CHAPTER 22

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Chemical Peel

A Facial Plastic Surgeon's Perspective

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In antiquity, our ancestors understood the ravages of the sun and its effect on the aging process. The Egyptian Pharaohs and their court could be easily distinguished by their lack of sun exposure and delicate skin. Sun damage was a cultural stigma that belonged to the working class, which today, ironically, is just the opposite. They used oils and alabaster and bathed in sour milk (lactic acid) to maintain their skin and social status. The Holy Scriptures also bore witness to the daily use of emollients and perfumes to soften and cleanse the skin among the wives of the Jewish and Babylonian kings. With time's progression, other cultures developed their own sine qua non remedies for the enhancement of their aged skin envelopes. For instance, Middle Eastern men and women used urine mixed with an abrasive compound and fire to exfoliate the outer layers of their skin. During the 1700s Madame Pompadour of France bathed in red wine (tartaric acid) to create a more supple look and feel to her countenance.

It has only been in the last 120 years or so that a more scientific approach was used to identify and measure the results of applying things to the skin's surface for its improvement. In 1882, German dermatologist P.G. Unna worked with and described different types of peeling agents such as trichloroacetic acid (TCA), phenol, and resorcinol.[1] Phenol was later used for war wounds during World War I, and that experience translated into techniques brought to the United States and practiced by "lay peelers" near Los Angeles and Southern Florida during the 30s and 40s. Others went on to work with these dermatologic methods over the next decade, culminating in the work of Brown and coworkers on the effectiveness of various phenol preparations.[2] In addition, Litton published his work on a nonsaponified phenol formula during the late 50s and early 60s.[3] Various results and complications followed that placed the practice of skin peels in a state of disrepute until Baker and Gordon presented their results with before and after photographs at a national conference in 1972.[4] This brought a resurgence of interest in facial peeling for the improvement of wrinkling, and their particular saponated peel formula has in some form or fashion become the mainstay of treatment for deep rhytids ever since.

TCA, resorcinol, and other chemical agents have had similar histories within the annals of skin resurfacing. They have been used with increasing frequency for the improvement of these and other skin problems, especially with the advent of national seminars that tout their results and teach their techniques.[5] In addition, the use of such products as Retin-A and the alpha hydroxy acids (glycolic and lactic) have received national attention over the past decade and a half. This widespread exposure has positioned chemical peels as a part of our armamentarium into the twenty-first century for the improvement of the skin's appearance.

EFFECTS OF AGING

Prerequisites to performing skin peels are a thorough knowledge of the anatomy of the epidermis and dermis, the depth of injury required to achieve the desired results (Fig. 22–1), and the series of events that follow the injury produced by the application of the "peel solution." In normal skin, the dermis can be 20 to 30 times thicker than the epidermis, and it is in this foundational layer that architectural changes are sought to obtain the desired enhancement for the overlying wrinkled epidermis. Simple changes in the epidermis alone do very little for wrinkled skin because dermal factors are largely responsible for this condition. As we age and our skin becomes photodamaged, the papillary dermis becomes thinner because of dermal collagen and elastin degeneration. The overlying epidermis will eventually come to rest either on the lower papillary or upper reticular dermis. This loss of upper dermal thickness requires the more *inelastic* epidermal layers to shrink, and its inability to do so helps produce wrinkling. (Simply stated, this physiologic process constitutes the main reason for the deeper chemical peels, i.e., to insult the remaining dermal components in a controlled fashion expecting a response of dermal regrowth with enhanced vertical thickness and a realignment of the collagen bundles parallel to the skin's surface, all of which help tighten the overlying skin.) This process of upper dermal degeneration is noted histologically by a decrease in dermal collagen production, an increase in collagen breakdown by collagenases, an increase in solar elastosis, a state of disarray within the melanocytic system producing variations in skin color, and a diminution of papillary dermal blood flow. Decrease in the number and length of the rete pegs at the dermal epidermal junction also create a more loose and mobile skin envelope. Coupled with the fact that as we age our facial skeleton gets smaller and the layer of subcutaneous fat may atrophy, it is a wonder that we are not all "prune faced."

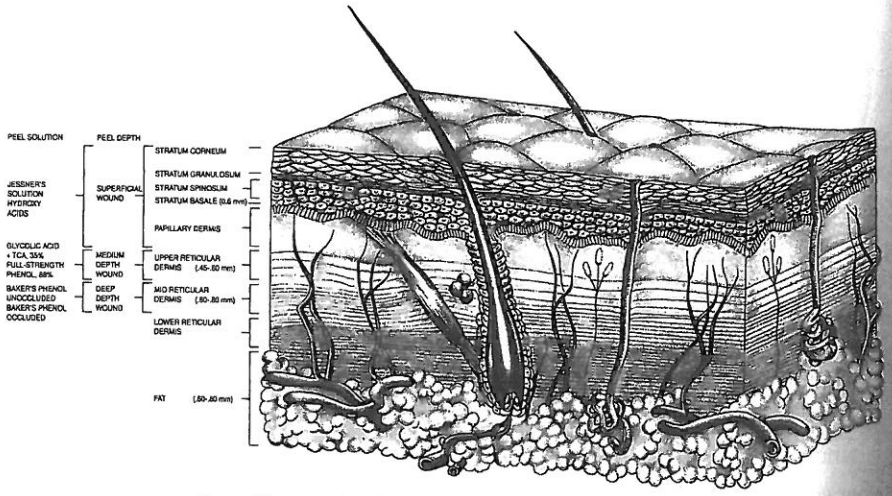

Figure 22–1. Note the depth of wounds and corresponding peel agents.

