressive liposuction in addition to a full rhytidectomy

Candidates for the neck-lift technique are those with significant laxity and redundancy of the submental skin in the absence of lateral jowling deformity. The focus of the operation is to recreate a defined cervicomental angle and smoother neck contour.^{17,18} To obtain consistently successful results, surgical treatment of the aging neck is centered on comprehensive anatomic diagnosis, appropriate cervicofacial aesthetics, and the relative importance of each structural component of the pathology. The degree to which the projection and contour of the anterior mandible, redundancy of cervical skin, presence of excess submental fat, and configuration of the platysma muscles affect the appearance of the neck should be determined preoperatively to develop an effective surgical plan.¹⁹

Step-by-step technique

Following induction of monitored intravenous sedation or general anesthesia, the previously described submental liposuction and platysmal plication procedures are performed.

The planned pre-and postauricular incisions are then infiltrated with lidocaine. The skin is removed in a posterolateral direction; therefore, the incision initiates in the preauricular crease extending downward around the lobule and superiorly along the posterior auricular skin. The decision to place the final segment of the incision within the hairline or along the hairline is dependent upon the preoperative evaluation and discussion with the patient (Figure 6.12). Once the incision is carried down through the skin and subcutaneous tissue, undermining is performed approximately 5 to 6 cm anteroinferiorly proceeding medially to identify the lateral edge of the platysma muscle. The lateral platysmal edge is then plicated over itself and back on to the postauricular mastoid periosteum, using layered 2-0 PDS and 3-0 Prolene. Alternatively, a conservative undermining of the lateral platysma muscle can be performed for approximately 2 to 3 cm medial with caution to avoid the marginal mandibular nerve which sits just under the platysma muscle near the lateral edge of the submandibular gland. The free edge of the lateral platysma can be transposed superior-laterally and imbricated down onto the mastoid fascia with a large PDS or Prolene suture. In either method, it is essential to yield a tension-free closure of the skin with all tension placed on the plicated(imbricated fixation). Once this is accomplished, the redundant skin is redraped tension-free over the preauricular incision and the ear in a posterosuperior

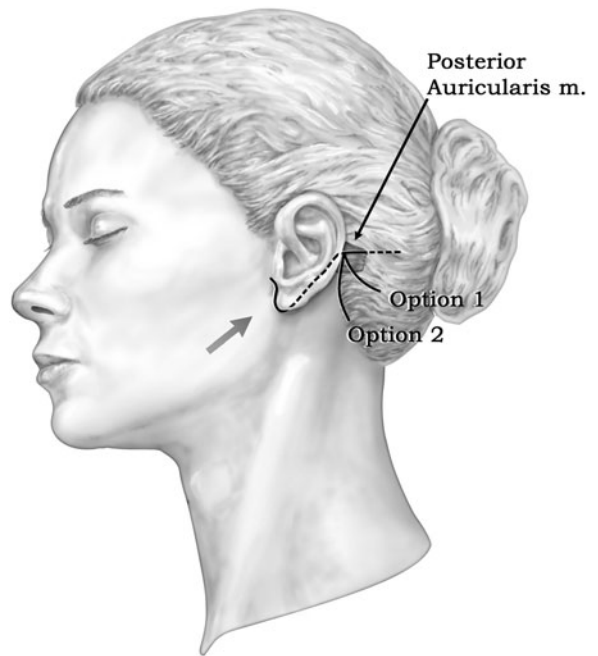


Figure 6.12 Postauricular incision is planned with care to achieve the maximum reduction in lax skin and also maintain a natural hairline

vector. The excess preauricular tissue is trimmed according to the planned incision. The tragal flap is left slightly redundant to avoid tension on the forthcoming closure.

The neck skin is lifted in the postauricular area in a posterosuperior fashion to circumvent a large step off deformity in the postauricular hairline. The preauricular incision is closed in two layers using 5-0 Monocryl and 6-0 Prolene for the deep and superficial aspects, respectively. The skin of the nonhair-bearing postauricular incision is sutured with 5-0 fast-absorbing gut suture. As there is no tension on the postauricular incision, no deep sutures are required. If the postauricular incision extends into the hairline, closure is accomplished with skin clips so no hair shaving is needed. Meticulous closure around the earlobe will prevent abnormal scarring. Upon closure, earlobe should be positioned in a slightly superior position so the ear will not be pulled inferiorly with the contractive forces of healing. Fluffs gauze, Kerlix roll gauze (Tyco Healthcare, Chicopee, MA), and Coban Self Adherent Wrap (3M, St. Paul, MN) are used to provide a mild to moderate pressure dressing.



Figure 6.13 *Following a hematoma evacuation, the skin is ecchymotic*

Complications to avoid

Hematoma or seroma formation is a common complication following neck-lift procedures with incidence in the literature ranging 2% to 15%^{20,21} (Figure 6.13). Hematoma can also occur following submental liposuction or platysmaplasty. Careful preoperative evaluation to discontinue all medications or supplements that increase the risk of bleeding is vital to prevent this complication. Seromas usually respond to aspiration and application of a pressure dressing. They have been reported to cause irregularities in the skin surface or subdermal fibrosis that can take weeks to months for resolution.²²

Chronic pain, which is rarely reported, becomes evident in 3 to 6 months following a rhytidectomy. Its etiology is not entirely clear but is suspected to be secondary to neuroma formation or possibly irritation to the sternocleidomastoid fibers. Treatment options to consider are referral for physical therapy and/or a neurologist. Success has been reported with oral gabapentine.²³

Infection can occur in approximately 4% to 5% of patients despite cephalosporin prophylaxis. Any signs of

unilateral erythema in the pre- or postauricular regions, fever, increasing pain, or delayed wound healing at the edges should be aggressively addressed with antibiotic therapy.

Skin necrosis and poor wound healing is a serious issue in cosmetic surgery patients who smoke tobacco. Smokers have a 12% higher risk of skin necrosis compared with nonsmokers.²¹ However, if the smoking patients refrain from smoking and have no nicotine in their system in the immediate perioperative period, the incidence of skin flap death drops to 2%. Honest patient counseling is imperative to avoid this severe complication in patients who use tobacco products.

Numbness of the neck is a common sequelae of neck procedures and gradually resolves over the ensuing weeks to months. Patients should be informed of the potential for hypoesthesia preoperatively. Facial nerve injury is a much more rare complication, with incidence ranging from 0.65% to 6%.¹⁸ Most often the injury is a paresis lasting between 3 and 6 months.

While patients seek an aesthetically improved neckline and jaw line, visible incisions and scarring betray the intervention of a neck-contouring procedure. Sequelae of poorly placed incisions include alopecia or an artificially created hairline pattern, conspicuous scars, an unnatural appearance of the tragus, skin dimpling or pulling, and a posterior hairline distortion. Careful attention to avoid excess tension on incision lines, meticulous preoperative analysis, and finely executed operative technique can help avoid these complications.

Postoperative instructions

In addition to 1 week of restricted activities and supportive care, the patients should wear compressive chin/neck strap dressings for 2 to 3 weeks postoperatively to promote healing and recontouring in the submental region. Pain is minimal and there are no dietary restrictions. Sleeping with the head of the bed elevated will promote lymphatic drainage and help with postoperative edema. Most patients return to work after 1 week.

■ Nonsurgical Approaches to Neck Rejuvenation

Patient selection

It is essential to understand the psyche of an aesthetically oriented patient. Patients seeking neck rejuvenation often use their hands to pull their neck skin back, behind

their ears obliterating all their fine wrinkles and expect a surgical treatment that can achieve such results. Unfortunately, this is exactly what surgery will not do. Surgery treats the underlying neck structures with little change to the superficial skin. The inadequately counseled neck-lift patient may be disappointed when after surgery their tight neck has no change in the skin texture or tone. Likewise, a patient with very redundant neck skin laxity may be disappointed following a nonsurgical approach with an improvement in their skin tone but no change in their “turkey gobbler” neck deformity. Nonsurgical approaches are much more focused on providing a benefit to the superficial dermis and supporting tissues rather than the deeper underlying neck structures. It is important to carefully listen to the patients’ goals and choose the best option or combination of options to meet their desires.

In the face and in the neck, nonsurgical treatments aimed at treating the dermal structures are important in achieving a complete rejuvenation and are frequently used in tandem with surgical treatments. However, neck skin is different from facial skin, lacking the appendage structures and thickness that is common to facial skin. Appendage structures such as sweat glands and hair follicles are reservoirs from which the dermis regenerates and heals wounds. But because the thinner neck skin lacks the density of appendage structures, care has to be taken while treating this area. Abbreviated and less aggressive treatments are necessary.

Significant photodamage is commonly seen on neck skin, which too often is neglected from photoprotection. This is easily demonstrated by identifying the contrast between the photoprotected submental area under the chin next to the photodamage lateral neck skin showing signs of chronic actinic changes (Figure 6.14). The lateral skin may appear leatherlike, dry, and mottled with red and brown discolorations. Rejuvenating treatments of the neck are not complete unless this skin has also been addressed. Regardless of a fantastic result from a surgical treatment, the crepe- cobblestoned appearing skin is not going to be improved. Often times a combination of nonsurgical and surgical treatments provides the best outcome. In the preceding section surgical approaches were discussed, below is an overview of some of the commonly used nonsurgical approaches to neck rejuvenation.

Nonablative skin rejuvenation

Step-by-step technique Intense pulsed light (IPL) devices and visible light lasers (VLL), most commonly the 532-nm KTP, are recognized as useful tools for cre-



Figure 6.14 Immediate submental region under the chin is photoprotected and exhibits less sun damage than the lateral neck

ating an epidermally spared, thermally damaged dermis producing new collagen, and repairing tissues defects associated with photoaging.^{24–33} IPL commonly offers light in the 450- to 1100-nm range and is absorbed by multiple chromophores, including melanin and hemoglobin. To reduce the strong affinity to melanin, various filters can be used to harness light wavelengths more specifically, depending on the device used. The KTP laser is specific to one wavelength, 532 nm, which falls in the green portion of the visible light spectrum. Much like the improvement noticed in the face, patients with lighter skin and dark dyschromias are the best candidates for treatments with the KTP and IPL. Following a series of treatments, the skin tone can reliably be expected to look better but unfortunately the efficacy and predictability for these devices to promote skin tightening may be less certain. Any ability to tighten the neck skin is modest if at all and is not routinely noticed by the patients. IPL and VLL are used predictably to improve

the skin tone and reduce the appearance of solar lentigines, erythema, and mottled pigmentation. When treating the neck, as compared to the face, energy settings should be reduced. The thin skin of the neck is at a greater risk for a heat generated injury. Devices with contact or parallel cooling may have an advantage in the neck area by protecting the thin dermis from heat damage.

All cosmetics and debris are removed from the face. A makeup cleanser pad will suffice as will an effective scrub with a 4 × 4 inch gauze pad and water. Alcohol or acetone scrub can also be used, but it is not necessary. It is important to be certain that if alcohol or acetone is used that they have dried prior to initiating the light-based treatment. Protective eyewear is worn by everyone in the room. Frequently for IPL and KTP laser treatments a topical water-based gel is applied to the skin facilitating movement of the device and providing a cool conductive medium. The treatment is initiated and usually one pass covering the entire neck is sufficient treatment. Treatments generally are not painful and are well tolerated without anesthesia.

Complications to avoid Complications in the neck following treatment with IPL and VLL lasers in the skin are rare and easily avoidable with the use of conservative energy settings and selection of appropriate candidates. Patients who have light skin (Fitzpatrick skin types I through III) and are not tan are most likely to achieve a satisfying effect and less likely to experience a burn or heat injury. Although, patients with skin type IV can be treated with caution by using significantly reduced energy settings and widened pulse widths to protect the epidermis. Patients on photosensitizing medications are either treated cautiously or not at all. If concerned about the patient's response to the device, a test spot can be done in an inconspicuous area (the posterior neck). The patient is instructed to return a week later to evaluate the tested skin. If a heat injury occurs, it often leads to a bullae formation and/or skin ulceration. Such injuries may result in temporary postinflammatory hyperpigmentation (PIH) or a permanent hypopigmented lesion. Additionally, although rare, a hypertrophic scar can occur in an area of injury. Keeping energies low, expanding the pulse width, and carefully cooling the skin are the measures taken to avoid such injuries. Those at higher risk, such as those with darker skin from the Pacific Rim or of Mediterranean background, can be pretreated with a skin-bleaching prod-

uct such as hydroquinone 4%. Permanent hypopigmented lesions are a possibility if the melanocytes are damaged. Although this is relatively rare, it should be avoided with careful patient selection and conservative treatments.

Postprocedure instructions Following the treatment, the patient is immediately provided with an ice pack to soothe the treated area. Patients are instructed to daily wear sunscreen of at least 15 SPF and with ingredients to block both ultraviolet A and B rays (UVA and UVB). Results tend to improve following multiple treatments and most patients are treated with a series of three to five treatments each separated by 3 to 4 weeks.

Nonsurgical skin tightening devices

There has been a lot of controversy surrounding the emergence of the nonsurgical nonablative skin tightening devices. Most use radio-frequency energies Thermage (Thermage, Inc., Hayward, CA).³⁴ Alma Accent and Harmony (Ceasera, Israel)³⁵ devices use radiofrequency plus diode laser energy (Syneron Medical Ltd, Yokneam, Israel).³⁶ However, some use infrared light such as Titan (Cutera Brisbane, CA).³⁷ Other devices use near infrared wavelengths, e.g., Gemini (Iridex).³⁸ There has been a favorable report using electrical muscle stimulation Pan G, (Pan Germinal Systems, Clearwater, FL).³⁹ Most of the devices are coupled with a parallel cooling and some with pre- and postcooling providing protection to the epidermis, while energy is delivered volumetrically to deeper tissues causing partial collagen denaturation.⁴⁰ Radiofrequency-generated heat results from tissue resistance to the electron flow and is known as impedance.⁴¹ Heat will follow the path of least resistance and the preferential heating has been experimentally deduced to extend along the collagenous fibrous septa connective tissue network interlaced between fat globules causing an immediate contraction of the subcutaneous tissues. This is believed to be the reason for the immediate contraction of the skin that is noted at the time of the treatment. This tightening allows lifting and remodeling of subcutaneous tissue and tightening of other attachments of the skin to the underlying muscle and bone.⁴² The epidermis is protected with a cooling device preventing the skin from overheating as the energy is directed to the deeper dermis. Because heat is nonspecific, there is little risk to darker skin color than to lighter skin color. Therefore, unlike IPL and VLL this device can be used in all skin colors.

The heat from the treatment causes collagen fibrils to denature and some contraction of the skin can be seen immediately, but most skin tightening becomes evident by 4 to 6 weeks and this continues over the next 3 to 12 months.^{43–45} Advocates recommend heating emphasis over anchoring points in the neck such as over the mastoid and following the direction of maximum lift of the skin.⁴²

However, caution is advised that the amount of tissue-tightening is variable and at times it can be unpredictable who is going to get the best result. It has been recommended that patients should be counseled that only modest improvement is likely to be achieved and is not equivalent to a surgical procedure.^{41,42} It seems this treatment is best suited for patients with early signs of aging with mild to moderate rhytides and skin laxity, and without significant structural ptosis.⁴²

In the early adoption of this procedure, during development of protocols, physicians attempting to maximize collagen denaturation and results were using high-energy settings; however, complications were frequent, and dissatisfying results were too frequently encountered.^{42,46} Recently more standardized protocols have been identified in which lower energy settings and multiple passes result in collagen contraction and tightening effects that are equivalent to or better than that with higher energy settings.⁴¹ Lower energy settings also mean less risk for a complication or heat induced injury. Additionally, patient satisfaction rates have also improved and are more reliably reported with an average of 75% to 100% satisfaction rates.^{42,47}

Patient selection There are few contraindications for RF heat skin tightening, but those with a permanent pacemaker or defibrillator are excluded from treatment. Those recently off isotretinoin (Accutane) or with a history of hypertrophic scarring may be excluded, or treated with caution.⁴¹

Step-by-step technique Patients are placed in a comfortable position. All jewelry is removed, as are cosmetics. If a monopolar RF is being used, then a grounding pad is applied to the patient. For bipolar RF heat, no grounding pad is necessary. A conductive gel is placed on the skin, followed by engagement of the device. Anesthesia is not generally warranted and general or intravenous monitored sedation is usually discouraged. A mild oral anxiolytic (diazepam 5–10 mg) or narcotic (hydrocodone 5 mg) 1 hour prior to the treatment is often sufficient to alleviate discomfort and anxiety in the anxious patient. A patient's response to the stimuli is an important commu-

nication feedback system. Too much heat-causing pain is not desired and can be associated with energy settings that are too high and put the patient at risk for injury. It is important that the device has complete contact with the skin. Partial contact can lead to ineffective treatment and even an injury as the tip has a cooling device within it. Additionally, with a bipolar RF device it is necessary for both ends of the conducting tip to be flushed with the skin or else the electricity may take the path of least resistance and jump across the skin surface to the other tip, causing an arcing of energy and development of a burn to the skin.

When performed properly, there is mild but tolerable discomfort. Although subjective, significant pain is considered an end point. Energy and subsequent heat is limited to what a patient can readily tolerate. One pass is performed over thinner skin areas with concentration over the anchoring points of the mastoid, mandibular angle, lateral occipital hairline in a band of approximately 1 to 2 cm wide.⁴¹ It takes approximately 30 minutes to do a full neck treatment.

Complications to avoid In the past high-energy fluences were used with damaging results, including potential for fat necrosis, skin color changes, and potential for scar formation.^{42,43} It was suspected that excess nonspecific heat reaching subdermal fat caused fat necrosis resulting in dimpling of the skin. Although it may resolve in some, the defect could be permanent in others requiring additional corrective procedures.

Additionally, there is a learning curve to using these devices. It has been suggested that operator error without complete contact with device to the skin may lead to tiny skin burns.⁴² Complications and skin texture changes have been estimated at 0.03% to 0.15%.^{41,42} However, with currently recommended lower settings and proper training complications are rare. With current protocols side effects are usually limited to mild transient edema, and redness typically resolves within hours and rarely extends beyond a day or two. Using settings that are common to the facial skin in the neck can lead to temporary numbness over the great auricular nerve which always resolves, and temporary bumps and ridging over the platysma. Fortunately, this too resolves over a month's period.⁴² Therefore, precautions are recommended to reduce energy settings by approximately 35% when treating the thin skin of the neck. Lower settings will help to avoid injuring the great auricular nerve or causing inflammation to the superficial platysmal muscle.⁴²

The main risk with these noninvasive skin tightening procedures is patient dissatisfaction. It is important that patient expectations are managed appropriately and the procedure is priced accordingly. Exaggerated definitions of “non-surgical face-lifting” may leave patients with the impression that such procedure replaces a surgical procedure. While objective skin tightening is frequently reported, patients expecting a surgical type result will certainly be disappointed with the subtle results. Additionally, a subset of patients may experience no result at all. It is this author's opinion if a cosmetic device does not reliably achieve a patient satisfaction rate of 90% or higher, the procedure should not be offered. Anything less than 90% may lead to an erosion of a cosmetic practice and professional reputation.

Postprocedure instructions Since the epidermis is not injured, patients experience limited downtime and may return to their regular schedule. To allow the maximal wound response and promotion of collagen production, anti-inflammatories are not recommended to be used. Anecdotally, it has been suggested that results last 14 months in most patients. However, some patients may have no results and others may not notice the results even if they have them.⁴¹ Multiple treatments repeated every 6 to 12 months may achieve better results especially in those with greater skin laxity.

Ablative procedures

Ablative procedures for neck rejuvenation have been recommended with blunted enthusiasm at best. However, if done with reliable and proven methods and in a conservative fashion, predictable and satisfying results can be achieved.

Removal of the epidermis can be performed with superficial to medium depth chemical peeling or with a low level erbium laser resurfacing. Rarely a low level CO₂ laser resurfacing is performed.⁴⁸

Chemical peels Chemical peeling of the neck will ablates the superficial layers of the epidermis and affects the dermis. Alpha hydroxy acids at strength up to 70% will removes superficial layers of the skin, and combat photoaging changes by decreasing corneocyte adhesion in the epidermis. Dermal effects include, increase in dermal fibroblast activity and an increase in glycosaminoglycans and collagen.⁴⁹ Following healing, the epidermal and dermal thickness increases as does the collagen density.⁵⁰ Clinically, the skin appears rejuvenated.

The results will take a few days to several weeks to appear. Lower strength peels are often done in a non-medical setting or by an extended provider and not the physician. Most states allow for glycolic acids of up to 35% to be performed by an esthetician. Treatments are often repeated and results are subtle and the risks for complications are relatively low.^{51–52}

Improvement in superficial dyschromias and texture of the skin can be appreciated but not by much. For a deeper extending peel and one that will produce a more extensive improvement in the appearance of the skin, a medium depth peel can be done using a trichloroacetic acid (TCA) solution. TCA peels are performed by a physician in a medically controlled environment. While TCA strengths of 35% are often used in the face, its strengths in the neck should be between 15% and 25%. The higher strength peels, above 25%, in the thin neck skin can result in an injury extending into the deep dermis and beyond. TCA peels seem to be particularly effective at improving deep-seated dyschromias and melasma. Similar to the superficial glycolic peels, treatments are generally repeated but not as often. A TCA peel can provide a modest, but significant improvement in the appearance of the neck skin.

Patient selection Ablative treatments are contraindicated in those on or recently off isotretinoin (Accutane) with connective tissue disease, open wounds, or previous radiation treatment for head and neck cancer.

Step-by-step technique Patients are placed in a comfortable reclining position for both peels. The skin should be cleansed of all debris. Preparation of the skin with a defatting acetone scrub with a 4 × 4 inch gauze pad is recommended to remove superficial debris, which will allow the peel to be placed more evenly.

For the superficial chemical peel the product is painted on the skin with a cotton tip, folded 2 × 2 inch gauze pad or a foam wedge applicator. Two passes are usually adequate after which the peel is allowed to sit in place for a few minutes (1–5 minutes). Patients often feel a mild burning discomfort after approximately 2 minutes. The peel is then neutralized and washed away with a topical water or basic solution. The neck is then soothed with a topical emollient and the patient is discharged soon after with posttreatment instructions.

For TCA peels patients are pretreated with an antiviral (valacyclovir 500 mg BID) and antibiotic (cefadroxil 500 mg qday) starting 1 day prior to treatment and extending for 6 days following the peel. After the patient is

placed in comfortable reclining position and all debris has been removed with an acetone scrub, the TCA solution is applied with a cotton tip applicator. One to two coats are applied evenly. Multiple coats cause the peel to extend deeper. As the solution starts to neutralize, it frosts coinciding with keratocoagulation. During this time period there is a mild burning sensation. The patient is allowed to hold a fan near his or her skin, which seems to help with the mild discomfort. Occasionally, oral analgesics are provided if needed such as ibuprofen and rarely a hydroxycodone (5 mg). The skin is not immediately ablated. The coagulated, frosted, and edematous skin provides a barrier to the slowly breaking down epidermis. Approximately 3 to 4 days after the treatment, the neck skin will start to exfoliate and peel off. In 1 to 2 weeks the skin will appear smoother, lighter in color with less dyschromias.

Postprocedure instructions In the posttreatment period as the skin exfoliates, patients are recommended to wear a petrolatum such as Aquaphor to protect the skin and promote rapid epithelization. The petrolatum is washed off three to four times a day and then replaced, until the skin epithelizes. Routine use of sunscreen is also recommended following recovery of the skin barrier.

Laser Ablative laser procedures of the neck have generally not been a routine treatment option for discoloration, dyschromias, and texture irregularity of the neck. In order to improve the texture of the skin, a deeper treatment is necessary and the neck with its thin nature and limited appendage structures is generally not an ideal site for an aggressive ablative laser procedure. CO₂ with a 10600-nm wavelength removes the entire epidermis and a portion of the dermis. Water, the chromophore, is heated over 100°C; sublethal thermal damage extends to deeper zones.⁵³ While this may be the most effective manner in which photodamaged skin of the face can be repaired downtime and risks for complications are rather high and even more risky in the thin skin of the neck. Erbium lasers 2940 nm with a much greater affinity to water than CO₂ is less aggressive with less thermal diffusion into deeper tissues.^{53,54} Erbium YAG can be expected to penetrate 4 μm/J/cm of erbium energy.⁵⁵ It has been noted that Erbium laser is extremely precise skin ablative tool that causes minimal thermal injury 5 to 10 μm and is a favored alternative to chemical peel for effective treatment of mild to moderate photodamage.^{55,56} But if a deeper effect is desired,

the erbium can be layered with multiple passes to achieve results similar to that of a CO₂ laser. This though is not simply performed as the erbium does not coagulate tissue as well as the CO₂ laser and when extending deep into the papillary and reticular dermis there is significant bleeding that can obscure signs indicating the depth of penetration. It is in the author's experience that a low level microablative erbium laser 2940 nm at 4 J/cm² can be an effective tool for rejuvenating the neck skin in patients with skin types I–IV.⁵⁷ The results are significant with an improvement in skin texture and superficial dyschromias. Deeper dyspigmentation and wrinkles will not be seen as significant improvements and results are comparable to that which is achieved with a precisely and evenly placed TCA 20% to 25% peel. Skin tightening improvements from this treatment are mild and not promoted. Rather the texture of the skin is where the greatest achievements are recognized.

Step-by-step technique Prior to the treatment an antiviral is often prescribed, (valacyclovir 500 mg bid), and an antibiotic (cefadroxil 500 mg BID) ideally taken prophylactically 24 hours prior to the procedure. Patients on or recently off isotretinoin (Accutane) CTD, previous radiation for head and neck cancer and open wounds are excluded from treatment. The patient is placed in a reclining position. Microablative erbium laser resurfacing can be painful; therefore, a topical anesthetic, commonly containing betacaine, tetracaine, and lidocaine, is applied for 20 to 30 minutes. Oral analgesics and anxiolytics such as hydrocodone 5 mg and diazepam 5–10 mg are prescribed to be taken 30 minutes prior to the procedure. It is important that the topical anesthetic cream is completely wiped off and the neck is “bone dry.” If the anesthetic is not completely removed, the laser will not penetrate the skin and the laser will be ineffective. Often an acetone scrub will effectively remove the anesthetic along with superficial debris and fat from the skin. Protective eyewear is provided and a vacuum to remove the plume is activated. The laser is initiated with a continuous one pass performed over the neck skin. The entire procedure takes approximately 5 minutes. Following the pass there is a slight ash char that covers the skin; this is left in place as a natural protective barrier. A topical petrolatum such as Aquaphor is applied and the patient is discharged.

Complications to avoid Complications for all ablative procedures in the neck are rare if staying within recommended conservative settings. Laser energy is decreased by 30% to 50% from those settings that are used in the face and chemical peels are kept to a lower concentration. For Erbium YAG not more than 15 J/cm is used and more often settings are kept between 4 and 7 J/cm².⁵⁶ Chemical peels are also used in more conservative strengths than that which are used on the face. Superficial glycolic peels are used at 50% or less and TCA peels at 25% or less. However, with all ablative procedures there is always a concern of skin color changes such as temporary hyperpigmentation, permanent hypopigmentation, or scar formation.⁵⁸ Such complications are fortunately uncommon but are a possibility if the settings are not kept conservative.

Disseminated topical herpes infection, although rare with a lower laser energy settings and conservative chemical peel strengths, is still a possibility if de-epithelized skin is contaminated. Patients are instructed on using good aseptic techniques when washing the petrolatum off their face and on the importance of taking their prophylactic antiviral medications. They are also instructed to call if they do recognize a fever blister surface. If herpes infection is recognized, then higher dose acyclovir 800 mg qid is used to treat the infection.

Postprocedure instructions Following the micro ablative procedures, the patient is recommended to cleanse the skin three times a day using gentle tap water followed by application of Aquaphor ointment. This helps to remove superficial debris exfoliating skin and bacteria. Epithelization is promoted and the skin peels for approximately 4 to 5 days. The renewed thin epidermis appears slightly pink for a few days there after returning to a normal skin color over the next week or two. Sunscreens are highly encouraged during the posttreatment period. The treatment can be repeated every 2 to 3 months if desired, but it is rarely performed more than three times a year. Multiple treatments tend to promote better results though.

Fractionated resurfacing One of the biggest breakthroughs in neck rejuvenating treatments in the last few years is the advent of fractionated resurfacing: Fraxel (Reliant Technologies, Palo Alto, CA) and Harmony and Pixel (Alma, Cesarea, Israel). Fractional resurfacing: Palomar (Burlington MA) 1540 nm TM LuxIR. Fraxel is the first to receive US Food and Drug Administration approval for treatment of pigmented lesions, periorbital

rhytides, skin resurfacing, melasma, and soft tissue coagulation. It is used “off label” for neck rejuvenation. This type of treatment is ideally suited for the neck. Using a fractionated handpiece with mid infrared wavelengths and a chromophore of water, energy can be provided deep into the neck skin. Heat creates injury in microscopic thermal treatment zones (MTZ). Using the Fraxel, collagen denaturation occurs to a depth of approximately 300 μm and 100 μm wide.⁵⁹ As fluence is increased, the laser can penetrate deeper into the dermis up to 1359 μm at 70 mJ.⁶⁰ The Pixel (Alma, Cesarea, Isreal) energy also working within MTZ does not reach the deep depths as that achieved with Fraxel, but similar islands of healthy tissue surrounding the MTZ of denatured collagen allow for rapid epithelization usually within a day and protection to the skin.⁶¹ Histological studies 3 months following Fraxel treatment reveal enhanced rete ridges, and increased mucin deposition in the superficial dermis. Such technology allows deep penetrating energy that breakdown collagen, then stimulates a wound response. The skin heals rapidly and the texture, wrinkles, and dyschromias are significantly improved.⁶¹ Unexpectedly, however, a significant improvement has been noted in epidermal and deeper dermal dyschromias like melasma.⁶² This technology is effective at reducing dyschromias without the downtime common to medium depth peels, microablative lasers, and with efficacy comparable, if not superior, to IPL and VLL (Figure 6.15). To date this technology has provided the biggest break through in improving texture, dyschromias and superficial crepe-ness, and rhytids of neck skin. Since downtime is minimal, patients are more apt to repeat treatments which can be done every 6 to 8 weeks apart but in clinical practice most patients are very satisfied after one or two treatments.

Patient selection Since treatment with fractional devices creates less heat damage and maintains islands of healthy tissue, the treatment is theoretically safer in darker skinned patients with compromised appendage structures. Nonetheless those on or recently off isotretinoin (Accutane) CTD or previous radiation treatments for head and neck cancer are excluded from treatment. While it is not recommended for treating darker skin types, it has been this author's experience that skin types IV and V can be treated using Alma pixilated device without significant sequelae. Skin type VI patients are not currently treated and those at risk for hyperpigmentation



Figure 6.15 Neck is rejuvenated following pixilated laser treatments

may be pretreated with hydroquinone 4% for 4 weeks prior to the procedure.

Step-by-step technique All patients are pretreated with an antiviral valacyclovir 500 mg bid starting 1 day before the treatment and extending into the next 6 days.

Patients are placed in a reclining position. This treatment is without discomfort; therefore, a topical anesthetic is not required. Topical cosmetics are removed and the skin is debrided with an acetone solution and 4 × 4 gauze pad. After the neck is thoroughly clean and dry, a smoke evacuator is initiated as is protective eyewear. The laser is turned on and aggressive settings are utilized (1400 mJ/cm²). One pass with two to three stacks of pulses at each site are done over the neck. The procedure takes approximately 20 minutes to perform after which a light layer of topical petrolatum is applied and the patient discharged.

Complications to avoid Complications are very rare when using this technology which seems ideally suited for the thin neck skin. There is a theoretical risk for hypo

and hyperpigmentation. If hyperpigmentation were to occur a topical hydroquinone 4% can be prescribed for up to 8 weeks. Ulcerations and hypertrophic scar formation are unlikely if the device is used according to manufacturer recommendations.

Postprocedure instructions Following treatment, the patient may feel a bit discomfort similar to a sunburn on their neck. Fraxel, which extends deeper than the Pixel, may cause more discomfort but is usually resolved within 60 minutes after the treatment.⁶¹ A topical emollient is effective at reducing the pain as is an over the counter analgesic. The skin can be expected to be erythematous for approximately 3 to 4 days. The skin routinely does not peel. After approximately a week the neck has a shiny sheen to it with a reduction in dyschromias clearly evident. For more than the next 8 to 12 weeks there is progressive improvement in the skin appearance. Sunscreens are highly encouraged.

Thread lifting Barbed sutures threaded thorough the dermis and superficial subcutaneous tissue of the face

and neck to anchor skin in a more vertical position had a limited period of popularity. Early results were promising,⁶³ but enthusiasm has waned as the results have not been as predictable or as lasting as desired. Barbed polypropylene suture threads marketed as Contour Threads™ received FDA approval for lifting ptotic skin of the face and neck. Although not currently available, these were highly anticipated as new, novel method for performing an in office quick 20 to 30 minutes lifting procedure that required local anesthetic only.

Step-by-step technique The thread is introduced proximally in the lateral neck and anchored to the thick confluence of fascia and muscle fibers of the sternocleidomastoid muscle. The long needle is then weaved thorough dermal and subdermal skin exiting just proximal to the platysmal midline. The skin is then massaged back over the thread engaging the barbs as the counter pressure is held on the distal end of suture. The skin is fixated in a vertically and laterally elevated position. Topical antibiotic ointment is applied to the exit and entry ports, and the patient is discharged soon after with little to no downtime. A mild bunching of the redundant skin can be noted on the lateral neck but usually flattened out over the next few days. If it does not completely resolve, the barbs can be released or disengaged from the skin by massaging the skin in the opposite direction to the barbs. However, if too many of the barbs are disengaged, the neck skin would immediately drop back into the native position.

Complications to avoid Complications most commonly include infection and extrusion (Figure 6.16). Many patients along with physicians have been greatly dissatisfied with the results. The results have been rather unpredictable and often not lasting beyond 6 months. As this method currently exists, it is rarely offered and has little indication. Perhaps future development in the manufacturing of such device will result in a more reliable method for temporarily or permanently mobilizing the superficial skin structures.

Botulinum toxin A treatment Botulinum Toxin A (BtxA) is the most popular nonsurgical cosmetic procedure in the US according to American Academy of Aesthetic Surgery 2006 statistics.⁶⁴ Thickened prominent platysmal banding can be temporarily reduced with BtxA injections.^{65,66}

Platysmal bands are vertically oriented muscular cords often extending from the mandibular border down to the



Figure 6.16 Subdermal sutures used to mobilize tissues can lead to infection and extrusion

clavicle. They are thought to occur from the result of persistently active platysmal muscle trying to support the sagging deeper neck and floor of mouth structures.⁶⁵

Patient selection The treatment is best suited for the patients with thick bands and a thin neck. Heavy necks with a turkey gobbler appearance may actually become worsened and more ptotic by decreasing tone in the platysmal muscles. BtxA injections also work very well in the postrhytidectomy patients with a prominent midline platysmal band. The bands can be identified by having the patient clench their teeth. The hyperdynamic bands can be improved for a period of time lasting approximately 12 weeks (Figure 6.17), and patients will then return to their pretreatment appearance.

Botulinum toxin A treatments in the neck can be offered to patients of all ages, and there are very few contraindications to BtxA treatments whether in the face or neck. BtxA for use in the neck is diluted with 2 mL or less of bacteriostatic saline/100 units of BtxA. A more concentrated solution preventing significant



Figure 6.17 A&B Midline platysmal banding is reduced following botulinum toxin injections into the platysmal muscle

diffusion into deeper structures of the neck may be beneficial for treating platysmal banding of the neck.

Patient positioning It is important to have the patient placed in the sitting position rather than a reclined position where the bands can become obscured.

Step-by-step technique The patient is asked to clench his or her teeth and neck and the bands are identified. An individual band is grasped between the forefinger and thumb. The neck is cleansed with an alcohol pad and is then allowed to dry. BtxA is injected directly into the muscle. Care is taken to not inject deep into the structures of the neck or below the platysma. Multiple injection sites spaced approximately 2-cm apart are performed along the bands most prominent position. Approximately 5 to 20 units are deposited along the band. Higher doses have not been found necessary, and a total treatment to neckbands rarely exceeds 100 units.

Complications to avoid Complications are rare with botulinum toxin A, which has been found to be one of

the safest products in medicine when used within recommended parameters.⁶⁷ However, if high doses of botulinum toxin A are placed (greater than 200 units) or injected deep into the supporting structures of the neck, there is a theoretical risk for neck weakness, laryngeal, or pharyngeal compromise with dysphagia and even dyspnea. There has been one report of dysphagia.⁶⁸ If botulinum toxin A is kept away from the area of thyroid cartilage then the risk for laryngeal effects are very unlikely.

Postprocedure instructions The patient is discharged soon after the treatment without any sequelae. The results will take 3 to 5 days to appear and will last approximately 4 months.

CONCLUSION

The triumvirate features of the aging neck combine accumulated submental fat, platysmal banding, and redundant dyspigmented skin in varying degrees. When these

anatomic changes associated with aging are astutely appreciated by the surgeon and patient, safe, appropriate, and effective aesthetic neck contouring surgery can be undertaken. Features of a youthful neck include an acute cervicomental angle, a distinct inferior mandibular border, subthyroid depression, a visible thyroid cartilage, anterior border of the sternocleidomastoid muscle along with evenly toned and textured skin. To restore these features the surgical armamentarium for neck contouring entails submental liposuction, platysmal plication, neck-lift along with chemical peeling ablative, nonablative laser (RF treatments, and chemodenervation. Selecting the appropriate procedure for the appropriate patient that will effectively meet the aesthetic goals and expectations is the core of successful neck rejuvenation.

REFERENCES

1. Vistnes LM, Souther SG. The platysmal muscle: anatomic consideration for aesthetic surgery of the anterior neck. *Clin Plas Surg*. 1983;10:441.
2. de Castro C. The anatomy of the platysma muscle. *Plast Reconstr Surg*. 1980;66:680.
3. Dayan SH, Bagal A, Tardy ME. Targeted solutions in submentoplasty. *Facial Plast Surg*. 2001;17:141-149.
4. Wang TD. Patient selection for aging face surgery. *Facial Plast Surg Clin North Am*. 2005;13(3):381-382.
5. Friedman O. Changes associated with the aging face. *Facial Plast Surg Clin North Am*. 2005;13:371-380.
6. Rorich RJ, Rios JL, Smith PD, et al. Neck rejuvenation revisited. *Plast Reconstr Surg*. 2006;118(5):1251-1263.
7. Haack J, Friedman O. Facial liposculpture. *Facial Plast Surg*. 2006;22(2):147-153.
8. Adamson PA, Litner JA. Surgical management of the aging neck. *Facial Plast Surg*. 2005;21(1):11-20.
9. McGraw-Wall B. Preoperative evaluation of the aesthetic surgery patient. In: Bailey B, ed. *Head and Neck Surgery-Otolaryngology*. Philadelphia: JB Lippincott; 1993:2070-2083.
10. Watson D. Submentoplasty. *Facial Plast Surg Clin North Am*. 2005;13(3):459-467.
11. Kridel RWH, Kelly PE. Liposuction of the face and neck. In: Papel ID et al, eds. *Facial Plastic and Reconstructive Surgery*. 2nd ed. New York, NY: Theime; 2002.
12. Jacob CI, Berkes DJ, Kaminer MS. Liposuction and surgical recontouring of the neck: a retrospective analysis. *Dermatol Surg*. 2000;26(7):625-632.
13. Morrison W, Salisbury M, Beckham P, et al. The minimal facelift: liposuction of the neck and jowls. *Aesthetic Plast Surg*. 2001;25:94-99.
14. Kamer FM. Isolated platysmaplasty. *Arch Facial Plast Surg*. 2003;5:184.
15. Sykes JM. Rejuvenation of the aging neck. *Facial Plast Surg*. 2001;17(2):99-107.
16. Dedo DD. Management of the platysma muscle after open and closed liposuction of the neck in facelift surgery. *Facial Plast Surg*. 1986;4:45-56.
17. Perkins S, Dayan S. Rhytidectomy. In: Papel ID et al, eds. *Facial Plastic and Reconstructive Surgery*. 2nd ed. New York, NY: Theime; 2002:153-170.
18. Cheng ET, Perkins SW. Rhytidectomy analysis: 20 years of experience. *Facial Plast Surg Clin North Am*. 2005;13(1):15-31.
19. Kamer FM, Pieper PG. Surgical treatment of the aging neck. *Facial Plast Surg*. 2001;17:123-128.
20. Perkins SW, Williams JD, MacDonald K, et al. Prevention of seromas and hematomas following facelift surgery with the use of postoperative vacuum drains. *Arch Otolaryngol Head Neck Surg*. 1997;123:743-745.
21. Strath R, Raju D, Hipps C. The study of hematomas in 500 conservative face lifts. *Plast Reconstr Surg*. 1983;52:694-698.
22. Jasin ME. Submentoplasty as an isolated rejuvenative procedure for the neck. *Arch Facial Plast Surg*. 2003;5:180-183.
23. Canter HI, Yilmaz B, Gurunluoglu R, Algan H. Use of gabapentine (neurontin) for relief of intractable pain developed after face-lift surgery. *Aesthetic Plast Surg*. 2006;30(6):709-711.
24. Goldberg DJ, Whitworth J. Laser skin resurfacing with the Q-switched Nd:YAG laser. *Dermatol Surg*. 1997;23:903-906.
25. Herne KB, Zachary CB. New facial rejuvenation techniques. *Semin Cutan Med Surg*. 2000;19:221-231.
26. Bjerring P, Clement M, Heickendorff L, Egevisst H, Kiernan M. Selective non ablative wrinkle reduction by laser. *J Cutan Laser Ther*. 2000;2:9-15.

