Chapter 32: Chemical Peel

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History

Chemical exfoliation of the skin dates as far back as the Egyptians, who used various animal oils and alabaster to improve skin texture. Formulas were developed using sulfur, mustards, and limestones (Brody and Alt, 1991). Through the ages in Europe, Gypsies passed several formulas for chemical exfoliation from generation to generation.

True exfoliation of the skin was first introduced in the early 1900s. In 1903 George MacKee used phenol to treat acne scarring (Brody and Hailey, 1989; Mackee, 1952). He performed superficial peeling by applying phenol to the skin over a 30- to 60-second period and then washing it away immediately with ethanol in four to six treatments at 2-month intervals (Kligman et al, 1985). Phenol, which is carbolic acid, was first used to remove the laxity of the lower eyelids for cosmetic purposes by Sir Harold Gillies (McCollough and Hillman, 1980). In the 1930s lay operators used phenol to remove wrinkles. It was during that time that peeling of the neck skin resulted in hypertrophic scarring. This scared many people away from chemical peel (Kligman et al, 1985). Eller and Wolf (1941) described using phenol and resorcinol paste along with carbon dioxide cryotherapy to desquamate and resurface the skin (Brody and Hailey, 1989). Urkov (1946) introduced salicylic acid as a skin surface exfoliant and added occlusion to the process. In 1960 Ayres applied phenol to actinically damaged skin and then studied the histologic dermal changes (Botta et al, 1988; Brody and Hailey, 1989; Kligman et al, 1985). He demonstrated a distinct new subepidermal layer of collagen 0.3 to 0.4 mm thick with parallel fibers arranged horizontally. He also noted that dermabrasion had the same effect. In 1961 Baker and Gordon developed a formula for phenol face peeling that is widely accepted and used today. Using essentially a 50% phenol solution, they advocated the use of a waterproof tape-mask occlusion in conjunction with the solution.

Litton (1962) followed with a formula for a phenol solution incorporating a different detergent. He recommended glycerin instead of Septisol. Litton was one of the first persons to describe the widening of the stratum papillare after phenol peeling. In 1980 McCollough and Hillman demonstrated that Baker's solution used without taping can give equivalent results to those obtained with taping but with much less discomfort and fewer complications. In 1982 Stegman did an in-depth paper further providing information on the histologic effects of chemical peel and dermabrasion in sun-damaged and non-sun-damaged skin (Brody and Hailey, 1989; Kligman et al, 1985). He noted the consistent development of enlarged papillary dermis.

During the 1980s there was a resurgence of interest in finding appropriate light- or medium-depth peel alternatives to the standard phenol solution. Stagnone (1989) noted that superficial peeling with mild acids resulted in a "freshening" of the skin. Brody (1986) advocated the use of trichloracetic acid (TCA) for light- or medium-depth peels (Brody and Hailey, 1988). Weiss et al (1988) demonstrated that by using retinoic acid (Retin-A) for 6 months or longer, one could achieve some resolution of the surface wrinkling and removal of actinic and pigmented solar keratoses (Stagnone, 1977). Stagnone and Brody advocated the use of Jessner's solution (essentially resorcinol and salicylic acid), a modified Unna's resorcin

paste, alpha-hydroxy acids, and retinoic acid for light peels. Brody further discussed TCA, 5-fluorouracil (5-FU), and carbon dioxide cryotherapy, and he pointed out how each could be used separately or in combination to perform light-, medium-, or deeper-depth peels (Brody and Hailey, 1988; Stagnone, 1987, 1989).

The evolution of chemical peels has come to the point where Baker et al (1989) changed their routine of water-proof mask taping for occlusion after phenol peeling. They have now found that moist occlusion with petroleum jelly (Vaseline) has fewer disadvantages and provides equal results.

Histology of Sun-Damaged Skin and Long-Term Effects of Phenol Peeling

Brown et al (1960) wrote the first scholarly report of phenol peel including the formula, the technique for applying it, and the histologic changes that were induced (Litton et al, 1986). Brown et al (1960) described laminated collagen in the epidermis, with fibrous strands consistently paralleling the newly formed epidermis.

Kligman et al (1985) studied the skin taken from Baker and Gordon's face-lift patients who had a chemical peel 1.5 to 20 years earlier. First they described histologic changes in the nonpeeled skin. These demonstrated changes were typical of actinic exposure with a loss of orderly differentiation in the epidermis and degeneration of the elastic network, along with some mottled pigmentation and lymphocytic infiltration. There was a decrease in collagen amounts as well as disordered degeneration of the dermal fibers, a flattening of the dermal-epidermal junction, and multiple actinic keratoses with atypia seen. The melanocytes were increased in this actinic skin, but they were unevenly distributed and contained variable amounts of melanin.