CHOICE OF SKIN PEEL

This histologic information helps the surgeon or peeler in choosing the appropriate wounding agent to carry them to the depth necessary to achieve the desired response. For instance, if superficial exfoliation is indicated, the alpha hydroxy acid peels with glycolic acid will suffice because only the epidermal layers need be involved. This particular acid tends to restore keratinocyte adhesion to normal by creating their detachment at the junction of the stratum granulosum and enhancing the process of desquamation. If depigmentation or a more homogenous pigmentary color of the face is desired, peels such as a 35% TCA with or without epidermal vesiculation can produce wounds down to and below the basement membrane of the epidermis, where alteration of the melanocytic system can occur. Of note, pigmentation can be epidermal, dermal, or both. In any of these the wound should not extend to the deepest levels of pigmentation so as to avoid unnecessary morbidity. For this particular problem, more frequent and more superficial skin peels are indicated to achieve improvement and avoid a significant loss of pigmentation. If wrinkle improvement of thin skin is desired where a flatter dermal-epidermal interface exists, such as the eyelids, a medium-depth peel is warranted and can be produced either by a 35% TCA in conjunction with glycolic acid or a straight 88% phenol (carboxylic acid) solution because the depth of injury required for this response is the mid papillary to upper reticular dermis (Fig. 22–2). (The latter is my preference and will be discussed later in this chapter. To place a deep peel solution on the lower lids within the confines of the orbit will lengthen the healing time and in some instances produce ectropion as a result of vertical contracture of the lower lid.) If, however, deeper rhytids exist as on the forehead or other areas of the face with thicker skin, a deep surgical peel such as a Baker-Gordon peel with or without occlusion is recommended because the level of response required is the midreticular dermis. Dermal wounds into and below the lower reticular dermis respond with scar formation, and this complication can be prevented by limiting the depth of injury above this level. Given today's range of peeling agents, it has become fashionable to diagnose and treat each cosmetic unit of the face with whatever modality is warranted. Such an approach will limit morbidity and unwanted complications but may produce differing lines of demarcation if the peel solution is not carefully applied and blended.

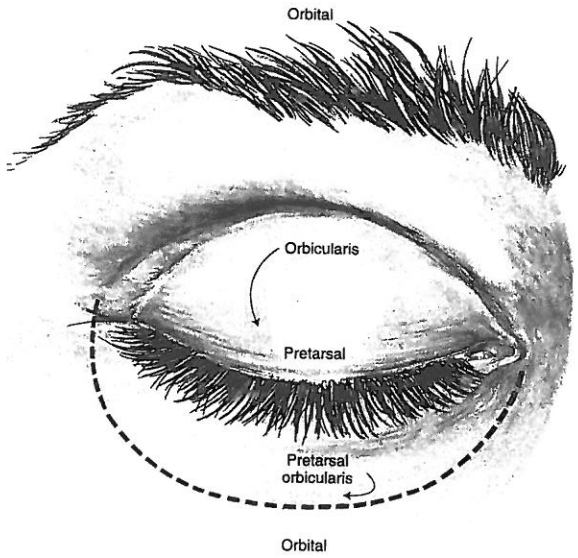

Figure 22–2. Anatomy of the eyelid with thinner skin in the pretarsal regions and thickening with outward migration.

THE HEALING PROCESS

Regardless of the depth of injury produced down to the midreticular dermis, the response of the individual to this controlled wounding is fairly predictable. The surgeon or peeler must understand this process completely so as to provide postoperative reassurance when needed and early intervention before a complication occurs if that is also needed. Patients need to understand that certain types of peels are very inconvenient and stressful procedures to undergo, but that they produce a type of result when nothing else can (Fig. 22–3). It is incumbent on the originator of the wound to walk the patient through this process as skillfully as possible. Much misinformation about chemical peels exists among the lay community, and one of the biggest challenges for the surgeon or peeler is in correcting these myths on the basis of a large body of existing knowledge and their own clinical experience. Wrinkling is within the substance of the skin, and the treatment modality needs to address that particular area. Surgery, per se, is not for wrinkling and so the treatment of rhytids lies within the domain of the chemical peel, laser resurfacing, or dermabrasion.

Chemical peels are partial-thickness injuries that heal rapidly by secondary intention provided that certain parameters are met. Because the first phase is coagulant and inflammatory, nothing else should be done to increase or for that matter to decrease this response after the initial insult. Although this phase is mandatory for proper healing, additional trauma such as chemical, mechanical, radiant, or infection should be avoided because an exacerbation at this time could be detrimental to the final result. In the past many who performed the deep Baker-Gordon peels attempted to prolong this initial phase by the occlusion of the wound with tape. It was thought that the tape trapped moisture in the wound bed and increased the absorption and penetration of the phenol through maceration. With this particular formula of phenol, its high 88% concentration had a keratocoagulant effect on the skin, but when diluted with any type of moisture such as tears or tissue fluid, it became keratolytic and went deeper. Recent data suggest that taping may not have this particular effect after all.

The body's response to these partial-thickness wounds is immediate, with the formation of the platelet fibrin clot and activation of the kinin and complement inflammatory pathways that produce chemotactic mediators at the wound site such as leukotrienes, kallikreins, growth factors, and fibrin lysis products. These factors increase local blood vessel permeability and attract neutrophils, monocytes, and lymphocytes.[6] This chemoattraction is necessary for stability, protection, and the initiation of wound healing.

Neutrophils are the first line of defense to a breach in the integumentary system and are present for at least 3 to 7 days. Careful attention with good wound management

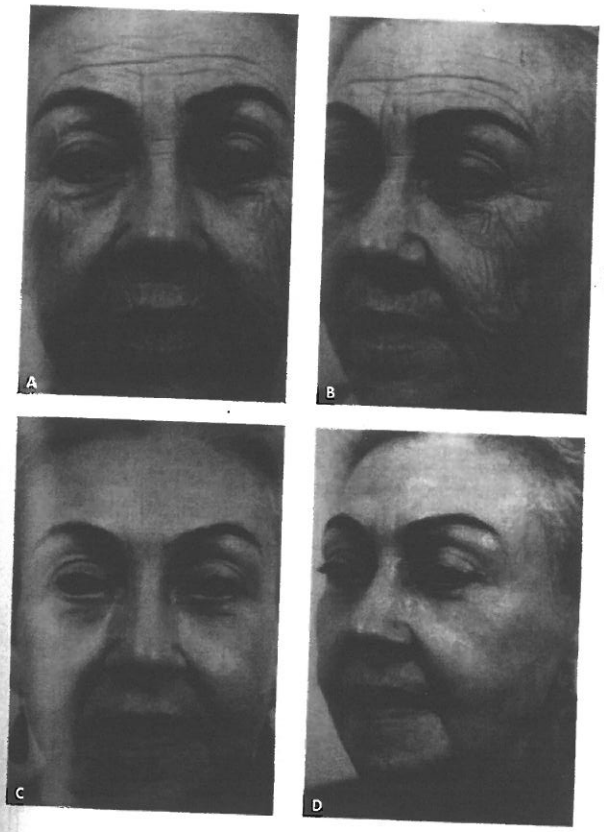

Figure 22–3. Preoperative frontal **(A)** and side **(B)** views. Note the "sleep lines" on each cheek. Five months postoperative frontal **(C)** and side **(D)** views from a full-face Baker-Gordon peel. Note slight thickening of each lower eyelid. This patient had a mild ectropion of each lower lid for approximately 3 months. It resolved with intralesional steroid injections and massages.