27. Ross EV, Sajben FP, Hsia J, et al. Non ablative skin remodeling: selective dermal heating with a mid infrared laser and contact cooling combination. *Laser Surg Med*. 2000;26:186-195.
28. Menaker GM, Wrone DA, Williams RM, et al. Treatment of facial rhytids with a non ablative laser: a clinical and histologic study. *Dermatol Surg*. 1999;25:440-444.
29. Kelly KM, Nelson JS, Lask G, et al. Cryogen spray cooling in combination with non ablative laser treatment of facial rhytides. *Arch Dermatol*. 1999;135: 691-694.
30. Goldberg DJ. Full face non ablative dermal remodeling with a 1320 nm Nd:YAG laser. *Dermatol Surg*. 2000;26:915-918.
31. Trelles MA, Allones I, Luna R. Facial rejuvenation with a non ablative 1320 Nd: YAG laser: a preliminary clinical and histologic evaluation. *Dermatol Surg*. 2001;47:482-488.
32. Levy JL, Trelles M, Lagarde JM, et al. Treatment of wrinkles with the non ablative 1320 nm Nd:YAG laser. *Ann Plast Surg*. 2001;47:482-488.
33. Lupton JR, William CM, Alster TS. Non ablative laser skin resurfacing using a 1540 nm erbium glass laser: a clinical and histologic analysis. *Dermatol Surg*. 2002;28:833-835.
34. Weiss RA, Weiss MA, Munavalli G, et al. Monopolar radiofrequency facial tightening: a retrospective analysis of efficacy and safety in over 600 treatments. *J Drugs Dermatol*. 2006;5:707-712.
35. Mayoral FA. Skin tightening with a combined unipolar and bipolar radiofrequency device. *J Drugs Dermatol*. 2007;2:212-215.
36. Doshi SN, Alster TS. Combination radiofrequency and diode laser for treatment of facial rhytides and skin laxity. *J Cosmet Laser Ther*. 2005;7(1):11-15.
37. Bunin LS, Carniol PJ. Cervical facial skin tightening with an infrared device. *Facial Plast Surg Clin North Am*. 2007;15(2):179-184.
38. Dayan SH, Vartanian AJ, Menaker G, et al. Non-ablative laser resurfacing using the long pulse (1064 nm) Nd:YAG laser. *Arch Facial Plast Surg*. 2003;5:310-315.
39. Taub AF. Evaluation of a non surgical muscle stimulating system to elevate soft tissue of the face and neck. *J Drugs of Dermatol*. 2006;5(5):446-450.
40. Zelickson BD, Kist K, Bernstein E, et al. Histological and ultrastructural evaluation of the effects of a radio frequency based non ablative dermal remodeling device: a pilot study. *Arch Dermatol*. 2004;140:240-209.
41. Ruiz Esperaza J. Non ablative radiofrequency for facial and neck rejuvenation: a faster safer and less painful procedure based on concentrating the heat in key areas. The Thermalift concept. *J Cosmetic Derm*. 2006;5:68-75.
42. Abraham MT, Ross EV. Current conception in non ablative radiofrequency of rejuvenation of the lower face and neck. *Facial Plas Surg*. 2005;21(1):65-73.
43. Fitzpatrick R, Geronemus R, Goldberg D, et al. Study of non invasive radiofrequency for periorbital skin tightening. *Lasers Surg Med*. 2003;33:232-242.
44. Narins DJ, Narins RS. Non surgical radiofrequency facelift. *J Drugs Dermatol*. 2003;2:495-500.
45. Alster TS, Tanzi E. Improvement of neck and cheek laxity with a non ablative radiofrequency device a lifting experience. *Dermatol Surg*. 2004;30:503-507.
46. Bassichis BA, Dayan SH, Thomas JR. Use of Non ablative radiofrequency device to rejuvenate the upper one third of the face. *Otolaryngol Head Neck Surg*. 2004;130:397-406.
47. Abraham M, Chiang S, Keller G, et al. Clinical evaluation of non ablative radiofrequency facial rejuvenation. *J Cosm Laser Ther*. 2004;6:136-144.
48. Fulton JE, Rahimi AD, Helton P, et al. Neck rejuvenation by combining Jessner/TCA peel dermasanding and Co₂ laser resurfacing. *Dermatol Surg*. 1999;25:745-750.
49. Bernstein Ef, Underhill CB, Lakkakorpi J, et al. Citric acid increases viable epidermal thickness and glycosaminoglycan content of sun-damaged skin. *Dermatol Surg*. 1997;23:689-694.
50. Newman N, Newman A, Moy LS, et al. Clinical improvement of photo-aged skin with 50% glycolic acid a double blind vehicle controlled study. *Dermatol Surg*. 1996;22:455-460.
51. Monheit GD, Chastain MA. Chemical peels. *Facial Plast Surg Clin* 2001;9(2):239-255.
52. Zakopoulou N, Kontochristopoulos G. Superficial chemical peels. *J Cosmet Dermatol*. 2006;5(3):246-253.

53. Khatri KA, Ross EV, Grevelink JM, et al. Comparison of Erbium YAG and Carbon dioxide lasers in resurfacing of facial rhytides. *Arch Dermatol*. 1999;135: 391-397.
54. Weinstein C. Why I abandoned CO₂ laser resurfacing: the dilemma of evolving technologies. *Aesthetic Surg J*. 1999;19:67-69.
55. Pozner J, Goldberg D. Superficial erbium YAG laser resurfacing of photodamaged skin resulting in a mild ablation without extensive heat injury to the deeper dermis. *J Cosmet Laser Ther*. 2006;8:89-91.
56. Pozner JN, Goldberg DJ. Histologic effect of a variable pulsed Er:YAG laser. *Dermatol Surg*. 2000;26: 733-746.
57. Dayan S, Rehl R. Microablative erbium laser resurfacing, Oral Presentation at the AAFPRS September 2004.
58. Fitzpatrick RF, Goldman MP. Resurfacing of the neck using the Ultrapulse CO₂ laser. *Lasers Med Surg*. 1997;9(suppl):33.
59. Burns AJ. Fractional resurfacing in Plast Surg. A Continuing medical Education Program 2005;1:6-12.
60. Chiu RJ, Kridel WH. Fractionated photo-thermolysis; The fraxel 1550 nm glass fiber laser treatment. *Facial Plast Surg Clin*. 2007;15:229-237.
61. Mannstein D, Herron GS, Sink RK, (et al.). Fractional photothermolysis: a new concept for cutaneous remodeling using microscopic patterns of thermal injury. *Laser Surg Med*. 2004;34:426-438.
62. Tannous ZS, Astner S. Utilizing fractional resurfacing in the treatment of therapy-resistant melasma. *J Cosmet Laser Ther*. 2005;7:39-43.
63. Horne DF, Kaminer MS. Reduction of face and neck laxity with anchored barbed polypropylene sutures (contour threads). *Skin Ther Lett*. 2006;11:5-7.
64. American Society of Aesthetic Plastic Surgery statistics 2006. Available from www.surgery.org.
65. Brandt FS, Boker A. Botulinum toxin for the treatment of neck lines and neck bands. *Dermatol Clin*. 2004;22(2):159-166.
66. Dayan SH, Maas CS. Botulinum toxins for facial wrinkles: beyond glabellar lines. *Facial Plast Surg Clin North Am*. 2007;15(1):41-49.
67. Vartanian AJ, Dayan SH. Complications of botulinum toxin A use in facial rejuvenation. *Fac Plast Surg Clin*. 2003;11:483-492.
68. Matarasso A, Matarasso SL, Brandt FS, et al. Botulinum toxin for the management of platysmal bands. *Plast Reconstr Surg*. 1999;103(2):645-652.

CHAPTER 7

Abdominal Rejuvenation

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KEY POINTS

- Abdominal rejuvenation is a growing interest, particularly as the incidence of obesity increases.
- Surface irregularities such as striae may be improved but not fully corrected.
- Tumescant liposuction is an excellent therapy for localized areas of increased adiposity.
- For patients with extremely large abdomens desiring improvement, or for patients post weight loss who have a significant amount of redundant skin, an abdominoplasty can aid in appearance improvement.
- Fat transplantation can be helpful in improving areas of soft tissue loss, such as traumatic fat necrosis.
- Lasers and radiofrequency devices are being investigated for their ability to improve the abdomen.

BACKGROUND

The wish for a better looking abdomen is common among aging patients. They often notice an increase in abdominal fat as well as a decreased lean body mass and decreased skeletal muscle mass. There is a worrisome phenomenon of increasing obesity in the United States.¹ In 2006, only 4 states had a prevalence of obesity less than 20%, 22 states had prevalence equal or greater than 25%, and 2 states had a prevalence of obesity greater than or equal to 30%. Because excessive body weight tends to accumulate in the abdomen, dermatologists and dermatologic surgeons will often be approached by patients during cutaneous examinations inquiring as to what can be done to rejuvenate the abdomen. Patients may also be bothered by abdominal striae or small areas of localized adiposity. Dermatologic science and surgery have brought about great advances in body improvements including tumescent liposuction, lasers, and energy-based devices.

■ Abdominal Striae

Many female patients inquire as to what can be done about striae, often called stretch marks, present on the abdomen (Figure 7.1) and treatment of these in general is covered in Chapter 11. These irregular bands or stripes appear on the skin after stretching the skin or gaining weight, for example, after pregnancy. Common areas include abdomen, breasts, hips, thigh, buttocks, and flanks.² They may be a result of abnormal collagen formation such as Ehlers-Danlos disease or as a result of chemical effects on collagen formation. In addition to pregnancy and abnormal collagen synthesis, stretch marks can be seen in disorders such as Cushing's disease, diabetes mellitus, obesity, or even after puberty. Striae can be divided into two types: striae rubra and striae distensae. Striae rubra are red areas that are inflamed. They may be slightly raised above the skin. Striae distensae or striae alba are whitish lines or bands which have a different texture than surrounding skin. They can be slightly depressed.

There are no methods to completely remove striae, but some improvements have been reported with topical retinoic acid. Striae rubra have been improved with the 585 nm pulsed dye laser. There have been reports of improvement of striae with radiofrequency plus pulsed dye laser and bipolar radiofrequency alone.^{3,4} See Chapter 11 for further details of these techniques.

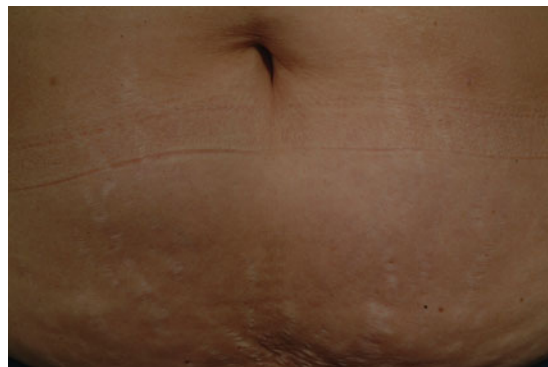


Figure 7.1 Abdominal striae in a 46-year-old female

LIPOSUCTION

■ Patient Selection and Preoperative Evaluation

Liposuction using the tumescent technique has been the mainstay of therapy for many years. Pioneered by physicians in Europe, the procedure has been revolutionized with the use of dilute lidocaine solutions containing small amounts of epinephrine. The developer, Dr. Jeff Klein, coined the term tumescent anesthesia. It has been proven safe and effective in thousands of patients. Complications are few and usually limited to local problems such as bruising.

Patients who are good candidates for liposuction ideally have limited areas of excess adiposity. They tend to be otherwise in good shape, have a relatively normal weight to height ratio, and are concerned mostly about small areas of localized fat excess.⁵ Patients who are likely to benefit from tumescent liposuction are those who

are in good general health, have appropriate expectations, and understand the limitations of liposuction surgery. When combined with a program of exercise and good nutrition, liposuction surgery can offer significant improvement in body contour for the abdomen (Figure 7.2). Patients benefit by looking better, feeling better about their appearance, and enjoying an improved fit of their clothing.

When considering issues of excess abdominal fat, it is important to remember that the ideal abdominal contour is determined by the correct proportionality of the upper and lower abdomen and flanks (“love handles”). It is important to evaluate whether the patient needs treatment of a single site or whether multiple areas need to be treated in order to obtain a balanced abdominal shape. It is also important to evaluate whether or not posture or skeletal abnormalities, such as kyphosis, could be responsible for the nonideal central shape. It is important to evaluate the patient to make sure that the areas that seem to contain excess adiposity are those having true

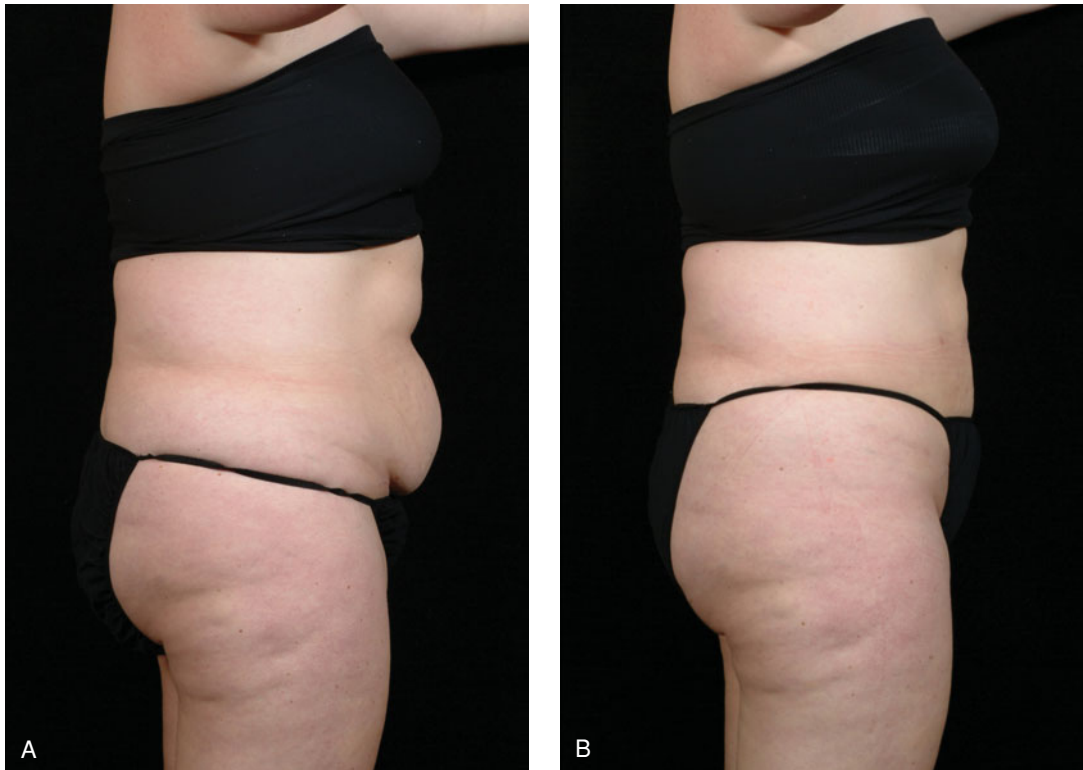


Figure 7.2 Before and 6 weeks after abdominal liposuction in a 36-year-old woman

subcutaneous fat excess. By pinching skin of the abdomen, the degree of subcutaneous fat is assessed. This allows some degree of a prediction as to how much improvement can be offered with tumescent liposuction.⁶ In men, the surgeon must often decide if the larger abdomen is composed mostly of subcutaneous fat or if the fat is omental. Intra-abdominal fat is clearly not approachable with tumescent liposuction, but the more common subcutaneous fat collections are areas where liposuction can make dramatic improvements.

Liposuction surgery is a cosmetic procedure and should be treated as such. Patients need to be in excellent overall general health, near or at their ideal body weight. It is important always to encourage patients considering liposuction to follow good nutritional habits and exercise, and thus they are usually put on this type of program for several months prior to liposuction to ensure they are “on board” with an overall program that will help to achieve good and hopefully long-term results. If the patient fails to follow these recommendations regarding calorie intake and caloric expenditure, it is doubtful that there will be any long-term benefit of the procedure. We have found that the low-carbohydrate diet can be helpful for many liposuction patients (for those patients medically cleared for this diet plan).

Liposuction is clearly not for the overweight patients. Overweight patients who have tried and failed past attempts to exercise and to lose weight by dieting may find benefit from a physical trainer and a nutritionist. Very obese patients who have slowly been gaining weight for years in spite of their approach toward diet and exercise will likely benefit most from an invasive bariatric surgery.

The liposuction surgeon in consultation emphasizes that patients will have an “improvement in silhouette” to describe changes in body shape. It is important that the surgeon must constantly emphasize that we are achieving improvement in overall body contour instead of perfection and should emphasize that liposuction goes along with diet and exercise in a program aimed at looking good for the long term. It is also important to inform the patient that while a single procedure can convey great benefit, occasionally subsequent procedures are necessary to achieve the desired outcome or fine-tune certain areas. Abdominal liposuction can sometimes even be performed in two sessions because of the frequent size of some patient's upper and lower abdomens.

Patients undergoing liposuction surgery using tumescent anesthesia will find the outpatient surgical procedure easy, straightforward, and relatively painless.⁷ Most

patients are informed that they can return to their work activities and gently resume their exercising over several days. They are also informed about the drainage of the dilute anesthesia, which is facilitated by compression garments and tell them that this oozing of fluid through the ports usually continues for approximately 3 days following the procedure. Patients are educated that they will be seen regularly, postoperatively, and their weight will be measured on each visit as well as continuing the discussion of their diet and exercise habits often facilitated through a patient diary.

It is important to give detailed, written-informed consent to any patient considering liposuction surgery. While there is a low risk of complications from tumescent liposuction, it is important to list additional serious complications such as thrombosis or pulmonary emboli, fat embolism, sepsis, or necrotizing fasciitis that accompany any elective surgical procedure. These have been observed but are fortunately uncommon complications in liposuction surgery. Common side effects, such as bruising and edema, from gravitational fluid movement over the first several days following the procedure are detailed on the consent form and reviewed with the patient.⁸

Patients are asked to refrain from the use of aspirin, nonsteroidal anti-inflammatory agents, herbal supplements, and vitamin E for 2 weeks prior to liposuction. Patients should expect fluid and blood-tinged drainage along with minor postoperative pain, which can usually be controlled with nonnarcotic medications. They are also informed that they will experience some transient numbness or minor contour irregularities from swelling, many of which resolve over a period of 6 months following the procedure.

It is essential to tell patients that the final result is not completely apparent until approximately 6 months following the surgery. Laboratory assessment before liposuction includes hepatitis serology, HIV status, complete blood count with differential and platelet count, metabolic panel, and coagulation studies.⁵

■ Technique

On the day of surgery, the patient arrives at the clinic having bathed the night before or morning of the surgery with a chlorhexidine shower. This procedure serves to reduce the bacterial count on the patient's skin before the procedure and before the in-office skin preparation. Patients usually come into the clinic wearing comfortable clothing such as a jogging suit. The nursing personnel and clinic

staff meet again with the patient and determine if there are any additional questions that the patient may have. The patient, having already given a copy of the informed consent before the procedure, signs the consent in the presence of the operating physician. The physician ensures that any additional questions or areas of patient concern have been addressed and asks if there are any additional questions that the patient would like to have answered. In essence, a small miniconsultation again occurs in which the expected outcomes are reviewed with the patient; all questions and concerns are answered regarding liposuction surgery, and the patient's medical history, medication use, cigarette smoking, alcohol use, and drug allergies are once again reviewed. Medical contraindications to liposuction of the abdomen are discussed. These include pregnancy, significant cardiovascular renal disease, coagulation disorders, immunosuppression, active hepatitis, or chronic liver failure. Morbid obesity, chemotherapy, and a history of malignant hyperthermia are contraindications.

It is vitally important to prevent drug interactions between lidocaine metabolism and any drugs the patient may be taking. A good list of the drugs which can interfere with lidocaine metabolism can be found in the text by Dr. Jeffrey Klein.⁹ As part of the physical examination, photographic documentation of areas of concern prior to liposuction surgery is vitally important. It is important to have pictures taken in several positions and angles to show the areas of excess fat. The patient's weight is again measured and documented.

Physical examination includes examining the patient again to delineate areas of fat excess and to look for hypertrophic or keloidal scars. Scars on the abdomen can indicate previous gynecologic or abdominal procedures. The abdominal areas which are to be suctioned are outlined with a surgical marker or black or red "Sharpie" (Figure 7.3). Having the patient verify that the areas marked are indeed the areas of concern for the patient, and it allows the liposuction surgeon and patient to review the process again. We discuss our markings with the patient and have them look at our "topographic map" that we have drawn on their body. The process of marking the patient also involves pinching the areas to get a sensation of the degree of subcutaneous fat with those areas that are greater "hills," needing additional passes of the cannula or suctioning in order to bring these larger areas down. After marking the patient, we obtain a second set of photographs. The patients are then escorted into the liposuction surgical suite.



Figure 7.3 *Topographic markings used preoperatively to mark the areas of excess adiposity*

Equipment/instrumentation

Monitoring equipment capable of continuously recording an EKG and intermittent blood pressure measurements, heart rate, body temperature, and pulse oximetry are used throughout the procedure. A warming blanket under the patient can provide significant comfort. The patient is dressed for the procedure in comfortable garments, which allows the surgeon full access to liposuctioning the abdomen (and often additional areas being treated the same day such as the thighs) but yet covers up the genitalia and perineum.

Patient positioning

A powered standard surgical table is helpful in assisting patients in terms of moving into position and provides greater comfort for the surgeon. If preoperative medications, such as lorazepam 1 mg, are to be used before the procedure, then these are given to the patient prior to initiating the procedure to help reduce anxiety. The patient is allowed to relax for approximately 20–30 minutes before infiltration of the tumescent anesthesia is begun.

Anesthesia

The patient's abdomen is prepped with a surgical scrub (for example, chlorhexidine or betadine). The skin entry sites that have been marked for infiltration of dilute anesthesia are numbed using stock 1% Xylocaine, with epinephrine injected through a 30-gauge needle. A 4-mm trephine punch can be used to open insertion sites in the skin. An electric-powered infusion pump is set up to allow instillation of the dilute local anesthesia. Bags of dilute anesthesia have been prepared ahead of time and contain 0.05% Xylocaine with 1:1000000 epinephrine.¹⁰ Sodium bicarbonate has been used to adjust the pH to physiologic. An electric-powered infusion pump greatly facilitates infiltration, and these peristaltic often allow for variable speeds. Anesthesia is usually infiltrated using a multiport infusion cannula that are blunt-tipped, approximately 3 mm in diameter and approximately 40 cm in length. Because of the length of the cannula a large area can be anesthetized from a single skin entry site. The assistant nurses should be instructed to take great care while doing tumescent anesthesia and to infiltrate the dilute solution in multiple planes. The infiltrate begins slowly at approximately 1 mL/min and the rate is increased as tolerated by the patient.

The maximum volume of anesthesia to be used during the procedure is precalculated based on the patient's weight in kilograms. The maximum lidocaine dose for the patient is 55 mg of lidocaine/kg body weight.¹¹ The chart records have precalculation of maximal dose so the patient will not receive dilute local anesthesia at a lidocaine dose exceeding 55 mg/kg.

Step-by-step technique

Once the patient has been completely anesthetized, the patient rests for 20–30 minutes to allow for the maximum epinephrine effect in order to minimize oozing. Suctioning of the subcutaneous fat is then ready to begin. A variety of electrically powered liposuction aspirators are available in the market, and most machines generate one atmosphere of negative pressure. The suctioning begins by selecting an appropriate cannula. This may be a manual cannula or a powered cannula. Powered cannulas are traditional liposuction cannulas that are connected to a moving hand piece which moves the tip of the cannula in a “to-and-fro” manner.¹² Though not all liposuction surgeons prefer powered cannulas, they usually allow for decreased work on the part of the surgeon and a more time-efficient fat harvest. The subcutaneous abdominal fat removed during liposuction is collected into canisters, which are self-contained disposable structures. The

biological material may be disposed of easily and the amount of aspirated fat may be measured by the graduations on the side. Some surgeons still use specially designed syringes (tulip system) in which a gentle vacuum in the syringe is manually used to withdraw the fat.

In their areas, a variety of surgical cannulas are available. It is advised that inexperienced dermatologic surgeons use a more conservative cannula, which includes smaller blunt-tipped cannulas with side ports. More experienced surgeons may prefer the more aggressive instruments having multiple openings at the tip of the cannula. These so-called more aggressive cannulas are more likely to rupture neurovascular bundles and increase bleeding. For smaller areas or for repeat touch-up procedures, the Klein microcannulas are helpful. These are very small in size and a multiple puncture technique can be used. The microcannulas are placed specifically in focally fatty areas in order to facilitate removal of abdominal fat. These are very helpful in small areas, especially around the periumbilical area. It is important to note that the fat aspirate is remarkably free of blood when the Klein microcannula methods are used.

The cannula is constantly moved in a “to-and-fro” fashion through the subcutaneous space with the dominant hand. The nondominant hand is used to palpate the area of the body undergoing liposuction surgery. This nondominant or “smart hand” can be used in a flat manner in order to stabilize the tissue with the dominant hand guiding the cannula underneath it. Alternatively, the nondominant hand can cup the skin in between it and the cannula used to suction the pocket of subcutaneous fat held in between the two skinfolds. It is important to remember the principles of crisscross cannula movement. A triangular pattern of suctioning occurs, and these fanning patterns are used to overlap so that there are multiple tunnels created from several entrance sites overlapping one another. Deep tunneling is often begun initially, later leading to superficial tunneling. It is important to remember when working in the abdomen to suction deep to Scarpa's fascia, which ensures complete fat removal and an even approach to the removal of the subcutaneous fat.

Complications to avoid

The most common side effect of liposuction surgery is pain and discoloration at the area.⁵ The pain is usually controlled with non-narcotic-based pain medications; however, narcotic-containing pain medications can be used in selective patients. Antibiotics are used to minimize the risk of postoperative infection, and these antibiotics can occasionally lead to cutaneous drug reactions. As with any surgical

procedure, it is important to have open lines of communication between the patient and clinic because during the first 4 days following liposuction surgery, an infection may become manifest. This will usually start as tenderness or pain at a cannulation site; erythema and swelling may be noted. Hypertrophic scars and keloids have been reported forming at the cannula insertion sites and are usually late complications. Hematomas or seromas can occur during the first few weeks during surgery. However, the potential for this is minimized with adequate compression garment use.

Several months postoperation, some dimples or postoperative depression can be seen and this can be treated with fat transfer, although this is rarely necessary. Areas of incomplete suctioning or asymmetry can be noted, and a touch-up procedure can be used to treat these areas.

The periumbilical site is an area that is often undertreated. This is because liposuction surgeons, when operating in large abdomens, often focus on the bigger lower abdominal roll underneath the umbilicus. This can lead to an “umbilical donut” which gives a ring of remaining adipose tissue around the umbilicus, producing a little torus. This responds very well to a touch-up procedure, which is relatively simple to do especially with a small cannula, and has great patient satisfaction. An entry site placed in the superior umbilicus is very helpful in allowing the cannula to travel up the midline helping to create an attractive midline sulcus. Patients have commented that they appreciate this slightly depressed trough.

When liposuction is performed on the abdomen, it is very important to pay attention to both the upper and lower abdominal areas. The lower area is often larger than the upper area and so the patient and liposuction surgeon may feel that the simple liposuction of the lower abdomen will give a satisfactory result. However, this allows the initially less heavy upper abdomen to then “fall down” upon and to produce a horizontal umbilicus. Improvement is best done when suctioning both the lower and upper abdominal areas can be undertaken. These two areas need not be done on the same day but are frequently done as two procedures 1 week apart.

Abdominal liposuction should also be accompanied by liberal feathering into adjacent fat that has not been fully suctioned. The male “spare tire” is a protuberant lower abdomen that moves into two “love handles.” If only the abdomen is treated, an abnormal or strikingly unnatural appearance can result. Men do well by having the flanks treated as well. It is important to remember to feather all around the areas being suctioned to help achieve a more natural and blended contour.

■ Postoperative Care and Instructions

At the end of the initial liposuction, the surgeon assesses the areas in which subcutaneous abdominal fat has been removed. Common methods of doing so are the pinch tests in which the skin is pinched between the fingers to assess the amount of subcutaneous fat left behind. Other techniques include a rolling procedure in which the skin is rolled back and forth using a pinch technique, which allows the surgeon to locate any individual pockets of adipose excess. If these individual pockets or uneven areas are noted, it is helpful to resuction these areas to attempt to remove the last amount of remaining fat and to achieve symmetry.

Once the liposuction procedure is complete, nurses help in compressing the areas suctioned to facilitate drainage. Occasionally, dependent 4 mm rents are placed in the skin to facilitate drainage. Absorbent pads are put on the insertion site areas. Compression garments are helpful in facilitating drainage of residual dilute anesthesia. A compression garment adds pressure, which compresses the skin and helps avoid seroma and hematoma formation. The pressure or tautness of the garment also helps diminish some of the discomfort over the following few days postprocedure. The patient wears the compression garment for 2 weeks minimum, with many patients preferring to wear it a few weeks longer as it offers comfort and support. The garments are often crotchless, which allows patients to tend to elimination needs while wearing them.

During the postoperative period, careful explanation with written documentation of postoperative wound care and the use of compression garments is very important.⁹ The patients are seen back in the office after 1 week, but are contacted by the staff the immediate few days following the surgery. It is important to remember to remind the patient that the final outcome will not be obtained for 6–12 months following the procedure (Figure 7.4). Review with the patient that the postoperative inflammatory process can often result in induration and firmness of the tissue in those areas having had liposuction. It is important to remind the patient that this will improve with time. Any hematomas or seromas which are seen in the treated areas often can resolve on their own; however, the surgeon may wish to drain these. Infection can follow any surgical procedure including liposuction surgery, and it is important that the surgeon remind the patient of the need to take postoperative antibiotics for the full length of their prescribed course.



Figure 7.4 Before and 1 year after abdominal liposuction in a 56-year-old female

■ Fat Transplantation

Larger subcutaneous soft tissue defects such as areas of adipocyte loss or soft tissue damage can often be improved with fat transplantation (Figure 7.5).

Patient positioning

Patient positioning is often the same as that used for tumescent liposuction. The room may contain or be in close proximity to a centrifuge, if one is used.

Anesthesia

This technique involves transplanting autologous adipocytes and using them as a filler substance.¹³ A donor area of adipose tissue is identified and tumesced using standard tumescent anesthetic consisting of 0.05% lidocaine with 1:1 000 000 epinephrine in normal saline. This small area is often tumesced with a large syringe instead of the mechanical pump.

Step-by-step technique

After waiting 15 minutes for the maximal epinephrine vasoconstrictive effect, a liposuction aspiration cannula is connected to a 10–30 mL syringe equipped with a Luer-Loc. By gently drawing the plunger backward and pro-

ducing only mild vacuum, “donor” fat can be aspirated through the small cutaneous incision. By the use of gravity or through centrifugation, the harvested pearls of fat are separated from the extra tissue and liquefied fat. This donor fat can then be implanted into the subcutaneous abdominal area using either a 16- or 18-gauge needle or a blunt tipped-fat implantation cannula equipped with a side port. It is important to note that the transplanted fat is not used as a dermal filler, but rather as a subcutaneous replacement of areas of fat atrophy. It is felt that between 30% and 40% of the graft is retained at 1 year, and multiple fat transfers to the same area can fill out areas of fat atrophy with long-lasting results.

SURGICAL WEIGHT LOSS

■ Patient Selection

Although severely overweight patients are not candidates for liposuction, these patients do have options for improvement. First, they should be advised of the benefits of nutritional counseling. Personal trainers are widely available in health clubs and are a good source to assist

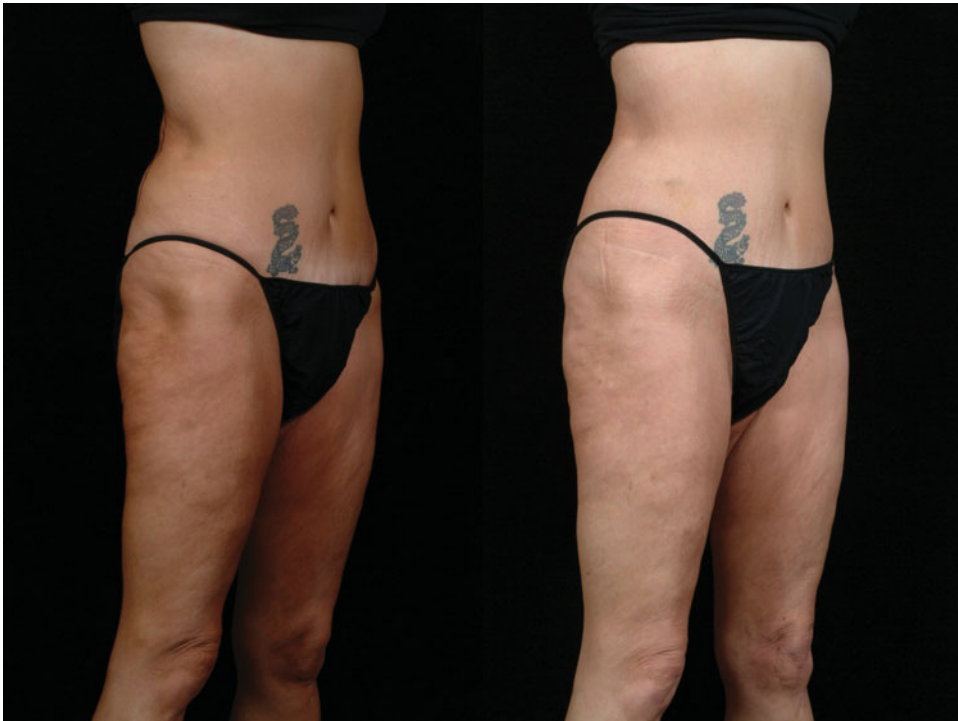


Figure 7.5 Patient with a lateral hip depression following liposuction. The area was corrected with one session of autologous fat transfer

the patient in beginning a custom-tailored exercise program. However, the reality is that many obese patients have failed diet and exercise programs and they may be helped with surgical approaches to obesity, such as a laparoscopic adjustable gastric band, or a gastric bypass.

■ Technique

The laparoscopic band is the least invasive operative bariatric surgery, being placed laparoscopically with only modest discomfort.¹⁴ Patients usually return to normal activities quickly in a few days. The band reduces the functional size of the gastric pouch allowing less food to be consumed.¹⁵ The stoma size is adjustable without reoperation, as the adjustments are made using injection or withdrawal of saline solution into a subcutaneous access port. The band is easily reversible and removable. The procedure is safer than the traditional Roux-en-Y gastric bypass. A systematic literature review in 2004 found that the gastric bands had an early mortality of 0.05%, one tenth that of gastric bypass. The operation is highly effective with patients losing 50% to 60% of their excess weight over a 2 to 3 year time period.¹⁴ Lap band placement and maintenance reduces the comorbidities of obesity as reported by the 2004 American Society for Bariatric Surgery Consensus Conference.¹⁴ In patients with type II diabetes mellitus and hypertension, 92% of the cases were resolved or improved. Cases of hypertension, sleep apnea, and obesity-related joint disorders were resolved or improved in 78%, 95%, and 83% respectively, as reported by this consensus.

In gastric bypass surgery, intestines are rerouted making the stomach appear smaller and allowing food to

bypass a portion of the small intestine. A small pouch is made by the bariatric surgeon at the top of the stomach, which can hold only a limited amount of food. The smaller pouch is connected to the jejunum, bypassing the duodenum. The procedure can also now be done laparoscopically. Gastric bypass can greatly assist obese patients with a body mass index above 40 or higher in losing weight. Patients usually lose weight for approximately 12 months and then stabilize. There are significant risks to gastric bypass surgery including dumping syndrome, gallstones, anemia, B₁₂ deficiency, ulcers, and stomal stenosis.

ABDOMINOPLASTY

■ Patient Selection

Liposuction using the tumescent technique has benefitted thousands of patients by reducing small areas of excess fat in the abdomen. However, some patients may need traditional scalpel surgery, particularly those patients with redundant skin or those patients who have lost a significant amount of weight following bariatric surgery.¹⁶

■ Technique

Abdominoplasty is traditionally performed with a hip-to-hip lower abdominal skin transverse incision. The incision is long and often shaped like a bicycle handle. The excess skin and fat is dissected superiorly, redraped, and removed. The umbilicus is reset. The abdominoplasty may be combined with liposuction, or liposuction may be beneficial as a refinement postabdominoplasty (Figure 7.6),

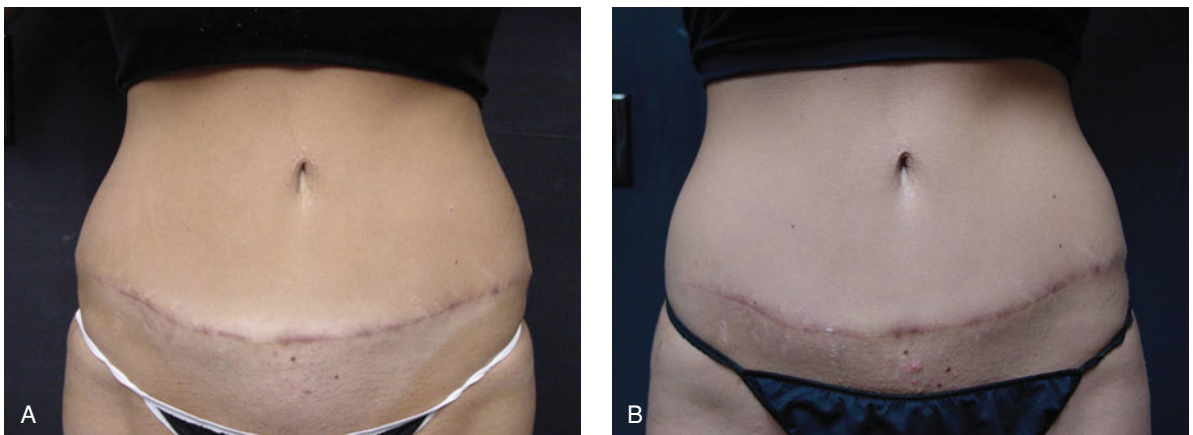


Figure 7.6 Abdominoplasty and liposuction

once the abdominoplasty surgeon gives clearance and full healing has occurred.

Complications to avoid

Patients should be aware that systemic complications from abdominoplasty are more frequent than with other aesthetic procedures. Referral of the patient to an experienced plastic surgeon should be considered.

LASERS AND RADIOFREQUENCY DEVICES

■ Abdominal Rejuvenation Through Lasers or Radiofrequency Devices

The goal of noninvasive skin tightening devices is skin tightening without surgery and its risks of complications and often lengthy recovery times. Noninvasive options are best suited for patients with not more than mild-to-moderate skin laxity without underlying structural ptosis. Patients with extreme skin redundancy will achieve only restricted contour improvement and are best referred for surgery.¹⁷

Noninvasive skin tightening devices, which currently include several different wavelengths and technologies, are believed to affect skin in two distinct ways. Immediately after treatment, with the rapid contraction of collagen fibers, there is a direct effect of skin tightening. Over time, with the creation of mild dermal injury, collagen production is stimulated which yields more dynamic dermal remodeling. The mechanism of this remodeling is the contraction of collagen fibrils, which, when heated to a specific temperature, yields tissue tightening via a wound response and the production of new dermal ground substance.¹⁸ Great care is needed with dosing as an excess of deposited heat can yield the breakage of intramolecular hydrogen bonds with complete denaturation of collagen fibrils, widespread cell death, and a significant risk of scarring.^{18–20}

Energy devices that may noninvasively tighten abdominal skin include the ThermoCool™, a Thermage™ (Hayward, CA) instrument, which uses monopolar radio frequency (MRF), the Cutera Titan™ (Brisbane, CA), which uses infrared (IR) light, and the Palomar Lux-IR™ (Burlington, MA), which uses fractional IR light.

In general, these alternative treatments deliver a substantially more modest tightening in comparison with surgery, as the skin contraction achieved with the latter is

approximately 1–3 mm. Noninvasive devices may be also be preferred over ablative treatments when there is no need for superficial skin peeling- as in the absence of acne damage, pigment problems, or texture abnormalities.²¹

■ MRF (Monopolar Radiofrequency)

Unlike laser technologies, in which light targets tissues using particular wavelengths absorbed by a specific target chromophore under the principle of selective photothermolysis, MRF heats the skin using a thin capacitive membrane which delivers MRF energy over a volume of tissue under its surface.¹⁷ The ThermoCool generates an electric field between two electrodes, and an AC current flows out of the MRF generator. The current travels through the treatment tip, into the skin through the body, and is collected with a return pad. In the skin, polar molecules move back and forth vibrating at 6 million times a second. Resistance to this movement creates heat within the tissue while a cryogen cooling system simultaneously protects the epidermis.^{17,21}

Unlike wavelength dependent laser dosing, the ThermoCool's heating depth depends on the tip used with the 3.00 cm² tip best suited for the abdominal area.¹⁷ The larger membrane allows the device to heat a large volume of skin, as in the case of the abdomen, as opposed to heating a specific point, a desirable option for more focal treatment sites. The ThermoCool appears to have two main effects. Immediately, it causes existing collagen fibrils to contract. Over a more prolonged response time it yields a delayed wound healing response, which is widely believed to cause neocollagenesis by stimulated fibroblasts.²¹ Tightening continues for 4–12 months after the procedure.¹⁷ Notably, the non-wavelength-based ThermoCool, with its attendant epidermal protections, is more safely used in treating all skin phototypes, unlike most laser systems, which run the risk of dyschromias with darker Fitzpatrick skin types and tanned skin.²¹ Common side effects of the ThermoCool include transient erythema, mild edema lasting 1–2 days, small superficial burns, and temporary skin numbness. Rare, more serious side effects include persistent edema lasting over a week¹⁷ and delayed contour irregularities which are generally attributed to overheating.²²

In 2004, Zelickson et al. examined the effects of the ThermoCool on the abdominal skin of two preabdominoplasty females. The first patient was treated with 95 J

using a 1-cm² tip for 2.1s. The second patient was involved in a dose–response study and was treated with energies of 104, 133, and 181 J with a 1-cm² tip for a duration of 2.3 s. Pre-, parallel-, and postcooling was applied. In this small pilot study, the authors observed increased fibril diameter and a loss of distinct fibril edges in biopsy samples from both patients. However, the dose–response patient exhibited greater changes, which repaired slowly over 8 weeks. The 181 J energy level caused significantly more damage to collagen fibrils than the 104 and 133 J levels, and the treatment effects were most apparent at a depth of 3–4 mm. Mild perivascular and perifollicular inflammation occurred 45 minutes after treatment with 104 and 181 J energy levels. However, this inflammatory response was not found at 8 weeks posttreatment.¹⁸

In both cases, collagen mRNA expression was elevated in treated skin immediately and 1 week after treatment. However, at 3 and 8 weeks after treatment, treated skin mRNA levels were just below control mRNA levels. Overall, while neither patient showed evidence of collagen contraction, the treatment did seem to cause thermal injury, which appeared to stimulate a wound-healing response, and extracellular matrix remodeling, as evidenced by elevated collagen mRNA expression.¹⁸

In 2007, Suh et al. reported on the effects of an MRF device on the abdomens of 37 patients with striae distensae.⁴ Striae duration ranged from 6 months to 30 years (mean 9.5 years). Thirty-six patients had striae alba, and one patient displayed striae rubra. Subjects (skin types III–VI) received combined Thermage and pulsed dye laser (PDL) as their first treatment, while PDL was used alone in the next two treatments, at 4 and 8 weeks. The ThermoCool operated at fluences between 53 and 97 J/cm² using a standard tip for 2–3 passes. The 585 nm PDL operated at a fluence of 3 J/cm², with a pulse duration of 0.5 ms and a 10 mm spot size. Elasticity improvements were graded as *very good* (76%–100%), *good* (51%–75%), *moderate* (26%–50%), *mild* (1%–25%), and *no improvement* (0%).