The skin in patients who had undergone a previous chemical peel showed a new band of dermis 2 to 3 mm thick just beneath the epidermis and lying on top of the old elastotic dermis. The epidermis had returned to orderly cellular differentiation without irregularities or microscopic actinic keratoses. Although there was an abundance of melanocytes present containing some fine melanin granules evenly distributed, there appeared to be impaired melanin synthesis with a generalized bleaching effect, or hypopigmentation. Lentigines were not seen. Further, the epidermal-dermal matrix was composed of thin, compact, parallel collagen bundles arranged horizontally in contrast to the usual wavy pattern. Elastotic fibers had actually regenerated, forming a network of fibers paralleling the new collagen. Finally, the lymphocytic infiltration was diminished as compared to untreated skin. Kligman et al (1985) felt that the dermal reconstruction lasted about 20 years based on his study. He further concluded that chemical peel reduced the development of new neoplasms. The laying down of a band of new connective tissue can adequately account for the effacement of the wrinkles seen clinically. The skin is smoother, fuller, and tighter. Stegman (1982) and Litton et al (1962; 1986) showed the chemical peel solution penetrating deeper in the dermis of actinically damaged skin than nonactinically damaged skin. Hayes and Stambaugh (1989) demonstrated that during the first 2 to 5 days of a chemical peel there is epidermal necrosis, edema, and homogenization with lymphocytic infiltration all the way into the reticular dermis (Hayes et al, 1990). At 2 weeks, new collagen formation had begun. Stegman (1982), Alt (1989), and Brody (1991) have illustrated that there is deeper penetration of phenol with occlusion than nonocclusion. According to Beeson and McCollough (1985), this is apparently true but not

necessarily desired.

Litton et al (1986) agreed with Kligman et al (1985) that the rate of appearance with precancerous and early cancerous lesions of photo-aged skin was decreased after a phenol chemical peel.

Brodland and Roenigk (1988) showed that TCA destroys the epidermis and upper dermis and further showed that the new epidermis migrated from the cutaneous adnexa beneath the destroyed tissue. This is similar to phenol peel. Histologically the atypical clones of keratinocytes are removed and replaced by normal epidermal cells.

Indications

The actual process or technique of chemical peel is fairly easily learned. However, it takes a great deal of experience with many different types of patients to learn the wide variation in skin types and how these respond to the peel solution (Brody and Hailey, 1989). It also takes a great deal of experience to predict the way in which each area of the face will respond to deep or light peeling in individual patients and how one can influence the outcome with the method of application used. Careful selection of the appropriate patients for chemical peel is the first and most important consideration. As McCollough and Hillman (1980) stated, "The ideal patient is a thin-skinned female with fair complexion and fine rhytids". Fitzpatrick (1988) described types of actinically damaged skin ranging type I to type VI (see box). Brody (1989b) stated that Fitzpatrick types I through III patients are suitable for a chemical peel. He describes the ideal patient as a light-complexed person of Celtic or Northern European descent with skin type I or II (Fig. 32-1) (Brody and Hailey, 1988).

Box 1. **Fitzpatrick sun-reactive types I to VI**

Type I Fair skinned, blue or hazel eyes, blond or red hair. Always burns, never tans.
Type II Fair skinned; blond, red, or brown hair. Usually burns, tans less than average.
Type III Fair skinned, largest group of US citizens. Sometimes burns mildly, tans about average.
Type IV Still considered white skinned. Rarely burns, tans more than average and with ease.
Type V Intermediate colored skin, that is, Asian, Latin, Indian. Brown skin.
Type VI Black-skinned individuals.

There are both aesthetic and therapeutic indications for chemical peel (see box).

Brody states that we need to further separate indications into whether a light, medium, or deep chemical peel is required (Brody and Hailey, 1988). The surgeon must understand skin types as well as the effect each agent will produce. Stagnone (1989) states that superficial peeling is indicated for the freshening of skin, for postinflammatory pigmentation changes, for minimal wrinkling, and for melasma.