is necessary during this critical phase of wound healing to prevent infection from monilial, herpetic, or opportunistic bacterial organisms, thus avoiding deeper wounds, delayed healing, and possible scar formation. Monocytes arrive 3 to 10 days after the initial wound and differentiate into macrophages. These are the most important cells in the resolution of inflammation and are largely responsible for initiating the proliferative phase of dermal wound healing by directing the development of granulation tissue. This type of tissue is a collection of cellular components such as fibroblasts, cellular by-products such as fibronectin and collagen, and ground substances, which include glycoaminoglycans (GAGs), sulfonated and nonsulfonated. The lymphocyte is found in the wound bed after the first week and continues the processes begun by the macrophages. Early intervention during this phase by the patient or physician can interfere with the natural healing process by slowing down the buildup of granulation tissue. Corticosteroids, which are a great treatment alternative later on, would adversely affect the accumulation of these "building blocks" and should not be used unless specifically indicated.

The next phase is re-epithelization of the defect, and this occurs initially by migration of the epithelium from the involved adnexal structures (Fig. 22–4 and 22–5) within the first 24 to 72 hours of a peel initiated by fibrin and platelet products. The density of the pilosebaceous units per square centimeter is important to the rapidity of re-epithelization, and this lack of density explains the differences in healing between the face and the neck because the neck has less units per area. Although this process is measured in hours, the next step that requires proliferation of epithelial cells at the periphery of the defect and continued growth from the adnexal remnants is measured in days. Keratinocytes migrate on a scaffold of dermal matrix consisting of fibronectin, which is cross linked to collagen, fibrin, and elastin. Fibronectin allows adhesion to these dermal structures and surrounding cells. After migration and coverage are obtained, cell proliferation at the wound edge shifts centrally, providing the needed depth to the epidermis (Fig. 22–6). This process continues for 60 to 90 days after a deep chemical peel. Early re-epithelization of the wound bed, although monocellular in initial thickness, is paramount to the prevention of complications. The longer a wound is left uncovered with epithelium, the greater the chances for healing abnormalities. Maibach and Rovee provided good insight into this early process of healing during their studies in the late 60s and early 70s.[7] They found that a moist wound with a high water content healed much faster than a dry wound. (This is not to be confused with the reasons for taping a deep surgical wound obtained by a peel agent such as Baker-Gordon. This technique is used solely for the purpose of driving the wounding agent

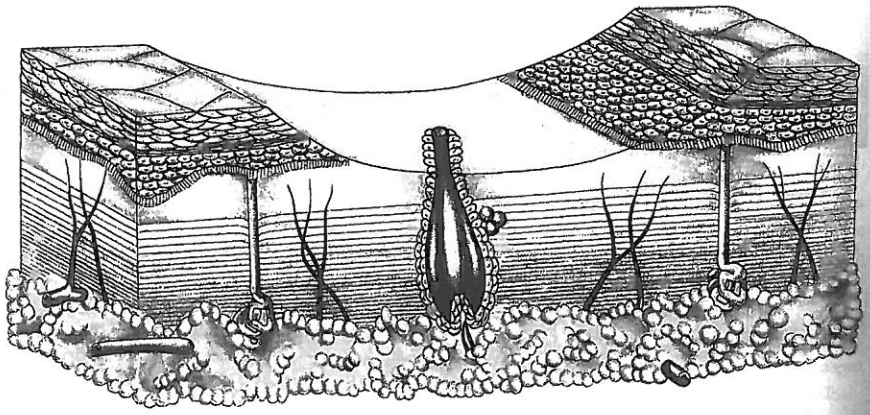

Figure 22–4. Suface of the epidermis-dermis immediately after a chemical peel.

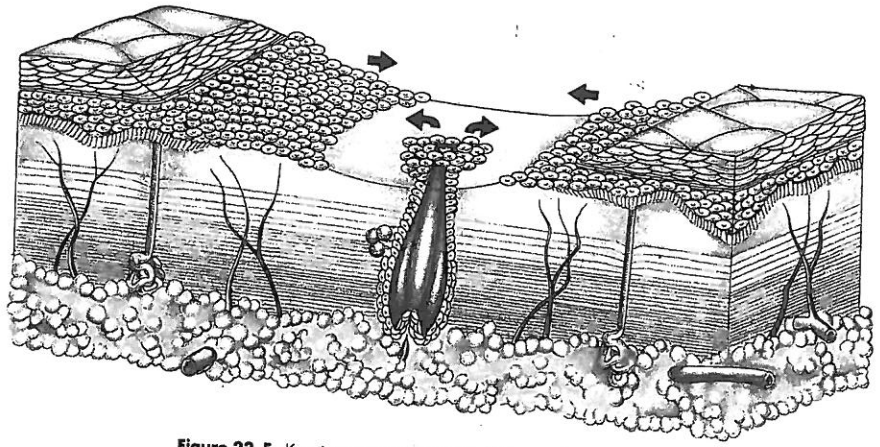

Figure 22–5. Keratinocyte migration within 72 hours of the chemical peel.