After treatment, 22/37 (59.1%) patients showed good or very good improvement in elasticity, while 12/37 (32.4%) patients had moderate improvement and 3/37 (8.1%) patients had mild improvement. Posttreatment, nine biopsy samples were taken. All nine specimens showed increased collagenesis, but only six had increased elastic fibers. Overall, this combined therapy seemed to both increase elasticity and promote neocollagenesis.⁴

■ Infrared Light

The Titan is a noncoherent, selectively filtered device that emits IR light in multisecond cycles for dermal heating. Wavelengths are set between 1100 and 1800 nm and the Titan's target chromophore is water, which absorbs the device's light energy to heat skin. The device penetrates 1–2 mm into the skin which represents an ideal depth for targeting the reticular dermis. The Titan uses multisecond cycles of up to 9.5 s and employs spot sizes of 1 × 1.5 cm and 1 × 3.0 cm. The epidermis is protected through contact cooling.²² As melanin absorbs wavelengths much lower than 1100–1800 nm, it is not likely to compete with water for light energy, and so the Titan device may be suitable for all skin types. Like the ThermoCool, the Titan claims to cause immediate skin contraction.²³

Zelickson et al. tested the efficacy of the Titan IR device on abdominal skin in 2006. One patient was treated with the Titan handpiece in the 1100–1800 nm range, at fluences of 30, 45, and 65 J/cm² with a 1 × 1.5 cm spot size. Each treated area received four passes, with 3–6 s exposure durations. Pre-, parallel-, and postcooling was applied. Collagen fibril changes were observed at all treatment levels at depths from 0–1 mm to 1–2 mm. Fibril contractions became more pronounced with increased fluence and depth, and partial collagen denaturation occurred in a dose-dependent manner, as more injury was found in deeper tissue where higher fluences were delivered. Maximal damage consistently occurred at the 1–2 mm depth. Collagen fibril changes were not found in control sites.²⁴

Tendon studies confirm the relationship between the extent of collagen denaturation and the contraction and tensile strength of the tendon. However, while researchers did observe collagen fibrils contracting, they only observed the immediate effects of the Titan device on abdominal skin. Because Zelickson et al. did not observe the long-term effects of the instrument on the patient; the immediate results may not have had any correlation with the long-term results.²⁴ Side effects of the Titan device include mild, temporary edema and erythema, and small superficial burns. Improper use or cooling can lead to more serious third-degree burns.¹⁷

■ Fractional IR

The Lux-IR is a near-IR lamp device that creates multiple, spatially confined thermal lesions in the dermis and hypodermis while avoiding epidermal damage. The

instrument uses a near-IR halogen lamp with an optically filtered emission spectrum ranging from 850 to 1350 nm. Its pulse duration lasts up to 10 s and can deliver a fluence of up to 200 J/cm². The device uses a spot size of 12 × 28 mm.¹⁷

Palomar reports that the Lux-IR operates like an IR device but has a higher safety margin and faster healing times because of the fractional delivery system. Because the Lux-IR has higher irradiance at the islets instead of uniform illumination, it can deliver a stronger effect at the islets without raising the output power. Additionally, fractional delivery of the IR light is reported to allow a greater ratio of the maximally acceptable fluence (with regard to patient tolerance and side effect profile) to the minimally effective fluence, ostensibly providing greater room for error and thus more safety in skin tightening procedures. Finally, the manufacturers claim faster recovery times through a higher surface-to-volume ratio of the microwounds. Limited side effects are reported with the Lux-IR; most commonly superficial burns if cooling is inadequate. A pilot study from Dierickx with the Lux-IR indicates promising results for abdominal skin tightening, but larger scale controlled clinical trials of the device are needed to assess its efficacy.¹⁷

While some of the preliminary data does show some promise for the role of nonablative skin tightening in the abdominal armamentarium, more studies are needed before the absolute efficacy of noninvasive abdominal skin tightening can truly be determined. A key limitation of all aforementioned studies were their extremely small sample sizes. Suggestions for future studies include controlled clinical trials of the Lux-IR's effects on abdominal skin as well as studies comparing the assorted modalities.

REFERENCES

- Centers for Disease Control and Prevention Website 2007. Downloadable from: <http://www.cdc.gov/nccdphp/dnpa/obesity/trend/>.
- Flynn TC, Coleman WP, IV. Body Rejuvenation. In: Parish LC, Ramos-e-Silva M, eds. *The Dermatology of Girls and Women*. WP Saunders; 2000.
- Montesi G, Calvieri S, Balzani A, Gold MH. Bipolar radiofrequency in the treatment of dermatologic imperfections: clinicopathological and immunohistochemical aspects. *J Drugs Dermatol*. 2007;6(9):890-896.
- Suh DH, Chang KY, Son HC, Ryu JH, Lee SJ, Song KY. Radiofrequency and 585-nm pulsed dye laser treatment of striae distensae: a report of 37 Asian patients. *Dermatol Surg*. 2007;33(1):29-34.
- Clark RE, Flynn TC. Liposuction of the torso, back, and abdomen. In: Hanke CW, Sattler G, eds. *Procedures in Cosmetic Dermatology*. Liposuction: China, Elsevier, Inc.; 2005:79-92.
- Flynn TC, Narins RS. Preoperative evaluation of the liposuction patient. *Dermatol Surg*. 1999;17:729-734.
- Johnson DS, Lillis PJ, Kaminer MS. Liposuction. In: Saminer MS, Dover JS, Arndt KA, eds. *Atlas of Cosmetic Surgery*. Philadelphia, PA: WB Saunders; 2002:194-230.
- Butterwick KJ. Liposuction: consultation and preoperative considerations. In: Narins RS, ed. *Safe Liposuction and Fat Transfer*. New York: Marcel Dekker; 2003:41-67.
- Klein JA. *Tumescent technique: Tumescent Anesthesia & Microcannular Liposuction*. Mosby: St. Louis; 2000.
- Klein JA. Anesthesia formulation of tumescent solutions. *Dermatol Clin*. 1999;17:751-759.
- Ostad A, Kazeyama N, Moy RL. Tumescent anesthesia with a lidocaine dose of 55 mg/kg is safe for liposuction. *Dermatol Surg*. 1996;22:921-927.
- Flynn TC. Powered liposuction: an evaluation of currently available instrumentation. *Dermatol Surg*. 2002;28:376-382.
- Coleman SR. Long-term survival of fat transplants: controlled demonstrations. *Aesthetic Plast Surg*. 1995;19:421-425.
- Ponce J, Dixon JB. Laparoscopic adjustable gastric banding. Surg for Obesity and Rel Diseases. 2004 ASBS Consensus Conference. 2005;1:310-316.
- Spivak H, Hewitt MF, Onn A, Half E. Weight loss and improvement of obesity-related illness in 500 US patients following laparoscopic adjustable gastric banding procedure [rapid communication]. *Am J Surg*. 2005;189:27-32.
- Matarasso A, Matarasso SL. When does your liposuction patient require an abdominoplasty? *Dermatol Surg*. 1997;23:1151-1160.
- Dierickx CC. The role of deep heating for noninvasive skin rejuvenation. *Lasers Surg Med*. 2006;38:799.
- Zelickson BD, Kist D, Bernstein E, et al. Histological and ultrastructural evaluation of the effects of a radiofrequency-based nonablative dermal

- remodeling device: a pilot study. *Arch Dermatol*. 2004;140:204.
19. Kirsch KM, Zelickson BD, Zachary CB, et al. Ultrastructure of collagen thermally denatured by microsecond domain pulsed carbon dioxide laser. *Arch Dermatol*. 1998;134:1255.
 20. Drew PJ, Watkins A, McGregor AD, et al. The effects of temperature and time on thermal bond strength in tendons. *Lasers Med Sci*. 2001;16:291.
 21. Koch RJ. Radiofrequency nonablative tissue tightening. *Facial Plast Surg Clin North Am*. 2004;12:339.
 22. Narins RS, Tope WD, Pope K, et al. Overtreatment effects associated with a radiofrequency tissue-tightening device: rare, preventable, and correctable with subcision and autologous fat transfer. *Dermatol Surg*. 2006;32:115.
 23. Ruiz-Esparza J. Near [corrected] painless, nonablative, immediate skin contraction induced by low-fluence irradiation with new infrared device: a report of 25 patients. *Dermatol Surg*. 2006;32:601.
 24. Zelickson B, Ross V, Kist D, et al. Ultrastructural effects of an infrared handpiece on forehead and abdominal skin. *Dermatol Surg*. 2006;32:897.

CHAPTER 8

Arm and Hand Rejuvenation

Neil Sadick

KEY POINTS

- Understand components of the aging hand.
- Recognition of anatomic landmarks (muscles, veins, nerves).
- Adequate photoprotection.
- At-home rejuvenation program.
- Matching appropriate technology to patient's concerns.
- Recognition of the increased bruising tendencies of hands and arms.
- Recognition of the prolonged wound healing capability of the hands vs. the face.
- Appropriate initial consultation and understanding patient's concerns.
- Recognition and appropriate management of complications.

BACKGROUND

In the ever-expanding baby-boomer population and as life expectancy increases, the demand for aesthetic procedures continues to grow exponentially. Improvement in facial rejuvenation technologies has allowed the aesthetic physician to produce reproducible results associated with great patient satisfaction.

However, in this regard, many individuals have been able to achieve excellent facial aesthetic results but continue to show signs of photoaging on other parts of the body including the neck, chest, hands, and arms.

Whole body rejuvenation remains the next great frontier of aesthetic technology development in individuals who do not want to have youthful appearing faces while other parts of the body continue to portray an aged appearance. In this setting, the present chapter on arm and hand rejuvenation is presented.

■ Components of the Aging Hand/Arm

Understanding a structural approach to the aging hand will enable the practicing aesthetic physician to choose

the appropriate clinical interventions based upon both patient's as well as physician's concerns. Clinical manifestations of the aging hand based upon pathophysiologic correlates are presented in Table 8.1.

Type I rejuvenation involves epidermal structures and presents clinically as pigment dyschromias, which may include discrete lentigines and ephelides, as well as diffuse inflammatory hyperpigmentation. In addition, epidermal contour irregularities and proliferation turnover aberrations may present as skin roughness.

Type II hand/arm aging involves dermal architectural changes involving collagen, elastin, and glycosamino glycan. Its major clinical correlates are rhytid expression.

Type III rejuvenation involves deeper structures including subcutaneous tissues, muscle, and bone. The major clinical correlates of hand aging involve skin laxity, volume loss, and the presence of protuberant hand veins.

ANATOMIC LANDMARKS

- Dorsal branch of radial nerve
- Dorsal branch of ulnar nerve
- Extensor pollicis longus tendon
- Extensor digitorum communis
- Extensor digiti minimi
- Superficial veins (dorsal arch veins)
- Metacarpal bones
- Dorsal interossei muscles
- Extensor indicis proprius

TABLE 8.1 ■ Components of the Aging Hand

Type I epidermal
Hyper-hypopigmentation
Roughness
Type II dermal
Rhytides
Type III subcutaneous/muscle/bone
Laxity
Volume loss/lipoatrophy
Protuberant veins

TABLE 8.2 ■ Treatment Algorithm for the Aging Hands and Arms**Type I****Pigmentation****Hyperpigmentation**

Hydroquinone 3%–4%, azelaic acid, kojic acid
 Q-switched laser (532 nm/1064 nm Nd:YAG
 695 nm ruby 755 nm alexandrite)
 Intense-pulsed light (500–1200 nm)
 Fractional laser resurfacing

Hypopigmentation

Excimer laser (308 nm)
 Targeted phototherapy (295–315 nm UVB,
 360–370 nm UVA1)

Roughness

Very superficial chemical peels (Lactic acid
 6%–12%, TCA 20%, 20%–50% glycolic acid)
 Fractional laser resurfacing

Type II**Rhytides**

Retinoids, glycolic acids 10%–40%
 Superficial chemical peels 50–70 glycolic acid
 Near infrared, infrared lasers 1064 nm, 1319 nm,
 1320 nm, 1450 nm, 1540 nm, diode laser 810
 nm/RF
 Low-energy, long-pulsed erbium laser micropeel
 1319 nm

Type III**Laxity**

High-energy radiofrequency technologies

Volume loss

Hyaluronic acid derivatives
 Poly-L-lactic acid derivatives
 Autologous fat

Veins

Endovenous hand laser (1320 nm)
 Sclerotherapy/foam sclerotherapy

PATIENT SELECTION

Patient selection is usually based upon those individuals with more severely photoaged skin as outlined in “Components of the Aging Hand/Arm.” Commonly, patients will have had procedures that have improved the aspects of the aging face and will be left with manifestations of the photoaged hands and neck, which is why recognition of the concept of whole body rejuvenation is so important.

PREOPERATIVE EVALUATION

Preoperative evaluation should include a thorough medication listing as well as bruising history because the thin-skinned hand is more susceptible to bruising phenomena. This includes discontinuance of aspirin, nonsteroidal anti-inflammatory agents, as well as Plavix (Sanofi-Aventis, Bridgewater, NJ) for 3 to 5 hours prior to a given procedure after discussions with the patient’s internist. In addition, arnica and bromelain supplements 1 to 2 days before and after hand/arm rejuvenation procedures may help to limit bruising.

When evaluating the hand under direct illumination, both physician as well as patient should decide on cosmetic concerns as well as a targeted treatment plan based upon these agreed upon concerns. Most patients require a combination of procedures, as they have multiple aesthetic issues (see Table 8.4). Thus, an orderly treatment plan should be devised in this setting.

TECHNIQUES**■ Type I Photoaging****Pigmentation abnormalities**

Most issues noted in this setting include either discrete pigment lesions, such as ephelides or lentigos, or patchy

TABLE 8.3 ■ Available Fractional Resurfacing Technologies

Company Name	Laser	Wavelength	Location
Reliant	Fraxel	1550	Mountain View, CA
Lumenis	ActiveFX	10600	Santa Clara, CA
Sciton	Profractional laser for profile	2940	Palo Alto, CA
Cynosure	Affirm	1320/1440	Westford, MA
Palomar	Starlux LuxLR	850–1350	Burlington, MA

zones of inflammatory vs. hypopigmentation. In addition, diffuse guttate hypopigmentation (idiopathic guttate hypomelanosis) may also commonly present on the hands and arms.

All hyperpigmentation treatment programs should include the use of a daily sunscreen (broad spectrum UVB/UVA coverage), as well as another at-home depigmentary regimen in order to prevent recurrence of pigmentation as well as to act synergistically with the in-office technology programs which will be discussed subsequently. Hydroquinone 3% to 4% applied nightly remains the gold standard, although products containing azelaic acid as well as kojic acid may be helpful in this setting.

For discrete pigmentary lesions, the Q-switched lasers and intense pulsed light sources remain the treatments of choice.

All of the green light sources produce comparable results in removing lentigines and ephelides. Ruby (694 nm), alexandrite (755 nm), and Nd:YAG (532 nm/1064 nm) options are most commonly employed. These tech-

nologies act by blasting melanosomes in these discrete lesions. Usually, a single treatment session is required. Multiple lesions tissue may be addressed simultaneously. In this technique, crusting or micropurpuric zones lasting 7 to 10 days may ensue. However, these treatments are relatively painless and are associated with great patient satisfaction. (Figure 8.1). In this setting as well, intense pulsed light sources with or without lower energy radiofrequency component may also be used. However, in our experience, the results after a single treatment session are not as predictable and thus the patient should be explained that multiple treatment sessions may be necessary if this approach is employed. The endpoint of therapy with this modality is a fine microcrusting or bronzing of the treated areas. A similar posttreatment course as noted with Q-switch lasers usually subsequently ensues.

We recommend using a bland ointment applied three to four times a day, such as Aquaphor Healing Ointment (Beiersdorf Inc., Wilton, CT), until the crusts are totally resolved.



Figure 8.1 Pre-, postQ-switched Nd:YAG laser 532 nm/1064 nm treatment of discrete hand lentigines 1.4 J/cm² fluence, 3-mm spot, single treatment session



Figure 8.2 Pre-, postfractional thermolysis (Fraxel, Reliant Technologies, Mountain View, CA) fluence 12 mJ, total density 2,000 MTZ/cm², five treatments, 3-month follow-up

Diffuse hyperpigmentation may be addressed by fractional resurfacing technologies. Although the Fraxel (Reliant Technologies, Mountain View, CA) is the pioneer of this technology, many laser companies have similar systems which may accomplish this goal.

At the time of publication the author is presently completing a study utilizing this approach for hand rejuvenation and has had excellent results. In this setting, patients were followed for up to 6 months, five to six treatment sessions were performed at 3-week intervals. Initial treatments were performed at 6 mJ, with an increase of 2 mJ at each treatment session as tolerated by patients. A total of 2000 micothermal zones (MTZ/cm²) were used on average. Technology works by deeply penetrating columns of light-energy with separate noninvolved zones, which allows for rapid healing. It is hypothesized that these columns diminish and disrupt melanin activity at the dermal-epidermal junction. This is a nonablative technology, although patients may have a sunburn-like reaction manifested by redness and branny scaling which may last for up to 2 to 3 days. In addition, the results of these treatments were also shown to improve wrinkling

and give patients the sensation of a smoother cutaneous surface.

Hypopigmentation is treated by lasers and light sources, which stimulate melanogenesis. The excimer laser (308 nm XTRAC, PhotoMedex, Montgomeryville, PA) and targeted combination UVB/UVA light sources (295–315 nm, UVB 360–370 UVA, MultiClear, Curelight, Gladstone, NJ) have been most helpful in this setting. Multiple treatments (up to 15–20) are necessary in this regard and may give more favorable results compared to the utilization of these technologies in treating facial hypopigmentation.

Roughness

Skin textural abnormalities is commonly part of the aging hand concern profile. This may be due to a combination of diminished water content associated with the aging process, photodamage associated with preskin cancers (actinic keratoses), and irregular epidermal kinetics. Very superficial chemical peeling agents are employed in this regard. Six to twelve percent lactic acid applications may be used nightly by the patient at home, as well as 20% to 40% TCA or 20% to 50% glycolic acid preparations may

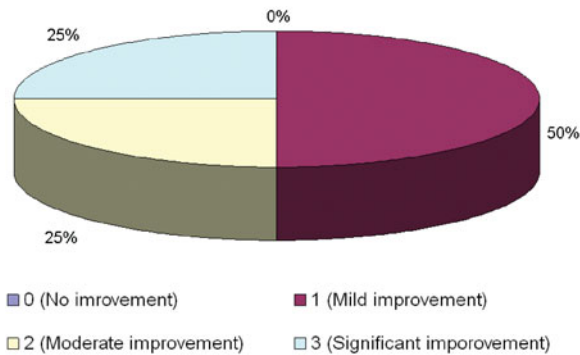


Figure 8.3 The utilization of a 1320 nm Nd:YAG laser for hand rejuvenation, in a recent study, is associated with positive reduction of rhytides and a relatively high-degree of patient satisfaction

be employed monthly or every 2 to 3 months in the physician's office to improve textural aberrations.

Finally, fractional laser resurfacing, as discussed in the previous section, also improves skin textural characteristics.

■ Type II Photoaging

Rhytides

Wrinkle formation is one of the major clinical manifestations of hand photoaging. It may be addressed by multiple product and technology treatments. Application of nightly

retinoids or glycolic acids lotions 10% to 40% in conjunction with in-office application of superficial chemical peels employing 50% to 70% glycolic acid at 1- to 3-month intervals may induce epidermal turnover as well as stimulate dermal collagen as part of a remodeling process collagen synthesis. In addition, near infrared and infrared laser technologies may also induce dermal remodeling and similarly lead to improvement in the collagen/elastin, matrix, and glycosamino glycans. Lasers with wavelengths of 1319, 1320, 1450, and 1540 nm as well as combination diode laser radiofrequency sources have all been shown to accomplish this goal.

In a recently completed study, the authors used the 1320 nm Nd:YAG laser (Cool Touch Inc, Mountain View, CA) and found significant improvement in hand wrinkling of average 50% after 6 monthly treatment sessions in the majority of individuals. This was associated with a high-degree of patient satisfaction as well (Figure 8.3). In this setting, two passes were employed with the first pass at 14–18 J/cm². Pre- and postcooling were employed. (Figure 8.4).

In addition, as previously stated, fractional resurfacing may improve fine wrinkling in this setting. Finally, low-energy laser micropeeling may be used to improve wrinkling and hyperpigmentation on both hands and arms. We prefer the long-pulsed erbium (1319 nm Nd:YAG) laser (Sciton, Palo Alto, CA). Usually a series of three treatment sessions spaced at 6- to 8-week intervals are performed. Increasing depths of ablation beginning at 20 μm and progressing to 40 μm may be used in this regard. In addition,



Figure 8.4 Pre-, post 6 monthly treatment with the 1320 Nd:YAG laser is associated with a reduction in rhytides, fluence 13–17 J/cm²

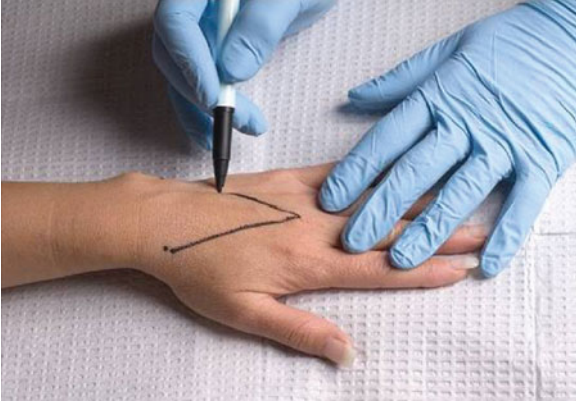


Figure 8.5 *The ThermoCool hand technology involves marking a dorsal angle with the apex at the head of the third metacarpal (first closed) and vectors extending to the base of the first and fifth metacarpal. Parallel vectors are then drawn at 1-cm intervals radially and ulnarly*

elevated epidermal keratoses may be simultaneously treated in the coagulation mode. The posttreatment course consists of erythema and microcrusting, similar to fractional resurfacing and may last for a period of 3 to 5 days. Again, bland emollient ointments may be applied three to four times per day in order to induce rapid re-epithelialization. Minimal complication profiles have been noted employing the previously outlined technologic approaches.

■ Type III Photoaging

Laxity

Skin laxity may be addressed by deep volumetric heating. High-energy unipolaris technologies such as the ThermoCool (Thermage, Heywood, CA) may be helpful in this regard. Usually a single treatment session is indicated. The more superficial 1.5-cm tip and the use of relatively low-energy levels makes this treatment relatively painless and with a low-complication profile compared to high-fluency, large tip, and deeper penetrating programs as previously described. Topical anesthetics employed (EMLA, EL-MAX, or Tricaine, Hopewell Pharmaceuticals, Hopewell, NJ) are the only preoperative pain reduction programs required. These topical anesthetics may be applied for 30 minutes under occlusion prior to performing this procedure.

The technique involves a vector-related approach. The physician should mark a dorsal angle with apex at the head of the third metacarpal with the first closed and vectors extending to the base of the first and fifth metacarpals. Then the user should draw parallel vectors at 1-cm intervals radially and ulnarly (Figure 8.5). A grid is subsequently applied to the hand in order to ensure uniformity of pulse application. The procedure involves following this combination grid/vector approach (Figure 8.6). The settings utilizing this approach are utilization of the 1.5 cm ST tip with 600 reps/300 reps/hand). From three to six passes may be employed following this vector approach.



Figure 8.6 *ThermoCool hand technique with a grid application ensures uniformity of energy application to improve skin laxity*

■ Volume Loss

Volume loss may be addressed with both autologous as well as biodegradable filler products.

Lipotransfer

Autologous fat remains the gold standard in this setting. The technique involves aspiration of fat from a donor site, usually the lateral thighs or abdomen. Tumescent anesthesia with 0.5% lidocaine may be used to numb the donor area.

Tumescent solution is injected into the area to be harvested (inner knee, thigh, lower abdomen) by a tumescent wand and possibly a Klein pump or by hand. After waiting for 5 minutes and massaging area, fat is harvested into a 60-cc syringe (or 30 cc syringe) by a harvesting cannula. Fat is then put into 6 to 5-cc syringes with Coleman sterile Luer-lock caps at the end and is centrifuged to take off excess serum and blood.

Once the fat is spun down, it is injected into the affected areas through small incisions or 18-gauge needle holes with a micro cannula (1 mm). An average of 5 to 6 ccs of fat is implanted per hand in a given treatment session. These volume replacements may last from 1 to 3 years on an average. The advantage of using this approach is that this material is autologous, natural, and nonantigenic. The disadvantage of this approach is that it requires donor harvesting and a more invasive surgical procedure associated with its inherent morbidity and infection.

Hyaluronic acid derivatives may also be used to replace lost hand volume. This is an easy technique. Both Restylane (Medicus, Phoenix, AZ) as well as Juvederm (Allergan, Irvine, CA) may be employed. Topical anesthesia with EMLA, ELA-Max, or Tricaine may be used in this setting left on 30 to 40 minutes with occlusion. Usually one to two syringes of hyaluronic acid are implanted to both hands, depending on the degree of volume repletion indicated. Hyaluronic acid derivatives usually last from 4 to 6 months. A deep dermal injection placement is indicated. Superficial placement may lead to a bluish discoloration on the skin surface because of the “Tyndall effect.”

Poly-L-lactic acid

We have taken a particular interest in the use of poly-L-lactic acid (Sculptra, Bridgewater, NJ) for volume hand rejuvenation because of its versatility, its ease of implantation, and longevity in this setting. Usually one vial is instilled per hand. Reconstitution of the product is usu-

ally done the night before the procedure so more dilute preparation is used. Reconstitution is performed with 6-cc sterile water and 2-cc 1% lidocaine. Topical anesthesia is recommended as above. With all fillers in the hand, which has thin skin, no aspirin, Motrin, or platelet inhibitors are taken 24–48 hours prior to treatment in order to minimize bruising. We use 26-gauge 0.5-inch needles and 1-cc syringes.

A tenting interosseous approach is employed with injection through the interosseous space of the elevation, and the involved areas are treated (Figures 8.7 and 8.8). We prefer a linear threading technique in order to minimize the number of needle punctures employed. Usually five 1-cc syringes are used per side. Molding and hand massage of the material is carried out in order to prevent nodule formation. The injection plane should be at the deep dermal subcutaneous junction. Two to three treatment sessions are usually carried out at 4-week intervals. The expected duration of improvement in this setting is 1 to 2 years.

Bruising manifested by ecchymoses is the most common side effect noted with poly-L-lactic acid as well as autologous fat implantation. Inappropriate or superficial implantation of material may lead to palpable or visible “bumps” on the skin. With poly-L-lactic acid, true granulomatous hypersensitivity reactions are extremely rare.

■ Veins

Protuberant hand veins may also be bothersome to patients. In the past, the gold standard for the treatment of these vessels has been sclerotherapy. The technique involves the use of sodium tetradecyl sulfate 0.25% (Sotradecol, Bioniche, Montreal CA) or, alternatively, a foam solution (foam sclerotherapy) with a weaker formulation of 0.2%. Either a direct needle or open cannulation approach may be used. A stretch elastic bandage should be applied and left on during waiting hours for the first 24 hours. Each hand is treated during a separate treatment session in order to avoid simultaneous dissolution in the dorsal arch vein system of both hands.

With all vein procedures, bruising and potential secondary hyperpigmentation are the most common adverse side effects noted. Arnica and bromelain supplements pretreatment and immediately posttreatment may help to minimize this phenomena. Usually one to two treatment sessions are necessary in order to see good clinical improvement.



Figure 8.7 *Technique of implantation of Sculptra in the hands employs attempting an interosseous approach. Usually one syringe per hand is employed for two to three treatment sessions at 4 to 6 weeks intervals*



Figure 8.8 *Pre- and postpoly-L-lactic acid hand treatment shows volume improvement after a single treatment session*

We have recently introduced a new endovenous laser technique (EVLH) to treat hand vessels. The concept is similarly utilized for treatment of saphenous vein incompetence of the lower extremity, but the hand vein fiber is of a shorter length. The laser fiber is of 1320-nm wavelength (CoolTouch, Roseville, CA). Usually a single treatment session is required. A small nick is made after injection of 1% lidocaine, then a 24-gauge 0.75-inch, radiopaque angiocath is introduced (Becton Dickinson, Franklin Lakes, NJ). The laser fiber is subsequently introduced through the lumina of the sheath. Tumescence anesthesia (lidocaine 0.05%; 15–20 cc per hand) is usually instilled to provide cushioning of treatment vessels (Figure 8.9). The treatment vein may be visualized by a HeNe beam, which detects

the location of the fiber placement. Settings at 3.5 W, 150 μm fiber, and total pulses 1450 (Figure 8.10) are recommended.

This approach is a novel way to treat dorsal hand veins. It is important to be sure that the vessel is well-cushioned from the skin surface in order to avoid thermal surface burns. An elastic stretch bandage is usually applied to the first 24 hours posttreatment.

Combination approaches

Most patients have a combination of aging concerns on their hands and arms and require multiple treatment modalities in order to address these concerns. Examples of combination therapies used in this regard are listed in Table 8.4.



Figure 8.9 Technique of endovenous hand laser (EVLH) involves introduction of a 1320-nm laser fiber with protection of the epidermis by tumescent anesthesia. A HeNE beam allows direct visualization of the laser fiber in order to ensure exact placement

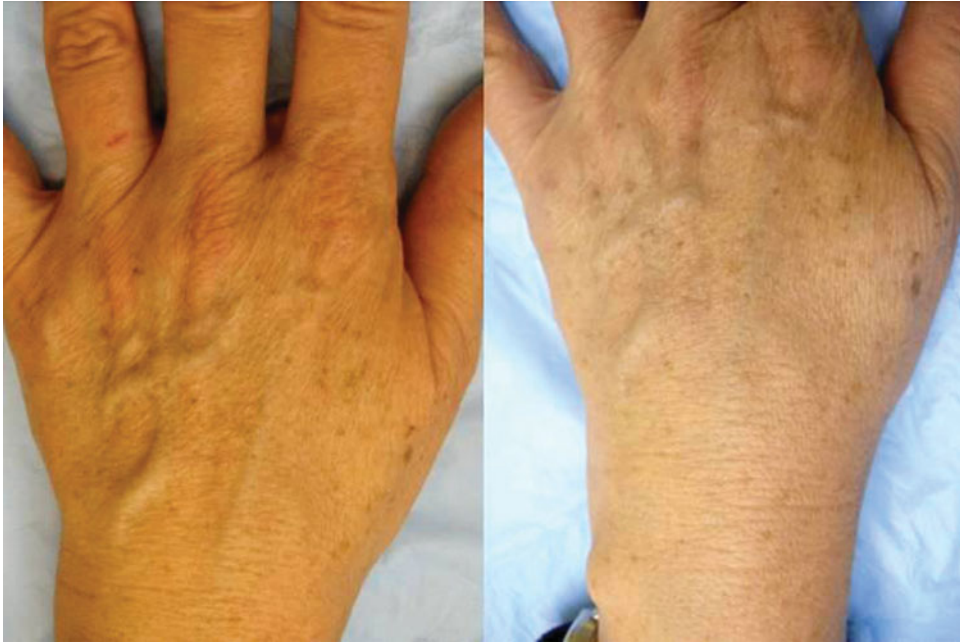


Figure 8.10 Pre-, postEVLH of dorsal arch hand vein (single treatment 3.5 W)

COMPLICATIONS TO AVOID

Complications and side effects associated with light source/laser and radiofrequency technologies usually result from the delivery of excessive thermal energy stacking of pulses or treating individuals with lasers when they are tanned or those who have had recent sun exposure. This may lead to the development of postinflammatory hyperpigmentation or epidermal contour irregularities leading to subsequent scarring. Appropriate settings may be helpful in this regard. Be sure to follow appropriate photoprotection practices and avoid pulse stacking.

TABLE 8.4 ■ Combination Therapies

Concern	Approach
Hyperpigmentation, veins, rhytides	ThermaCool, Fraxel, EVLH
Wrinkles, rhytides, lentigines, atrophy	ThermaCool, Q-switched laser, poly-L-lactic acid
Roughness, veins, atrophy	Fraxel, EVLH, poly-L-lactic acid
Rhytides, atrophy	CoolTouch, poly-L-lactic acid

Appropriate wound care measures are indicated for crusted ulcerative zones. Hyperpigmentation should be treated with 3% to 4% hydroquinone preparations. The major sequelae of filler treatments are bruising and secondary infection. Topical application of vitamin K creams and arnica and bromelain supplements posttreatment may be helpful in this regard.

After hand vein sclerotherapy, if extravasation occurs necrosis may ensue if the solution is inadvertently implanted into the soft tissue. Again, appropriate wound care measures are indicated if this occurs.

Finally, thermal burns may occur after endovenous hand laser treatment if too much energy is delivered too close to the skin surface. Adequate tumescent anesthesia to cushion the vein will minimize this adverse event (Figure 8.11).

Contact dermatitis occurring due to adhesive derivative is not uncommon and may be treated by a medium potency corticosteroid cream. In summary, fastidious aseptic technique will minimize the incidence of adverse events after hand rejuvenation.

POSTPROCEDURE INSTRUCTIONS AND CARE

The postoperative care for hand rejuvenation patients is uncomplicated. After any procedure for rejuvenation,



Figure 8.11 Thermal burn following endovenous laser treatment often results from inadequately cushioning a vein against the skin's surface with tumescent anesthesia

appropriate broad-spectrum photoprotection is of importance. After microablative laser procedures, such as fractional photothermolysis, laser micropeels, and Q-switched laser, application of a bland healing ointment, or an ointment with mild antibacterial properties such as mupirocin (Bactroban, GlaxoSmithKline, Middlesex, UK), or Aquaphor healing ointment applied three to four times per day is indicated.

After autologous fat transplantation or injection of a filler, such as hyaluronic acid derivatives or poly-L-lactic acid in the hands, bruising is the major side effect encountered. Topical application of vitamin K cream (Hopewell Pharmaceutical, Hopewell, NJ) arnica or bromelain supplements bid may be helpful in this regard. Similar postoperative discourses may be considered with hand vein sclerotherapy or endovenous hand vein ablation procedures as well. An elastic bandage such as Coban (3M Healthcare, St. Paul, MN) is applied for 24 to 48 hours in order to provide adequate compression as well as reduce swelling and bruising following these procedures. Patients can usually resume normal activities following these procedures.

CONCLUSION

Treatment of photoaged skin remains a challenge and is a visual marker of the aging process. Understanding and matching the patient concerns with targeted treat-

ment plans based upon a structural approach to hand rejuvenation leads to optimal patient results and subsequently a high-degree of patient satisfaction. Combination approaches using multiple modalities are often beneficial in this setting.

REFERENCES

1. Sadick NS, Burgess, C. Clinical experience of adverse outcomes associated with poly-L-lactic acid. *J Drugs Dermatol*. 2007;6(1) Supplement:S3-S8.
2. Redaelli A. Cosmetic use of polylactic acid for hand rejuvenation: report on 27 patients. *J Cosmet Dermatol*. 2006;5:233-238.
3. Schell BJ. Nonfacial soft tissue restoration with poly-L-lactic acid. *Cosmet Dermatol*. 2006;(4) S2: 25-28.
4. Sadick NS, Schechter AK. Utilization of the 1320-nm Nd:YAG laser for the reduction of photoaging of the hands. *Dermatol Surg*. 2004;30:1140-1144.
5. Wanner M, Tanzi EL, Alster TS. Fractional photothermolysis: treatment of facial and nonfacial cutaneous photodamage with a 1550-nm erbium-doped fiber laser. *Dermatol Surg*. 2007;33:23-28.
6. Laubach H, Chang HH, Rius F, Rox Anderson R. Effects of skin temperature on lesion size in fractional thermolysis. *Lasers Surg Med*. 2007;39:14-18.
7. Goldberg DJ, Silapunt S. Histologic evaluation of a q-switched Nd:YAG laser in the nonablative treatment of wrinkles. *Dermatol Surg*. 2001;27:744-746.
8. Abergel RP, David LM. Aging hands: a technique of hand rejuvenation by laser resurfacing and autologous fat transfer. *Dermatol Surg Oncol*. 1989;7: 725-728.
9. Shiffman MA. Autologous fat transplantation. *Am J Cosmet Surg*. 1997;4:433-443.
10. De Felipe I, Redondo P. Animal model to explain fat atrophy using nonablative radiofrequency. *Dermatol Surg*. 2007;33:141-145.
11. Andre P. Hyaluronic acid and its use as a "Rejuvenation" agent in cosmetic dermatology. *Semin Cutan Med Surg*. 2004;9:218-222.
12. Monheit GD, Coleman KM. Hyaluronic acid fillers. *Derm Therapy*. 2006;19:141-150.

CHAPTER 9

Leg Rejuvenation

Karen L. Beasley and Robert A. Weiss

KEY POINTS

- Treatment of spider veins and varicose veins not only improves the cosmetic appearance of the leg, but may also
 - help relieve symptoms of leg pain and swelling and
 - halt the progression of serious venous disease.
- By addressing and correcting photodamage of the leg, you can also
 - screen for skin cancers and precancers and
 - educate your patients about protecting their skin from the sun.
- Improving loose skin and fatty deposits of the legs makes patients feel better about themselves and may lead them to be more self-confident in social situations.

PATIENT SELECTION

Cosmetic leg concerns affect millions of patients worldwide. Many patients will choose to address their leg concerns before their face because they feel that the cosmetic disfigurement of their legs is more socially inhibiting. They report embarrassment to the point that they no longer wear shorts, short skirts, or bathing suits and have changed their lifestyles to avoid situations in which they would normally bare their legs. The overwhelming majority of our patients' leg rejuvenation concerns is regarding elimination of spider and varicose veins, followed by correction of photodamage and tightening of loose skin and fat.

Millions of patients around the world are affected by bulging varicose veins and unsightly "roadmap" telangiectatic webs. The incidence is highest in Caucasian patients in which telangiectases comprise the most common of all cosmetic complaints. This is borne out by epidemiologic surveys in which leg telangiectasia is reported in 70% of women.¹ Women are at least four times more likely than men to develop telangiectasia, while males have double the risk of developing large varicose veins.² The main techniques employed in the dermatologist's

practice for treatment of cosmetic spider veins are sclerotherapy and, occasionally, lasers.

SCLEROTHERAPY

Sclerotherapy gained acceptance in the United States as a highly effective treatment during the early 1990s, and it can be utilized for veins of all sizes.³ It remains the treatment of choice for telangiectasia (spider veins) and reticular veins. With the addition of foam sclerotherapy, leg vein treatment is now even more effective.⁴

■ Pretreatment Evaluation

A complete medical and surgical history is obtained. Contraindications to sclerotherapy include hypercoagulable states, inability to ambulate or comply with compression, arterial insufficiency, allergy to sclerosing solutions, pregnancy, or a significant medical issue that would interfere with healing, such as poorly controlled diabetes. Physical examination is performed by viewing the patient's legs in a 360-degree rotation while they are standing. On the basis of the history and physical examination, noninvasive diagnostic vascular tests are performed as necessary.⁵ Patients who only have small telangiectasia, especially on the lateral thigh, typically need no diagnostic testing for reflux. However, those patients with a family history of large varicose veins are more likely to have early axial (saphenous) reflux even when presenting with telangiectasias alone.⁶ Previous venous surgery always warrants further testing before treatment. Any bulging veins or dense patches of smaller vessels around the ankle or inner thigh that is suggestive of saphenous system involvement should be minimally evaluated by handheld Doppler ultrasound, which is equivalent to using an enhanced "stethoscope" to hear vein flow. In order to generate or augment an audible signal of flow, a maneuver such as manual compression of the calf must be performed by the examiner. When compression is released, gravitational hydrostatic pressure causes reverse flow to cease within 0.5 to 1 second when valves are competent, but a long flow sound is audible when valves are incompetent. The most essential part of the Doppler examination is the examination of

the saphenofemoral junction below the inguinal fold just medial to the femoral arterial signal. During a Valsalva maneuver, a continuous and pronounced reflux signal is a reliable sign of valvular insufficiency. An equivocal result may require a duplex ultrasound examination for a definitive answer. If any part of the diagnostic examination reveals evidence of reflux in the saphenous system, sclerotherapy will be ineffective, and an endovenous closure of the affected vein will need to be performed before successful treatment of spider veins with sclerotherapy.

If the physical examination does not reveal any significant venous insufficiency and the patient is judged to be a candidate for sclerotherapy, pretreatment photographs are taken and an informed consent is obtained; this includes a discussion of all potential side effects.

Postsclerotherapy hyperpigmentation is the most common adverse effect occurring in approximately 30% of patients; it gradually resolves in 70% of cases within 6 months and rarely persists for greater than a year.^{7,8} Telangiectatic matting is the second most common adverse effect and a retrospective analysis of over 2000 patients reports an incidence of 16% in patients treated with sclerotherapy.⁹ Resolution usually occurs spontaneously within a period of 3 to 12 months with 70% to 80% spontaneous resolution within the first 6 months.¹⁰ Cutaneous necrosis with skin ulceration happens rarely but occurs more commonly when hypertonic sclerosing solutions are used. Significant superficial thrombophlebitis, deep vein thrombosis, pulmonary embolism, and accidental arterial injection are extremely rare when performing sclerotherapy, especially when treating superficial spider veins.

During the initial consultation, patient expectations should always be discussed. It is important for them to understand that they will need a series of treatment for slow, gradual improvement of their leg veins. The same vein may have to be injected upon several times before it shows improvement. New veins will form in the future and the improvement they achieved from their initial sclerotherapy series will need to be maintained with periodic maintenance sclerotherapy.

■ Technique

Endovenous chemoablation sclerotherapy solutions

Numerous solutions are available for sclerotherapy and are summarized in Table 9.1. The three most common solutions we employ in our practice are sodium tetradecyl sulfate (STS), hypertonic saline and dextrose (HSD)

(Sclerodex), and glycerin. Test injections of the sclerosing solutions are always given during the initial consultation before sclerotherapy sessions begin, to evaluate for allergy, side effects, and efficacy.

Sodium tetradecyl sulfate STS is an anionic detergent that occurs as a white, waxy solid; it must be carefully purified for sterile injection. This requires pharmaceutical grade purification and is difficult (almost impossible) for compounding pharmacies to manufacture reliably. It is a highly effective sclerosing agent used worldwide. Appropriate concentrations for superficial telangiectases are 0.1% to 0.2%. Other concentrations are 0.2% to 0.5% in reticular veins or small varicosities (1–3 mm diameter) and 0.5% to 3% in larger varicosities related to major sites of valvular reflux. The maximum dose per session is 10 mL of the 3% solution, which is never approached when treating spider veins. When foamed, this solution is used in half the concentrations discussed earlier. However, the foamed solution is too strong for telangiectases that are less than 1 mm in diameter. In repeated studies, both STS and the other detergent solution, Polidocanol (POL), are found to be roughly equivalent.¹¹ There is one legal source of STS—as Sotradecol through AngioDynamics® (AngioDynamics®, Queensbury, NY).

Hypertonic saline and dextrose HSD is a viscous mixture of dextrose (250 mg/mL), sodium chloride (100 mg/mL), propylene glycol (100 mg/mL), and phenethyl alcohol (8 mg/mL). HSD is a relatively weak sclerosant for local treatment of small vessels; the total volume of injection should not exceed 10 mL per visit with 0.1 to 1.0 mL per injection site. Although a slight burning sensation occurs, pain is far less compared to that with hypertonic saline (HS).