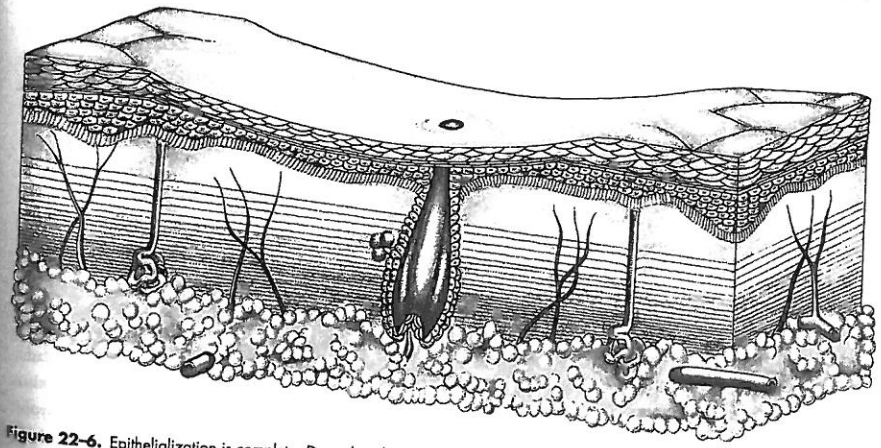

Figure 22–6. Epithelialization is complete. Dermal and epidermal height regeneration will be ongoing for 2 to 3 months.

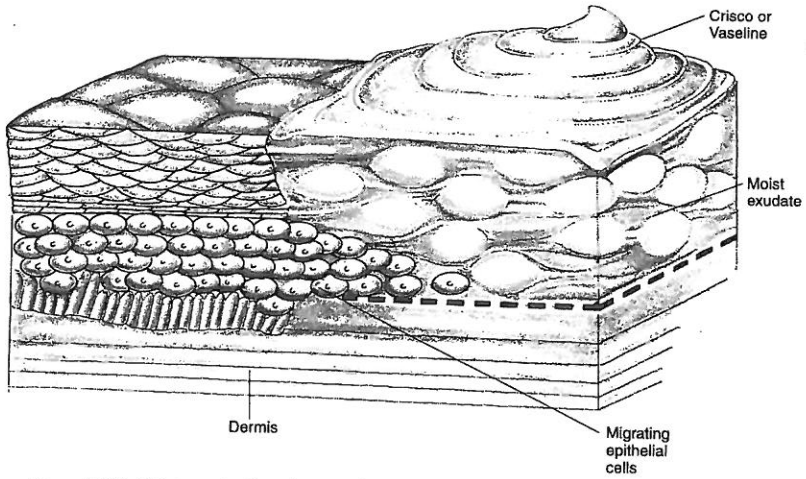

Figure 22–7. Moist wound with overlying emollient and a more superficial layer of bridging keratinocytes.

deeper through maceration. For this reason, I do not place an emollient on any deep surgical peels for 4 to 6 hours to prevent dilution of residual phenol and possibly a deeper wound.)

Because the leading edge of epithelium must seek a sustaining layer of hydration to bridge the defect, it need not travel as deeply when an emollient or a bio-occlusive dressing is kept on the wound bed in such a way as to trap moisture. This allows the advancing epithelium to bridge the defect more efficiently and more superficially (Fig. 22–7). A desiccated wound makes it necessary for the advancing epithelial cells to descend further down into the dermis and subsequently delays healing and runs the possibility of compromising the final results (Fig. 22–8). Placing a desiccant such as thymol powder on a deep peel is physiologically incorrect and should be avoided. Also, a dry wound is a rich breeding ground for facultative microaerobes that will interfere with the advancing edge of keratinocytes in their attempt to cross this denuded expanse of epidermis and dermis. Good wound management dictates frequent washings and application of a suitable emollient or the use of a bio-occlusive dressing in an effort to trap dermal moisture. This in turn enhances the substance within the dermis previously alluded to as GAGs. This material absorbs water up to 1000 times its own volume and is critical to the health of the wound. Without this hygroscopic material, the wound would desiccate and the healing process would be delayed and possibly compromised.

Because the acute phases of inflammation are ongoing for the first 2 weeks, the dermal matrix cannot begin to replicate until this inflammatory smolder has decreased. The first signs of replication are noted by the increased presence of granulation material that continues to proliferate until the wound has epithelialized. One of the tissue's components as previously mentioned is fibroblasts, and their activity produces collagen, elastin, and other needed materials. A differentiated fibroblast, the myofibroblast, produces wound bed contracture. Studies have

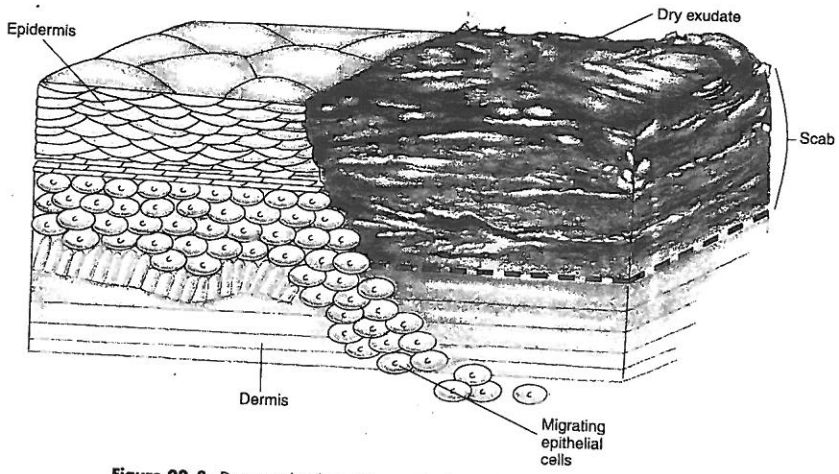

Figure 22–8. Dry wound with scabbing and a deeper layer of bridging keratinocytes.

suggested that these steps can be improved with the preoperative use of tretinoin[8,9] and the postoperative use of a zinc-containing compound such as bacitracin.[10] Topical retinoids increase fibroblast and epidermal migration, possibly by decreasing the desmosomal and tonofilament attachments. They also increase mitosis in regenerating epidermal cells. Systemic retinoids on the other hand increase collagen synthesis, decrease collagenase production, and increase fibronectin production. This alteration in collagen degradation may be the factor in the accumulation of excessive collagen and hypertrophic scarring.[11] Head and neck studies have also suggested that vitamins with additional zinc may have added benefit in this early postoperative healing phase as well. During this stage of granulation tissue buildup, close attention must be paid to excessive fibroblastic activity, especially in an area where regrowth of epidermis has been slow. These areas can produce prolonged erythema and potential scarring if not closely monitored and appropriately dealt with. Once the wound bed is covered with epidermis, the formation of granulation tissue essentially stops, and the regrowth of dermal components begins. This regrowth of vertical dermal height and its remodeling is ongoing for 2 to 3 months and needs to be monitored and encouraged because this buildup of dermal thickness, its alteration, and reorientation of existing structures are the main histologic reason for the chemical peel when the improvement of wrinkles is desired. Good nutrition, lack of stress, and the absence of sun exposure combined with good common sense can make this phase most fruitful and create the desired outcome with an enhanced dermal foundation for the overlying epidermis.

Angiogenesis is extremely important during this phase and is noted within the first 72 hours of wounding with endothelial buds that travel along the dermal matrix, providing oxygen to the area of healing. Smoking at this time is very harmful to this neoproliferative process because it not only impedes vascular bud generation but also robs the hemoglobin of its transported cargo, oxygen. A sensitization of the raw area to smoke also will

create symptoms of increased burning and stinging if allowed to come in contact with the areas peeled before complete epithelization has occurred.

A later concern for patient and physician is that of prolonged erythema or hyperpigmentation. Although erythema may represent a sentinel finding of trouble, it need not always be such an indicator. Regardless, however, it can be a concern to the patient, and they need to be reassured that this is a normal healing process and should subside when the healing is completed. An exception would be that of telangiectasias. This vascular abnormality is more noticeable with the thinner layers of regenerating dermis and epidermis, and the patient needs to be encouraged that they should obtain a prepeel appearance. However, their number maybe increased at the conclusion of a medium to deep peel when healing is completed. This is an issue that must be addressed with the patient preoperatively; however, the advent of newer lasers have made this sequela less problematic. Hyperpigmentation, on the other hand, is not a favored response to the peel process and can persist if not prevented with good patient selection and adequate preoperative preparation with complete sun avoidance. If developed, the problem is managed with retinoic acid and hydroquinone with or without steroids. The process of lightening with these agents is slow, but the enzymatic disruption of tyrosine to melanin is certain if the patient is consistent in their use.[11]

Collagen remodeling is the last healing phase to start and finish. This process usually manifests itself 60 to 90 days after surgery, depending on the depth of the wound; the deeper the wound the longer it is before remodeling begins and the longer it takes to complete the process. Corticosteroids to help decrease swelling during these phases delays all aspects of wound healing and should be used sparingly unless an overproduction of fibroblastic material or of pigmentation occurs. Also, it stands to reason that immunosuppressive agents are to be avoided during and after a peel for at least 3 to 6 months.

PHENOL

PATIENT SELECTION

Patient selection is extremely important to the successful outcome of a facial peel. The normal prepeel evaluation should include a thorough head and neck examination, especially noting the amount of photodamage, sebaceous activity, and the presence or lack of pilosebaceous units. The Fitzpatrick classification of sun-reactive skin types and Glogau classification of photoaging are two of the best tools used today for categorizing patients for two purposes: first, to help in choosing the best peel formula and technique, and second, to help in predicting the postoperative response. The Fitzpatrick classification gives a subjective but reliable measure of the skin's responsiveness to sun exposure; when considering the patient's eye color and the choice of wounding agent, the postoperative course is fairly predictable (Table 22–1). Types I through III skin types are ideal candidates for all the favored chemical peel solutions. Type IV skin begins that gray area when the surgeon or peeler must anticipate and attempt to avoid pigmentary changes such as postinflammatory hyperpigmentation, noticeable lines of demarcation, and hypopigmentation. These concerns are paramount in skin types V and VI, and these patients should undergo test spots before a medium or deep facial peel, although the test spot's response is not a guarantee of the face's response.

The Glogau classification describes the structure of the rhytids with and without expression and the

Table 22–1 The Fitzpatrick Classification

Skin Type/Color	Reaction of First Summer Exposure
I—White	Always burn, never tan
II—White	Usually burn, tan with difficulty
III—White	Sometimes mild burn, tan average
IV—Moderate brown	Rarely burn, tan with ease
V—Dark brown	Very rarely burn, tan very easily
VI—Black	No burn, tan very easily

patient's attempt to overcome their appearance (i.e., through the use of makeup) (Table 22–2). Although each group is individual, overlap exists, and the surgeon or peeler should use common sense in assigning a patient to a particular class. As previously mentioned, it may also be necessary to assign each of the cosmetic units a particular classification to tailor each to the appropriate wounding agent.

Another important aspect of performing chemical peels is assessment of lifestyle and emotional stability. The superficial to medium depth peels provide little-to-moderate disruption to either, whereas the deeper peels are indicated only when the patient and physician can come to a mutual understanding concerning the amount of morbidity and encroachment on the patient's day-to-day routine. Additional information obtained before medium and deep peels should include a prior history of isotretinoin (Accutane), previous surgery, radiation or peels, a history of smoking, future plans for sun exposure, the use of hormones, a history of herpes simplex, and the patient's cardiac and renal status.