Glycerin The creation of a specially compounded solution of 72% glycerin diluted with 1% lidocaine with epinephrine (1:100000) in a 2:1 solution has been a tremendous advance in the treatment of telangiectasia. In Europe, chromated glycerin sclerosing solution has been available for years. Because of concerns that the chromium moiety that is added to the glycerin to induce coagulation may be carcinogenic, phlebologists began to experiment with pure glycerin as a chemical-irritant type of sclerosant. They found it necessary to dilute it with lidocaine to make it less viscous and painful. In 2003, Drs. Leach and Goldman published a small trial comparing the treatment of telangiectasia using a 0.25% STS compound with that using a 72% glycerin compound.¹² Patients treated with glycerin demonstrated a significant

TABLE 9.1 ■ Commonly Used Sclerosing Solutions in the United States

Solution— Chemical Name	Brand Names	Category	Advantages	Disadvantages
Sodium tetradecyl sulfate	Sotradecol Fibro-Vein Thrombovar	Detergent (emulsifier)— rapid dissolution of endothelium	Painless intravascular Painful extravascular Strong for varicose veins Effective at low concentration FDA approved for sclerotherapy	Skin necrosis with extravasation of concentrations >0.25% Expensive Pigmentation— postsclerosis Dissolves rubber— must use latex-free syringes to avoid latex allergic response
Polidocanol	Aethoxysklerol Sclero-Vein	Detergent (emulsifier)	Always painless Cutaneous necrosis low Effective at low concentration	Urticaria (immediate) at injection site Skin necrosis from painless arteriolar injection Not FDA approved
Glycerin (72%) diluted with 1% lidocaine with epinephrine	None	Chemical irritant	Painless Low risk of allergic reaction Decreased risk of hyperpigmentation and matting No reported ulceration	Too weak for reticular and varicose veins FDA approved for reduction of cerebral edema
Hypertonic saline (23.4%)	None	Hyperosmolar— slow crenation of endothelium	Low risk of allergic reaction Readily available Rapid action	Painful stinging and cramping Highly ulcerogenic— high risk for skin necrosis FDA approved as abortifacient
Saline and dextrose	Sclerodex	Hyperosmolar	High viscosity— remains in treated veins Low allergic risk Low risk of necrosis	Too weak for larger varicosities Slight stinging One concentration only Not FDA approved

decrease in bruising, swelling, and hyperpigmentation. No cutaneous necrosis from glycerin was noted in this trial or has been reported in the phlebology literature. It also works very well in the treatment of cutaneous blushing (matting) that can occur as an adverse effect of

sclerotherapy. The maximum recommended amount per injection session is 10 mL of pure solution. The total amount of glycerin injected intravenously during an average sclerotherapy session is so minimal that there are no reports of anaphylaxis or systemic toxicity.

Hypertonic saline Although approved by the Food and Drug Administration (FDA) only for use as an abortifacient, HS is still commonly used in the United States in spite of its shortcomings. Used at a concentration of 23.4%, a theoretical advantage of HS is its total lack of allergenicity when unadulterated. With hypertonic solutions, damage of tissue adjacent to injection sites may easily occur. The pain of injection along with risks of ulceration makes it highly undesirable for modern vein treatment.

Polidocanol POL contains hydroxypolyethoxydodecane dissolved in distilled water with 5% ethanol as a stabilizer. The detergent-based POL was originally developed as an anesthetic, but was found to have the property of sclerosing small-diameter vessels after intradermal injection. POL is popular worldwide for smaller vessels due to painless injection and lowest incidence of cutaneous necrosis with intradermal injection. The US FDA has not yet approved POL.

Foam sclerotherapy Sclerotherapy with a foamed solution has become a standard of treatment of larger spider veins (>1 mm) and reticular veins and even some varicose veins (Figure 9.1). Currently, the only two solutions that may be foamed are the detergent solutions, STS and POL. Foam sclerotherapy is more effective than liquid sclerotherapy because the foam completely fills and dis-

places the blood in the vein, maximizing endothelial cell injury and subsequent fibrosis. This more effective form of sclerotherapy requires fewer treatment sessions. Since the foamed solution is stronger than liquid sclerotherapy, the concentration can also be decreased, reducing the side effects of hyperpigmentation and necrosis. In addition, less solution is required, which decreases potential systemic toxicity and the cost of supplies to the physician and patient. Duplex-guided sclerotherapy is also technically easier and safer since the foam is very echogenic and easily visualized on ultrasound. Transient visual disturbances and headaches occur infrequently but are more common with foam sclerotherapy than liquid sclerotherapy.

The most utilized technique for foam sclerotherapy was developed by Lorenzo Tessari, an Italian phlebologist.¹³ He connected two 3 mL Luer lock syringes with a disposable three-way stopcock. One syringe is filled with air and the other is filled with a detergent-type sclerosant (Figure 9.2). He found the optimal ratio of solution to air to be 1:4. For example, our practice uses 0.5 mL of STS to 2 mL of air. The contents of the syringes are shifted back and forth quickly 10 to 20 times and the turbulent flow generates the foam. The foamed solution should be used immediately since it degrades to approximately half the initial concentration within 60 to 90 seconds. The nature of the foam allows for the same therapeutic effect



Figure 9.1 Foamed sclerotherapy solution. The consistency of the foamed solution allows it to completely displace the blood within the vein during sclerotherapy, which maximizes contact and subsequent fibrosis of the internal vein wall



Figure 9.2 Tessari technique of foam sclerotherapy. Vigorous agitation between two syringes results in a frothy material for injection. Reduced total amounts of sclerosing solution with decreased total volumes result in increased safety

with a lower concentration of sclerosant. For instance, one can substitute 0.1% STS foam for 0.2% STS liquid. Lower concentrations are used for veins of all sizes. Foamed solution should only be used for varicosities, perforating veins, reticular veins, and larger telangiectasias (1.0 to 2 mm). Smaller telangiectasia (<1 mm) run the risk of increased pigmentation and matting as more inflammation occurs when relatively increased concentration interacts with the vein wall.¹⁴

Step-by-step technique

During the treatment of spider veins, it is important to first treat any of the blue reticular veins that are feeding the telangiectasias in the hope of decreasing the number of needle sticks and the potential number of treatments.¹⁵ When treating blue reticular veins, always pull the syringe plunger back to see the flashback of blood, to ensure you are in the vessel before injection. If there is no associated reticular vein, then start injecting at the base of the telangiectatic web. The sclerotherapy tray is prepared with the necessary equipment (Figure 9.3 and Table 9.2). A 30-gauge needle, bent to an angle of 10 to 30 degrees with the bevel up, is placed on the skin so that the needle is parallel to the skin surface. A 3-mL syringe filled with 1.5 to 2 mL of solution is held between the index and middle fingers (Figure 9.4). The nondominant hand is used to stretch the skin around the needle and may offer

TABLE 9.2 ■ Sclerotherapy Tray

Cotton balls soaked with 70% isopropyl alcohol
Protective gloves
3 mL disposable syringes
30 gauge disposable transparent hub needles
Cotton balls or STD pads for compression
Transpore™ and/or paper tape
2% nitroglycerine paste (for prolonged blanching or extravasation)
Sclerosing solutions in labeled syringes:
Sodium tetradecyl sulfate (various concentrations)
Hypertonic saline (10%) and dextrose (25%)
Glycerin (72%) diluted with 1% lidocaine with epinephrine (1:100 000) in a 2:1 solution
3-way stopcock to foam solutions (1:4—sclerosant:air)

additional support for the syringe. Magnifying lenses or operating loops on the order of 1.5 to 3 X may help cannulation of the smallest telangiectases. The initial treatment of telangiectatic webs begins with the lowest possible concentration that will cause a telangiectases to sclerose over a period of 1 to 6 months postinjection. This typically is 0.1% STS liquid or 72% glycerin for telangiectasia and 0.1% to 0.2% STS foam for reticular veins. Posttreatment compression consists of use of a graduated 20 to



Figure 9.3 Sclerotherapy tray. The tray is composed of clearly labeled sclerotherapy solutions in 3 mL syringes, alcohol-saturated cotton balls, metal cup for sharps, three-way stopcock for foaming, extra 30 gauge needles, cotton balls and tape for compression, and nitropaste



Figure 9.4 Position of the hands for sclerotherapy. While the dominant hand holds the syringe and creates a platform with the fifth digit, the nondominant hand stretches the skin and acts as a support for the needle hub so that fine changes in position are permitted



Figure 9.5 Sclerotherapy results: Before and after sclerotherapy with foamed 0.1% STS and glycerin

30 mmHg support hose for 2 weeks for smaller veins. Treatment intervals vary, but allowing at least 4 weeks between treatments helps to minimize the number of necessary sessions. Typical results are shown in Figure 9.5.

LASER AND LIGHT TREATMENTS

■ Patient Selection

In general, treatment with lasers and light sources is more expensive, less predictable, associated with inconsistent responses, and more painful than sclerotherapy. However, these devices are occasionally used in certain circumstances. For example, lasers or light sources may be used if the patient is afraid of needles, not interested in wearing postsclerotherapy compression hose, has telangiectatic matting that cannot be cannulated with a needle, or is nonresponsive to sclerotherapy.

The basic requirement for a laser or light source used for the treatment of leg veins is a wavelength that is proportionately better absorbed by the target (hemoglobin)

than surrounding chromophores, and which penetrates to the full depth of the target blood vessel with energy sufficient to damage the vessel without damaging the overlying skin. The pulse duration must be long enough to slowly coagulate the vessel and its lining without damaging the surrounding tissue. Over the years, the development of devices with longer wavelengths, improved cooling technology, and variable pulse durations have improved the results of laser or light treatment of leg veins, but, in general, they are not as effective as sclerotherapy.

■ Pretreatment Evaluation

Reverse pressure from associated reticular or varicose veins must always be recognized and treated or laser treatment will be ineffective. Pretreatment photographs are taken. Informed consent is obtained with a discussion of the adverse effects of pain, erythema, swelling, bruising, hyper- or hypopigmentation, lack of efficacy, and rarely crusting, blistering, and potential infection or scars. A discussion with the patient informing him or her that a series of treatment will be necessary for improvement and will not prevent the formation of new vessels should also be included in the consultation. Posttreatment compression is unnecessary. Test sites may be considered with different laser and light sources to test the response of the vessels.

■ Treatment

Potential devices to treat spider veins

Although many lasers and light devices have been attempted to clear leg veins, the majority of those tried have been unsuccessful. This chapter reviews the laser or light sources with the most reported efficacy. These devices have wavelengths that are close to the hemoglobin absorption peaks at 541 nm and 800 to 1000 nm. These light sources include potassium titanyl phosphate (KTP) laser or frequency doubled neodymium:yttrium–aluminum garnet (Nd:YAG) laser (532 nm), pulsed dye laser (PDL) (595 nm), intense pulsed light (IPL) (560–1200 nm), and Nd:YAG laser (1064 nm). Diode and alexandrite lasers also have wavelengths within the hemoglobin absorption spectrum and have been used to treat leg veins, but these would not be our first choice to treat blood vessels.

532-nm lasers The 532-nm wavelength is absorbed by hemoglobin and penetrates just as well as 585-nm light, which is well known to treat vascular lesions. For leg

vessels less than 1 mm in diameter that are not directly connected to a feeding reticular vein, and with use of contact cooling to protect the epidermis, this laser can be quite effective. Two to three treatments are necessary for maximal vessel improvement although some have reported 100% resolution of the treated leg vein with one treatment.¹⁶ Patients with darker or tanned skin should avoid this type of treatment secondary to a relatively high risk of temporary hypopigmentation in our experience.

Pulsed dye laser The traditional PDL (585 nm), with a 450- μ sec pulse duration, can penetrate 1.2 mm to reach the typical depths of leg telangiectasia.¹⁷ However, the pulse duration is too short to effectively damage all vessels; it can damage only the most superficial fine vessels. Theoretically, the new PDL devices, which have long pulse durations, some up to 40 milliseconds, should be more effective. However, in general, veins treated with traditional or long-pulse PDLs are less responsive and more prone to posttherapy hyperpigmentation than when treated with sclerotherapy.¹⁸

Intense pulsed light The initial report on treating leg telangiectasias with IPL was very optimistic; clearance of 75% to 100% was achieved in 79% of treated lesions and better than 50% clearance was achieved in 94% of cases.¹⁹ However, this data was obtained from dozens of centers with widely different techniques of treatment and data recording and therefore could not be reproduced on

a regular basis by some users.²⁰ For many years, we have treated several thousand patients with multiple parameters using multiple combinations of short and long pulses in an attempt to obtain the most consistent results. We have found IPL to be most useful for the telangiectatic matting that occurs on the inner thighs following sclerotherapy.

Nd:YAG laser The first report of 1064-nm Nd:YAG laser indicated that 75% improvement was possible after a single treatment at 3 months.²¹ These findings were confirmed and the mechanism of action was explained as heat-induced vessel damage and subsequent fibrosis.²² The primary benefit of this wavelength is deep penetration and the absence of absorption in melanin thus allowing treatment even in deeply pigmented individuals. However, high energies must be utilized for adequate penetration and heat dissipation to affect the posterior wall of a larger diameter (1–2 mm) vessel filled with deoxygenated hemoglobin. The larger the vein, the more painful would be the treatment, due to increased absorption of infrared energy.²³ For patient comfort, epidermal cooling must be provided. Contact and cryogen cooling are both effective means of protecting the skin and reducing pain. Using smaller spot sizes with moderate fluences to reduce excess dermal heating and pain has also been reported to be an effective way to treat leg veins with the 1064-nm laser.²⁴ A clinical photograph of leg vein response before and after treatment with a 1064-nm laser is shown in Figure 9.6.

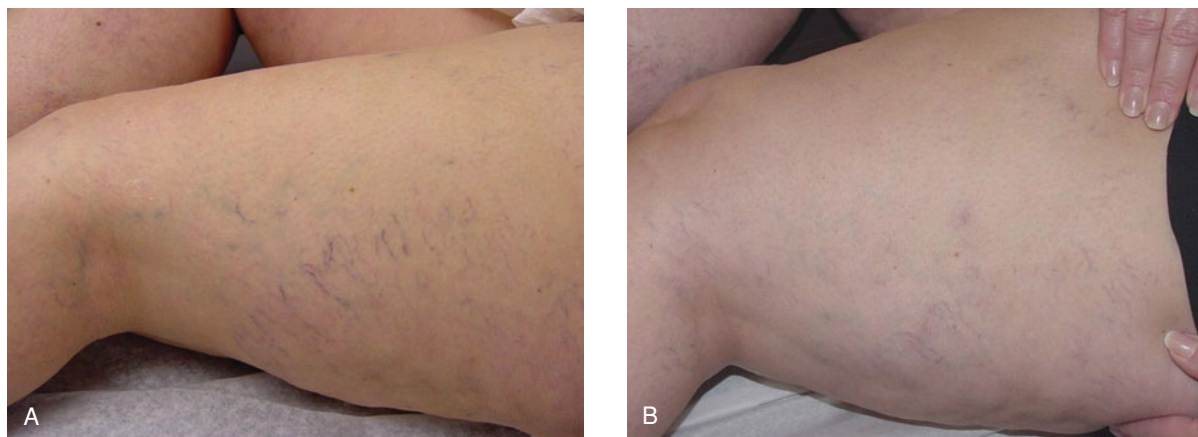


Figure 9.6 Clinical results with 1064-nm laser. A small isolated group of telangiectasias is treated with 1064-nm laser, (Vasculight, Lumenis, Santa Clara, CA). (A) Before and (B) 3 months after treatment. Treatment parameters are a single 16-millisecond pulse, 6-mm spot size, and fluence of 120 J/cm²

VARICOSE VEINS

■ Patient Selection

More than 24 million people are affected by varicose veins and the incidence of varicose veins increases with each decade of life. With an increasing life span within the United States, the rate of varicose veins is on the rise. While 41% of women in the fifth decade have varicose veins, this number rises to 72% in the seventh decade.²⁵ Statistics for men are similar with 24% incidence in the fourth decade, increasing to 43% by the seventh decade. Pregnancy, increased body mass, and family history increase the risk of varicose vein development. It is estimated that 6 million workdays per year may be lost in the United States due to complications of varicose veins.²⁶ Obviously, varicose veins are not just cosmetically disfiguring, they may also cause significant morbidity including chronic stasis dermatitis, ankle edema, spontaneous bleeding, superficial thrombophlebitis, recurrent cellulitis, lipodermatosclerosis, and skin ulceration on the ankle and foot.

■ Pretreatment Evaluation

When a patient presents with bulging varicosities, complaints of leg pain or swelling, has a previous history of vein surgery, has a strong family history of varicose veins, and an abundance of veins in the area served by the saphenous veins, a duplex ultrasound is indicated. Duplex ultrasound is the gold standard of noninvasive examination of the venous system. It allows direct visualization of the veins and identification of flow through venous valves. The Duplex examination may also help to delineate reflux sources when patients experience poor results from sclerotherapy. If venous insufficiency is found in the saphenous system, dermatologic surgeons can employ endovenous occlusion of the vessel by radiofrequency (RF) or laser, and then address the remaining veins depending on their size and location by ambulatory phlebectomy (AP) and duplex-guided or standard sclerotherapy.

■ Technique

Endovenous ablation techniques

When saphenous system reflux is present, the endovenous occlusion techniques must be performed prior to treatment of visible and associated varicosities, reticular veins, or telangiectases. Without elimination of reflux, the

patient will experience repeated recurrences. Previously, the only treatment option for these patients with varicose veins from saphenous insufficiency was surgical ligation and stripping of the vessels under general anesthesia. This procedure resulted in significant postoperative downtime with surgical scars, swelling, bruising, and pain. The expense of a hospital stay and missed work, along with an unacceptable rate of recurrence has decreased the popularity of this procedure. While RF-mediated elimination started the noninvasive varicose vein trend, endovenous obliteration with lasers has revolutionized varicose vein treatment.

Radiofrequency This method involves the placement of a catheter within the varicose vein through a small puncture or incision. The catheter is threaded up to the saphenofemoral junction typically under duplex ultrasound guidance. Critical to the safety and success of the technique is the placement of tumescent local anesthesia, which involves first infiltrating the tissue under the skin including the thigh muscles to push the saphenous vein further away from the skin and then creating a space between the fascia and the saphenous vein so as to hydrodissect the vein away from structures such as nerves and arteries. Once this hydrodissection has been performed with high volume and low concentration of local anesthetic (0.1% lidocaine), energy is applied as the catheter is slowly withdrawn. This results in heat being directly applied to the vein wall via a computer-controlled thermocouple feedback loop, which leads to a collagen shrinkage accompanied by complete vein wall occlusion. The efficacy for RF elimination of reflux is 90% at 2 years and 80% at 5 years.^{27,28} Recent advances in the delivery of the RF energy to the vein will hopefully increase the efficacy of this procedure.

Lasers with hemoglobin-predominant absorption (810 nm, 940 nm, 980 nm) Endovenous laser treatment with these wavelengths are similar to RF in that both devices produce endothelial and vein wall shrinkage by nonspecific heating of the vessel. The target for lasers with wavelengths of 810, 940, and 980 nm is the intravascular red blood cell. The success of these lasers is highly dependent on correct placement of tumescent local anesthesia to have a thin layer of blood surrounding the laser fiber while having enough anesthesia to protect the surrounding nerves and muscles from injury. Direct thermal effects on the vein wall without the presence of blood probably do not occur. Consistent results may be hard to duplicate given that these lasers are dependent on so

many different factors including the amount of blood in the lumen, the rate of pullback and the amount of tumescent anesthesia placed around the vein. Histologic examination of one excised vein demonstrated thermal damage along the entire treated vein with evidence of perforations at the point of laser application described as “explosive-like” photodisruption of the vein wall. Formation of bubbling blood is the proposed mechanism of action for heating surrounding tissue.²⁹ We have found superheating of blood during these procedures to reach a maximum temperature of approximately 1200°C.³⁰ These endovenous lasers have short-term efficacy and 90% occlusion occurs over the first year, but the rate decreases with time.^{31,32} In addition, most patients experience major degrees of postoperative ecchymosis and discomfort. Saphenous nerve injury, skin burns, and deep venous thrombosis have occurred due to the high temperatures of blood heating from these wavelengths, although the overall safety and efficacy record is better than for stripping.^{33–35}

Lasers with water-predominant absorption (1320 nm)

A variation of the endovenous laser with a wavelength of 1320 nm was developed by dermatologic surgeons in an attempt to bypass the problems associated with the lasers with wavelengths that absorb hemoglobin. Tissue water within the vein wall is the specific target of the 1320-nm laser and the presence or absence of red blood cells within the vessels is unimportant. For a more controlled release of laser energy, it is coupled with an automatic pullback device, which can pull back at the rate 0.5 or 1 mm/s. The penetration of the 1320-nm wavelength is unique in that it effectively heats and contracts the vein with far less heat generation and risks than with the hemoglobin-absorbing wavelengths. Proebstle and colleagues demonstrated a statistically reduced rate of postoperative pain accompanied by a higher initial success rate using 1320-nm laser versus 940-nm laser.³⁶ Our own experience reflects this, with a reduction in pain and bruising of 80% when switching from 810-nm endovenous to 1320-nm endovenous. Having treated over 200 greater saphenous veins with 1320 nm laser, our incidence of mild pain is 5%. No significant pain interfering with walking has been observed with the 1320-nm laser but has been observed in up to 50% of patients who underwent 810-nm laser treatment. A clinical example of varicose veins resulting from reflux at the saphenofemoral junction pre- and posttreatment with a 1320-nm laser is shown in Figure 9.7.

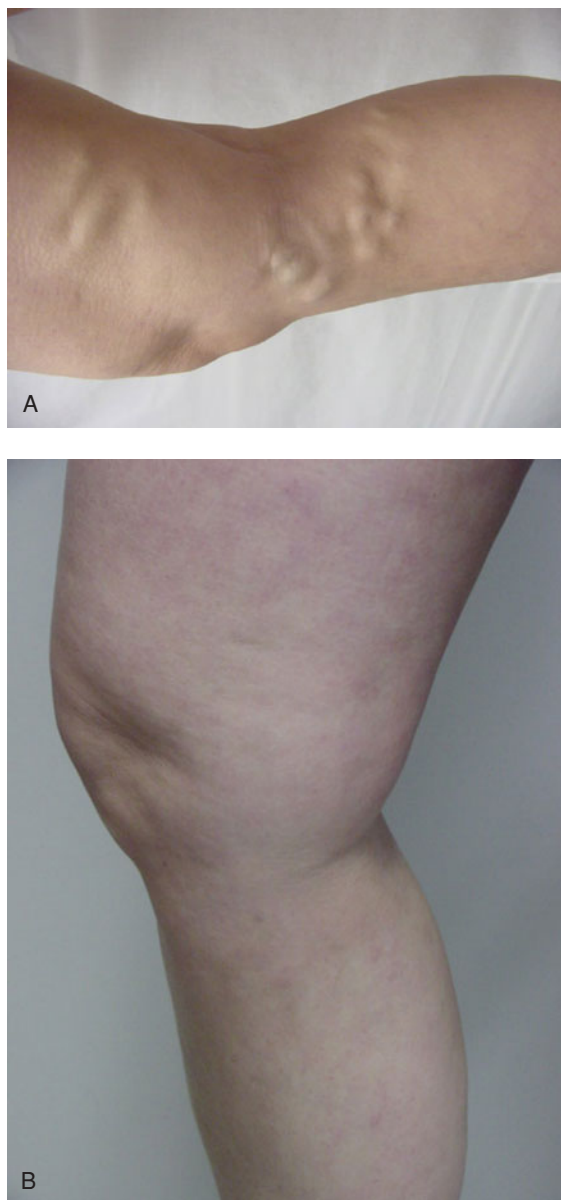


Figure 9.7 Varicose tributaries arising from an incompetent greater saphenous vein (GSV): **(A)** Before treatment and **(B)** 6 weeks after endovenous ablation of the GSV with 1320-nm laser results in the improvement of associated varicosities

Endovenous ablation technique with 1320-nm laser

Preoperative history and physical examination, photographs, and informed consent are obtained. The procedure is performed in an outpatient setting with local

tumescent anesthesia. A small catheter is threaded percutaneously and the laser fiber is placed directly into the refluxing vein. Correct placement of the laser fiber at least 2 cm distal to the saphenofemoral junction is confirmed with ultrasound. Under ultrasound guidance and with an automatic pullback system, the laser seals the varicose vein from the inside out. Although the patient has to use compression stockings for 3 days, normal activity is encouraged immediately after the procedure. Patients can return to work the next day, discomfort is minimal and the rate of successful long-term vein closure is 95% at 2 years.³⁷ Adverse effects like deep vein thrombus and nerve damage are very rare.

Ambulatory phlebectomy This technique, originally described by Robert Muller and further refined by another Swiss dermatologist (Albert-Adrien Ramelet) involves the use of tiny incisions through which the varicose vein is removed by a small hook.^{38,39} Saphenous vein insufficiency must be corrected or not present before AP is performed or it may be ineffective. Superficial bulging varicosities, veins that are resistant to foam sclerotherapy or are located in areas that are hard to compress after sclerotherapy like around the knee joint are particularly indicated for AP. The supplies used during phlebectomy are given in Table 9.3.

Technique The varicose vein is first marked to view the full extent of the bulging vein when the patient is standing, and again when the patient is supine. Since the vein collapses when the patient becomes supine, the area is marked utilizing transillumination to make sure that there are no positional shifts in the vein (Figure 9.8). The skin

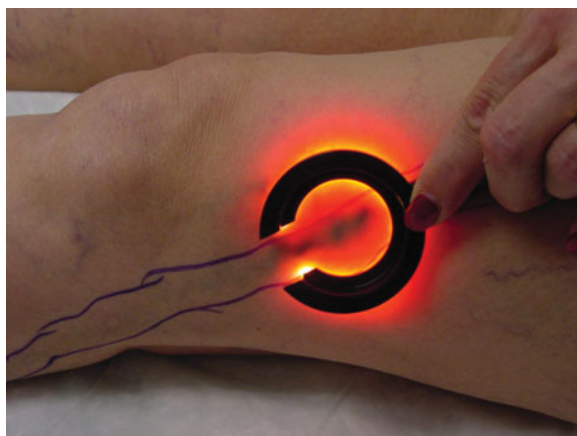


Figure 9.8 *Transillumination: The vein is easily visualized utilizing this lighting technique and is easily marked preambulatory phlebectomy*

around the vein is painted with iodine and anesthetized with tumescent anesthesia. Small incisions adjacent to the vein are created with an 18-gauge NoCor needle. A phlebectomy hook is inserted in the small incision, which is used to harpoon the vein or hook around the vein and pull it out to the surface (Figure 9.9). This process is repeated down the length of the vein and it is removed in segments. A variety of hooks is manufactured for this procedure and may be placed on the AP tray to be selected according to the physician's personal preference



Figure 9.9 *Beasley ambulatory phlebectomy technique: A loop of varicose vein has been externalized with a phlebectomy hook and is now gently removed with mosquito clamps*

TABLE 9.3 ■ Phlebectomy Supplies

Skin marking pen
Iodine prepping solution
Disposable face mask, sterile gloves
Local anesthetic, syringes, needles
Ambulatory phlebectomy hooks: Muller, Oesch, Ramelet, Varady
Clamps, sterile 4 × 4 gauze pads
NoCor 18-gauge needle, scalpel No. 11
Mosquito forceps (one dozen)
Hydrogen peroxide for postoperative washing of the leg
Absorbent dressings
Inelastic compression wrap
Elastic graduated compression stockings



Figure 9.10 Ambulatory phlebectomy tray: The sterile tray is composed of an 18-gauge NoCor needle, gauze, mosquito clamps, and a variety of phlebectomy hooks

(Figure 9.10). Postoperative care includes covering the incision sites with absorbent pads, securing the pads with a short stretch bandage, and covering the leg in 30 to 40 mm Hg compression hose for 24 hours. Then the pads are removed; the patient has to wear the compression hose during the day for a total of 2 weeks. Adverse effects consist of minor discomfort and bruising. The small incisions usually heal with a very small or unapparent scar. Nerve damage, lymphocele, and infection are rare. This procedure is very gratifying for the patient and the physician because the results are impressive and immediate. Typical results are shown in Figures 9.11 and 9.12.

Correction of photodamage It is always important that when a patient schedules a consultation for improvement in sun damage, a careful physical examination is performed first in order to detect any skin cancers or precancers. These must be treated appropriately before any



Figure 9.11 Ambulatory phlebectomy of large shin varicosity: (A) Pretreatment and (B) 6 weeks post AP with dramatic improvement

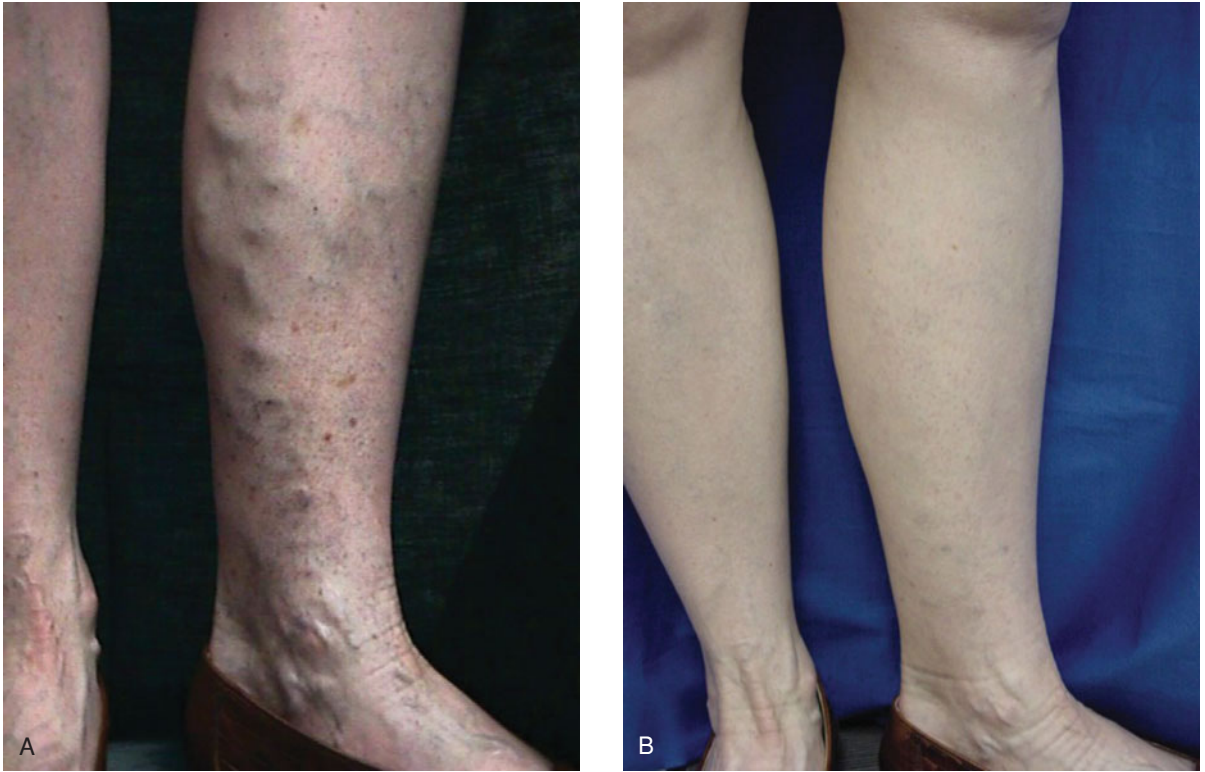


Figure 9.12 Ambulatory phlebectomy of a large truncal varicose vein. **(A)** Pretreatment **(B)** Clinical results with complete disappearance at 6 weeks. Bruising usually lasts no longer than 2 weeks

cosmetic rejuvenation of the leg can occur. Benign seborrheic keratoses of the leg are common cosmetic complaints and these can be treated with gentle liquid nitrogen therapy. Performing cryosurgery in a nonaggressive manner helps in avoiding permanent hypopigmentation and scarring that can easily occur on this part of the body. Photodamage of the legs may be addressed in a similar fashion as photodamage of the face. Sun damage may be improved by spot treatment of lentigines with a laser (such as a ruby or an alexandrite laser) that specifically targets the pigment (Figure 9.13). If the sun damage is diffuse, the legs may be treated overall with IPL photorejuvenation. New advances in lasers, such as fractional resurfacing, can now be applied to the skin of the leg. Leg rejuvenation by resurfacing the skin was previously impossible with the older ablative resurfacing lasers because of the unacceptable side effects of poor healing, scarring, and permanent hypopigmentation. Both IPL photorejuvenation and fractional resurfacing are success-

ful forms of leg rejuvenation but they are both very time consuming to perform. Fluences lower than what are recommended for facial rejuvenation must be used, and multiple treatments after every 6 weeks are necessary.

TIGHTENING OF LOOSE SKIN AND FAT

■ Patient Selection

This cosmetic complaint is one that we see almost exclusively in women. Fatty deposits along the hip and inner thighs are especially problematic. For best results, patients must be of a stable weight, have an established exercise routine, and have localized areas of fat or loose skin. Obese patients or those who have undergone dramatic weight loss with poor skin elasticity are poor candidates for treatment. Obese patients should be encouraged to lose weight and maintain a healthy diet and exercise program before treatment is considered. Those

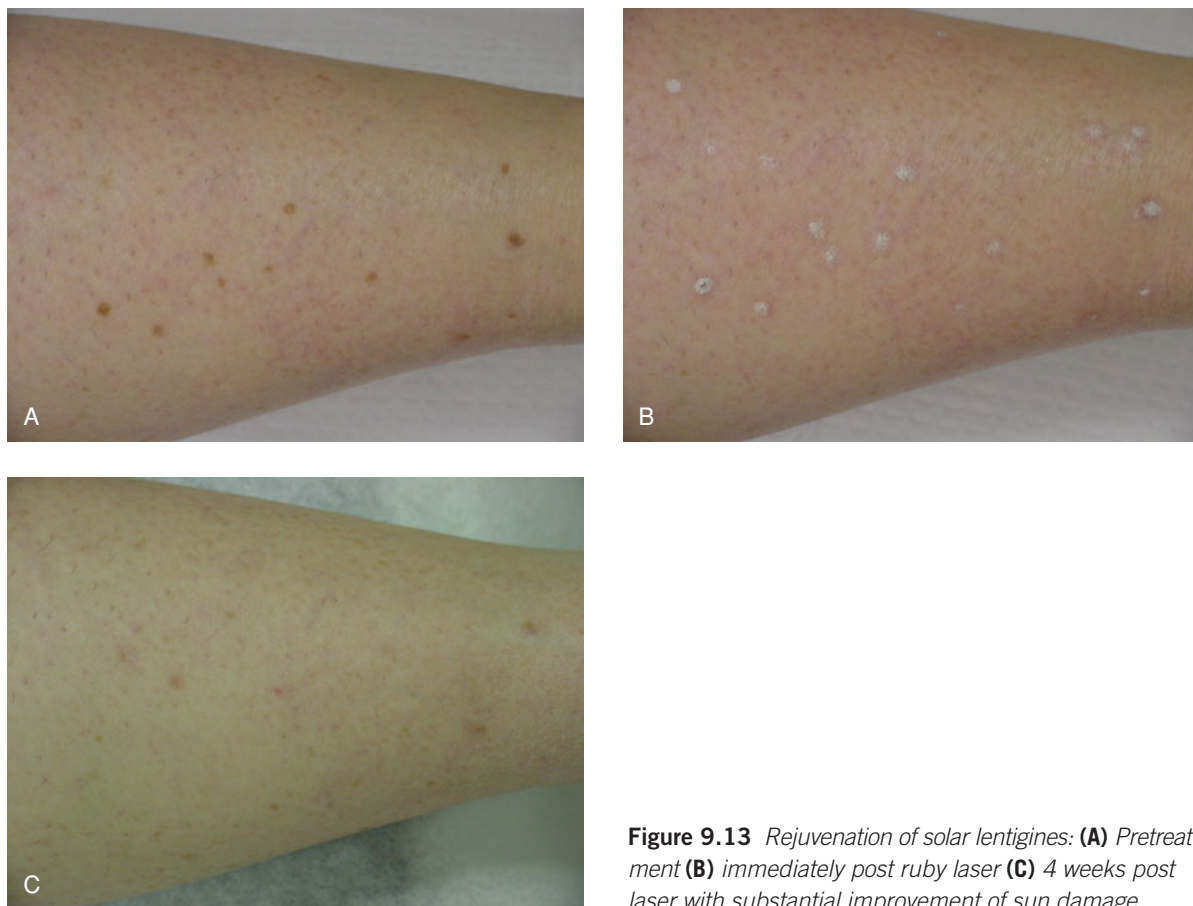


Figure 9.13 Rejuvenation of solar lentigines: (A) Pretreatment (B) immediately post ruby laser (C) 4 weeks post laser with substantial improvement of sun damage

patients with poor skin elasticity or a moderate to severe amount of loose skin are better candidates for a leg-lifting surgical procedure with a plastic surgeon. Outpatient tumescent liposuction has been the mainstay of treatment in patients with localized fatty deposits. The safety record of this procedure when performed by dermatologists using tumescent anesthesia is excellent.⁴⁰ A detailed discussion of tumescent liposuction is beyond the scope of this chapter. Other procedures that are currently evolving for leg rejuvenation are RF tightening of skin and laser lipolysis with lipoaspiration.

■ Technique

RF tightening of loose skin and fat

Patients who do not have an abundance of loose skin and fat and are interested in a noninvasive alternative to induce skin tightening with no cosmetic downtime are

candidates for RF tightening. The first RF device approved for a cosmetic indication was ThermoCool™ (Thermage, Hayward, California, USA). This device emits a novel form of monopolar RF energy to create a uniform field of dermal and subdermal heating, while the epidermis is protected by contact cooling. Originally, this procedure was approved by the FDA for the reduction of periorbital rhytides, but is commonly used for facial and neck rejuvenation; its use has recently been expanded to include body rejuvenation. Clinical tissue tightening of the dermis and subdermis following RF treatment is thought to result from heat-induced immediate collagen contraction, subsequent collagen remodeling, neocollagenesis, and the normal wound healing response. Patients experience heat in the region of each pulse, but it is much less uncomfortable when performed on the leg as opposed to the face. Mild self-limited erythema and edema are the most common



Figure 9.14 *Leg rejuvenation with radiofrequency: Pre- and 3 months postradiofrequency tightening of the skin of the upper leg and knee*

treatment responses. The side effect profile and safety of the procedure is similar to what has been reported for RF treatments of the face and neck.⁴¹ A specific algorithm of multiple passes at low energy, concentrating on the area of the leg where the skin laxity is most apparent, has lead to consistent tightening of the skin (Figures 9.14 and 9.15). However, the patients must be appropriately selected and have realistic expectations. RF treatment of the leg does not seem to be as predictable as RF treatment of the face. Other devices of delivering RF to the skin have been reported but definitive studies are pending.

Laser lipolysis with lipospiration

Patient selection is similar to that of tumescent liposuction. The procedure involves the removal of fat using a laser to melt fat cells and tighten skin. Three laser wavelengths (1064 nm, 1319 nm and 1320 nm) are available, with 1064 nm being the first to obtain FDA clearance. Less downtime and absence of the side effects associated with plain tumescent liposuction have been reported by all the manufacturers. These lasers appear to deliver effective, reproducible results safely and effec-

tively; however, there are currently no studies that directly compare the different wavelengths. The advantages of this procedure are excellent patient tolerance, quick recovery time, and the benefit of dermal tightening. A very small cannula, approximately 1.0 mm in diameter, contains the laser fiber and is inserted into the skin. The cannula is moved back and forth as the laser's energy is delivered directly to the fat cells, making them liquefy. Then they are aspirated or gently suctioned out of the skin. Because the fat cells are ruptured, they are easier to drain away. In addition, as the laser energy interacts with the dermis it results in skin shrinkage or tightening. Due to the small size of the cannula, this procedure can be performed under tumescent anesthesia. It usually requires less infiltration of tumescent anesthesia than traditional tumescent liposuction, which reduces the chance of lidocaine toxicity. It is postulated that these procedure causes less bleeding, swelling, and bruising, resulting in a shorter recovery time than conventional liposuction methods since the laser causes small blood vessels to coagulate immediately on contact. More than one procedure may be necessary especially if the treated area needs more skin tightening. After the treatment,



Figure 9.15 Rejuvenation of the leg with radiofrequency: (A) Pre and (B) one month posttreatment with smoothing of the inner thigh

patients wear a compression garment for several days to several weeks, depending on the body area being treated. Rest is recommended after the procedure and activity may be resumed gradually. Patients should refrain from hot tubs or strenuous exercise for 2 weeks.

Laser lipolysis is still an emerging technology. The most efficacious technique and laser settings have yet to be determined. A patient with irregularity after conventional liposuction shows improvement after laser lipolysis as seen in Figure 9.16.

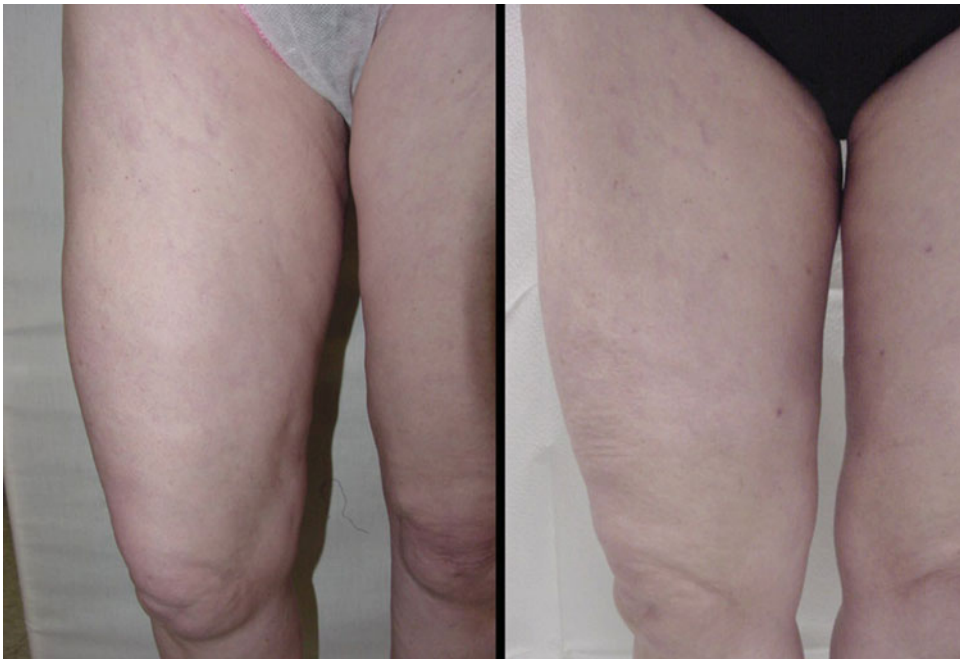


Figure 9.16 Laser lipolysis: Surface irregularity of inner thigh treated with 1320-nm laser lipolysis with lipoaspiration showing smoothing of the inner thigh

REFERENCES

- Chiesa R, Marone EM, Limoni C, Volonte M, Schaefer E, Petrini O. Chronic venous insufficiency in Italy: The 24-cities cohort study. *Eur J Vasc Endovasc Surg*. 2005;30(4):422-429.
- Chiesa R, Marone EM, Limoni C, Volonte M, Schaefer E, Petrini O. Demographic factors and their relationship with the presence of CVI signs in Italy: The 24-cities cohort study. *Eur J Vasc Endovasc Surg*. 2005;30(6):674-680.
- Weiss RA, Weiss MA, Goldman MP. Physicians' negative perception of sclerotherapy for venous disorders: Review of a 7-year experience with modern sclerotherapy. *South Med J*. 1992;85:1101-1106.
- Darke SG, Baker SJ. Ultrasound-guided foam sclerotherapy for the treatment of varicose veins. *Br J Surg*. 2006;93:969-974.
- Weiss RA. Vascular studies of the legs for venous or arterial disease. [Review]. *Dermatol Clin*. 1994;12(1):175-190.
- Goldman MP, Weiss RA, Bergan JJ. Diagnosis and treatment of varicose veins—A review. [Review]. *J Am Acad Dermatol*. 1994;31(3:Part 1):393-413.
- Goldman PM. Sclerotherapy for superficial venules and telangiectasias of the lower extremities. *Dermatol Clin*. 1987;5:369-379.
- Weiss RA, Weiss MA. Incidence of side effects in the treatment of telangiectasias by compression sclerotherapy: Hypertonic saline vs. polidocanol. *J Dermatol Surg Oncol*. 1990;16:800-804.
- Davis LT, Duffy DM. Determination of incidence and risk factors for postsclerotherapy telangiectatic matting of the lower extremity: A retrospective analysis. *J Dermatol Surg Oncol*. 1990;16:327-330.
- Goldman MP, Sadick NS, Weiss RA. Cutaneous necrosis, telangiectatic matting, and hyperpigmentation following sclerotherapy. Etiology, prevention, and treatment. [Review]. *Dermatol Surg*. 1995;21(1):19-29.
- Rao J, Wildemore JK, Goldman MP. Double-blind prospective comparative trial between foamed and liquid polidocanol and sodium tetradecyl sulfate in the treatment of varicose and telangiectatic leg veins. *Dermatol Surg*. 2005;31(6):631-635.
- Brian C. Leach, Mitchel P. Goldman. Comparative trial between sodium tetradecyl sulfate and glycerin in the treatment of telangiectatic leg veins. *Dermatol Surg*. 2003;29(6):612-615.
- Tessari L, Cavezzi A, Frullini A. Preliminary experience with a new sclerosing foam in the treatment of varicose veins. *Dermatol Surg*. 2001;27(1):58-60.
- Alos J, Carreno P, Lopez JA, Estadella B, Serra-Prat M, Marinell-Lo J. Efficacy and safety of sclerotherapy using polidocanol foam: A controlled clinical trial. *Eur J Vasc Endovasc Surg*. 2006;31(1):101-107.
- Weiss RA, Weiss MA. Painful telangiectasias: Diagnosis and treatment. In: Bergan JJ, Weiss RA, Goldman MP, eds. *Varicose Veins and Telangiectasias: Diagnosis and Treatment*. 2nd ed. St. Louis, MO: Quality Medical Publishing, Inc.; 1999:389-406.
- Adrian RM. Treatment of leg telangiectasias using a long-pulse frequency-doubled neodymium: YAG laser at 532 nm. *Dermatol Surg*. 1998;24(1):19-23.
- Garden JM, Tan OT, Kerschmann R, et al. Effect of dye laser pulse duration on selective cutaneous vascular injury. *J Invest Dermatol*. 1986;87(5):653-657.
- Polla LL, Tan OT, Garden JM, Parrish JA. Tunable pulsed dye laser for the treatment of benign cutaneous vascular ectasia. *Dermatologica*. 1987;174(1):11-17.
- Goldman MP, Eckhouse S. Photothermal sclerosis of leg veins. ESC Medical Systems, LTD Photoderm VL Cooperative Study Group. *Dermatol Surg*. 1996;22(4):323-330.
- Green D. Photothermal sclerosis of leg veins [letter; comment]. *Dermatol Surg*. 1997;23(4):303-305.
- Weiss RA, Weiss MA. Early clinical results with a multiple synchronized pulse 1064 nm laser for leg telangiectasias and reticular veins. *Dermatol Surg*. In press; 1998.
- Sadick NS, Prieto VG, Shea CR, Nicholson J, McCaffrey T. Clinical and pathophysiologic correlates of 1064-nm Nd:YAG laser treatment of reticular veins and venulectasias. *Arch Dermatol*. 2001;137(5):613-617.
- Omura NE, Dover JS, Arndt KA, Kauvar AN. Treatment of reticular leg veins with a 1064 nm long-pulsed Nd:YAG laser. *J Am Acad Dermatol*. 2003;48(1):76-81.
- Baumler W, Ulrich H, Hartl A, et al. *Br J Dermatol*. 2006;155(2):364-371.

25. Coon WW, Willis PW, Keller JB. Venous thromboembolism and other venous disease in the Tecumseh community health study. *Circulation*. 1973;48:839-846.
26. Lofgren KA. Varicose veins: their symptoms, complications, and management. *Postgrad Med*. 1979; 65(6):131-139.
27. Weiss RA, Weiss MA. Controlled RF endovenous occlusion using a unique RF catheter under duplex guidance to eliminate saphenous reflux: 2 years follow-up. *Dermatol Surg*. 2002;28(1):38-42.
28. Merchant RF, Pichot O. Long-term outcomes of endovenous radiofrequency obliteration of saphenous reflux as a treatment for superficial venous insufficiency. *J Vasc Surg*. 2005;42(3):502-509.
29. Proebstle TM, Lehr HA, Kargl A, et al. Endovenous treatment of the greater saphenous vein with a 940-nm diode laser: Thrombotic occlusion after endoluminal thermal damage by laser-generated steam bubbles. *J Vasc Surg*. 2002;35(4):729-736.
30. Weiss RA. Comparison of endovenous radiofrequency versus 810 nm diode laser occlusion of large veins in an animal model. *Dermatol Surg*. 2002;28(1):56-61.
31. Min RJ, Zimmet SE, Isaacs MN, Forrestal MD. Endovenous laser treatment of the incompetent greater saphenous vein. *J Vasc Interv Radiol*. 2001; 12(10):1167-1171.
32. Navarro L, Min RJ, Bone C. Endovenous laser: A new minimally invasive method of treatment for varicose veins—Preliminary observations using an 810 nm diode laser. *Dermatol Surg*. 2001;27(2): 117-122.
33. Min RJ, Khilnani N, Zimmet SE. Endovenous laser treatment of saphenous vein reflux: Long-term results. *J Vasc Interv Radiol*. 2003;14(8):991-996.
34. Sharif MA, Soong CV, Lau LL, Corvan R, Lee B, Hannon RJ. Endovenous laser treatment for long saphenous vein incompetence. *Br J Surg*. 2006; 93(7): 831-835.
35. Mozes G, Kalra M, Carmo M, Swenson L, Gloviczki P. Extension of saphenous thrombus into the femoral vein: A potential complication of new endovenous ablation techniques. *J Vasc Surg*. 2005;41(1):130-135.
36. Proebstle TM, Moehler T, Gul D, Herdemann S. Endovenous treatment of the great saphenous vein using a 1,320 nm Nd:YAG laser causes fewer side effects than using a 940 nm diode laser. *Dermatol Surg*. 2005;31(12):1678-1683.
37. Munavalli GS, Weiss RA. Advances in techniques for endovenous ablation of truncal veins. *Skin Therapy Letter*. 2006;11(3):4-7.
38. Ramelet AA. Muller phlebectomy. A new phlebectomy hook. *J Dermatol Surg Oncol*. 1991;17:814-816.
39. Ramelet AA. Le traitement des telangiectasies: Indications de la phlebectomie selon muller. *Phlebol* 1995;47(4):377-381.
40. Housman TS, Lawrence N, Mellen BG, et al. The safety of liposuction: Results of a national survey. *Dermatol Surg* 2002;28(11):971-978.
41. Weiss RA, Weiss MA, Munavalli G, Beasley KL. Monopolar radiofrequency facial tightening: A retrospective analysis of efficacy and safety in over 600 treatments. *J Drugs Dermatol*. 2006;5(8):707-712.

CHAPTER 10 Breast Rejuvenation

Theodore Diktiban and Joan L. Monaco

KEY POINTS

- The breast form confers a significant degree of physical and mental well-being for a female patient.
- Pregnancy, minor weight fluctuations, and massive weight losses or gains are contributing factors to the aging process.
- Rejuvenation consists of restoring a youthful breast contour and volume in the surgically appropriate patient.

ANATOMIC LANDMARKS

The female breast is situated over the pectoralis major muscle between the second and sixth ribs, and is anchored to the pectoralis fascia by suspensory ligaments referred to as Cooper's ligaments.¹ These ligaments and the skin envelope both determine how the breast will change with weight fluctuations, pregnancy, and aging. The nipples lie above the inframammary fold at the level of the fourth rib and immediately lateral to the midclavicular line. A common surgical measurement is from the sternal notch to the nipple, which is typically 21 cm. The inframammary-fold-to-nipple distance is also an important presurgical measurement and averages approximately 7 cm.² The breast is innervated by the second to sixth intercostal nerves (Figure 10.1a), whereas the nipple–areola complex derives sensation from the third through fifth anterior and lateral cutaneous nerves.³ The blood supply to the breast (Figure 10.1b) is through the second to fifth perforators of the internal mammary artery, which provides roughly 60% of the total blood supply. In addition, the thoracocromial, the serratus anterior, the lateral thoracic, and the terminal branches of the third to eighth intercostal vessels provide the remaining blood supply to the breast.^{4,5}

Venous drainage of the breast consists of the superficial and the deep systems. The superficial venous system is divided into two types: transverse and longitudinal. The transverse veins run medially and empty into the internal mammary veins, while the longitudinal system ascends

to the suprasternal area. Deep venous drainage is primarily through perforating branches of the internal mammary vein. There are anastomotic connections across the midline in the superficial system.⁶

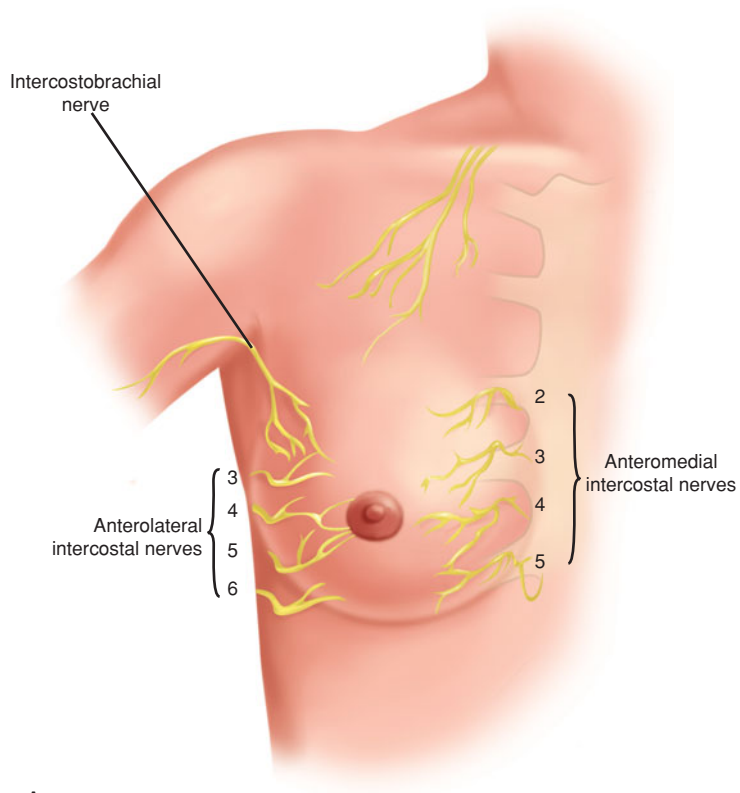
Breast ptosis tends to increase the sternal-notch-to-nipple distance and the inframammary-fold-to-nipple distance. Breast rejuvenation surgery attempts to reestablish preptosis measurements as well as breast volume and contour. Subsequently, crucial measurements during preoperative evaluation include sternal-notch-to-nipple distances (measured bilaterally), inframammary-fold-to-nipple distances, internipple distance, and base breast width. These breast measurements with careful examination of the breast itself and the underlying chest wall help determine any discrepancies between the two breasts in order to tailor surgery to a patient's particular anatomic needs.

PATIENT SELECTION AND PREOPERATIVE EVALUATION

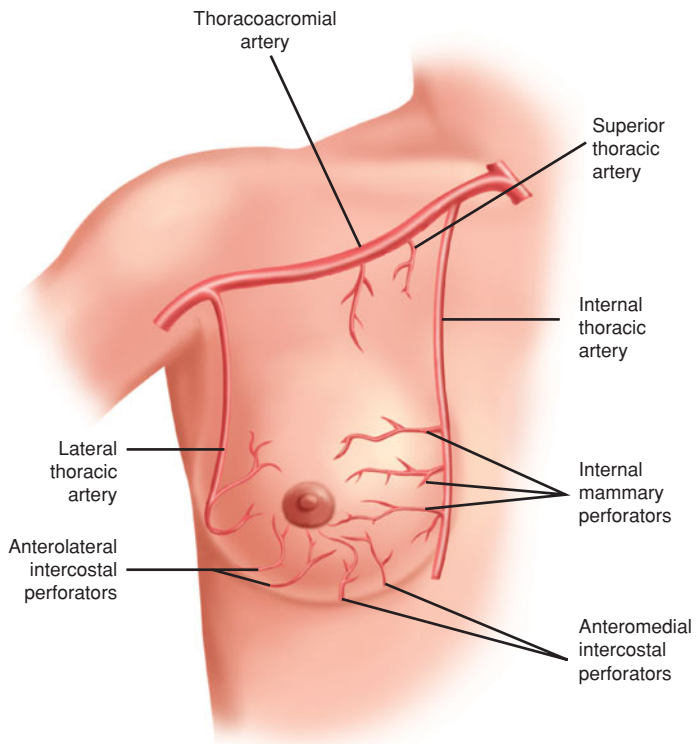
Patients who experience pregnancy with or without breast-feeding commonly develop ptosis and loss of upper pole breast volume. These patients often desire rejuvenation of the breast that raises the aged breast and restores the nipple–areola complex to a higher, more youthful size, position, and upward direction. They also desire to reestablish a prepregnancy breast volume or prefer to enlarge their breasts to a desired size.

Patients who have symptomatic macromastia since puberty or early adulthood, or those who have gained weight with aging often choose to have their breasts reduced to a size that is manageable to alleviate potential back and neck pain or inframammary skin irritations from constant skin-to-skin contact from pendulous breasts. Others who are massive weight loss patients after gastric bypass, laparoscopic banding, or traditional diet and exercise regimens choose to raise their breasts to a higher and more youthful appearance.

The scope of patients seeking breast rejuvenation is broad, with many surgical options available to tailor to an individual patient's body habitus and desires. Breast rejuvenation procedures are typically quite safe barring other medical comorbidities, and are most commonly performed on an outpatient basis with minimal postoperative



A



B

Figure 10.1 (A) *Innervation* and (B) *blood supply of the breast*

pain. Satisfaction of patients is high and the results after breast rejuvenation surgery are reasonably predictable.

Of the nearly 11 million cosmetic procedures performed, augmentation mammoplasty was the most commonly performed cosmetic surgery in women in the United States in 2006.⁷ It is estimated that more than 1% of the adult female population in the United States has undergone augmentation mammoplasty.⁸ The popularity of the procedure is thought to be related to patient satisfaction⁹ and with the availability of silicone gel implants, as of late 2006, after a 14-year restriction by the Food and Drug Administration (FDA), the procedure is projected to become even more popular in the coming years.

According to a survey conducted by the FDA,¹⁰ 91% of women undergoing augmentation mammoplasty wanted to look better without clothes, while 8% wanted to feel better about themselves. A self-motivated patient will, most commonly, experience enhanced self-image, increased self-assurance, and improved sexual functioning after breast rejuvenation, particularly after breast augmentation.¹¹ Yet, appropriate patient selection is crucial for surgical planning, and ultimately patient satisfaction. Patients with significant psychiatric history or patients who have unreasonable expectations should be evaluated with caution.

Accurate preoperative evaluation should be performed for each breast surgery candidate. Mammograms for patients over the age of 35 are crucial for preoperative detection and baseline documentation. Comorbidities that could escalate a patient's surgical risk should be taken into account. Patients should also receive a post-operative mammogram at a minimum of 6 months after surgery to establish a new baseline study. Important areas of investigation prior to any breast surgery include review of the patient and family history for breast and/or ovarian cancers. The review of the general medical background should assess the patient's history of bleeding disorders, diabetes, collagen disorders, autoimmune diseases, abnormal scarring tendencies, and lastly, a history of smoking. The patient's motivations and expectations are important to illicit prior to surgery.

Breast asymmetry, breast ptosis, patient height, shape of the patient's thorax, and length of the patient's thorax to trunk are evaluated on initial assessment (Figure 10.2A).¹² Varying degrees of breast asymmetry are quite common and subtle asymmetries must be pointed out to the patient preoperatively to help establish reasonable goals and expectations.

Compliance of the patient's breast tissue envelope and the presence of breast scars and striae, in addition to the degree of breast ptosis, will help determine implant size, position, and incisional approach. A soft tissue pinch of the upper pole of the breast will determine if there is sufficient soft tissue coverage necessary to cover a subglandular implant. A soft tissue pinch of 2 cm is recognized as the acceptable amount for subglandular implant placement.^{13,14} A patient with a smaller volume of soft tissue pinch can experience visible implant rippling with subglandular placement. This particular candidate might benefit more from a subpectoral placement.

Similar preoperative measurements are recorded for breast reduction or mastopexy procedures. For these rejuvenating procedures, the new breast position is ideally placed between 18 and 26 cm from the sternal notch. The nipple diameter is often grossly enlarged in patients with large breasts. A reduction in the size of the areola is a required step in reduction or mastopexy procedures. The areola is designed with a diameter of 38 to 50 mm.¹⁵ Careful attention is placed on positioning the nipple because an overly elevated nipple position is a difficult problem to correct. Pitanguy recommended use of the midhumeral point plus 2 to 3 cm for determining the new nipple level.¹⁶ The desired nipple position can also be determined by locating it on the breast skin at the level of the inframammary fold (Figure 10.2b).

Final breast size can often be difficult for a patient to verbalize for both augmentation and reduction procedures. With lingerie marketing strategies that market bras at inflated cup sizes, patients' cup measurements are often quite different from accepted surgical standards. Cup volume is estimated by first measuring the patient's chest circumference. The chest circumference over the most prominent portion of the patient's breast is then measured (chest or breast circumference). A 1-in. increased measurement in the chest or breast circumference compared with the chest alone measurement is regarded as an "A" sized cup. A 2-in. increase would be a "B" cup and so forth. Guaranteeing a patient a particular cup size after surgery is not always predictable and not advised. It has been an accepted surgical standard that a 250-mL volume increase will increase a patient's breast by one cup size, although this volume should be used only as a frame of reference for the patient and the surgeon. Some other reports suggest that a volume increase of 100 mL will increase a patient's cup volume by one size.¹⁷ Having patients place different size implants in a sports bra

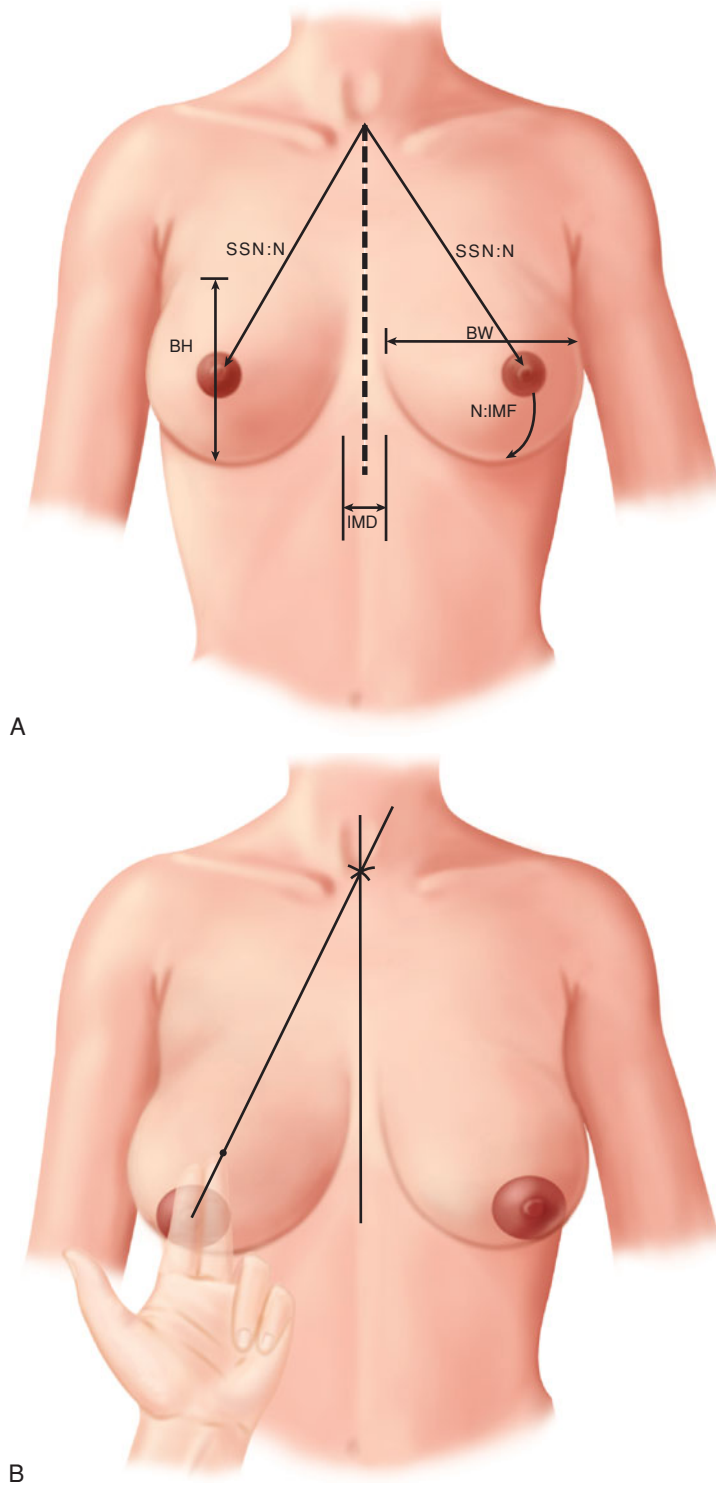


Figure 10.2 (A) Preoperative measurements of the breast and (B) nipple placement

can help them to determine the final look they are hoping to achieve during an augmentation procedure.

On the other hand, women planning to have a breast reduction also need to express their desired shape and volume. For these patients, the use of photographs depicting different breasts might facilitate the decision-making process. The volume that is safe to resect in order to preserve flap and nipple viability is at times less than what the patients would ultimately want resected. This discrepancy must be explained to the patient in order to create reasonable surgical goals for both the surgeon and patient.

Breast hypertrophy may be categorized into mild, moderate, major, and gigantic, which are represented by resections of less than 200 g, 200 to 500 g, 500 to 1500 g, and more than 1500 g per side.¹⁸ The amount of breast hypertrophy determines the size of safe reduction to preserve nipple viability. Once resections approach 1800 to 2000 g per side, breast amputation with free nipple graft may be considered.

TECHNIQUE

■ Breast Augmentation

Axillary, periareolar, inframammary, and transumbilical incisions are the standard choices for placement of breast prostheses. The surgeon's experience, the patient's preference, and the patient's body habitus/breast shape will determine the appropriate approach. Patients with asymmetric breasts or uneven inframammary folds that need to be corrected at the time of augmentation are often better treated with an inframammary or periareolar approach. Periareolar approaches may not be suitable for patients with small areolar diameters. The transaxillary approach that was described in the early 1970s underwent a significant transformation with the introduction of endoscopic instrumentation in the mid 1990s. The endoscope changed a formerly blind approach into a fully visualized one. The transumbilical approach is typically avoided in patients with long thoraces or with uneven inframammary folds, as correcting this can be difficult from this more remote access incision. Each surgical approach has advantages and disadvantages and requires a consensus between patient and surgeon for the determination of preoperative surgical goals and ultimately patient satisfaction.

Breast implants

Saline versus silicone With the FDA's approval of silicone implants in 2006, patients now have more choices when selecting an implant for breast augmentation. The use of silicone implants in the United States was possible without any restrictions from the mid 1960s until January 1992 when the FDA placed a moratorium on the use of these implants.¹⁹ From 1992 until 2006, women were able to have silicone implants only for reconstructive conditions. The decision to make them available to the public came after many years of lawsuits, bankruptcies, petitions, and supporting statistics from several accredited research groups. Currently, the choice between saline and silicone gel implants is one of patient's preferences after appropriate relay of information by the surgeon. Both saline and silicone implants are made with a silicone elastomer shell. Silicone gel implants are thought to feel softer and look more natural with less rippling. Saline implants, on the other hand, have a lower incidence of capsular contracture and require a smaller incision for their placement.²⁰ Importantly, a failed saline implant will be clinically detectable, whereas a failed silicone implant may be associated with a silent rupture. This has led the FDA to educate patients into having MRIs of the breasts on a periodic basis in order to detect any ruptures.

Textured versus smooth The distinction between textured versus smooth implants is made to minimize capsular contracture. There are numerous reports in the literature stating a lower rate of capsular contracture in textured implants compared to smooth implants.²⁰⁻²³ Various surveys over the years, obtained from plastic surgeons, revealed that round, smooth saline implants were the commonly used implant for augmentation purposes. Both textured and smooth implants come in different projection and width profiles that are individualized to the patient.

When an implant is placed in a subpectoral plane, either a textured or a smooth implant can be used with comparable results and contracture rates. When an implant is placed in a subglandular plane, textured implants have been found to have a lower capsular contracture rate compared to smooth implants. Textured implants should be used in a subglandular plane if the patient has adequate breast tissue for coverage from visible rippling. Textured implants have thicker shells thereby making visible rippling more likely over smooth implants.

The anatomic textured implant was designed to imitate the natural breast. It provides the patient the benefit of overall breast enlargement without excessive

upper pole fullness. Anatomic textured implants would benefit a patient with highly mobile parenchyma that may potentially slide off the surface of a round implant and produce a double-bubble deformity.²⁴ Anatomic implants have been reported to produce greater projection and height.²⁵ However, the recent release of saline implants with “moderate plus” categorization of height provides additional implant height without increasing the base diameter of the implant significantly. If a patient only requires an increase in volume, a round implant would be easier to use and would provide an aesthetic result. Anatomic implants are slightly more challenging to implant as the parenchymal pocket must be the appropriate size and not too large as rotation of the implant would produce an altered breast shape.

Implant position Implant placement in a subpectoral plane (Figure 10.3) confers the lowest risk for capsular contracture, and using the pectoralis muscle to completely or partially cover the implant provides an additional layer of biologic coverage against implant extrusion.²⁶ Subpectoral placement also provides a smoother breast contour with less rippling as the edges of the implant are blunted by the muscle itself providing additional soft tissue coverage. Furthermore, creating a subpectoral pocket provides greater protection of nipple sensation over a subglandular dissection.

Placement of a breast implant in a subglandular plane will help correct mild ptosis in a patient who does not require a formal mastopexy or wishes to avoid this procedure for additional cost or scarring concerns. In patients with a pinch test of greater than 2 cm, implants can be

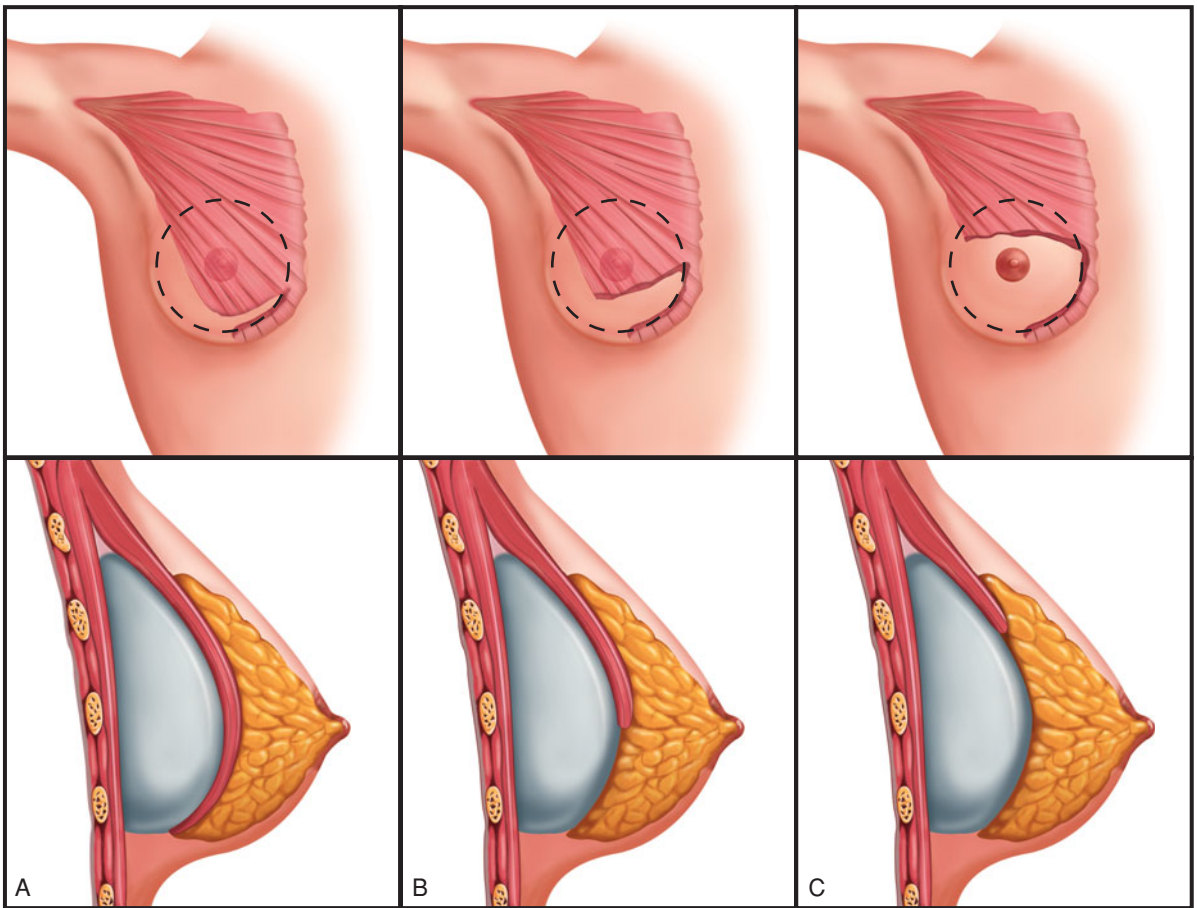


Figure 10.3 Subpectoral implant placement results in varying degrees of muscle coverage of the upper portion of the implant and glandular coverage of the lower portion

safely placed in a subglandular plane with minimal implant visibility or rippling visibility.

The decision to place an implant in a subpectoral versus a subglandular plane is made in conjunction with the patient's body habitus or breast volume as well as the implant choice. Saline implants placed in the subpectoral plane lessen the incidence of visible rippling. Smooth implants are safer in the subpectoral plane to protect against higher capsular contracture rates in the subglandular plane. Capsular contracture rates have been reported by numerous authors to be lower in the subpectoral plane over the subglandular plane.^{27–31} Dual-plane maneuvers where the surgeon creates an implant pocket with varying degrees of subpectoral to subglandular implant coverage have also been described.³² These techniques permit alteration of the muscle–parenchyma interface that helps to protect against a double-bubble phenomenon. When performing a dual-plane technique, it is of paramount importance to preserve the medial attachments of the pectoralis in order to protect against symmastia and visibility of the edges of the implant.

The subpectoral fascial pocket has also been described recently.³³ The subpectoral fascial approach is thought to offer the advantages of a subglandular placement with a slightly thicker soft tissue cover. The dissection in this tissue plane requires a more tedious technique and the fascial layer itself varies in thickness over the pectoral muscle and varies from patient to patient.

■ Breast Reduction

Numerous techniques have been described to perform the appropriate reduction mammoplasty or mastopexy procedure for a patient. The specific technique is chosen on the basis of the breast size, estimated resection volume, breast shape, and minimization of surgical scar. Most commonly, the skin resection pattern for reduction mammoplasty entails the use of an inverted-T scar or the vertical mammoplasty technique. The choice of the skin pattern depends upon surgeon preference and the amount of resection volume planned.

Liposuction-assisted reduction

The use of liposuction alone or in conjunction with excisional reduction (Figure 10.4) has been in patients with predominantly fatty breasts and nipples in a normal, nonptotic position. Patients are typically satisfied with the lack of breast scars; however, breast volume is often not dramatically changed, the breast is not reshaped, and nipple elevation is modest at best. Other concerns include the formation of fat necrosis after suction lipectomy and the difficulty in examining the liposuction aspirate from a histological point of view. Most examiners believe that breast tissue proper is not predictably aspirated during a conventional liposuction procedure.

Dermal-parenchymal pedicle reduction

Many variations in technique have been described to reduce the breast while preserving the subdermal plexus

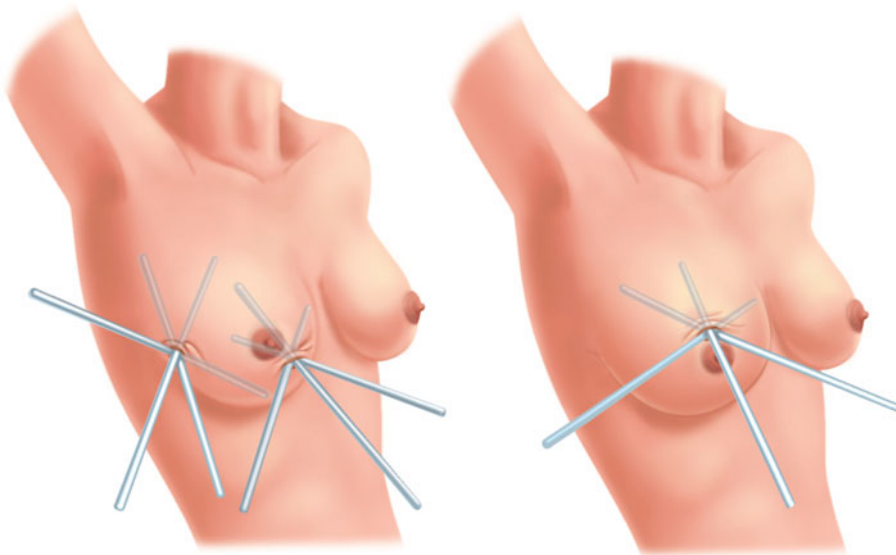


Figure 10.4 Breast reduction liposuction technique

that supplies the nipple–areola complex. Pedicles can be based superiorly, superomedially, medially, superolaterally, laterally, and inferiorly. The ultimate shape of the reduced breast is determined by the parenchymal resection pattern and not the pedicle. All pedicle variations have been described in the literature with varied long-term results and complication rates. Reliability and long-term preservation of breast shape are essential with any pedicle chosen. The degree of breast hypertrophy, ptosis, the character of the skin envelope, and preference of the surgeon contribute to the pedicle choice and skin resection pattern.

The superior pedicle technique (Figure 10.5) is limited by increased difficulty in moving the nipple longer distances in larger breasts. The inferior pedicle technique is considered very versatile and reliable and is very commonly used. Nipple insensitivity has been reported in as low as 1.3% of patients with as high as 72% being able to secrete milk postoperatively after inferior pedicle reduction.³⁴ Other variations include the vertical bipedicle, horizontal bipedicle, lateral pedicle, and medial pedicle techniques. All techniques have been met with both acceptance and criticism and ultimately are chosen by the surgeon on the basis of patient's specific anatomy.

Skin reduction patterns

The Wise pattern skin reduction remains the most commonly used skin reduction pattern in reduction mammoplasty. Patients are marked preoperatively in the upright position and the new nipple position is marked by transposing the level of the inframammary fold onto the anterior breast skin. Careful attention is paid toward not marking the new nipple–areola complex position too high as this is a difficult problem to fix postoperatively. The final scar consists of a periareolar scar with a descending scar down the central breast mound and a horizontal inframammary scar, often referred to as the inverted-T or anchor scar.

Short-scar techniques have been widely accepted outside of the United States for decades and now are commonly used in the appropriate patient in the United States. This skin resection pattern has been modified to reduce the amount of scar on the breast. These techniques fall into five categories including the vertical mammoplasty, the vertical mammoplasty with horizontal extension, the L-scar mammoplasty, the horizontal mammoplasty, and the periareolar mammoplasty.

The vertical mammoplasty technique (Figure 10.6) was first described in 1964 as the “resection en bloc of skin, fat, and gland; transposition of the areola on a supe-

riorly based flap; no undermining; and a vertical scar.”³⁵ A 30-year follow up of this technique reported minimal nipple necrosis, minimal nipple insensitivity, and stable long-term results.^{36,37} Modification of this early technique has been described. One common variation included skin undermining adjacent to the vertical parenchymal pillar edges, suture suspension of the pedicle to the pectoralis major fascia, total breast liposuction, and tumescent fluid infiltration into the breast gland.³⁸ Follow up of this technique reported excellent shape retention.³⁹ Another reported modification included a medial pedicle, no skin undermining, targeted liposuction, and no pectoralis fascia suturing.⁴⁰ Furthermore, another modification entailed wide undermining of the skin of the lower pole to allow gathering of the skin along the vertical closure.⁴¹ The vertical mammoplasty with horizontal extension is similar in technique to the dog-ear that is formed after resection of the vertical closure at the new submammary fold by converting it to a short horizontal scar. The horizontal limb varies from as a short as 3 cm to almost 7 cm in length.

The L-scar reduction mammoplasty technique eliminates the medial horizontal limb of the inverted-T closure and when originally described, was tailored for a patient who required less than 7 cm of nipple transposition with a mild to moderate reduction.⁴² This technique has been modified by several authors with excellent reported long-term results.