Whether isotretinoin is a culprit remains to be seen, however, because it is in the literature as a potential contraindication, planning a medium-to-deep peel within a 6-to 18-month time frame after its use needs to be discussed with the patient, including its possible ramifications, the need for test spots, or a delay of the procedure.[12] Previous peels and facial plastic surgery are usually good indicators of history and compliance. Neither are contraindications if sufficient time has elapsed. A history of radiation should alert the surgeon or peeler that normal skin anatomy may not exist. The number of pilosebaceous units should confirm that suspicion if less than normal and lead to a more superficial attempt to improve the skin's surface if the decision to proceed with a chemical peel is acceptable to both parties. Smoking is detrimental to the immediate and the long-term healing process in the form of oxygen depletion and the formation of free radicals within the healing skin that will damage existing and regenerating dermal components such as collagen. Sun exposure can also produce these free radical scavengers and stimulate the less covered melanocytes to produce melanin and cause subsequent postinflammatory hyperpigmentation. A good sunscreen both before and as soon after a peel is always indicated. Hormones may also exacerbate the stimulation of the exposed melanocytic system and should be discontinued at least 2 weeks before and 2 to 6 weeks after a peel. This request to forego hormone therapy for 4 to 8 weeks or longer seems to be one of the biggest hardships for the

Table 22–2 Glogau's Classification

Group I (Mild)	Group II (Moderate)	Group III (Advanced)	Group IV (Severe)
No keratoses	Early actinic keratoses—slight yellow skin discoloration	Actinic keratoses—obvious yellow skin discoloration with telangiectasis	Actinic keratoses and skin cancers have occurred
Little wrinkling	Early wrinkling—parallel smile lines	Wrinkling present at rest	Wrinkling—much cutis laxa of actinic, gravitational, and dynamic origin
No scarring	Mild scarring	Moderate acne scarring	Severe acne scarring
Little or no makeup	Little makeup	Wears makeup always	Wears makeup that does not cover, but cakes on

patient and their family to endure. If depression or anxiety is a postoperative complaint, the decision to restart the hormones versus their potential harm should be weighed and acted on accordingly. Any history of herpetic infections of the face should necessitate the addition of an antiviral agent to be given with their postoperative medications. If a blister is present or the suspicion of one is evident the day of the procedure, it is prudent to cancel and reschedule. Routine laboratory studies should include a complete blood count, an electrolyte panel, liver enzymes, and a creatinine level to assess kidney function. An electrocardiogram and medical clearance is always indicated if prior history of renal and/or cardiac problems exists.

Preoperative Preparation

Preparation of the skin before a peel is necessary to prevent or to reduce the chances of postoperative complications in all skin types, especially in Fitzpatrick IV through VI types or Glogau groups III and IV. Once scheduled, the patient needs to be taking retinoic acid for 2 to 6 weeks before surgery to enhance wound healing and decrease the possibility of postinflammatory hyperpigmentation. If the skin type suggests, twice daily additions of a hydroquinone 2 to 4% are indicated. Higher concentrations initially can produce skin irritation. If either or both are not tolerated, then decrease their frequency of use. These preoperative difficulties may lead to choosing a lighter peel if the skin's inability to tolerate these products is significant. It is also a good idea to have the patient on a proper suncreen before the peel to determine compliance and to protect the skin before wounding. These proactive precautions are helpful to ensure good wounding at the time of the procedure, lack of postoperative sequelae, and adaptation of the patient's schedule and skin to ensure its good health and maintenance.

Technique

With the introduction of the transconjunctival blepharoplasty, the need for lower eyelid skin tightening at the time of surgery has led me to modify my existing technique for this cosmetic unit. In the past, when a "pinch technique" was not indicated and skin tightening was still required, the peel of choice was the Baker-Gordon peel with minimal skin preparation because peeling in this area with the thin dermal-epidermal interface would produce a deeper wound than necessary, resulting in delayed healing and on occasion a mild ectropion. Also, the length of time for healing was in weeks or months if the depth of peel matched that of the rest of the face where thicker skin predominated (Fig. 22–3C, D). Because the standard formula for the Baker-Gordon peel solution included distilled water, septisol soap, croton oil, and 88% phenol, simply removing all adulterants and using only the straight phenol prevented many of these previous concerns. Instead of weeks or months in healing, the time was cut to days. The concentration of the phenol was such that when applied to the thinner skin of the lower eyelid, frosting was immediate, and the process of keratocoagulation limited its penetration. When I first performed this procedure, the initial concern was the speed and the amount of frosting for fear of too much skin destruction. However, experience proved that the straight 88% phenol was so concentrated that its destructive properties were self limited and created the desired wound without going too deep. The result was that of a medium-depth wound, and healing was usually complete in 7 to 10 days if the wound was kept moist and clean. In retrospect, the results may not be quite as good as a Baker-Gordon peel in this particular area, but the significant decrease in morbidity outweighed the small loss in improvement. Also, if necessary, this type of medium depth peel can be repeated within 4 to 6 months if additional skin tightening is needed and no contraindications exist. It is best to repeel with a medium-depth peel than contend with the sequelae left from peeling with a deep peel in an area that may not tolerate it without increased morbidity and increased healing time. The other good thing about this type of wound is that no appreciable change occurs in skin color or texture in the Fitzpatrick type I to IV. Skin tests are performed on types V to VI.

As far as the technique is concerned, after the transconjunctival blepharoplasty, any blood or residual moisture on the lower eyelids is removed and the dry skin is degreased lightly with acetone, taking precautions not to get the liquid or fumes in the patient's eyes. Because the surgical plane is beneath the orbicularis oculi muscle, peeling the lids concomitantly is not a concern. An 88% phenol solution to be used is poured from the stock bottle into a glass container on the back table and a cotton-tipped applicator is dipped into the phenol and the excess solution is wrung out by rimming the applicator on the lip of the glass. The first stroke is at the orbital rim, where the junction of lid and cheek skin exists and the skin is thicker. This first pass dispenses the most peel solution and subsequent strokes cephalically will contain less solution as the strokes are carried from lateral to medial and within 2 to 3 mm of the free lash margin. At all times, a dry cotton-tipped applicator is kept ready to absorb any tearing that might dilute the peel solution and create a keratolytic effect especially in the lateral fornix of the eyelid. Tearing can be minimized by anesthetizing the conjunctiva with an appropriate topical anesthetic. Before the other lower lid is peeled, the lowest stroke of peel is feathered onto the upper cheek and over the zygomatic region to prevent any lines of demarcation even though this medium-depth peel is not usually known for producing any significant variation in pigment.

Postoperative Course

After both lids are peeled, the patient should stay on the table for at least another 15 minutes to watch for tearing and to monitor them for cardiac dysrhythmias, although the amount of phenolic uptake is negligible for such a small area peeled. When released from the recovery room, the patient is instructed to apply an emollient on the skin surface in approximately 4 hours. The facial rinsings are not required until the next morning when they are instructed to allow a gentle stream of clean water to lightly rinse the area for 3 to 5 minutes followed by drying with unscented facial tissues. The emollient is then applied, and the process is repeated four to six times daily. Patients are seen in follow-up the day after surgery and either at day 6 or 7. If delayed healing is suspected after 10 to 14 days, Duoderm, a hydrocolloid dressing that is occlusive and oxygen impermeable, should be applied to the raw areas for 48 to 72 hours. In most instances, this will complete the re-epithelization.

Complications and Sequelae

Although hypopigmentation is not an issue, postinflammatory hyperpigmentation is a concern and may be prevented with good preoperative management and proper patient selection. If there is any redness or pinkness turns light brown after 2 or 3 weeks, a 2% hydroquinone should be applied twice daily with close follow-up visits. Most of the hyperpigmentation that develops is gone after this type of treatment in 2 to 4 weeks. If not, increase the percentage of hydroquinone and consider the concomitant use of retinoic acid or a nonfluorinated steroid. The patient should be reassured that the final result is forthcoming in approximately 3 months and that the patient needs to minimize sun exposure for that period and avoid squinting, which can induce mechanical disruption and encourage increased fibroblastic activity. Again, if agreed on by both parties, repeeling for additional improvement can be done in 4 to 6 months if no contraindications exist.

Baker-Gordon Deep Peel

The "Cadillac" of chemical resurfacing is the Baker-Gordon peel. The formula is 3 mL of 88% phenol (carboxyclic acid), 3 gtts, of croton oil, 2 mL of distilled water, and 8 gtts of Septisol soap. Although this formula has been modified over the years, most surgeons or peelers find this particular concentration fairly predictable in results and satisfaction.

The technique is operator dependent and requires the aforementioned body of knowledge, some type of previous training with a skilled surgeon or peeler, and the experience that comes with a graduated and conservative

approach to peeling one's own patients. To take the position of simply applying the solution to the skin and expecting the same results every time is foolhardy and unrewarding for both patient and physician in the postoperative stages of healing. Although the actual application is matter of fact, the preparation before and the care after its application is paramount to consistent results and requires the most experience.

Patient Selection

Before a deep peel with a Baker-Gordon solution, a thorough evaluation should include a prior history of isotretinoin; previous surgery, radiation, or peels; a history of smoking; the use of hormones; a history of fever blisters; and the cardiac and renal status with appropriate medical clearance. All candidates should be adequately photographed and thoroughly educated in the follow-up care.

Preoperative Preparation

Two to 6 weeks before peeling, the patient is placed on a formula of Retin A that had been handed down to me by Jack Anderson during a conference in 1986.[11] That formula contains 0.05% Retin A and Shepherd's lotion, which is an emollient that offsets the skin's reactivity to the retinoic acid. This is applied to the skin at night and rinsed off in the morning. If indicated, the patient is also placed on a skin bleacher such as 2 or 4% hydroquinone in the morning to be followed by an application of a sunscreen that protects against both ultraviolet A and ultraviolet B radiation. If patients are not compliant with these preoperative requests, it is a safe assumption that they will be noncompliant with the postoperative instructions.

Technique

On the day of the peel, the patient needs to have been NPO from the night before, have showered well, and on arrival, will need to perform the first of that day's skin preparation. This includes the patient washing his or her own face with a degreasing solution such as Septisol soap.[12] This removes the oils and dirt along with any type of makeup that may still remain on the facial skin. An oral preoperative medication is then given an hour before their transfer into the operating room, where an IV is started and cardiac monitoring is begun. With appropriate IV push sedation and regional nerve blocks, the surgeon or peeler begins to prepare the patient's face to his or her satisfaction. This includes aggressive corneocyte epiabrasion with a 2 × 2 gauze soaked in acetone and wrung out. Some use alcohol first to be followed by acetone, but acetone alone is sufficient. A fan to blow away the fumes from the acetone is necessary to prevent inhalation and eye irritation.

The peel solution does have a shelf life; however, the low cost of the ingredients should be reason enough to have the formula constituted fresh each morning before a peel of any sufficient size on the patient's face. When the skin has been adequately prepared by degreasing with the acetone (i.e., when the surgeon or peeler can feel and hear a light scraping with the 2 × 2 gauze without inducing purpura), the peel solution can be applied. Most advocate the use of a cotton-tipped applicator. However, broad strokes with these can place a lot of solution at the beginning of the stroke and little at its end. For that reason, small "blocks" of facial skin are peeled within a cosmetic unit at a time (Anderson J, personal communication, 1986), waiting 15 to 20 minutes per cosmetic unit to allow uptake of the phenol and its sequestration by the bladder to prevent cardiac dysrhythmias. Where deep rhytids exist as around the mouth, I break the cotton stick applicator at an angle, dip the wooden end into the solution, and apply it directly into the depth of the rhytid with light pressure. Visible signs of retained solution may be present within the depth of the rhytid, but if no pooling exists, one should not be alarmed. With constant monitoring, the areas of the face are sequentially peeled. If full-face peeling is performed, the entire process should take about 2 hours. By then, approximately 1000 to 1500 mL of IV fluids should have been infused and processed by the body if not medically contraindicated. The patient is allowed to stay on the table for an additional length of time while monitoring is ongoing. After an additional 15 to 30 minutes, if the patient can tolerate transfer, he or she is taken to recovery. In keeping with the need not to dilute the remaining phenol on the face, no emollient is placed on the face for at least 4 to 6 hours. The patient is allowed to fully recover and sent home. Follow-up consists of a phone call that night and an office visit the next morning for further instructions and reassurance.

Postoperative Course

The daily care consists of showers with body temperature water for 5 to 10 minutes every 4 to 6 hours. Drying is accomplished either by room air or unscented facial tissue. After these two steps, petroleum or nonbuttered flavored shortening is applied to the peeled areas. Eucerin will not stick to a raw wound early on and, if used, needs to be reserved until after epithelization. The head should be elevated at 30 to 40 degrees during sleep to retard swelling, and eating consists of soft foods with care not to get any on the face.