The horizontal mammoplasty (Figure 10.7) was originally described using a central parenchymal mound or an inferior pedicle followed by moving the nipple–areola complex to its new higher position with inset through a circular incision. It was initially described for patients requiring moderate to large reductions and vertical repositioning of the nipple complex by at least 7.1 cm. The results were initially referred to as “standing” cones where the skin of the vertical limbs was gathered at the inferior pole with 20% of these patients requiring revision in the following months.⁴³

The periareolar reduction technique involves creating a surgical resection plane at the areola–skin junction with a purse-string closure around the newly reduced areolar diameter. The purse-string suture technique is commonly associated with an intraoperative scalloping effect that flattens with time provided that skin tension forces and geometric principles are adhered to. Patients who are appropriate candidates for this skin resection pattern will benefit from minimal scarring. The technique, like a similar mastopexy procedure for ptosis, has been criticized as

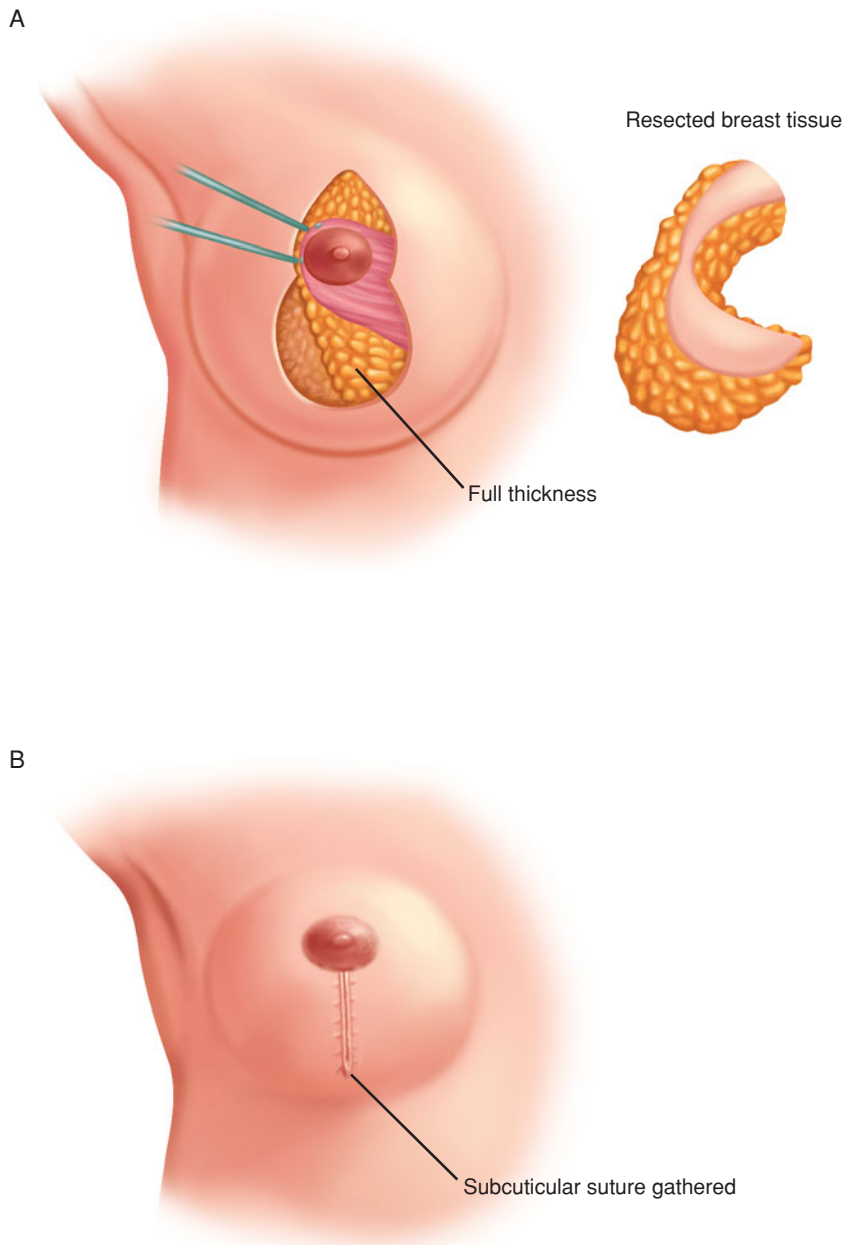


Figure 10.5 Vertical breast reduction using a superomedial pedicle

forming a flat, underprojected breast and is also associated with the possibility of areolar widening with time despite the use of a permanent suture for purse-stringing. This technique is considered best for patients who require limited skin resection and small to moderate volume reductions. The problem of widened areolae led to the use

of a Gore-Tex purse-string suture. The short scar periareolar inferior pedicle reduction mammoplasty consists of an inferior pedicle with medial, superior, and lateral glandular resection, parenchymal suspension sutures, permanent periareolar purse-string sutures with Gore-Tex, and inferior pole skin resection.⁴⁴

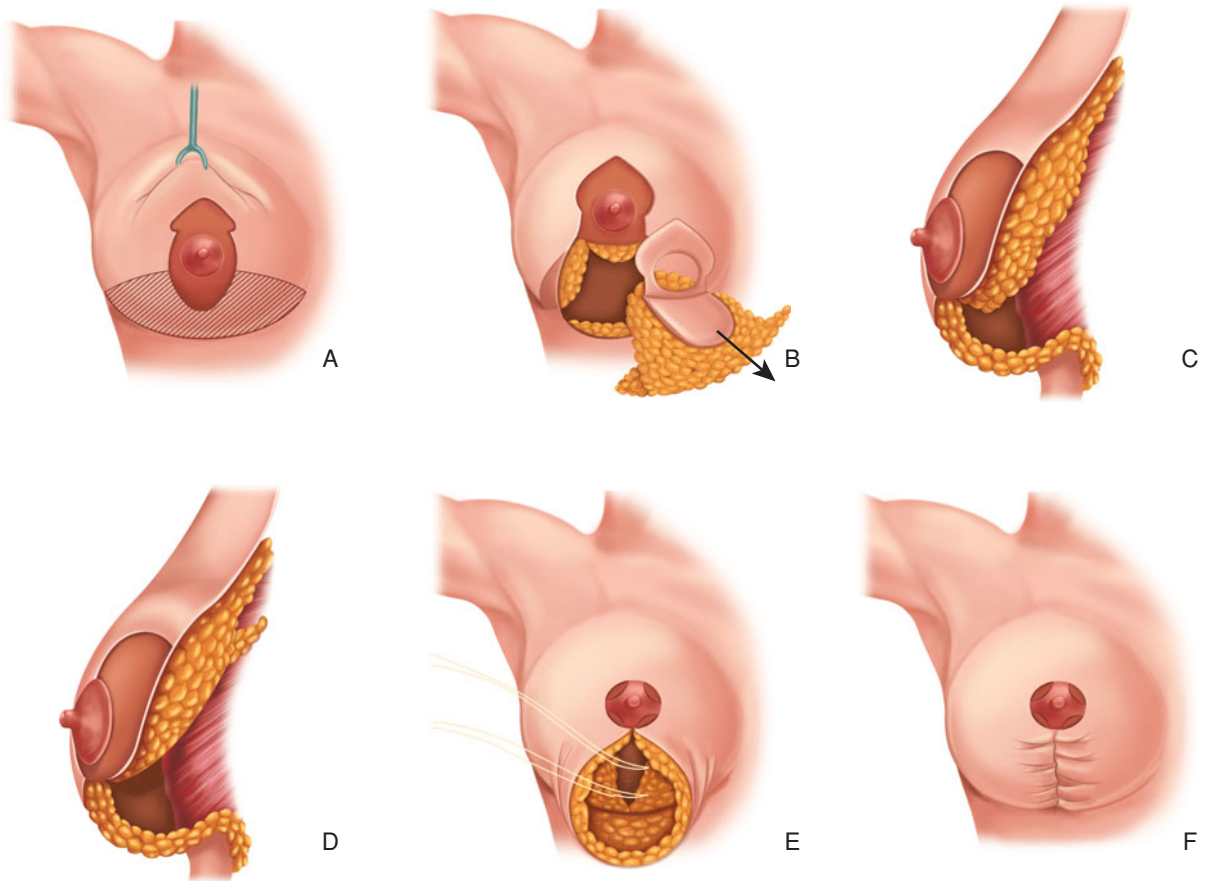


Figure 10.6 Modified vertical breast reduction. (A) Preoperative marking, (B) deepithelialization and glandular resection, (C) removal of glandular tissue, (D) elevation of breast by plication and fixation to muscle fascia, (E) suture of lateral pillars of breast tissue to create breast cone, and (F) vertical skin closure

Free nipple grafting has been performed for women with gigantomastia, severe hypertrophy, or systemic disease that could affect the vascularity of the skin flaps. This technique was first described in 1922⁴⁵ and has been criticized for creating a flattened breast as well as the obvious criticism of no nipple sensation, although later reports have established that a moderate number of patients have some sensation after 6 months or more. Another problem is depigmentation of the nipple graft in non-white patients. Tattooing has been performed as a salvage method with modest improvement. Some surgeons argue that patients with gigantomastia can be safely and effectively reduced using a pedicled technique and free nipple grafting need not be considered. The advantages of this technique include shorter operating time, reduced blood loss, and fewer wound-healing

complications as the technique classically does not involve flap undermining.⁴⁶

■ Breast Mastopexy

A mastopexy procedure entails lifting of the breast to a natural youthful position (Figure 10.8). The breast reduction procedure involves reduction in breast volume and the overlying skin envelope, whereas the mastopexy procedure essentially entails reducing only the skin envelope while maintaining the inherent breast volume. Performed concurrently with breast augmentation, the mastopexy/augmentation proves to be one of the more challenging of plastic surgery procedures because the surgeon is now balancing two operations into one.

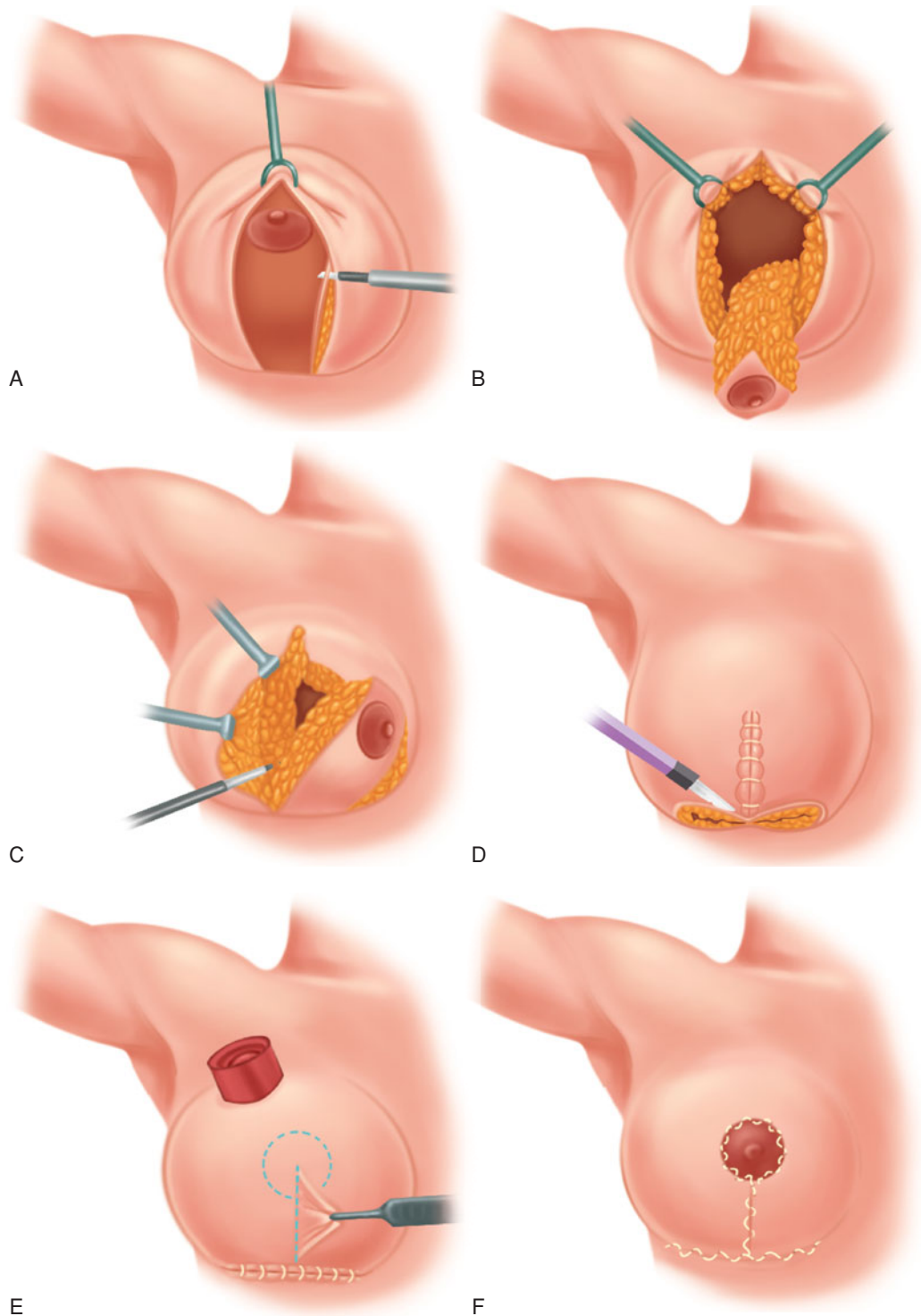


Figure 10.7 Horizontal breast reduction using central pedicle. (A) Deepithelialization, (B) flaps are freed from surrounding tissue, (C) excision of glandular tissue, (D) temporary closure of vertical limb with staples and excision of “dog-ears,” (E) marking of nipple–areola site, and (F) final closure

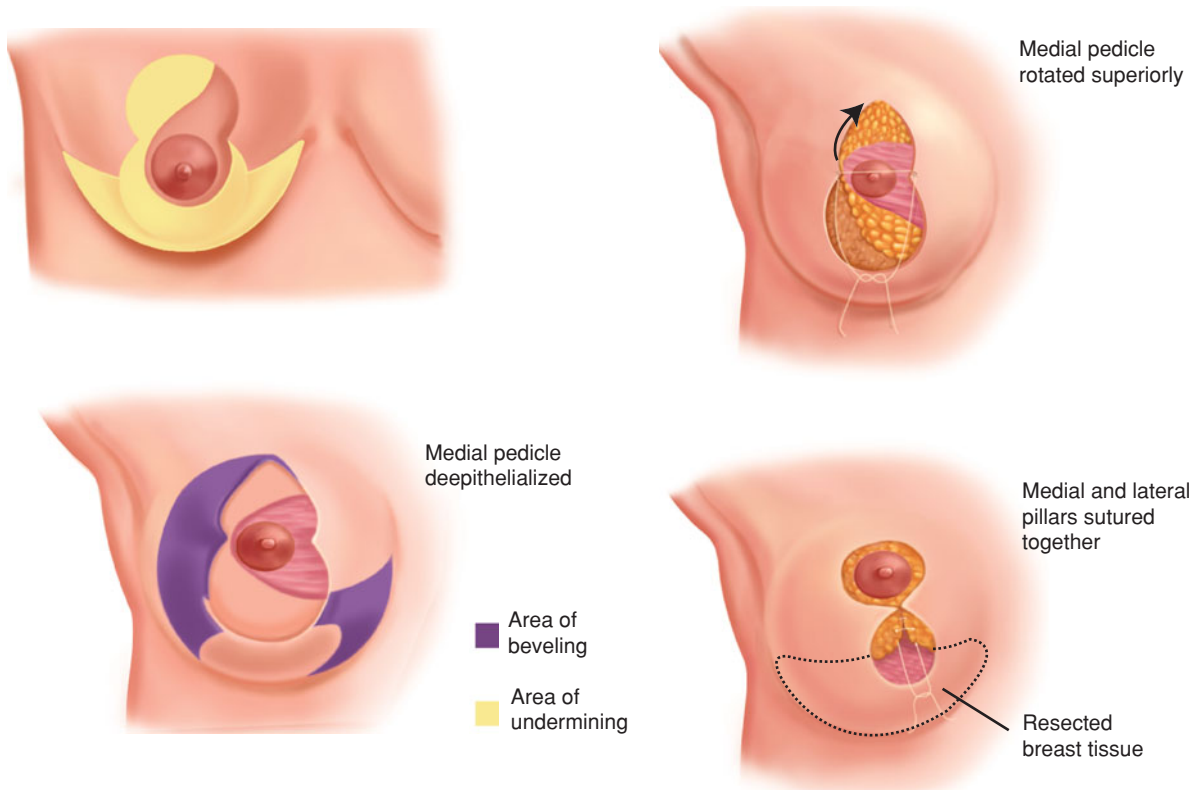


Figure 10.8 *Mastopexy technique using medial pedicle*

Breast ptosis was first classified by Regnault in 1976.⁴⁷ First-degree ptosis is a mild ptosis where the nipple is within 1 cm of the level of the inframammary fold and above the lower contour of the gland. Second-degree ptosis is a moderate ptosis where the nipple is 1 to 3 cm below the level of the inframammary fold but above the lower contour of the gland and skin envelope. Third-degree ptosis is a severe ptosis where the nipple is more than 3 cm below the lower contour of the breast and skin envelope. The fourth classification is pseudoptosis where the nipple is at or above the level of the inframammary fold with the majority of the breast volume sitting below the inframammary fold. Ptosis is primarily caused by involution after pregnancy or weight loss. There is a loss of skin elasticity secondary to aging, which also contributes to the problem.

Numerous procedures have been described to correct breast ptosis but many are similar to breast reduction skin resection patterns. The varied techniques can be categorized into three basic incisional patterns: periareolar, vertical scar, and inverted-T scar. Similar to breast

reduction technique, careful attention must be paid toward not elevating the nipple to an unnaturally high final position. The skin pattern can be determined by infolding the central and inferior skin and bringing together the key points to assess tension and final breast appearance.

The periareolar technique for ptosis correction creates limited scars on the breast mound and confines the scar to the areola-skin interface, which is usually a minimally noticeable scar. The difficulty with this approach is the probability of nipple sensitivity change as the initial incision is circumferential around the areola. Furthermore, slight variation of the incision into the areola or away from the areola-skin interface onto the skin will produce a noticeable scar. The periareolar or donut mastopexy^{48,49} is useful for mild to moderate ptosis and can be tailored from a circumferential scar to a crescent-shaped scar depending on the patient's needs. The advantage of this approach is camouflaged scarring. Disadvantages include the possibility of poor scarring from poor incision placement as well as the possibility of a widened

scar from excess tension at the time of closure. Another potential problem is a flattened breast contour at the apex.

A modification of this technique is the perioareolar Bennelli mastopexy first described in 1990⁵⁰ that treats the skin and the breast gland as two separate components. The gland is accessed through the periareolar incision and is restructured by internal breast contouring sutures. The lower pole is tightened and then the skin envelope is redraped over this newly formed glandular scaffold. Criss-cross sutures are placed subdermally beneath the nipple–areola complex to diminish the pouting that occurs after purse-string techniques. A permanent purse-string suture of Gore-Tex or Mersilene is placed in the deep dermis of the skin edge as in the donut mastopexy to help control the closure tension and prevent areolar widening. This technique permits access to the gland and subsequently permits flexibility in gland reshaping. It is not recommended for breasts that have a large amount of skin excess especially if the envelope itself is of poor quality. It can be performed concurrently with a permanent breast implant.⁵¹

Vertical and short-scar mastopexy techniques entail a perioareolar scar with extension down the breast meridian to excise excess skin envelope. The vertical scar technique described by Lassus does not undermine the skin, permits a wedge resection of the gland if needed and transposes the nipple–areola complex to a higher position based on a superiorly based flap.⁵² This technique permits central wedge resection if needed and does not impair the blood supply to the gland. The use of a superiorly based flap preserves the neurovascular supply of the areola, thereby reducing the occurrence of necrosis or decreased nipple sensation. The risk for skin necrosis is minimal as this technique does not require skin undermining. It does, however, require a vertical scar and several months are often required for the breast to achieve final aesthetic shape.⁵³ This technique also relies upon adjusting the skin envelope with multiple temporary sutures or staples to determine how much skin is to be resected. Careful attention must be paid toward this repeated handling of the tissue.

Modification of this technique was described by Lejour. It entails using adjustable skin markings, a superior pedicle for the areola, central pedicle reduction, when necessary, with lower skin undermining. Undermining is thought to promote skin retraction and to reduce the amount of scarring. Liposuction of the breast mound is performed to facilitate shaping and to remove unnecessary fatty tissue,

which creates a more compact breast that will be less likely to contribute to the return of ptosis.⁵⁴

The short-scar mammaplasty is more commonly used for breast reduction but can be adapted for a mastopexy by taking the tissue that would be normally resected and folding it inward onto itself to provide superior pole fullness. This technique described by Chiari⁵⁵ is best for patients with moderate ptosis but ample breast volume where a loose skin envelope requires resection. The final scar does not have a medial inframammary component and the technique itself confers the ability to achieve a good final shape with adequate breast projection without distorting the areola. The preoperative markings can be difficult at first, making this one of the biggest drawbacks to this approach.

The combination of mastopexy with augmentation is useful for patients with deficient glandular tissue with skin envelope excess. This combined procedure is also useful in cases of breast asymmetry with deficient glandular volume. The addition of an implant will help fill the skin envelope but creates a larger risk for incision breakdown because of increased tension on the suture lines. Any mastopexy technique that requires undermining of skin flaps should not be combined with subglandular augmentation because this can contribute to glandular necrosis.

COMPLICATIONS TO AVOID

■ Operative Complications

Hematoma, seroma, and wound infection are the most commonly reported operative complications after augmentation mammaplasty, and mastopexy or reduction mammaplasty; the rates of these events may be made quite low with appropriate surgical technique, patient selection, and patient compliance. Hematoma development has both short-term and long-term consequences. Short-term consequences include pain, blood loss, need for reoperation, and breast disfigurement. Patients found to have a hematoma after breast augmentation in the perioperative period are treated with operative reexploration, implant removal, irrigation, and control of hemostasis before the implant is reintroduced into the breast pocket. Hematoma rates after augmentation mammaplasty have been reported at 3%.⁵⁶ Hematoma formation is related to higher rates of capsular contracture as high as 86%.⁵⁷ Postoperative management remains the same.

Delayed hematoma formation at 1 to 2 weeks postoperatively, or on rare occasions, of several years after initial surgery, is often related to a history of trauma. An expanding hematoma causing breast envelope tightness must be treated with surgical exploration. The rate of risk of developing capsular contracture after augmentation mammoplasty remains as high as in the perioperative hematoma formation.

Seroma formation of a small degree in the perioperative period is often reabsorbed by the body within the first week after surgery. Persistent seromas may require ultrasound-guided drainage or drain placement in refractory cases. The formation of perioperative seroma has been linked to overuse of electrocautery or the use of concentrated antibacterial irrigation fluid.^{58,59} It occurs more frequently with textured than with smooth implants.

Wound infection after augmentation mammoplasty can range from mild cellulitis to a severe purulent periprosthetic space infection with eventual implant extrusion if not aggressively treated. Infection after breast augmentation has been reported at 2.2%.⁶⁰ The most commonly associated organism in wound infection is *Staphylococcus epidermidis* and the most feared infectious concern is the development of toxic shock syndrome in the presence of *S. aureus*.^{61,62} Salvage of an infected implant is possible in a small percentage of cases but advanced or late infections are treated with surgical explantation and antibiotic therapy. Prevention begins with the appropriate careful surgical technique, minimal touch manipulation of the implant prior to placement, antibiotic irrigation of the breast pocket, and a single dose of intravenous first- or second-generation cephalosporin prior to incision. Any question of the severity of a wound infection should be treated with implant removal and aggressive antibiotic therapy. Reinsertion of the implants can take place in 3 to 6 months provided the soft tissues have healed satisfactorily.

The connection of the lactiferous ducts directly to the surface of the nipple contributes to colonization with microorganisms in as high as 90% of patients.⁶³ The nipple–areola complex has been thought of as a source of contamination during breast surgery and some surgeons advocate the use of an adhesive film barrier during augmentation⁶⁴ to avoid this infectious source, but this is not routinely practiced.

Mondor's disease is a rare self-limiting complication after breast augmentation that consists of a superficial thrombophlebitis in the veins on the inferior aspect of the breast. It may occur in as few as 1% to 2% of breast aug-

mentation patients and is treated with warm compresses.⁶⁵

Complications after reduction mammoplasty or mastopexy procedures are varied but the most common error in planning is placing the nipple too high. The nipple must be planned at the level of the inframammary fold as previously described. The nipple can also be misplaced to either side of the breast meridian.⁶⁶ Inadequate skin resection or overresection of the gland may produce a boxy, irregular-looking breast. An overly resected gland may leave insufficient gland for adequate projection. Asymmetry in size, shape, or nipple position is also a problem post breast reduction whether the breasts were symmetric or not to begin with. Symmastia can occur with breast augmentation if medial dissection is excessive or soft tissue attachments are tenuous. Every effort must be made at not trying to overly narrow the intermammary distance to achieve cleavage even with a demanding patient.

An overly narrow pedicle can lead to nipple loss or partial nipple ischemia producing a range of sequelae from nipple–areola depigmentation and scar hypertrophy to possible total nipple loss. Nipple–areola complex ischemia can be linked to hematoma and infection. Prior breast operations on the nipple–areola complex can predispose to ischemic or necrotic complications. Nipple loss after breast reduction is a reported phenomenon that is most commonly related to patient smoking. A 6% incidence after inferior pedicle and bipedicle techniques has been reported,⁶⁷ whereas, more commonly, superficial sloughing and epidermolysis is seen. Patients who present with this should be allowed to heal secondarily, but monitored wound care is essential along with comforting the patient.

Alterations in nipple sensation vary with the surgical technique used. The periareolar approach is generally associated with the highest rate of change in nipple sensation, and the transumbilical approach is thought to have the lowest rate after augmentation mammoplasty. A permanent change in nipple sensibility is roughly 3% to 5% overall after breast augmentation.⁶⁸ Preservation of the third, fourth, and fifth lateral intercostal nerves during surgical dissection is needed to maintain nipple sensibility postoperatively.

Change in nipple sensation is a well-known complication after reduction mammoplasty and incidences correlate with the technique used for the reduction. Women with gigantomastia have been reported to have lower sensory thresholds compared with small-breasted women⁶⁹

possibly due to traction injury to the intercostal nerves, and reports of improved nipple sensibility after breast reduction have been reviewed.⁷⁰ When comparing nipple sensation using superior versus inferior pedicle techniques, the breast skin has been reported to have better sensation after superior pedicle technique, whereas the areola has slightly better sensation after inferior pedicle technique; however, at 6 months, both groups had comparable nipple–areola complex sensitivities, although 50% of patients had less sensation than preoperative levels.⁴¹

Breast implants do not interfere with breast-feeding and silicone has not been identified in the milk of lactating women.⁷¹ Breast feeding after reduction mammoplasty is possible with different studies stating varied percentages of success. A woman should also be informed that the quantity of milk production might prove to be inadequate for nursing.^{72,73} Another study reported that only 19% of patients breast-fed successfully without requiring formula supplementation after inferior pedicle reduction.⁷⁴

Fat necrosis has been seen after breast reduction and has been loosely correlated with the overuse of electrocautery whereby many surgeons use knife or scissor dissection in place of electrocautery. Finally, minor to major skin flap loss can occur, particularly at the inverted-T junction, from overresection of the skin with a tight closure. In addition, the shear weight of the patient's breast creates a pressure point for wound breakdown.

■ Implant Complications

Rupture, deflation, displacement, and rippling are implant-related complications to augmentation mammoplasty. Saline implant rupture occurs less often with the improvement of the silicone elastomer shell but rupture rates are still reported to be as high as 5.5%.⁷⁵ Treatment consists of implant removal and replacement. Factors that contribute to saline implant rupture include trauma, underfilling, intraluminal antibiotics, implant irrigation with betadine solution, and the use of intraluminal steroids. Rupture with saline implants is easily noticeable by the patient as complete deflation of the implant will occur.

Rupture rates of silicone gel implants significantly depending on the type of implant used. Risk factors include age of the implant, history of trauma (including trauma from mammography), degree of capsular contracture, and type of implant. Silicone implant rupture

may be subclinical, but any ruptured implant, even without distortion of the breast shape, should be removed. Diagnosis may require MRI as mammography alone does not adequately diagnosis implant rupture.⁷⁶ MRI with a radiographic “linguine sign” representing fragments of a ruptured shell is the most accurate imaging modality for detection of rupture, followed by ultrasonography and mammography.⁷⁷

Silicone rupture has been categorized by the degree of silicone escape from the elastomer shell. Leak is classified as a small amount of gel passing from the elastomer shell through a defect that is less than 0.5 mm thick on the external surface of the shell. Rupture consists of a major tear in the elastomer shell where a significant amount of silicone has escaped the confines of the implant but remains within the breast capsule. Extrusion of the implant entails displacement of silicone gel through a tear in the breast capsule to an adjoining area of breast parenchyma.⁷⁸ Silicone “bleed” is considered a normal process whereby a small amount of silicone diffuses out of the elastomer membrane. Many silicone ruptures are subclinical and are only noted at time of mammography or implant exchange, thereby making prevalence of implant rupture unknown. Implants older than 10 years are often considered silently ruptured even if no breast distortion is apparent.⁷⁹

Implant displacement and asymmetric positioning are second to capsular contracture as the most frequent complaint by the patient after augmentation. Implant positioning that is too high, too low, or too lateral after an appropriate waiting period may require further pocket revision to improve patient satisfaction. Postoperative elastic banding to the upper poles of the breasts helps to prevent implants from remaining high after subpectoral placement. A period of 1 to 3 months may be required for elastic banding to improve this problem before considering any surgical intervention. Routinely, a secondary revisionary surgery is not performed before 6 months and, at times, even longer. Rotation of anatomic implants that produces a noticeable deformity may be treated with taping of the breast mound for several weeks to allow for a subclinical capsule to form around the textured implant. If this fails to improve the deformity to an acceptable degree, a revision may be required surgically where the implant is either secured to the pectoral fascia or the pocket is sutured to create a tighter fit around the implant.

Rippling from subglandular placement in a thin patient is typically only improved upon with removal of the

implant and placement in a subpectoral plane. Rippling in a subpectoral plane is often not treatable other than by removing the implants and replacing with a smaller size or switching to silicone gel implants, but unfortunately, a thin chest wall with little to no breast mound coverage will produce similar rippling even after careful subpectoral placement with a smaller implant. Rippling is also a consequence of underfilling and traction on the implant. Underfilling an implant below recommended manufacturer volumes will lead to implant folding and at times to visible rippling. This can also contribute to higher rupture rates as the two surfaces of the implant shell come in contact and lead to frictional damage. Traction on an implant is often seen with textured implants where the textured surface of the implant is tethered by the overlying glandular plane. Adequate breast coverage and pocket size creation help avoid this complication with textured implants.

POSTOPERATIVE INSTRUCTIONS AND CARE

Augmentation mammoplasty is performed on an outpatient basis and patients are sent home with oral analgesics and a 3-day course of an oral first-generation cephalosporin. Many surgeons will also send the patient home with a 3-day course of a muscle relaxant to protect against muscular spasm pain. Patients are allowed to shower on postoperative day 1 to 3 and the first postoperative visit is usually scheduled within 7 days of surgery. If there is any suggestion of upward implant positioning, as is typical in a subpectoral placement, massage exercises of the upper poles are initiated as soon as the patient can tolerate them from a pain perspective. In conjunction with the massage instructions, the patient is placed in an upper pole compressive elastic wrap that is worn to provide downward pressure. Patients are typically placed in a sports bra garment for support for the first few postoperative weeks and rigorous exercises and lifting greater than 10 lbs are avoided in the first few postoperative weeks.

After reduction mammoplasty and mastopexy procedures, the use of drains and antibiotics is controversial. Drains do not prevent hematoma formation, but they aid in removing fluid from the dead space and are particularly helpful and necessary if tumescence is performed for the reduction. The development of seromas is moderately common but many do not require treatment.⁸⁰ As with any breast reduction technique, there

is the concern for nipple loss but this is monitored clinically during surgery and after appropriate nonsmoking patient selection. If nipple loss is due to congestion, removal of sutures immediately may improve blood flow.

Breast reduction and mastopexy patients require postoperative care similar to breast augmentation patients. Patients are instructed to wear a supportive sports bra garment for the first few weeks postoperatively and are to avoid heavy lifting or rigorous exercises for the first few weeks postoperatively. Patients are instructed to avoid showering while drains are in place although this has been debated. Postoperative drains are usually only kept in place for 24 hours postoperatively after tumescent technique. Patients need preoperative instruction that final breast position and shape will take several months to achieve after any reduction mammoplasty technique.

■ Capsular Contracture

Capsular contracture is the most common complication after breast augmentation with rates ranging from 0.5% to as high as 30%.⁸¹ Changes in implant design were made to address this complication. The focus was then shifted to surgical technique as a potential source for capsular contracture. The no-touch technique for implant placement became popular with the thought that a subclinical infection introduced at the time of implantation was a potential cause for capsule formation.

Other theories of capsular contracture formation include foreign body reaction, autoimmune-connective tissue reaction, a genetic predisposition to form encapsulation, hematoma formation, and infection. Multiple studies have reported a lower incidence of capsular contracture rates with the use of textured implants over smooth implants.^{82,21} Careful surgical technique and antibacterial irrigation containing 50,000 units of bacitracin, 1 g of cefazolin, and 80 mg of gentamicin in 500 mL of normal saline are currently recommended to minimize capsular contracture formation.⁸³

Treatment of capsular contracture ranges from open capsulotomy to partial or total capsulectomy, and even surgical site change (i.e., subglandular to subpectoral, etc). Closed capsulotomy in which manually applied external pressure is placed on the breast mound to rupture a formed capsule is no longer recommended because of high rates of implant rupture and even physical injury to the person performing the capsulotomy.

■ Oncologic Considerations

Several studies report no significant difference in tumor detection in augmented versus nonaugmented breasts.^{84,85} Earlier studies lent suspicion to the theory that the presence of an implant can lead to tumorigenesis but this has since been demonstrated as inaccurate. There have also been several reports of women with augmented breasts experiencing a lower than expected incidence of breast cancer and no delay in tumor detection in a 14-year follow-up period.⁸⁶ Augmentation mammoplasty is no longer considered to be correlated with an increased risk of breast cancer or other forms of cancer. There is no increased risk in delayed tumor detection, staging of breast cancer, or the prognosis for those patients with breast cancer and implants.

The extensive dissection that can be needed for reduction mammoplasty has created ongoing concern about the possibility that postoperative fibrosis and scar formation may interfere with the detection of breast cancer. It is imperative that patients continue to perform breast self-examinations and have regular mammograms at the appropriate age. Breast cancer has been diagnosed at the time of breast reduction and these patients were found to have earlier staged lesions with a higher incidence of node-negative lesions and a higher 5-year survival rate.⁸⁷

■ Autoimmune and Connective Tissue Disorders

Numerous collagen and autoimmune disorders such as progressive systemic sclerosis, rheumatoid arthritis, lupus erythematosus, and scleroderma have been investigated as potentially being correlated with the presence of silicone gel breast implants. Silicone gel bleed is a common phenomenon, yet the fate of this small amount of silicone remains disputed, so does its correlation with autoimmune and connective tissue disorders. Detectable amounts of silicone in nearby lymph nodes and lung parenchyma have been reported⁸⁸ and the presence of this silicone within lymph tissue remains debated.

Despite FDA approval of silicone implants in late 2006, the debate over silicone gel implants and autoimmune processes will persist. Several studies report no association between breast implants and various connective tissue disorders.^{89–91}

CONCLUSION

Any surgeon performing augmentation mammoplasty must have a thorough understanding of current surgical technique as well as recent literature on the safety and longevity of current saline and silicone implants. With the FDA approval of silicone implants in late 2006, projected numbers for the upcoming years will continue the current trend in increasing breast augmentation popularity. In 2006, more than 329,000 augmentation mammoplasty procedures⁷ were performed making it the most commonly performed cosmetic surgical procedure in women in the United States. The popularity of this procedure will continue to rise with persistently high patient satisfaction rates and lower complication rates.

Breast reduction and mastopexy procedures are consistent techniques to reduce breast volume and raise the nipple-areola complex to a less ptotic, more youthful position. Patients have a high degree of satisfaction after reduction mammoplasty and experience fewer complications associated with mammary hypertrophy such as neck and back pain. Outcome studies have demonstrated the positive impact of breast reduction procedures on patients' physical and psychological well-being. There are also reports that reduction in overall breast volume has been correlated with a reduced risk in breast cancer thereby making breast reduction not only a functional improvement in patient lifestyle but a protective technique against future breast cancer development.⁹²

REFERENCES

1. Cooper AP. *On the Anatomy of the Breast*. Vol 2. London, UK: Longmans; 1840:1-89.
2. Penn J. Breast reduction. *Br J Plast Surg*. 1955; 7:357.
3. Craig RDP, Sykes PA. Nipple sensitivity following reduction mammoplasty. *Br J Plast Surg*. 1970; 23:165.
4. Palmer JH, Taylor GI. The vascular territories of the anterior chest wall. *Br J Plast Surg*. 1986;39:287.
5. Jones G. Breast reduction. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006:551.
6. Spear SL, Little JW. Reduction mammoplasty and mastopexy. In: Aston SJ, Beasley RW, Thorne CHM, eds. *Grabb and Smith's Plastic Surgery*. 5th ed. Philadelphia, PA: Lippincott-Raven; 1997:725.

7. American Society of Plastic Surgeons. Press release. March 22, 2007.
8. Terry MD, Skovron ML, Garbers S, et al. The estimated frequency of cosmetic breast augmentation among U.S. women 1963 through 1988. *Am J Public Health*. 1995;85:1122.
9. Hetter GP. Satisfaction and dissatisfaction of patients with augmentation mammoplasty. *Plast Reconstr Surg*. 1979;64:151.
10. FDA, Breast Implant Patient Survey, n = 5000, 2003.
11. Druss R. Changes in body image following augmentation breast surgery. *Int J Psychoanal Psychother*. 1973;2:248.
12. Gerow FJ. Augmentation mammoplasty: Variation on a theme. In: Owsley JQ, Peterson RA, eds. *Symposium on Aesthetic Surgery of the Breast*. Vol 18. St Louis, MO: CV Mosby; 1978.
13. Maxwell GP, Hartley RW. Breast augmentation. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006:25.
14. Maxwell GP. Discussion of “transaxillary subpectoral augmentation mammoplasty: Long-term follow-up and refinements” by JB Tebbetts. *Plast Reconstr Surg*. 1984;74:648.
15. Hall-Findlay E. Breast reduction. In: McCarthy JG, Galiano RD, Boutros SG, eds. *Current Therapy in Plastic Surgery*. Philadelphia, PA: Saunders Elsevier; 2006:405.
16. Pitanguy I. Surgical correction of breast hypertrophy. *Br J Plast Surg*. 1967;20:78.
17. Regnault P, Baker TJ, Gleason MC, et al. Clinical trial and evaluation of a proposed new inflatable mammary prosthesis. *Plast Reconstr Surg*. 1972;50:220.
18. Jones G. Breast reduction. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006:554.
19. Maxwell GP, Hartley RW. Breast augmentation. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006:26.
20. Malata CM, Feldberg L, Coleman DJ, et al. Textured or smooth implants for breast augmentation? Three year follow-up of a prospective randomized controlled trial. *Br J Plast Surg*. 1997;50:99-182.
21. Pollock H. Breast capsular contracture: A retrospective study of textured versus smooth silicone implants. *Plast Reconstr Surg*. 1993;91:404.
22. Hammerstad M, Hansteen DB, Rindal R, et al. Quality of the capsule in reconstructions with textured or smooth silicone implants after mastectomy. *Scand J Plast Reconstr Hand Surg*. 1996;30:33. [SRPS. 9(28):40.]
23. Bronz G. How reliable are textured implants used in breast surgery? A review of 510 implants. *Aesthetic Plast Surg*. 1999;23:424.
24. Tebbetts JB. Comment on “Experience with anatomic breast implants”, by PC Hobar and K Gutowski. *Clin Plast Surg*. 2001;28(3):559.
25. Hobar PC, Gutowski K. Experience with anatomic breast implants. *Clin Plast Surg*. 2001;28(3):553. [Hunt J, Solomon J. Augmentation mammoplasty. *Plastic Surgery*. In selected readings. University of Texas, Southwestern Medical Center. 2002;9(28).
26. McKinney P, Shedbaker AR. Augmentation mammoplasty using a non-inflatable prosthesis through a circumareolar incision. *Br J Plast Surg*. 1974;27:35.
27. Pickrell KL, Puckett CL, Given KS. Subpectoral augmentation mammoplasty. *Plast Reconstr Surg*. 1977;60:325.
28. Truppmann ES, Ellenby JD. A 13-year evaluation of subpectoral augmentation mammoplasty. In: Owsley JQ Jr, Peterson RA, eds. *Symposium on Aesthetic Surgery of the Breast*. Vol 18. St Louis, MO: CV Mosby; 1978.
29. Scully SJ. Augmentation mammoplasty without contracture. *Ann Plast Surg*. 1981;6:262.
30. Mahler D, Hauben DJ. Retromammary versus retropectoral breast augmentation—A comparative study. *Ann Plast Surg*. 1982;8:370.
31. Rohrich RJ. Safety of silicone breast implants: Scientific validation/vindication at last. *Plast Reconstr Surg*. 1999;104:1786.
32. Tebbetts JB. Dual plane breast augmentation: Optimizing implant–soft tissue relationships in a wide range of breast types. *Plast Reconstr Surg*. 2001;107:1255.
33. Graf RM, Bernardes A, Rippel R, et al. Subfascial breast implant: A new procedure. *Plast Reconstr Surg*. 2003;111:904.
34. Mandrekas AS, Zambacos GJ, Anastasopoulos A, et al. Reduction mammoplasty with the inferior pedicle technique: early and late complications in 371 patients. *Br J Plast Surg*. 1996;49:442.

35. Lassus C. A technique for breast reduction. *Int Surg*. 1970;53:69.
36. Lassus C. A 30-year experience with vertical mammaplasty. *Plast Reconstr Surg*. 1996;97:373.
37. Lassus C. Update on vertical mammaplasty. *Plast Reconstr Surg*. 1999;104:2289.
38. Lejour M, Abboud M. Vertical mammaplasty without inframammary scar and with breast liposuction. *Perspect Plast Surg*. 1990;4:67.
39. Lejour M. Vertical mammaplasty: Early complications after 250 personal consecutive cases. *Plast Reconstr Surg*. 1999;104:764.
40. Hall-Findlay EJ. A simplified vertical reduction mammaplasty: Shortening the learning curve. *Plast Reconstr Surg*. 1999;104:748.
41. Hamdi M, Greuse M, DeMey A, et al. A prospective quantitative comparison of breast sensation after superior and inferior pedicle mammaplasty. *Br J Plast Surg*. 2001;54:39.
42. Bozola AR. Breast reduction with short L scar. *Plast Reconstr Surg*. 1990;85:728.
43. Yousif NJ, et al. Elimination of the vertical scar in reduction mammaplasty. *Plast Reconstr Surg*. 1992; 89: 459.
44. Hammond DC. Short scar perioareolar inferior pedicle reduction (SPAIR) mammaplasty. *Plast Reconstr Surg*. 1999;103:890.
45. Thorek M. Possibilities in the reconstruction of the human form. *NY Med J*. 1922;116:572.
46. O'Neal RM, et al. Reduction mammaplasty with free-nipple transplantation: Indications and technical refinements. *Ann Plast Surg*. 1991;26:117.
47. Regnault P. Breast ptosis. Definition and treatment. *Clin Plast Surg*. 1976;3:193.
48. Bartels RJ, Strickland DM, Douglas WM. A new mastopexy operation for mild or moderate breast ptosis. *Plast Reconstr Surg*. 1976;57:687.
49. Gruber RP, Jones HW. The "donut" mastopexy: Indications and complications. *Plast Reconstr Surg*. 1980;65:34.
50. Benelli L. A new periareolar mammaplasty: the "round block" technique. *Aesthetic Plast Surg*. 1990;14:93.
51. De la Fuente A, Martin del Yerro JL. Periareolar mastopexy with mammary implants. *Aesthetic Plast Surg*. 1992;16:337.
52. Lassus C. Vertical scar breast reduction and mastopexy without undermining. In: Spear SL, ed. *Surgery of the Breast: Principles and Art*. Philadelphia, PA: Lippincott-Raven; 1998:717.
53. Grotting JC, Marx AP, Chen SM. Mastopexy. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006:60.
54. Lejour M. Vertical mammaplasty for breast hypertrophy and ptosis. *Operative Techniques Plast Surg*. 1996;3:189.
55. Chair AJ. The L short scar mammaplasty: A new approach. *Plast Reconstr Surg*. 1992;90:233.
56. Williams JE. Experiences with a large series of slapstick breast implants. *Plast Reconstr Surg*. 1972;49:253.
57. Williams C, Aston S, Rees TD. The effect of hematoma on the thickness of pseudo sheaths around silicone implants. *Plast Reconstr Surg*. 1975;56:194.
58. deCholnoky T. Augmentation mammaplasty: Survey of complications in 10,941 patients by 265 surgeons. *Plast Reconstr Surg*. 1970;45:573.
59. Snyder GB. Augmentation mammaplasty. In: Owsley JQ, Peterson RA, eds. *Symposium on Aesthetic Surgery of the Breast*. St Louis, MO: CV Mosby; 1984:303.
60. Courtiss EH, Goldwyn RM, Anastasi GW. The fate of breast implants with infections around them. *Plast Reconstr Surg*. 1979;63:812.
61. Olesen LL, Ejlersen T, Nielsen J. Toxic shock syndrome following insertion of breast prostheses. *Br J Surg*. 1991;78:585.
62. Poblete JV, Rodgers JA, Wolford FG. Toxic shock syndrome as a complication of breast prostheses. *Plast Reconstr Surg*. 1995;96:1702.
63. Ransjo U, Asplund OA, Gylbert L, Jurell G. Bacteria in the female breast. *Scand J Plast Reconstr Surg*. 1985;19:87.
64. Collis N, Mirza S, Stanley PRW, et al. Reduction of potential contamination of breast implants by the use of 'nipple shields'. *Br J Plast Surg*. 1999;52: 445.
65. Maxwell GP, Hartley RW. Breast augmentation. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006.
66. Spear SL, Little JW. Reduction mammaplasty and mastopexy. In: Aston SJ, Beasley RW, Thorne CHM,

- eds. *Grabb and Smith's Plastic Surgery*. 5th ed. Philadelphia, PA: Lippincott-Raven; 1997:750.
67. Davis GM, Ringler SL, Short K, et al. Reduction mammoplasty: Long-term efficacy, morbidity and patient satisfaction. *Plast Reconstr Surg*. 1995;96:1106.
68. Maxwell GP, Hartley RW. Breast augmentation. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006:30.
69. Slezak S, Dellon AL. Quantitation of sensibility in gigantomastia and alteration following reduction mammoplasty. *Plast Reconstr Surg*. 1993;91:1265.
70. Temple CLF, Hurst LN. Reduction mammoplasty improves breast sensibility. *Plast Reconstr Surg*. 1999;104:72.
71. Slavin SA. Augmentation mammoplasty and its complications. In: Aston SJ, Beasley RW, Thorne CH, eds. *Grabb and Smith's Plastic Surgery*. 5th ed. New York, NY: Lippincott-Raven; 1997:715-720.
72. Aboudib JH, de Castro CC, Coelho RS, et al. Analysis of late results in post-pregnancy mammoplasty. *Ann Plast Surg*. 1991;26:111.
73. Marshall DR, Callan PP, Nicholson W. Breastfeeding after reduction mammoplasty. *Br J Plast Surg*. 1994;47:167.
74. Brzozowski D, Niessen M, Evans GB, et al. Breast feeding after inferior pedicle reduction mammoplasty. *Plast Reconstr Surg*. 2000;105:530.
75. Maxwell GP, Hartley RW. Breast augmentation. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006:46.
76. Rohrich RJ, Adams WP Jr, Beran SJ, et al. An analysis of silicone gel-filled breast implants: Diagnosis and failure rates. *Plast Reconstr Surg*. 1998;102:2304.
77. Slavin SA. Augmentation mammoplasty and its complications. In: Aston SJ, Beasley RW, Thorne CH, eds. *Grabb and Smith's Plastic Surgery*. 5th ed. New York, NY: Lippincott-Raven; 1997:720.
78. Dowden RV. Definition of terms for describing loss of gel from breast implants. *AJR*. 1993;160:1360.
79. Camara DL de, Sheridan JM, Kammer BA. Rupture and aging of silicone gel breast implants. *Plast Reconstr Surg*. 1993;91:828.
80. Hall-Findlay EJ. Breast reduction. In: McCarthy JG, Galiano RD, Bourtos SG, eds. *Current Therapy in Plastic Surgery*. Philadelphia, PA: Saunders Elsevier; 2006:409.
81. Maxwell GP, Hartley RW. Breast augmentation. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006.
82. Embrey M, Adams EE, Cunningham B, et al. A review of the literature on the etiology of capsular contracture and a pilot study to determine the outcome of capsular contracture interventions. *Aesthetic Plast Surg*. 1999;23:197.
83. Adams WP Jr, Conner WCH, Barton FE Jr, Rohrich RJ. Optimizing breast pocket irrigation: The post-Betadine era. *Plas Reconstr Surg*. 2001;107:1596.
84. Carlson GW, Curley SA, Martin JE, et al. The detection of breast cancer after augmentation mammoplasty. *Plast Reconstr Surg*. 1993;91:837.
85. Birdsell DC, Jenkins H, Berkel H. Breast cancer diagnosis and survival in women with and without breast implants. *Plast Reconstr Surg*. 1993;92:795.
86. Deapen DM, Bernstein L, Brody GS. Are breast implants anticarcinogenic? A 14-year follow-up of the los angeles study. *Plast Reconstr Surg*. 1997;99:1346.
87. Tang C, Brown M, Levine R, et al. Breast cancer found at the time of breast reduction. *Plast Reconstr Surg*. 1999;103:1682.
88. Kao CC, Rand RP, Holt CA, et al. Internal mammary silicone lymphadenopathy mimicking recurrent breast cancer. *Plast Reconstr Surg*. 1997;99:225.
89. Sanchez-Guerrero J, Colditz GA, Karlson EW, et al. Silicone breast implants and the risk of connective-tissue diseases and symptoms. *N Engl J Med*. 1995;332:1660-1670.
90. Gabriel SE, O'Fallon WM, Kurland LT, et al. Risk of connective-tissue diseases and other disorders after breast implantation. *N Engl J Med*. 1994;330:1697-1702.
91. Schottenfeld D, Burns CJ, Gillespie BW, et al. The design of a population-based case-control study of systemic sclerosis (scleroderma): Commentary on the University of Michigan study. *J Clin Epidemiol*. 1995;48(4):583-586.
92. Jones G. Breast reduction. In: Mathes SJ, ed. *Plastic Surgery*. 2nd ed. Vol 6. Philadelphia, PA: Saunders Elsevier; 2006:539.