Medication consists of a cephalosporin four times daily with a Medrol dose pack and a nonsteroidal anti-inflammatory (NSAID) such as generic ibuprofen, 800 mg, every 8 to 12 hours as needed for discomfort. A narcotic can also be dispensed for any discomfort not controlled by the NSAIDs. Follow-up visits include the day after, postpeel day 4 and 7, and then weekly for at least a month for reassurance and troubleshooting. The visits become biweekly or as indicated for an additional 2 months and then monthly, depending on the patient and their needs. The final result may be as long as 6 to 9 months in coming, and the patient is observed for at least that length of time to ensure that all is well (Fig. 22–9).

Complications and Sequelae

The most common mistake or postoperative problem is uneven uptake of the peel solution and subsequent skip areas of varying degrees of texture and color. The use of preoperative retinoic acid and hydroquinone coupled with good preoperative degreasing will limit this problem but will not totally eliminate it. If the areas of skip are noticeable after 9 to 12 months, a light feathering across the borders of these areas will help camouflage the situation. Although this problem is potentially distressful for patient and physician, nothing is more troublesome than scar formation that is obvious to the naked eye. This happens when the depth of injury proceeds below the lower layer of reticular dermis either from the initial insult or by subsequent trauma from the patient such as scratching or some type of infectious process from the lack of good wound care. As mentioned previously, frequent visits and topical or intralesional steroids can minimize or eliminate this condition if dealt with in an appropriate time frame.

The other sequela that is more common that all others and can only be predicted but not totally eliminated is that of skin lightening. Most of the time in the Fitzpatrick type I through IV, the degree of lightening is one-half shade. This is more pronounced in darker skinned individuals and proper patient selection will keep this problem to an acceptable level.

CONCLUSION

Time and space have prevented a complete exegesis of the known peel techniques that are in existence today. If further information is required, Brody[14] has an excellent compendium concerning most of the peels that are presently performed. Also, specific articles exist for other types of peels not covered in this chapter such as Monheit's articles on the combined use of TCA and Jessner's solution.[16,17]

Chemical peels can produce some of the best results and some of the worst long-term complications for patient and surgeon or peeler. However, with proper attention to detail, patient selection, good skin preparation, and common sense from proper training and clinical experience, the risks and complications are few and far between and should not be a significant factor for either party with good education and appropriate follow-up.

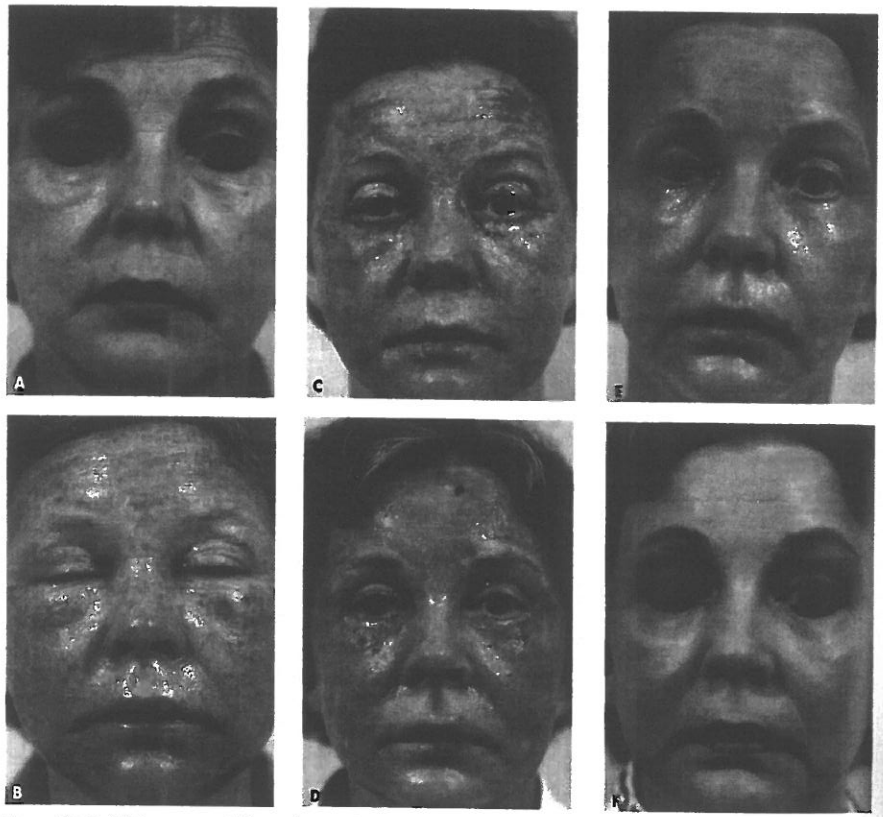

Figure 22–9. **(A)** Preoperative full-face Baker-Gordon chemical peel. This patient had had previous surgery but continued to have facial rhytids. **(B)** Postpeel day 1. Swelling normally peaks in 48 to 72 hours. **(C)** Postpeel day 5. The patient has allowed crusting to form on each of the upper and lower lids. This is secondary to the use of full-strength Baker-Gordon solution. These and other incidences led the author to alter the depth of peel in these areas to that of a medium depth. **(D)** Postpeel day 14. Note the lower eyelids are delayed in their healing. **(E)** Postpeel day 21. Duoderm application to the lower lids resulted in faster reepithelization. **(F)** Postpeel day 28. Note the persistant erythema that will subside over the next 3 to 6 months.

PEARLS

- If depigmentation or a more homogenous pigmentary color of the face is desired, peels such as a 35% TCA with or without epidermal vesiculation can produce wounds down to and below the basement membrane, where alteration of the melanocytic system can occur.
- Wrinkling is within the substance of the skin, especially the dermis, and the treatment modality must address that particular area. Surgery, per se, is not for wrinkling and so the treatment of rhytids lies within the domain of the chemical peel, laser resurfacing, or dermabrasion.
- My patients wear $\frac{1}{2}$-inch brown paper tape on their scar revisions for several months postoperatively. This provides good wound apposition and traps moisture, providing a better milieu for the healing process.

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