CHAPTER 11

Rejuvenation of Scars and Striae

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KEY POINTS

- Wound healing process is highly ordered and complex.
- Malfunctions may result in hypertrophic scars (HS), keloids, or striae.
- Abnormal or conspicuous scars or striae may require medical treatment, and most serious malformations are also accompanied by psychological distress.
- The rejuvenation of HS, keloids, and striae can not only heal the abnormal appearance of these scars but can ease the psychological burden of an unsightly wound as well.
- Rejuvenation of HS yields the most dramatic improvements, while keloid and striae treatments have yet to provide such extensive benefits.

HYPERTROPHIC SCARS

■ Definition, Cellular Anatomy, and Etiology

HS are defined as raised, firm, erythematous scars that remain within the boundaries of the original injury.¹

The etiology of hypertrophic scarring is poorly understood as cutaneous wound healing is an extremely complex procedure that offers countless paths for deviation.² When normal wound healing occurs after injury, hemostasis begins at once, as platelets begin plugging vessels and fibrin polymerizes through the wound area.² Next is the inflammatory stage, with the arrival of neutrophils, macrophages, and synthesis lymphocytes to the wound region. Synthesis events follow as cytokines from the injured tissues, platelets, and cellular migrants start the processes for angiogenesis and fibrogenesis. In the remodeling phase, continuing over the next 6 to 12 months, tissue proliferation and reepithelialization begin, with matrix deposition and collagen synthesis. The final mature scar will have approximately 80% of the strength of the initial, uninjured tissue.²

HS often appear within 6 to 8 weeks after reepithelialization of a wound, and continue to grow rapidly until

reaching maturity at approximately 1 year.³ They are the result of extreme collagen production and reduced collagen degradation during wound remodeling.¹ HS may follow acne, infections, burns, or piercings.^{3,4}

■ Epidemiology

HS affect 1.5% to 4.5% of the general population.³ They present equally among males and females.⁵ HS occur most often in areas where the skin is significantly anatomically stretched, such as the presternal area, upper back, and deltoid region, usually within the first month post surgery or trauma.⁶ HS and keloids tend to develop between the ages of 10 and 30.⁷

■ Patient Selection and Preprocedure Evaluation

HS, by definition, are confined to the original region of injury. They are raised, erythematous, and may be painful or pruritic. Fortunately, many HS gradually resolve; first erythema decreases, followed by scar flattening months to years later.² In some cases, scars can grow further and become permanent.³ However, overall, HS are expected to have a good response to treatment.²

Histologically, normal skin shows distinct collagen bundles that are oriented parallel to the epithelial surface. In contrast, collagen bundles in HS are flatter than those in normal skin, less distinct, and run in a wavy pattern.⁴ These thin collagen bundles have unique nodules containing alpha smooth muscle actin expressing myofibroblasts,⁴ as well as the presence of increased mast cells.²

Although HS can resolve over time, they should be treated when they cause functional or cosmetic deformities, discomfort or pain, or psychological stress.⁸

■ Techniques

Treatments for HS (Table 11.1) are many and include pressure garments, silicone gel sheeting, corticosteroid injections, 5-fluorouracil (5-FU), interferon (α , β , and γ), bleomycin, radiotherapy, surgery, cryosurgery, and laser therapy.^{8,20}

Intralesional corticosteroid treatment is the current gold standard and is often used alone or in combination with pressure treatment or surgery. Triamcinolone

TABLE 11.1 ■ Hypertrophic Scars Treatment Studies

Author	# Patients	Treatment	Cooling Devices	# Treatments	Results	Side Effects
PDL Studies						
Alster et al. (1993)	10	PDL: 585 nm, 6.0–7.0 J/cm ² , 360 μs. Spot size not given.	Not noted.	5	All treated scars improved. Dilated vascular channels cleared and regular-sized dermal vessels replaced these. Unexpected improvements in scar texture, height, and pliability.	Not noted.
Dierickx et al. (1995)	15	PDL: 585 nm, 6.0–7.5 J/cm ² , 450 μs, 5 mm.	Chemical ice pack applied posttreatment.	1–4, average of 1.8.	Color, texture, height, hypertrophy, and erythema improved. Younger scars improved more than older scars. Facial scars improved more than body scars.	Minimal crusting, temporary purpura.
Reiken et al. (1997)	30 mice with HS implants	PDL: 585–600 nm, 2–10 J/cm ² , 450 μs. Spot size not given.	Not noted.	1	Maximum scar growth inhibition at 585 nm, minimal scar growth inhibition at 600 nm. Fluences of 6 J/cm ² and 10 J/cm ² yielded best results with 585 nm.	Not noted.
McCraw et al. (1999)	106	PDL: 585 nm, 2.5–8.0 J/cm ² , 3 or 10 mm	Not noted.	Approximately between 3 and 6.	124(171 (72%) sites had minimal white scar with good pliability and no contour abnormalities. Analyzing PDL as a preventative therapy for HS, researchers advocated more aggressive treatment.	Hypertrophic scarring, blistering, and scar widening.

(continued)

TABLE 11-1 ■ Hypertrophic Scars Treatment Studies (Continued)

Author	# Patients	Treatment	Cooling Devices	# Treatments	Results	Side Effects
Manuskiatti et al. (2002)	10	PDL: 585 nm, 5 J/cm ² , 7 mm. No pulse duration given. Other treatments: Intralesional triamcinolone acetonide (TAC) alone (20 mg/mL), intralesional 5-fluorouracil (5-FU) alone (50 mg/mL), or combination of TAC (1 mg/mL) and 5-FU (45 mg/mL).	No cooling	6 or 10, depending on treatment type.	All treated segments showed significant improvement. All treatments improved scar height; intralesional therapies also improved pliability. Only laser treatment improved skin texture. Researchers deemed results between treatments comparable. Study did not distinguish between HS and keloid results.	Hypopigmentation, telangiectasia, and skin atrophy.
Alster (2003)	22	PDL: 585 nm, 4.5–5.5 J/cm ² , 1.5 ms, 10 mm, alone or in combination with 10–20 mg intralesional TAC.	Concomitant cryogenic cooling.	2	PDL treatment appeared responsible for most therapeutic benefits. Corticosteroids did not seem to greatly enhance effects, although the combination treatment had greater symptomatic reduction.	Mild purpura, temporary hyperpigmentation, and intraoperative pain.
Nouri et al. (2003)	11	PDL: 585 nm, 3.5 J/cm ² , 450 μs, 10 mm.	Not noted.	3	Surgical scars started treatment on suture removal day as a preventative measure. At end of treatments, vascularity and pliability improved more in treated scars.	No side effects.
Chan et al. (2004)	56	PDL: 585 nm, 7–8 J/cm ² , 1.5 ms, 5 mm.	Not noted.	3–6.	Chinese patients separated into prevention and treatment groups. 19/35 (54%) of prevention patients and 24/36 (67%) treatment patients judged scars as	Blisters occurred on approximately 3/56 (6%) patients.

“better” or “much better.”
Treatment group symptoms improved significantly.
Preventative PDL treatment started on suture removal day. Treated scars improved mainly in vascularity, pliability. Researchers determined early PDL treatment was safe and effective for surgical scars.
PDL and Q-switched Nd:YAG delivered comparable improvements.

VM decreased in volume, color, hemorrhaging, and pain.
Rare. Included blistering, ulceration, edema, pain, and scarring.

Majority of HS (80%) resolved after procedure, while 20% of HS tended to recur and were treated with additional intralesional steroid injections and silicone gel sheeting. Exact number of HS not given. CO₂ laser effective in camouflaging self-inflicted razor scars.

Conologue et al. (2006)	13	PDL: 595 nm, 8.0 J/cm ² , 1.5 ms, 7 mm.	Cryogen cooling spray.	3		
Bowes et al. (2002)	6	Nd:YAG: Frequency-doubled 532-nm Q-switched at 2.8 J, 10 ns, 3 mm. Variable-pulsed at 9.5 J, 10 ms, 4 mm. PDL: 585 nm, 3.5 J, 450 μs, 10 mm.	Not noted.	Average of 3.3.		
Bhardwaj et al. (2006)	26 patients with cutaneous or mucosal VM	Nd:YAG: long-pulsed 1064 nm, 40–250 J/cm ² , 3–50 ms, 7–18 mm.	Not noted.	1–5.		
Nd:YAG Studies						
CO₂ Study						
Acikel et al. (2005)	16	Elevated HS portions excised, followed by CO ₂ laser resurfacing, intralesional TAC injections (20 mg/mL), and a skin graft. CO ₂ : 34 W, 9 mm.	Not noted.	1		

PDL, pulsed dye laser; HS, hypertrophic scars; VM, venous malformations.

acetoneide (TAC) is used most often, at 5 to 10 mg/mL, beginning 1 month postoperation, and repeated monthly, with clinical improvement as the long-term end point.⁸

The mechanical compression of pressure garments has been shown to reduce HS, especially in burn patients. Pressure garments lead to an improved organization of the dermal extracellular matrix compared with nonpressurized treatments, and lead to a restoration of ultrastructure at the dermal or epidermal junction.⁸ They have also been shown to induce apoptosis and moderate cytokine release in HS, limiting the hyperproliferation involved in abnormal healing.⁸

Silicone gel sheeting is now part of standard care for HS, as in some studies it has been shown to be safe, effective, and especially helpful for treating children and patients with a low threshold for pain. However, results are not always dramatic and intralesional corticosteroids remain the primary standard of care.²⁰

Surgical excision of HS is occasionally used with silicone gel sheeting or surgical taping. The best way to splint the scars is by surgical closure with intradermal sutures for a minimum of 6 weeks and up to 6 months if the tension is substantial. W-plasty and Z-plasty techniques, while useful in treating burn scars, are not optimal for immature HS.²⁰ However, surgery also involves the risk of leaving another scar.

Other treatment options include the antimetabolite 5-FU, which can be used as an intralesional injection for HS, either alone or with laser treatment. It targets the fast-proliferating and metabolizing fibroblasts in dermal wounds responsible for extra collagen production.⁸ Injections of interferon α , β , and γ can increase collagen breakdown but can also be painful enough to necessitate anesthesia.²⁰ Bleomycin injections can be useful for HS resistant to intralesional corticosteroids, and in some cases have caused significant or complete scar flattening.

Cryosurgery, via spray or intralesional needle, has also been shown to decrease the volume of the scar, and improve pain, itching, hardness, and scar color.⁸

HS can also be successfully treated with the pulsed dye laser (PDL). Operating on the principles of selective photothermolysis,²¹ the PDL was designed specifically to treat vascular lesions. HS treatment parameters include a wavelength of 595 nm, fluences between 6 to 10 J/cm², pulse durations of 450 μ s, and spot sizes between 2 to 10 mm.^{1,22,23}

PDL treatment is analogous to snapping a rubber band on the patient, but it is usually endured with only mild

discomfort.¹ Treatments should begin at lower fluences to determine scar response, and the starting fluence can be adjusted upwards as necessary. In general, less fibrotic scars in sensitive areas such as the anterior chest or breast begin treatment with lower fluences (4.5–5.5 J/cm² with a 10-mm spot size) as the skin is more delicate. Thicker or darkly pigmented scars (where epidermal pigment will compete with hemoglobin for absorption of the laser energy) can be treated with slightly higher fluences (6.5–7.5 J/cm² with a 5-mm spot size) as the net amount of energy effectively delivered to this tissue is lower.

In the treatment of HS, PDL treatment also has many other advantages. It can be used on the entire body, with caution on the neck and chest, and it is minimally invasive with no anticoagulants needed. Simple postoperative care includes using a mild cleanser and topical antibiotic ointment as well as strict sun protection.²²

Early studies suggested the argon laser was ineffective and damaging to HS, and there is little evidence supporting the use of continuous wave (CW) CO₂ lasers for HS treatment.^{24–26} By the late 1980s, the PDL was regularly used for treating vascular lesions such as port wine stain (PWS). However, the marked improvement the PDL made in the treatment of scars was still unexpected when Alster and colleagues published a study on argon laser-induced scars in 1993. Ten patients (skin types I and II) previously treated for PWS and subsequently scarred by argon laser treatment were treated with a 585-nm PDL. All treated scars displayed clinical improvement, shown by flattening of hypertrophic sections and/or return of skin surface markings in atrophic areas. PDL-treated scars showed skin texture closer to normal, non-argon-treated PWS skin. In control PWSs, PDL treatment lightened skin but did not affect the original texture. After five PDL treatments, dilated vascular channels cleared and a normal amount of regularly sized dermal vessels replaced these channels.⁹ Unexpected improvements in the texture, size, and pliability of the scars occurred after PDL treatment, but researchers could not explain the precise mechanisms causing the results.²²

In 1995, Dierickx and colleagues reported on the treatment of resistant HS with a 585-nm PDL. The original goal of the study was to improve HS color, but researchers also discovered that the PDL drastically changed the texture of the scar, creating a softer, flatter wound. Researchers also found that scars less than a year old responded better than older scars, and facial scars responded better than body scars. Dierickx and

colleagues theorized that targeting the blood supply caused laser-induced hypoxia, which may have altered collagen regulation, lowering production and increasing catabolism.¹⁰

In 1997, based on earlier efforts, Reiken and colleagues attempted to determine the precise wavelength and fluence that would achieve maximum results. Testing on mice, they found maximum significant scar growth inhibition resulted at 585 nm, with significant growth inhibition at 590 nm and 595 nm but minimal inhibition at 600 nm. This suggested that 585 nm was the closest approximation possible to the absorption peak of hemoglobin, with the laser causing a direct effect to the scar's blood vessels.¹¹ Energy density also had a significant impact on growth inhibition. When the laser wavelength was kept at 585 nm, a fluence of 2 J/cm² did not provoke histological changes in the implant. However, fluences of 6 J/cm² and 10 J/cm² yielded implants 70% and 92% smaller than untreated controls at day 17, respectively.¹¹

In 2002, Manuskiatti and colleagues compared the clinical response of keloids and HS after treatments with a 585-nm PDL, intralesional TAC alone, intralesional 5-FU alone, and intralesional TAC combined with intralesional 5-FU. All treated segments showed statistically significant flattening and reduced erythema across all treatment modalities. Skin texture improved in laser-treated sections only. Overall, Manuskiatti and colleagues deemed the results between treatments comparable.¹³

A 2003 combination treatment study confirmed the strength of the PDL's effects on the scars. Alster compared a HS combination treatment of PDL and intralesional corticosteroids to a treatment of PDL alone. The purpose was to determine whether combination therapy could outperform the PDL. As intralesional corticosteroids are known to improve scar symptoms such as pruritis, it was hypothesized that a combination therapy with the PDL might achieve the desired results in fewer treatment sessions. All scars showed clinical improvement after each of the two treatments, with increased scar flexibility and decreased symptoms. Patients receiving only PDL treatment showed clinical improvement of 36% 6 weeks after the first treatment and 61% improvement 6 weeks after the second treatment. Patients receiving the PDL or TAC combination therapy showed 40% clinical improvement 6 weeks after the first treatment and 63% improvement 6 weeks after the second treatment. Scar pliability increased by an average of 50% after two sessions with either treatment. Symptoms such as itching and burning were reduced by 50% after two

sessions with PDL and 70% after two sessions with the combination treatment. Histologic examination showed that the additional use of corticosteroids did not change the appearance of the treated scars. The PDL treatment seemed responsible for the most significant improvements, but combination therapy may be helpful for patients with very symptomatic scars.³

Like the PDL, the Nd:YAG laser is a nonablative laser that operates primarily at a wavelength of 1064 nm. It can be frequency-doubled to 532 nm to target epidermal melanin or hemoglobin. It is thought to selectively inhibit collagen production and establish tissue infarctions.²⁷ The Nd:YAG laser has been used to treat HS and keloids with varying results.²⁷ While some studies report success, others find initial improvement but common recurrences.²²

In 2002, Bowes and colleagues compared the effects of the 585-nm PDL to the effects of the 532-nm frequency-doubled Nd:YAG laser on pigmented HS. Scars were graded on a scale that evaluated pigmentation, vascularity, pliability, and height. The Q-switched Nd:YAG scored the best with a 38% improvement overall. The 585-nm PDL scored almost as high with a 36% improvement, not significantly different from the Nd:YAG outcome. The 532-nm variable pulse Nd:YAG showed a 19% improvement overall, but this was not significantly different from the 16% improvement shown in control scars in the last follow up. Researchers determined that the 585-nm PDL and the 532-nm Q-switched Nd:YAG laser offered comparable favorable outcomes.¹⁷

A 2006 study by Bhardwaj and colleagues also indicated the efficacy of the long-pulsed Nd:YAG laser for treating venous malformation (VM). VMs, such as PWS, are slow-flowing vascular malformations that exist at birth and grow larger through time. Researchers noted these VMs often do not respond sufficiently to 595-nm PDL treatment, even at larger pulse durations, given the size and depth of the targeted vessels. A long-pulsed Nd:YAG appeared to be a potentially promising alternative treatment, given the deep penetration of 1064-nm light, longer pulse durations, and high fluences that made up for a smaller absorption coefficient. In 25 patients, the VMs decreased in volume, color, hemorrhaging, and pain. Significant functional improvements occurred in several patients, although these were not described in detail. Negative effects were rare, but included blistering, ulceration, edema, pain, and atrophic or hypertrophic scarring. Overall, researchers determined the long-pulsed Nd:YAG laser to be an effective treatment for cutaneous and mucosal VM.¹⁸

CO₂ lasers have also been shown to improve HS in limited studies. CO₂ lasers, unlike PDLs, do not target hemoglobin, but instead resurface skin by vaporizing cutaneous water. In 2005, Acikel and colleagues examined the effect of this resurfacing laser on HS caused by self-mutilation with razors. The goal was to trim the scars down to intact skin level with a blade, then to use the CO₂ laser to provide a better surface for grafts. HS received two passes of the laser after trimming, followed by intralesional TAC (20 mg/mL) injection. Ablating was bloodless, well-controlled, and time-efficient, and 80% of the 26 scarred regions resolved completely post operation. Approximately 20% of the HS tended to recur and responded well to a series of intralesional steroid injections and silicone gel sheeting. No new scar formation developed in any treated areas. The results were successful in that the scars were well-camouflaged and looked like burn scars, which were more socially acceptable.¹⁹

Complications to avoid

Adverse effects of corticosteroid injection can include pain upon injection, skin atrophy, pigmentation problems, and telangiectases.²⁰ No side effects with the use of bleomycin for HS treatment have been recorded, although when used for wart treatment, bleomycin can cause nail loss and Raynaud's phenomenon.

The increased fluences on darker skin may also increase the risk of pigmentation side effects.²² If only minimal results occur after clinical observation, fluences should increase by 10%. Alternatively, if postoperative vesiculation or crusting occur, a lower fluence should be used with careful avoidance of overlapping pulses.²²

Purpura is the main adverse effect of PDL treatment, the product of immediate microvascular hemorrhage, thrombosis, and delayed appearance of vasculitis. Purpura is dependent on fluence, spot size, and pulse duration.²⁸ Because very short pulses confine thermal damage to the target, purpura occurs more quickly with a very short pulse duration as opposed to a longer pulse duration. A relatively longer pulse produces a gentler but still selective heating of the microvasculature that is more efficient and better suited to treating vascular abnormalities, and minimizes scarring.²⁹

Hyperpigmentation is a concern for tanned and dark-skinned patients as well. The laser surgeon must show great care to cool the skin carefully and avoid overlapping pulses, as these conditions can lead to postoperative vesiculation and crusting.²² PDL scar treatment is usually

given at 6 to 8 week intervals, although longer intervals may be necessary for dark-skinned patients prone to postoperative hyperpigmentation.¹

KELOIDS

Definition, Cellular Anatomy, and Etiology

Keloids are dark, raised scars that by definition extend beyond the original boundaries of injury. They are firmer than HS but have a similar prolonged growth phase.¹

There are many theories behind keloid formation. Their exact cause is unknown; events causing inflammation, such as infections, excessive wound tension, and foreign material, may be responsible in some cases, although keloids can also develop without an obvious injury.² They are often believed to occur due to increased fibroblast activity leading to excess collagen production.³⁰ These excess deposits of collagen slow oxygen circulation with a reduction in vascularization yielding selective tissue hypoxia. The net result is a marked overproduction of collagen. Transforming growth factor beta-1 (TGF- β 1) is thought to play a role in fibroblasts' excess collagen and is found at higher than average levels in keloids and HS. Beer and colleagues also theorized that the reduced vascularization of keloids stimulates collagen production due to suppressed inhibitory factor XIIIa-positive dermal dendrocytes.³¹

Hormonal issues may also contribute to keloid formation. Keloids are known to emerge during puberty, enlarge during pregnancy, and regress after menopause.³² Stern and Lucente discovered that pregnant women tended to report an onset of keloids during pregnancy, even though two-thirds of the women had stable wounds for many years prior.³³

Genetics may also influence keloid formation. A 2001 pedigree study by Marneros and colleagues suggested keloids often develop in families with an autosomal recessive or dominant transmission.³⁴ A 2004 study by Marneros and colleagues studied two families with an autosomal dominant inheritance pattern of keloids and discovered evidence for keloid susceptibility loci on two chromosomes (7p11 and 2q23). Researchers concluded that additional loci for keloids were likely.³⁵ Keloids have also been associated with patients with blood type A or patients with certain connective tissue diseases.³²

■ Epidemiology

Keloids occur across all ethnicities but they are 15 times more likely to appear in dark-skinned patients, particularly those of African, Spanish, and Asian origin.³² The most common regions for keloid occurrence are the anterior chest, shoulders, earlobes, upper arms, and cheeks.³² There is equal prevalence among men and women, although women tend to outnumber men in studies that include keloids resulting from pierced ears.² Keloids, like HS, tend to occur between the ages of 10 and 30 years⁷ and are less common in small children and older patients.²

■ Patient Selection and Preprocedure Evaluation

Keloids extend outside the original area of injury, and the collagen production is invasive and aggressive as the keloid branches out to occupy uninjured tissue.³⁶ They are raised, erythematous, and symptomatic² and can form at any time, from weeks to years after the original injury.²² They can be soft and dough-like or firm and banded.⁷ They tend to develop in regions where skin is thicker or where there is increased tension, such as the presternal and deltoid areas.³⁷ Keloids very rarely spontaneously resolve, and they have a poor treatment response.²

Histologically, keloids have almost no collagen bundles, and collagen fibers are randomly arranged in loose sheets. Keloids exhibit increased mast cells, but unlike HS, they have no myofibroblasts. Also unlike HS, keloids have a reduced blood supply as the developing collagen is poorly vascularized with neither lymphatics nor elastin.³⁸ Research has linked keloids with elevated levels of growth factors, such as TGF- β 1, but their precise mechanism of development has yet to be determined.

Keloids, with their excessive collagen synthesis, signify a permanent change in the body's ability to regulate the balance between collagen production and destruction in the process of wound healing.³⁶

As keloids usually do not resolve over time and may continue to grow to disfiguring proportions, they generally always require treatment. Like HS, keloids can cause cosmetic or functional deformities, discomfort, pain, and psychological distress.⁸

■ Techniques

The best treatment for keloids is that which leaves the keloid region functional, asymptomatic, and cosmetically acceptable with no recurrence.³⁷ However, because the

threat of keloid recurrence is so high, there is no one treatment that is considered ideal for this condition (Table 11.2). Keloid treatments include surgery, often in conjunction with corticosteroids or pressure therapy, cryosurgery, silicone gel sheeting, and laser therapy.

Intralesional TAC can produce marked flattening and symptom reduction in keloids. A 1966 study by Griffith treated 56 keloids with TAC injections alone, keloid excision followed immediately by TAC injections, or keloid excision followed immediately by TAC injections and 3 weeks postoperatively. The TAC injections alone produced complete or partial dissolution of keloids in 34 out of 37 patients (92%). The 19 excised keloids had only 1 recurrence (5%), and the study found only 5 recurrences out of all 56 treated keloids (9%).³⁹ In a 1970 follow-up study, Griffith and colleagues recommended total excision of the keloid followed by immediate TAC injection, with postoperative TAC injections as needed.⁴⁰ Dosage of postoperative TAC varies from 10 to 40 mg/mL between studies; low recurrence rates are associated with stronger corticosteroid concentrations, but this also increases the risk of adverse effects such as atrophy.⁵¹ Conventional surgery when performed without additional combination therapy yields high recurrence rates, ranging from 45% to 100%.⁵¹

Pressure dressings can be used alone or in combination with surgery. The dressings most likely work by causing fibroblast modification and collagen breakdown.⁵² However, because pressure garments may be cumbersome and conspicuous, patient compliance may be lower.⁵²

Cryosurgery is another option. Cryosurgery directly damages cells and changes microcirculation. Very low temperatures create vascular damage, causing blood stasis in the keloid tissue, which leads to cell anoxia. Blood flow slows, white thrombi form, and the lumens of smaller vessels close, leading to tissue necrosis and sloughing. Results from keloid cryotherapy range from complete scar flattening to keloid recurrence. Adverse effects include hypopigmentation and slight-to-moderate atrophy.⁴¹ In 1993, Rusciani and colleagues used cryotherapy to flatten 48/65 (74%) keloids, with only 1/65 (1.5%) recurrence.⁴¹ A later cryotherapy study by Rusciani and colleagues in 2006 found similar results, as researchers found that approximately 133/166 lesions (80%) reduced in volume by more than 80%, with no recurrences noted.⁴² A 1994 keloid study comparing cryosurgery with corticosteroid injections determined cryosurgery to be the more effective treatment, particularly with younger keloids and keloids located on the back.⁵³

TABLE 11.2 ■ Keloid Treatment Studies

Author	# Patients	Treatment	Cooling Devices	# Treatments	Results	Side Effects
Nonlaser Treatment Studies						
Griffith (1966)	41	Intralesional TAC alone or with keloid excision and/or postoperative TAC. Varying dosages.	N/A	1–5 doses	34/37 (92%) keloids given only injections showed complete or partial dissolution. 19 excised keloids with or without postoperative injections showed better dissolution and elimination of symptoms with only 1 (5%) recurrence. 5/56 (9%) recurrences overall.	Recurrence, risk of atrophy, scar spreading. ^{39,40}
Rusciani et al. (1993)	40	Cryotherapy.	N/A	Maximum of 2.	Treatment flattened 48/65 (74%) keloids, with 1/65 (1.5%) recurrence.	Recurrence, hypopigmentation, atrophy.
Rusciani et al. (2006)	166	Cryotherapy.	N/A	Varied between patients.	Treatment flattened 133/166 (80%) keloids by at least 80% with no recurrences noted.	Hypopigmentation, atrophy.
Mercer et al. (1989)	22 scars	Silicone gel sheeting	Not noted.	N/A	9/22 (41%) keloids improved in height, color, and texture, while another 9/22 (41%) improved in 2 out of these 3 areas.	Mild skin irritation, 1 scar showed regrowth after sheeting removed for prolonged period.
PDL Studies						
Alster and Williams (1995)	16 patients with HS and keloids	PDL: 585 nm, 6.5–7.25 J/cm ² , 450 μs, 5 mm.	Not noted.	2	All 16 patients showed improvements in scar height, texture, erythema, and pruritis. Study did not distinguish between patients with HS and keloids.	Not noted.
Kuo et al. (2004)	30	PDL: 585 nm, 10–18 J/cm ² , 450 μs, 5 mm.	Not noted.	Maximum of 11.	Keloid regression seen in 26/30 (87%) patients. Keloids with more than 6 treatments regressed more than keloids	Not noted.

					with 6 or less treatments. Researchers concluded PDL treatment possibly induces keloid regression by inhibiting TGF- β 1 expression and limiting fibroblast proliferation and collagen-3 deposition.	
Kuo et al. (2005)	10	PDL: 585 nm, 10–18 J/cm ² , 450 μ s, 5 mm.	Not noted.	1	7 d after treatment, samples had decreased TGF- β 1 and increased MMP-13. The selective increase of MMP-13 but not MMP-1 suggests MMP-13 may help with keloid regression.	Not noted.
Kuo et al. (2005)	10	PDL: 585 nm, 10–18 J/cm ² , 450 μ s, 5 mm.	Not noted.	1	7 d after treatment, levels of apoptotic bodies in the keloid fibroblast had increased, as had levels of MAP kinases and caspase-3. Researchers concluded PDL treatment may induce keloid regression by suppressing keloid fibroblast proliferation, inducing apoptosis, and increasing the levels of ERK and p38 MAP kinase activity.	Not noted.
Nd:YAG and CO₂ Studies						
Abergel et al. (1984)	8	Nd:YAG: 1064-nm continuous wave, 70 W, 60 J/cm ² , 1 cm ² .	Not noted.	Varied according to patient.	Results summarized in a case study. Patient treated 12 times and keloid lightened and flattened, with no recurrence at 3-yr follow up.	Not noted.
Apfelberg et al. (1984)	13	CO ₂ : 500 W/cm ² . Argon: 10000–12000 W/cm ² .	Not noted.	3	All keloids except 1 recurred or showed no clinical improvement. Temporary relief from pain and itching. No long-term benefits.	All symptoms returned to original intensity with time.

(continued)

TABLE 11.2 ■ Keloid Treatment Studies (Continued)

Author	# Patients	Treatment	Cooling Devices	# Treatments	Results	Side Effects
Apfelberg et al. (1989)	7	CO ₂ : 10600 nm, 1120–73211 W/cm ² .	Not noted.	Varied according to patient	All patients showed flatter, softer, less pruritic scars 12 months after treatment. However, 8/9 keloids (89%) recurred within 22 months.	Recurrence.
Stern and Lucente (1989)	18	CO ₂ : 10600 nm, 20 W in continuous mode or 12 W in 0.2 s pulse mode.	Not noted.	1	17/23 (74%) keloids recurred. Researchers concluded CO ₂ treatment was not superior to surgical excision due to potential HS caused by the laser and keloid recurrences.	Recurrences, hypertrophic scarring.
Norris (1991)	23	CO ₂ : continuous, 8–14 W, 0.2, 0.3, 0.75 mm.	Not noted.	1	Only 1/23 (4%) had no recurrences. 9/23 (39%) had keloids suppressed with corticosteroids, and 13/23 (57%) patients had recurrences regardless of extra corticosteroid therapy.	Recurrences.
Nowak et al. (2000)	Dermal fibroblast cultures.	CO ₂ : superpulsed, 2.4, 4.7, 7.3 J/cm ² , 16 Hz, 730 μs.	N/A	Not noted.	CO ₂ laser shrank keloid cell growth to normal dermal fibroblast levels and increased keloid bFGF levels.	N/A

TAC, triamcinolone acetoneide; PDL, pulsed dye laser; TGF-β1, Transforming growth factor beta-1; MMP, matrix metallo proteinase; MAP; ERK; bFGF.

As with HS, silicon gel sheeting can also be used to improve the texture, color, and height of the keloid. Silicone gel dressings increase pressure on the wound, hydrate the stratum corneum, and increase the temperature of the scar, which potentially elevates collagenase activity.^{52,54} A 1989 study by Mercer analyzed the effects of silicone gel sheeting on 22 scars over a period of months, testing changes in height, color, and texture. 19/22 (86%) exhibited a response to the scars, and 9/22 (41%) improved in all 3 areas, while another 9/22 (41%) improved in 2 out of 3 areas.⁴³ However, silicone gel sheeting is generally only useful on small scars that can be entirely covered by the gel. It is not very useful on larger, more obtrusive keloids. Adverse effects include mild skin irritation.⁴³

■ Laser Treatment of Extant Keloids

Keloids are notoriously difficult to treat given their high rate of recurrence. Nonablative lasers such as the PDL, although very useful in treating HS, have proven far less effective with keloids. While HS display an overproduction of vascular targets, the presence of keloids indicates the suppression of vascular targets. Ablative lasers such as CO₂ often remove keloids successfully, but recurrence rates are unacceptably high. Both ablative and nonablative lasers have been used to varying degrees of success.

PDL treatment

In 1995, Alster and Williams reported on 16 pale-skinned patients (skin types I – III) with median sternectomy keloid and HS post heart surgery with two 585-nm PDL treatments, 6 to 8 weeks apart.⁴⁴ After 6 months, all 16 patients showed significant improvement in erythema, scar height, skin texture, and pruritis in laser-treated areas. Erythema and mean scar height both improved significantly after just the first and second treatments. Prior to treatment, 12 patients reported pruritis, which ceased for 11 patients after treatment.⁴⁴

A number of studies have suggested that TGF- β 1 may induce keloid formation. In 2004, Kuo and colleagues explored whether PDL treatment caused keloid regression by decreasing TGF- β 1, thus decreasing keloid fibroblast proliferation and collagen deposition. TGF- β 1 is a growth factor that stimulates matrix proteins such as collagen, enhances mitogenesis, and inhibits protease production. Using a 585-nm PDL, researchers administered up to 11 treatments, every 2 months, to 30 keloid patients, depending on keloid response.

Improvement measured 12 months after treatment was based upon increases of skin surface uniformity and erythema reduction. Overall, keloid growth regressed or was arrested in all 30 patients after treatment. Twelve months after treatment, 26 patients had keloid regression of 50% or more, while 4 patients did not experience a change. Keloids that were treated more than 6 times regressed much more (79%) than keloids treated 6 times or less (50%).⁴⁵ Keloids in different locations responded equally well to treatments. Twelve months after final treatments, no patients exhibited signs of keloid progression.⁴⁵

At a microscopic level, TGF- β 1, PCNA, and collagen-3 were significantly reduced in fibroblasts. Results suggested the PDL treatment induces keloid regression by suppressing TGF- β 1 expression and by decreasing fibroblast proliferation and collagen-3 deposition. However, Kuo and colleagues could not determine exactly how the PDL inhibits these factors, although they stated similar results had not been seen with ablative lasers.^{33,45}

The PDL may also increase keloid-suppressing enzymes. A separate study by Kuo and colleagues in 2005 investigated whether the PDL decreased TGF- β 1 induction and upregulation of matrix metallo proteinase (MMP) expression in keloid reduction. MMPs are enzymes responsible for destroying connective tissue. Collagenases are a subfamily of MMPs and those studied (MMP-1, also known as collagenase-1, and MMP-13, also known as collagenase-3) play a key role in degrading the collagen matrix. Ten patients were treated with 585 nm PDL.

Seven days after treatment, all keloid tissue samples exhibited decreased TGF- β 1 and increased MMP-13. In an average count, TGF- β 1 expression decreased by 42%, while MMP-13 increased by 89%. There was no significant difference in MMP-1 expression before and after treatment. Results indicate PDL appears to suppress fibroblast TGF- β 1 expression and selectively increases MMP-13, but not MMP-1, in keloid tissue. This selective increase indicates that MMP-13, but not MMP-1, might play an important role in keloid regression after PDL treatment.⁴⁶ Researchers concluded that the regression of keloids after PDL treatments was associated with downregulation of TGF- β 1 expression and upregulation of MMP-13 activity.

Fibroblast studies have identified key mechanisms and cellular components responsible for keloid development and regression. In 2005, Kuo and colleagues examined whether the PDL prompted cell apoptosis and reduced fibroblast production. They hypothesized that keloid

fibroblasts had lower apoptosis rates than normal fibroblasts. They proposed that with the help of a 585-nm PDL laser targeting the fibroblasts' vascular and nutritional supply, the keloid fibroblasts could undergo apoptosis through the MAP kinase pathway, which is thought to control cell growth, differentiation, and apoptosis.⁷ Specifically, studies have shown that MAP kinases ERK and p38 are modulators of fibroblast differentiation.^{55,56} The theory was that this would lead to keloid regression after the PDL treatment.

Seven days after PDL therapy, results showed an increase in apoptotic bodies in the keloid fibroblast and elevated levels of MAP kinases and caspase-3, which is thought integral to apoptotic signal transduction. There was no significant difference in MAP kinase JNK expression before and after treatment. Researchers concluded that PDL treatment appeared to induce keloid regression through suppression of keloid fibroblast proliferation, induction of apoptosis, and upregulation of ERK and p38 MAP kinase activity.³⁰

However, while these studies depict positive possibilities for PDL treatment of keloids, it is critical to remember that no long-term follow up was included with these results. Since keloids can thrive with a suppressed blood supply, the PDL is far from a panacea.

Nd:YAG treatment

Nd:YAG lasers have also been explored as treatment possibilities for keloids. In 1984, Abergel and colleagues treated keloid fibroblast cultures and control cultures with the CW 1064-nm Nd:YAG. Keloid fibroblasts initially had twice the amount of collagen production as the controls, and when treated with a fluence of 700 J/cm², collagen production dropped to approximately the same level measured in controls. A fluence of 1100 J/cm² further inhibited collagen production in keloids. Abergel and colleagues reported collagen production was notably repressed with the 1100 J/cm² fluence, without affecting the viability or DNA replication of the cells. However, collagen production was not suppressed in controls using lower fluences.²⁷ Based on the fibroblast culture studies, a case study of Nd:YAG treatment of resistant keloids showed progressing lightening and flattening, with no recurrence noted at the 3-year follow up.²⁷

Researchers believed that the Nd:YAG laser most likely acts through a process involving a subtle biological modulation of the fibroblast functions, resulting in selective suppression of the procollagen production by the cells. The

treatment is relatively benign, as cell viability and DNA replication are not affected at the energy levels used for suppressing procollagen production. Optimizing treatment schedules and using relatively low energy densities might permanently suppress keloids, although data remains sparse. Abergel and colleagues concluded that sequential treatment, using the CO₂ laser to remove keloids followed by the Nd:YAG laser to suppress recurrence, may be an even more effective treatment. Unfortunately, most of the data was qualitative, as percent improvements of the patients and histology were not published. Patient success was described in only one case study.²⁷

For many years, keloids have been treated with ablative CO₂ lasers with mixed results. The CO₂ laser emits a wavelength of 10600 nm in the far infrared spectrum, and destroys tissue by vaporizing intracellular water. It also photocoagulates blood vessels and seals small lymphatics and nerve endings virtually bloodlessly. Unlike PDL treatment, the CO₂ laser penetration depends only on the tissue's water content, making vascularity irrelevant, and the treatment less specific and more prone to widespread tissue damage.⁵⁷

The original CW CO₂ lasers caused extensive thermal damage with unacceptable aesthetic results.⁵⁸ However, it has since been redesigned as a superpulsed laser to deliver higher fluences (often greater than 7 J/cm²) in much shorter pulse durations (1 ms). It is now possible to vaporize superficial tissue layers with a reduced area of thermal damage compared to what a CW CO₂ laser would produce, although in practice this is generally achieved using a spot size of 0.8 mm or smaller.⁵⁹ It would seem the ideal laser to treat the undervascularized keloid. However, despite these redesigns, CO₂ lasers still carry a high recurrence rate when treating keloids, and are currently not widely accepted for their treatment.²⁰ Also, because the intensity of postoperative erythema relates directly to the amount of thermal necrosis, CO₂ lasers will cause more severe, prolonged erythema than other ablative lasers, such as the Er:YAG.⁶⁰

The evidence supporting CO₂ laser use for keloids is sparse. A 1984 study by Apfelberg and colleagues measured the effects of ablative lasers argon and CO₂ lasers on 13 patients with well-established keloids. Unfortunately, all except one earlobe keloid either recurred or showed no clinical improvement. There was a minor temporary relief of pain and itching, but all symptoms returned to their original intensity over a period of weeks to months. Researchers found no long-term beneficial effects with either treatment.⁴⁷

Further studies echo keloid recurrence problems. For a 1989 study by Apfelberg and colleagues, nine keloids were excised on seven patients. The majority of treatments were given with a continuous CO₂ laser, although a superpulsed laser was used on two patients. Steroids were administered to five patients. Results appeared promising 12 months after treatment as all 8 patients initially demonstrated flatter, softer, and less pruritic scars. However, 8 of these 9 keloids recurred to original or nearly original size as early as 10 months and as late as 22 months post treatment. The only patient who did not experience a recurrence had only been followed for 9 months and required pressure dressings for her earlobe keloid constantly. Researchers determined that the laser had limited benefit over prolonged periods.⁴⁸

In 1989, Stern and Lucente excised 27 keloids on 18 patients. Four patients returned for more than one follow up, and these patients received 0.2 mL of corticosteroid triamcinolone (40 mg/mL), which was repeated up to 4 times at 3-week intervals depending on the severity of hypertrophic scarring.³³ Of the 23 keloids that received CO₂ laser therapy, 17 had recurrences. Stern and Lucente concluded that although CO₂ laser keloid excision followed by injections may improve the overall success rate of the laser treatment, it is unlikely that CO₂ laser excision would be superior to scalpel excision, due to the known occurrence of hypertrophic scarring and keloid formation associated with the treatment.³³

In other studies, combination therapy has been shown to help, albeit with limited results. In 1991, Norris treated 23 patients with the continuous CO₂ laser in combination with steroids. Out of the 23 patients, only 1 patient had no recurrence, 9 patients had recurrences suppressed with steroids, and 13 patients had recurrences with or without concomitant steroid therapy. Norris concluded that CO₂ laser excision alone of keloids failed to suppress keloid growth or recurrence. It is noted that failures may also be tied to poor patient compliance, the inability to control the keloid with steroids, and the involved area being too large to be effectively treated with steroids.⁴⁹

Some limited success in treating keloids has been achieved with the CO₂ device. Results of other studies indicate keloid fibroblasts secrete more TGF- β 1 than normal fibroblasts.⁶¹ Nowak and colleagues published an in vitro study in 2000 that examined the effects of the superpulsed CO₂ laser on fibroblast proliferation and growth factors.⁵⁰ Results showed the CO₂ laser caused keloid cell growth to regress to normal dermal fibroblast levels. CO₂ laser treatment caused statistically significant

growth rates in both treated groups, and a statistically significant increase in keloid bFGF levels compared to keloid controls. TGF- β 1 also increased in both groups, but not significantly. A stabilized cellular phenotype and decreased collagen production suggested that the superpulsed CO₂ laser may be a promising keloid treatment. Researchers recognized that the growth factors do not act in isolation, although they suggested that the effect of the superpulsed CO₂ laser on growth factors may help normalize wound healing in the future.⁵⁰

It is possible that the disappointing results in early studies of CO₂ laser treatment were mainly due to the damaging effects of the early CW laser design. Unfortunately, since the laser has been reconfigured as a superpulsed device, there is still little evidence suggesting that the CO₂ laser is an effective long-term treatment for keloids.

Overall, literature for laser treatment of keloids does not reflect the same enthusiastic results as studies for the laser treatment of HS.

■ Complications to Avoid

Laser treatment of HS and keloids carries substantial risks. Adverse effects can be temporarily inconvenient or permanently damaging. Mild complications from dermatologic laser treatment include prolonged erythema, acne, milia formation, contact dermatitis, and pruritis. Moderate complications include the risk of bacterial and fungal infections, postinflammatory hyperpigmentation, delayed-onset hypopigmentation, and reactivation of herpes simplex virus. Severe complications include widespread infection and additional hypertrophic scarring. A successful treatment depends on the skill of the laser surgeon, intraoperative technique, strict adherence to postoperation wound treatment, and proper patient selection.⁶⁰

Regarding postoperative care, the lased area must be cleaned and dressed daily and strict sun protection must be enforced. Treated tissue should be evaluated 6 to 8 weeks after the original treatment to evaluate the benefits of future treatments. If hyperpigmentation is present after treatment, future treatments must be postponed until pigmentation is resolved, to avoid absorbance interference from melanin.²² Bleaching cream, sunblock, and strict sun avoidance can help resolve excess pigmentation. In terms of patient selection, skin types I and II generally fare best, due to a lack of melanin, a competing chromophore.⁶²

In terms of preventing HS and keloids from forming or recurring, studies have suggested that extended angiogenesis is a major contributing factor to HS formation.³ Reiken and colleagues indicated that the PDL could be used during these early healing stages to regulate the amount of new blood vessels in the wound.¹¹ It has also been suggested that laser treatment can protect against abnormal scar formation by encouraging the development of young, growing collagen fibers that are sensitive to intralesional corticosteroids. However, if scars are allowed to mature, these injections will not be as effective, as collagen synthesis will have reached a close balance with collagen lysis and the fibers will no longer respond to steroids.³⁶

Studies have shown lasers to be safe and efficient methods of preventing hypertrophic scarring. Researchers suggest that because HS have increased microvasculature, using selective photothermolysis to target blood vessels will decrease cell function and nutrition in the developing scar.¹⁰ The majority of positive prevention results occurred using the PDL on lighter-skinned patients (Fitzpatrick skin types I–IV). McCraw and colleagues conducted the first study examining HS prevention by early PDL treatment on 106 patients and concluded that early HS would need aggressive treatment (fluences between 4 and 4.5 J/cm² with a spot size of 10 mm and a 20% pulse overlap).⁴⁴ Overall, the prophylactic treatment resulted in faster repair of stiffness and erythema, less frequent HS development, and excellent color matches between scar tissue and skin.¹²

In 2003, Nouri and colleagues examined the effects of 585-nm PDL treatment on surgical scars beginning on suture removal day. Vascularity in treated scars improved 54% compared with 8% in controls. Pliability improved by 64% in treated scars compared with 1% in controls. There were no significant differences in scar pigmentation or height between treated and control scars.¹⁴ A scar-grading scale used biopsies to determine the degree of scarring on a scale of 0 (normal dermis, normal elastic tissue network) to 4 (complete scarring, disruption of 75%–100% of elastic tissue network).¹⁴ Treated scar halves scored better, with an average score of 1.3, compared with the average control score of 3.3. Treated scars also scored significantly better in cosmetic appearance.¹⁴

A 2006 study by Conologue and colleagues confirmed these results. Starting on suture removal day, 13 patients (skin types I–IV) received three treatments in 4- to 8-week intervals with a 595-nm cryogen-cooled PDL. Treated scars improved significantly, on an average, with the most marked effects in vascularity and pliability. There

were no differences in pigmentation between treated and untreated scars. Additionally, treated scars scored on average 2 points higher on the cosmetic scale than untreated scars, with an average improvement of 24%, compared with untreated scars, which averaged a 7.8% cosmetic improvement. Like Nouri and colleagues, researchers determined early PDL treatment a safe, successful option to improve the appearance of surgical scars in type I to IV skin.¹⁶

Prophylactic laser treatment has not shown as promising results in patients with darker skin tones. Little research has measured the effects of PDL treatment on darker skin because melanin can act as a competing chromophore, heightening the risk of undesirable side effects. In 2004, Chan and colleagues examined the efficacy and side effects of the PDL regarding HS prevention and treatment in 56 Chinese patients. Approximately 19/35 (54%) of prevention group scars (less than 6 months old) and 24/36 (67%) of the treatment group scars (over 6 months old) were noted as “better” or “much better” after laser treatment, according to a patient questionnaire. However, overall changes (especially regarding scar thickness and viscoelasticity), when measured against controls, were not significant. Chan and colleagues also advocated the use of adjunct therapies such as hydroquinones, but did not specify when or how these should be delivered for a more successful outcome.¹⁵

STRIAE

■ Definition, Cellular Anatomy, and Etiology

Striae distensae, commonly known as stretch marks, are atrophic dermal scars with epidermal atrophy.⁶³ Striae rubra (red) are early phase stretch marks, while striae alba (white) are mature stretch marks.

Striae are caused by breaks in connective tissue due to excessive stretching, which lead to collagen ruptures, dermal atrophy, and scarring.⁶⁴ Additional mechanisms may include fibroblast malfunctioning due to rapid tissue growth, as in growth spurts or pregnancy,⁶⁵ as well as mast cell degranulation with elastolysis.⁶ It is not fully understood why striae occur, but hormones (especially corticosteroids and estrogen), mechanical stress, excessive exercise, rapid weight gain or loss, obesity, and genetic predisposition all seem to play a role.^{6,63,66} It is also thought that striae are a special case of connective dystrophy.⁶⁵

■ Epidemiology

Striae are extremely common, and develop between ages 5 and 50, although they predominate during growth periods such as puberty and pregnancy. There is a 25% to 35% overall incidence during puberty, and a 77% overall incidence during pregnancy.⁶⁶ They are more common in younger women experiencing their first pregnancy than in older pregnant women. Striae are more prevalent among Caucasians than other races, and occur twice as frequently in women as in men. They are also associated with conditions causing excess cortisol, such as Cushing syndrome.⁶⁶

■ Patient Selection and Preprocedure Evaluation

Striae occur as multiple and symmetric linear atrophic lesions that follow the lines of cleavage. They occur in regions where the dermis has been injured due to the skin stretching,⁶⁶ perpendicular to the direction of largest tissue tension.⁶⁴ During puberty, striae often develop on the breasts, hips, thighs, and buttocks of females, and on the shoulders, lumbosacral region, and thighs of males. They occur less commonly on the abdomen, upper arms, neck, and axillae.⁶⁶ During pregnancy, they are common on the abdomen, and less common on the breasts and thighs.⁶⁶ In pregnant women, stretch marks are also statistically significant predictors of lacerations during vaginal delivery.⁶⁶ Striae occurring from systemic corticosteroid treatment and Cushing syndrome, with its attendant overproduction of cortisol, appear wider and larger than normal and unlike physiologic striae, are more common in flexural and intertriginous areas.⁶⁶

Striae first appear as red or purple elevated lines on the skin. These early striae are known as striae rubra and can be pruritic,⁶⁶ erythematous, fibrotic, and contain dilated capillaries. Clinically and histologically, they are similar to early scars.⁶ With time, the color diminishes and the lesions become atrophic. Mature striae cause the skin to look delicately wrinkled and are known as striae alba.⁶⁶ These are generally permanent, but sometimes fade with age. They are hypopigmented and fibrotic, like older scars, and exhibit fragmented elastic fibers and epidermal atrophy.⁶ Mature striae can be several centimeters in length and between a few millimeters to a few centimeters in width.⁶⁶

The diagnosis of striae is generally uncomplicated, but should be distinguished from linear focal elastosis, which presents with conspicuous yellow striae-like bands on the lower back, a condition which most often affects elderly men.⁶⁶

Histologically, striae cause notable changes in affected skin. Fibroblasts are globular and quiescent with no signs of fibrillar secretion.⁶⁵ Collagen becomes thin and fragmented while ground substance is plentiful. Collagen bundles in the dermis are thin, short, uncoiled, and parallel to the epidermis. The few elastic fibers are thin and fragmented as well. There is no conversion between normal and striated skin; at meeting points, the elastic fibers form heaps and appear retracted and dystrophic.⁶⁵ Striae usually appear to reach into the mid-dermis or a little deeper before normal randomly arranged collagen reemerges.⁶⁷ In comparison, the dermis of normal skin is compact, elastin fibers are thick and organized, and collagen bundles are thick, wavy, and densely packed.^{65,67} The histological appearance of striae is possibly due to mast cell degranulation, which destroys collagen and elastin fibers.⁶³

Although striae share some characteristics with scars and aging skin, they are considerably different. In scars, active fibrillogenesis is observed, especially when intracytoplasmic collagen is present, a sign of greater collagen synthesis. Striae fibroblasts never display this, as their fibroblasts are quiescent and do not have secretory organelles.⁶⁵ In aging skin, collagen is much less displaced and fragmented than in striae. Aging skin also has a lot of ground substance, but it does not inhabit large regions, and fibroblasts show signs of secretion.⁶⁵

Striae usually pose no medical danger and are generally a cosmetic concern, but they can be psychologically worrisome for patients, and, in rare circumstances, they can ulcerate.⁶⁶ Treatment response tends to be superior in rubrae, while older, deeper striae have a less noticeable response. Fitzpatrick skin types I to III are suitable for all types of striae treatment, while tanned skin type III and darker respond better to nonablative lasers with cooling, topical treatment, microdermabrasion, and ultrasonic sonophoretic treatment than to PDL.⁶³

■ Techniques

The goal of striae treatment is improvement, as complete elimination of striae is not realistic.⁶³ Possible treatments include PDL (585 nm), short-pulsed CO₂ and Er:YAG lasers, nonablative Nd:YAG (1320 nm), IPL Quantum SR, topical retinoic acid or L-ascorbic acid mixtures, microdermabrasion, and ultrasonic sonophoretic therapy. There is no monotherapy for striae, and none of these treatments is drastically better than the others (Table 11.3).⁶⁶ It is important to note the difficulty in assessing the effectiveness of treatments as striae tend to spontaneously regress over time.⁶⁶

TABLE 11.3 ■ Striae Treatment Studies

Author	# Patients	Treatment	Cooling Devices	# Treatments	Results	Side Effects
Topical Treatment Studies						
Elson (1990)	16	Topical: 0.1% Retin-A cream.	N/A	Applied daily for 12 weeks.	Some benefit for majority of patients, results neither quantified nor discussed in detail.	Erythema, vesicular dermatitis.
Pribanich et al. (1994)	11	Topical: 0.025% tretinoin cream.	N/A	Applied daily for 32 weeks.	Double-blind, placebo-controlled study. No differences between treatment and control groups. 0.025% tretinoin cream deemed ineffective.	Mild pruritis.
Ash et al. (1998)	10	Topical: 10% L-ascorbic acid mix applied to one treatment half, 0.05% tretinoin emollient cream (Renova) applied to other treatment half. 20% glycolic acid applied to both treatment halves.	N/A	Applied daily for 12 weeks.	Both L-ascorbic acid mixture and tretinoin cream improved appearance of mature striae when combined with glycolic acid. Both treatments increased epidermal thickness and decreased papillary dermal thickness. Tretinoin increased elastin content in reticular and papillary dermis.	Minor skin irritation, mild dermatitis.
Laser Studies						
McDaniel et al. (1996)	39	PDL: 585 nm, 2.0–4.0 J/cm ² , 450 μs, 7 or 10 mm.	Not noted	1	Best results occurred with a fluence of 3 J/cm ² and a spot size of 10 mm. Striae treated with low fluences showed elastin content virtually indistinguishable from normal nonstriae skin.	Temporary hypopigmentation or hyperpigmentation seen in 2 patients. Mild purpura and erythema occurred but cleared mainly by 4 wks.

Nouri et al. (1999)	4	PDL: 585 nm, 3 J/cm ² , 10 mm. CO ₂ : 350 or 400 mJ, 3 mm.	Not noted.	1	Patients had skin types IV and VI. Striae remained unchanged or worsened. Researchers cancelled further trials due to poor results and emphasized treating darker skin types extremely cautiously with lasers.	Erythema, hyperpigmentation.
Jimenez et al. (2003)	20	PDL: 585 nm, 3.0 J/cm ² , 450 μs, 10 mm.	Not noted.	2	No dramatic changes. 4/9 (44%) striae rubra patients showed color changes. No color changes in striae alba. Researchers concluded PDL treatment best reserved for pink or red striae, and used cautiously on patients with darker skin types.	Hyperpigmentation

PDL, pulsed dye laser.

Topical treatments are often cited as an alternative to laser therapy, especially for those patients who may experience pigmentation problems from laser treatment. Anecdotal success has been reported from use of topical tretinoin and alpha hydroxy acids such as glycolic acid.²³ Tretinoin is thought to affect the fibroblasts, potentially decreasing proliferation and collagen synthesis.⁶⁷ It is often used in treating photodamaged skin, and has been shown to increase collagen production, fibroblast activity, and angiogenesis.⁷⁰ Glycolic acid has been shown to be a useful peeling agent, and can increase epidermal and papillary dermal thickness, increase acid mucopolysaccharide, and improve elastic fibers and the density of collagen.⁷⁰ In combination with tretinoin, it has been shown to improve cutaneous photodamage and acne vulgaris.⁷⁰ Skin irritation is a possible adverse effect when using topical therapies.

In 1990, Elson tested the effects of topical tretinoin on striae. Sixteen patients (15 female, 1 male) with stretch marks resulting from puberty or pregnancy were given 0.1% Retin-A cream and instructed to apply the treatment daily to the affected regions. Elson did not specify whether treated striae were striae rubra, striae alba, or a mix. Patients were assessed at 4, 6, 8, and 12 weeks. Results were anecdotal and were neither quantified nor discussed in detail, but Elson reported that the Retin-A provided some benefit for all patients except one. Adverse effects included erythema and vesicular dermatitis. Elson concluded that the Retin-A Cream 0.1% was the most effective dosage level.⁶⁸

In 1994, Pribanich and colleagues assessed the effects of a 0.025% tretinoin cream on striae in a double-blind, placebo-controlled study. All striae were initially assessed as severe (more than four striae of any width, or too many to count). Striae were a mix of striae rubra and alba, with ages 2 weeks to 13 years. At the end of the study, all striae were still graded as severe. Even when variables such as patient perception and weight gain or loss were controlled for, there were no differences or improvements between the treatment group and the placebo group. None of the patients mentioned an exfoliative reaction while using the 0.025% cream. Adverse effects included mild pruritis, which was reported in both the treatment and placebo group. Researchers concluded that the 0.025% tretinoin cream was ineffective and suggested a 1% tretinoin cream might be more effective, although there was no objective data to date.⁶⁹

A 1998 study by Ash and colleagues compared topical treatments for mature striae. Ten female patients (skin

types I–V) with abdominal striae alba (6 patients had additional thigh striae) participated. Topical 20% glycolic acid was applied daily to both treatment halves. Patients also applied a mixture of topical 10% L-ascorbic acid, shown to be an effective treatment for photodamaged skin,^{70,73} 2% zinc sulfate, and 0.5% tyrosine to one treatment half, and 0.05% tretinoin emollient cream (Renova) to the other treatment half.

Histopathologically, when compared to untreated striae sites, L-ascorbic acid-treated sites showed no improvement in the elastin content of the papillary dermis, decreased papillary dermis thickness by a mean of 28%, showed no improvement in the reticular dermis elastin content, and increased epidermal thickness by a mean score of 116%.

When compared to untreated striae sites, 0.05% tretinoin-treated sites increased papillary dermal elastin content by a mean score of 107%, decreased papillary dermis thickness by a mean score of 27%, increased reticular dermis elastin content by a mean score of 17%, and increased epidermal thickness by a mean score of 5.9%. Adverse effects were mild and included minor skin irritation and one case of mild irritant dermatitis.

Researchers concluded that while the sample size was very small, both the 10% L-ascorbic acid and the 0.05% tretinoin treatments improved the appearance of mature stretch marks when combined with 20% glycolic acid. Both regimens also increased epidermal thickness, decreased papillary dermal thickness, and caused combined epidermal and papillary dermal thickness in mature striae to approach normal skin. The 0.05% tretinoin combined with 20% glycolic acid increased elastin content in the reticular and papillary dermis.⁷⁰

■ Laser Treatment of Extant Striae

Because striae are dermal scars, and laser treatment has shown significant efficacy treating HS, the efficacy of striae laser treatment was examined. Researchers have had modest success treating striae with the PDL, but results were not nearly as successful as PDL HS treatment. However, early striae (less than a year old), like early scars, often do not necessarily need laser treatment, as it is possible that symptoms such as erythema will fade over time.⁶

In 1996, McDaniel and colleagues studied the effect of a 585-nm PDL on striae. Thirty-nine striae were treated from a group of female patients (mean age of striae was 14 years). Researchers found that striae appearance improved with different treatment protocols,

but a fluence of 3.0 J/cm^2 and a 10-mm spot size caused the greatest normalization. Histopathologically, while untreated striae appeared thinner but otherwise similar to normal skin, treated striae showed a reduction in the normal papillary patterns of the epidermis. Treated striae also showed normal levels of elastin stain in papillary and mid-dermis, revealing thick and thin elastic fibers. However, untreated striae showed very little uptake of elastin stain, signaling that there were markedly fewer elastin fibers present.

Adverse effects were minimal (transient hypopigmentation, transient hyperpigmentation, mild purpura, mild erythema) but resolved spontaneously.

Overall, researchers recommended treatment at low-energy densities and advocated optical profilometry as a more effective way of assessing improvement.⁶⁷

In 1999, Nouri and colleagues examined the efficacy of the short-pulsed CO_2 laser and the PDL on striae in four patients with skin types IV and VI. The CO_2 section was treated with the short-pulsed CO_2 laser, with a 3-mm spot size at 400 mJ on the first pass, followed by a second pass at 350 mJ after a saline wash to remove any eschar. The second section was a control section. The third section was treated with a 585-nm PDL with a 10-mm spot size, and a fluence of 3 J/cm^2 . Results were unfavorable: striae showed no changes or worsened. Adverse effects included persistent erythema and hyperpigmentation. Because results were so poor, researchers cancelled further trials with additional patients. Researchers concluded that for skin types IV to VI, treatment of striae with the short-pulsed CO_2 laser or the 585-nm PDL should be avoided or used extremely carefully. Researchers advised narrow use of the lasers for striae on skin types I to III.⁷¹

In 2003, Jimenez and colleagues examined whether the 585-nm PDL would be an effective treatment for striae rubra and alba after two treatments and whether this treatment would affect collagen density. Researchers found mixed results. At week 12, there was no significant difference in decreased area of striae between treated and control groups. Less than half of the striae rubra (4/9) and none of the striae alba showed color changes after PDL treatment. No significant differences were found regarding striae location or age. Biopsy samples revealed treated striae had a net increase in collagen content and a net decrease in striae area, while control striae showed a net decrease in collagen content at 12 weeks. Adverse effects included hyperpigmentation.

Researchers admitted that there was not a striking difference in striae appearance after two treatments with the PDL. The skin types of the patients (II–IV) were suggested as a possible interference with results, as melanin competes with hemoglobin for the PDL laser light. Striae color at the beginning of treatment was an important factor—only striae that were pink or red in color showed any improvement, while no improvement in color was observed in white striae. Researchers concluded that striae PDL treatment should be reserved for pink or red striae, used with caution on darker skin types, and include a follow-up longer than 12 weeks, which would focus on the changes in collagen as compared to other extracellular matrix components and clinical parameter changes after laser treatment.⁷²

CONCLUSION

Table 11.4 shows the comparison between HS, keloids, and striae.

Clinical Recommendations: HS

Intralesional TAC injections are the preferred HS treatment and are often used alone or in combination with pressure treatment or surgery. Dosages are administered at 5 to 10 mg/mL and repeated monthly, with clinical improvement as the long-term end point.⁸ Adverse effects include atrophy, pain upon injection, and pigmentation problems.

HS can also be treated with the PDL. Parameters include a wavelength between 585 and 595 nm, fluences from 6 to 10 J/cm^2 , pulse duration of 450 μs , and spot sizes between 2 and 10 mm.^{1,22,23} For sensitive skin, lower fluences with a larger spot size are recommended ($4.5\text{--}5.5 \text{ J/cm}^2$ and 10 mm), while thicker, more pigmented scars can be treated with a higher fluence and a smaller spot size ($6.5\text{--}10 \text{ J/cm}^2$ and 5 mm).²² PDL treatment is best for patients with skin types I to III. The most common adverse effects are purpura and pigmentation problems. Steroids can be used concomitantly, but this combination has not proven to be completely effective.

Clinical Recommendations: Keloids

There is no optimal treatment for keloids. One option is to excise the lesion followed immediately by intralesional TAC injection, with postoperative TAC injections every 2 to 6 weeks until clinical resolution or significant side

TABLE 11.4 ■ Comparison Between Hypertrophic Scars, Keloids, and Striae

	Hypertrophic Scars	Keloids	Striae
Etiology	Exact origin unknown. Appear 6–8 wks after wound reepithelialization. Mature by 1 yr.	Exact origin unknown. Possible links to hormones, family inheritance, blood type A, and certain connective tissue diseases. ^{32,34,35} Can appear at any time, from weeks to years after original injury.	Occur due to bodily expansion (puberty, pregnancy), hormones, excessive exercise, or family history. ^{6,63,65,66}
Prevalence	1.5%–4.5% of general population. ³ Equal prevalence between males and females. Often develop between ages 10 and 30. Usually appear in presternal area, upper back, and deltoid region. ⁶	Higher prevalence among darker skin types: 15 times as likely to appear in patients of African, Spanish, and Asian origin than in patients with fairer skin types. ³² Equal prevalence among men and women. Often develop between ages of 10 and 30. ⁷	Higher prevalence among Caucasians and females. Usually develop between ages of 5 and 50, especially during puberty and pregnancy. ⁶⁶ More common in younger women experiencing first pregnancy. Also more common in obese individuals and weight lifters.
Diagnosis	Raised, erythematic, symptomatic, confined to original region of injury. Histologically, collagen bundles are flatter than normal skin and set in wavy pattern. Express myofibroblasts, increased mast cells. ^{2,4} Generally resolve over time. Good treatment response.	Raised, red–purple scars that extend beyond original boundaries of injury. Histologically, few collagen bundles arranged randomly, poorly vascularized. No myofibroblasts. Increased mast cells. No resolution over time. Poor treatment response.	Multiple and symmetric linear atrophic lesions following lines of cleavage. Occur in regions of injury due to skin stretching: breasts, hips, thighs, buttocks, upper arms (puberty), abdomen (pregnancy and puberty), armpit, groin, underside of breasts (cortisol imbalance, Cushing's syndrome). Bright red or purple fading to white over time. Histologically, collagen is thin and fragmented, few elastic fibers, globular quiescent fibroblasts. Poor treatment response.
Who Requires Treatment	Patients experiencing functional or cosmetic deformities, discomfort, pain, psychological stress. ⁸	Patients experiencing functional or cosmetic deformities, discomfort, pain, and psychological stress. ⁸	Patients experiencing psychological distress or ulceration. ⁶⁶
Treatment Types	Corticosteroid injections, 5-fluorouracil, interferon (α , β , and γ), bleomycin, radiotherapy, surgery, cryosurgery, pressure garments, silicone gel sheeting, laser therapy.	Conventional surgery in combination with intralesional corticosteroids, silicone gel sheeting, pressure garments, cryosurgery, laser therapy. Preferred treatment: unknown.	Topical retinoic acid, topical L-ascorbic acid, laser therapy, microdermabrasion, ultrasonophoretic therapy. Preferred treatment: unknown.

TABLE 11.4 ■ Comparison Between Hypertrophic Scars, Keloids, and Striae (Continued)

	Hypertrophic Scars	Keloids	Striae
	Preferred treatment: Intralesional TAC or laser therapy.		
Laser Treatment Types	595-nm PDL, with fluences 5–10 J/cm ² , spot size 2–10 mm, pulse duration 450 μ s. ²	595-nm PDL, with fluences 6.5–7.25 J/cm ² , spot size 2–10 mm, pulse duration 450 μ s. ² 1064-nm Nd:YAG, power of 60 W, fluence 60 J/cm ² , spot size 1 cm. ² Superpulsed 10600 nm CO ₂ , power 4–8 W, pulse duration 730 μ s–1 ms, fluences 2–7 J/cm ² , spot size 0.8 mm or lower.	585-nm PDL, short pulsed CO ₂ and Er:YAG lasers, nonablative 1320 nm Nd:YAG, IPL Quantum SR.

TAC, triamcinolone acetonide; PDL, pulsed dye laser; IPL, intense pulsed light.

effects. Recommended TAC dosage is 10 mg per linear centimeter of keloid.⁷⁴ Possible adverse effects include pain upon injection, skin atrophy, pigmentation problems, and telangiectases.²⁰ Pressure dressings may be used concurrently. Laser treatment is not advised.

■ Clinical Recommendations: Striae

Like keloids, striae have yet to show strong, consistent results with treatment options. Striae rubra may be treated with the 585-nm PDL, with a fluence of 3 J/cm², pulse duration of 450 μ s, and spot size of 10 mm adverse effects include erythema and pigmentation problems. Striae alba may be treated with either 10% L-ascorbic acid or 0.05% tretinoin cream. Creams should be used with 20% glycolic acid and may be applied daily until resolution of striae. Adverse effects include minor skin irritation.

Future studies should avoid flaws in past literature. Many studies had a very limited sample size, resulting in data that were more observational than scientifically valid.^{9,10,13,14,17,27,33,44,47,48} Some studies left out hard data, and other literature did not present the data clearly or used charts as an approximation of their findings.⁹ Some methods and discussion sections were too short and unclear,⁶⁷ other studies omitted methodological parameters such as spot size and pulse duration,^{9,11,13,48} and still more relied too heavily on subjective assessment to gauge patient improvement.¹³ A few studies did not even distinguish between HS and

keloids.^{13,24} Adverse effects were also frequently unmentioned.^{9,11,27}

In the future, research will continue to focus on more effective treatments for keloids and striae, as well as better preventative measures for each.

REFERENCES

1. Tanzi EL, Alster TS. Laser treatment of scars. *Skin Therapy Lett.* 2004;9:4-7.
2. Burton. *Dermatology Book.*
3. Alster T. Laser scar revision: Comparison study of 585-nm pulsed dye laser with and without intralesional corticosteroids. *Dermatol Surg.* 2003;29:25-29.
4. English RS, Shenefelt PD. Keloids and hypertrophic scars. *Dermatol Surg.* 1999;25:631-638.
5. Wolff K, Johnson RA, Suurmond D. *Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology [e-book]*. 5th ed. New York, NY: McGraw-Hill; c2005 [cited 2006 Aug 17]. Available at <http://www.library.tufts.edu/hsl/>.
6. Alster TS. Laser treatment of hypertrophic scars, keloids, and striae. *Dermatol Clin.* 1997;15:419-429.
7. Alster TS, Tanzi EL. Hypertrophic scars and keloids: Etiology and management. *Am J Clin Dermatol.* 2003;4:235-243.

8. Chen MA, Davidson TM. Scar management: Prevention and treatment strategies. *Curr Opin Otolaryngol Head Neck Surg*. 2005;13:242-247.
9. Mustoe TA, Cooter RD, Gold MH, et al. International clinical recommendations on scar management. *Plast Reconstr Surg*. 2002;110:560-571.
10. Anderson RR, Parrish JA. Selective photothermolysis: Precise microsurgery by selective absorption of pulsed radiation. *Science*. 1983;220:524-527.
11. Groover IJ, Alster TS. Laser revision of scars and striae. *Dermatol Ther*. 2000;13:50-59.
12. Stratigos AJ, Dover JS, Arndt KA. Laser therapy. In: Bologna JL, Jorizzo JL, Rapini RP Sr, et al., eds. *Dermatology*. London, UK: Mosby; 2003:2153-2175.
13. Hirsch RJ, Anderson RR. Principles of laser-skin interactions. In: Bologna JL, Jorizzo JL, Rapini RP Sr, et al., eds. *Dermatology*. London, UK: Mosby; 2003:2143-2151.
14. Garden JM, Tan OT, Kerschmann R, et al. Effect of dye laser pulse duration on selective cutaneous vascular injury. *J Invest Dermatol*. 1986;87:653-657.
15. Alster TS, Kurban AK, Grove GL, Grove MJ, Tan OT. Alteration of argon laser-induced scars by the pulsed dye laser. *Lasers Surg Med*. 1993;13:368-373.
16. Dierickx C, Goldman MP, Fitzpatrick RE. Laser treatment of erythematous/hypertrophic and pigmented scars in 26 patients. *Plast Reconstr Surg*. 1995;95:84-90; discussion 91-92.
17. Reiken SR, Wolfort SF, Berthiaume F, Compton C, Tompkins RG, Yarmush ML. Control of hypertrophic scar growth using selective photothermolysis. *Lasers Surg Med*. 1997;21:7-12.
18. McCraw JB, McCraw JA, McMellin A, Bettencourt N. Prevention of unfavorable scars using early pulse dye laser treatments: A preliminary report. *Ann Plast Surg*. 1999;42:7-14.
19. Manuskiatti W, Fitzpatrick RE. Treatment response of keloidal and hypertrophic sternotomy scars: Comparison among intralesional corticosteroid, 5-fluorouracil, and 585-nm flashlamp-pumped pulsed-dye laser treatments. *Arch Dermatol*. 2002;138:1149-1155.
20. Nouri K, Jimenez GP, Harrison-Balestra C, Elgart GW. 585-nm pulsed dye laser in the treatment of surgical scars starting on the suture removal day. *Dermatol Surg*. 2003;29:65-73; discussion 73.
21. Chan HH, Wong DS, Ho WS, Lam LK, Wei W. The use of pulsed dye laser for the prevention and treatment of hypertrophic scars in Chinese persons. *Dermatol Surg*. 2004;30:987-994; discussion 994.
22. Conologue TD, Norwood C. Treatment of surgical scars with the cryogen-cooled 595 nm pulsed dye laser starting on the day of suture removal. *Dermatol Surg*. 2006;32:13-20.
23. Bowes LE, Nouri K, Berman B, et al. Treatment of pigmented hypertrophic scars with the 585 nm pulsed dye laser and the 532 nm frequency-doubled Nd:YAG laser in the Q-switched and variable pulse modes: A comparative study. *Dermatol Surg*. 2002;28:714-719.
24. Bhardwaj S, Tope WD, Frieden IJ. Long-pulsed Nd:YAG laser treatment of venous malformations [abstract]. *Lasers Surg Med*. 2006;38(suppl):22. Abstract 67.
25. Acikel C, Ergun O, Ulkur E, Servet E, Celikoz B. Camouflage of self-inflicted razor blade incision scars with carbon dioxide laser resurfacing and thin skin grafting. *Plast Reconstr Surg*. 2005;116:798-804.
26. Hulsbergen Henning JP, Roskam Y, van Gemert MJ. Treatment of keloids and hypertrophic scars with an argon laser. *Lasers Surg Med*. 1986;6:72-75.
27. Apfelberg DB, Maser MR, Lash H. Argon laser treatment of cutaneous vascular abnormalities: progress report. *Ann Plast Surg*. 1978;1:14-18.
28. Dixon JA, Huether S, Rotering R. Hypertrophic scarring in argon laser treatment of port-wine stains. *Plast Reconstr Surg*. 1984;73:771-779.
29. Abergel RP, Dwyer RM, Meeker CA, Lask G, Kelly AP, Uitto J. Laser treatment of keloids: A clinical trial and an in vitro study with Nd:YAG laser. *Lasers Surg Med*. 1984;4:291-295.
30. Kuo YR, Wu WS, Jeng SF, et al. Activation of ERK and p38 kinase mediated keloid fibroblast apoptosis after flashlamp pulsed-dye laser treatment. *Lasers Surg Med*. 2005;36:31-37.
31. Beer TW, Baldwin HC, Goddard JR, Gallagher PJ, Wright DH. Angiogenesis in pathological and surgical scars. *Hum Pathol*. 1998;29:1273-1278.
32. Niessen FB, Spauwen PH, Schalkwijk J, Kon M. On the nature of hypertrophic scars and keloids: a review. *Plast Reconstr Surg*. 1999;104:1435-1458.
33. Stern JC, Lucente FE. Carbon dioxide laser excision of earlobe keloids. A prospective study and critical

- analysis of existing data. *Arch Otolaryngol Head Neck Surg*. 1989;115:1107-1111.
34. Marneros AG, Norris JE, Olsen BR, Reichenberger E. Clinical genetics of familial keloids. *Arch Dermatol*. 2001;137:1429-1434.
 35. Marneros AG, Norris JE, Watanabe S, Reichenberger E, Olsen BR. Genome scans provide evidence for keloid susceptibility loci on chromosomes 2q23 and 7p11. *J Invest Dermatol*. 2004;122:1126-1132.
 36. Henderson DL. In discussion of: Norris JE. Effect of carbon dioxide laser surgery on the recurrence of keloids. *Plast Reconstr Surg*. 1991;87:50-53.
 37. Brown LA, Jr., Pierce HE. Keloids: Scar revision. *J Dermatol Surg Oncol*. 1986;12:51-56.
 38. Burd A, Huang L. Hypertrophic response and keloid diathesis: Two very different forms of scar. *Plast Reconstr Surg*. 2005;116:150e-157e.
 39. Griffith BH. The treatment of keloids with triamcinolone acetonide. *Plast Reconstr Surg*. 1966;38:202-208.
 40. Griffith BH, Monroe CW, McKinney P. A follow-up study on the treatment of keloids with triamcinolone acetonide. *Plast Reconstr Surg*. 1970;46:145-150.
 41. Berman B, Bielewicz HC. Adjunct therapies to surgical management of keloids. *Dermatol Surg*. 1996;22:126-130.
 42. Leventhal D, Furr M, Reiter D. Treatment of keloids and hypertrophic scars: A meta-analysis and review of the literature. *Arch Facial Plast Surg*. 2006;8:362-368.
 43. Rusciani L, Rossi G, Bono R. Use of cryotherapy in the treatment of keloids. *J Dermatol Surg Oncol*. 1993;19:529-534.
 44. Rusciani L, Paradisi A, Alfano C, Chiummariello S, Rusciani A. Cryotherapy in the treatment of keloids. *J Drugs Dermatol*. 2006;5:591-595.
 45. Layton AM, Yip J, Cunliffe WJ. A comparison of intralesional triamcinolone and cryosurgery in the treatment of acne keloids. *Br J Dermatol*. 1994;130:498-501.
 46. Newsome E, Bolling R, Langston K, Wang A, Jansen D. *Wound healing, keloids*. Emedicine Web site. March 2006. Available at <http://www.emedicine.com/plastic/topic404.htm>. Accessed Jan 3, 2007.
 47. Mercer NS. Silicone gel in the treatment of keloid scars. *Br J Plast Surg*. 1989;42:83-87.
 48. Alster TS, Williams CM. Treatment of keloid sternotomy scars with 585 nm flashlamp-pumped pulsed-dye laser. *Lancet*. 1995;345:1198-1200.
 49. Kuo YR, Jeng SF, Wang FS, et al. Flashlamp pulsed dye laser (PDL) suppression of keloid proliferation through down-regulation of TGF-beta1 expression and extracellular matrix expression. *Lasers Surg Med*. 2004;34:104-108.
 50. Kuo YR, Wu WS, Jeng SF, et al. Suppressed TGF-beta1 expression is correlated with up-regulation of matrix metalloproteinase-13 in keloid regression after flashlamp pulsed-dye laser treatment. *Lasers Surg Med*. 2005;36:38-42.
 51. Apfelberg DB, Maser MR, Lash H, White D, Weston J. Preliminary results of argon and carbon dioxide laser treatment of keloid scars. *Lasers Surg Med*. 1984;4:283-290.
 52. Apfelberg DB, Maser MR, White DN, Lash H. Failure of carbon dioxide laser excision of keloids. *Lasers Surg Med*. 1989;9:382-388.
 53. Norris JE. The effect of carbon dioxide laser surgery on the recurrence of keloids. *Plast Reconstr Surg*. 1991;87:44-49; discussion 50-53.
 54. Nowak KC, McCormack M, Koch RJ. The effect of superpulsed carbon dioxide laser energy on keloid and normal dermal fibroblast secretion of growth factors: A serum-free study. *Plast Reconstr Surg*. 2000;105:2039-2048.
 55. Pearson G, Robinson F, Beers Gibson T, et al. Mitogen-activated protein (MAP) kinase pathways: Regulation and physiological functions. *Endocr Rev*. 2001;22:153-183.
 56. Chang NS. TGF-beta-induced matrix proteins inhibit p42/44 MAPK and JNK activation and suppress TNF-mediated I κ B degradation and NF-kappaB nuclear translocation in L929 fibroblasts. *Biochem Biophys Res Commun*. 2000;267:194-200.
 57. Dover JS, Hruza G. Lasers in skin resurfacing. *Australas J Dermatol*. 2000;41:72-85.
 58. Cantatore JL, Kriegel DA. Laser surgery: An approach to the pediatric patient. *J Am Acad Dermatol*. 2004;50:165-184; quiz 185-188.
 59. Dover JS, Arndt KA, Dinehart SM, Fitzpatrick RE, Gonzalez E. Guidelines of care for laser surgery. American Academy of Dermatology. Guidelines/Outcomes Committee. *J Am Acad Dermatol*. 1999;41:484-495.

60. Alster TS, Lupton JR. Prevention and treatment of side effects and complications of cutaneous laser resurfacing. *Plast Reconstr Surg*. 2002;109:308-316; discussion 317-318.
61. Chau D, Mancoll JS, Lee S, et al. Tamoxifen down-regulates TGF-beta production in keloid fibroblasts. *Ann Plast Surg*. 1998;40:490-493.
62. Battle EF Jr, Hobbs LM. Laser therapy on darker ethnic skin. *Dermatol Clin*. 2003;21:713-723.
63. McDaniel DH. Laser therapy of stretch marks. *Dermatol Clin*. 2002;20:67-76, viii.
64. Hahler B. An overview of dermatological conditions commonly associated with the obese patient. *Ostomy Wound Manage*. 2006;52:34-36, 38, 40 passim.
65. Pieraggi MT, Julian M, Delmas M, Bouissou H. Striae: morphological aspects of connective tissue. *Virchows Arch*. 1982;396:279-289.
66. Maari C, Powell J. Atrophies of connective tissue. In: Bolognia JL, Jorizzo JL, Rapini RP Sr, et al., eds. *Dermatology*. London, UK: Mosby; 2003:1539-1548.
67. McDaniel DH, Ash K, Zukowski M. Treatment of stretch marks with the 585-nm flashlamp-pumped pulsed dye laser. *Dermatol Surg*. 1996;22:332-337.
68. Ash K, Lord J, Zukowski M, McDaniel DH. Comparison of topical therapy for striae alba (20% glycolic acid/0.05% tretinoin versus 20% glycolic acid/10% L-ascorbic acid). *Dermatol Surg*. 1998;24:849-856.
69. Elson ML. Treatment of striae distensae with topical tretinoin. *J Dermatol Surg Oncol*. 1990;16:267-270.
70. Pribanich S, Simpson FG, Held B, Yarbrough CL, White SN. Low-dose tretinoin does not improve striae distensae: a double-blind, placebo-controlled study. *Cutis*. 1994;54:121-124.
71. Tajima S, Pinnell SR. Ascorbic acid preferentially enhances type I and III collagen gene transcription in human skin fibroblasts. *J Dermatol Sci*. 1996;11:250-253.
72. Nouri K, Romagosa R, Chartier T, Bowes L, Spencer JM. Comparison of the 585 nm pulse dye laser and the short pulsed CO₂ laser in the treatment of striae distensae in skin types IV and VI. *Dermatol Surg*. 1999;25:368-370.
73. Jimenez GP, Flores F, Berman B, Gunja-Smith Z. Treatment of striae rubra and striae alba with the 585-nm pulsed-dye laser. *Dermatol Surg*. 2003;29:362-365.
74. Al-Attar A, Mess S, Thomassen JM, Kauffman CL, Davison SP. Keloid pathogenesis and treatment. *Plast Reconstr Surg*. 2006;117:286-300.

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