Box 2. **Aesthetic and therapeutic indications**

Aesthetic indications

Fine facial rhytids
Atrophic changes in skin caused by excessive sun exposure
Spotty or splotchy hyperpigmentation
Chataigne skin (sailor's or farmer's skin)
Multiple actinic and solar keratoses
Superficial acne scarring
Melasma
Excessively wrinkled skin
After blepharoplasty or face-lift

Therapeutic indications

Multiple actinic, seborrheic, and solar pigmented keratoses
Superficial basal cell carcinomas
Lentigo maligna lentigines
Melasma (discoloration of skin secondary to pregnancy).

Contraindications and Relative Contraindications

There are a few relative contraindications to chemical peel and several very specific contraindications. It has been stated that a history of previous herpes simplex infection is a contraindication to chemical peel (Farber, 1979). However, with the use of acyclovir (Zovirax) antiviral medicine, only active herpes at the time of chemical peel would be a contraindication. Telangiectasias are a relative contraindication in that they not only will not be better after chemical peel, but also they may be more apparent. Malignant lesions should not be treated with chemical peel unless they are very superficial basal cell carcinomas. Nevoid or nevus lesions may become darker or actually stimulated to grow, and port-wine stains, hemangiomas, and neurofibromatoses are not effectively treated with chemical peel. Contraindications include the presence of hepatorenal disease or cardiac disease, unless approved by an appropriate specialist, or an allergy to the agent to be used. Patients who are unstable psychologically should not be treated with any aesthetic procedure, but particularly not chemical peel, since this procedure requires intense patient involvement, education, and understanding.

Brody's list of relative and absolute contraindications are given in the box (Brody, 1989b).

Patient Evaluation and Selection

Selecting and preparing a patient for chemical peel begin with preconsultation written materials. The patient normally calls the office for consultation seeking help with wrinkles and other aging changes about the face. A consultation booklet that provides information about chemical peel as well as other rejuvenation procedures can be sent to the patient, who

can begin to understand what surgery will do and what it will not do. This allows the patient to understand how chemical peel can be an ideal primary or adjunctive procedure. Often a patient assumes that a rhytidectomy will remove wrinkles. It is imperative that the physician help the patient understand that although the sags will be lifted with a rhytidectomy, the wrinkles and other surface irregularities will still be present. Chemical peel is a technique that rejuvenates sun-damaged skin and reduces other effects of aging.

Box 3. **Relative and absolute contraindications**

Relative contraindications

Darker skin type, such as Fitzpatrick IV, V, and VI
Keloid formation
History of herpes infection
Cardiac abnormalities
History of previous facial irradiation
Marked amount of vellus hairs present
Unrealistic patient expectations
Physical inability to properly care for the face postoperatively
Telangiectases
Anticipation of inadequate photo protection because of job, vocation, or recreation

Absolute contraindications

Hepatorenal disease
An HIV-positive patient
An immunosuppressed patient (ie, hypogammaglobulinemia)
Emotional instability or mental illness
Ehlers-Danlos syndrome
Scleroderma or collagen vascular diseases
Recent isotretinoin (Accutane) treatment (less than 6 months before).

During consultation, the surgeon must determine whether the patient wears makeup routinely and whether the patient's life-style indicates heavy sun exposure (McCollough and Langsdon, 1988). Female patients who are unwilling to wear makeup or males who are generally not able to use makeup to cover postoperative hypopigmentation are not good candidates for this procedure. Those patients who are unwilling to decrease their actinic exposure are likewise poor candidates for chemical peel, since the procedure does in fact reduce their melanin protection.

Litton et al (1986) believe it is imperative that the patient have appropriate motivation and expectations to achieve a successful result. The patient must understand that large pores will be essentially unchanged. He or she also needs to have a clear understanding of the postoperative discomfort, appearance, and care that will ensue. Informed consent about the risks is imperative. At the end of the consultation, photographic documentation is essential as for any aesthetic procedure. This is the only objective way that the surgeon can later

determine whether or not he or she is achieving satisfactory results. Litton (1962, 1986), as well as Farber (1979), feels that urinalysis, EKG, and liver and kidney function tests are necessary in the preoperative medical workup for chemical peel. If the patient is over 40 years of age, a call is made preoperatively to the general physician confirming the suitability of sedation anesthesia and the general good health of the patient (McCollough and Langsdon, 1988).

Any impairment of liver or kidney function would impair the excretion of phenol and potentially increase the concentration of phenol in the bloodstream, which could result in cardiac irregularities and even death.

Chemical Agents

Depending on the surgical goals, an appropriate agent is selected for each individual patient. There are a variety of agents available that will produce the depth of peel desired. The surgeon must be able to predict the results likely with each and select the most appropriate agents for each patient (Brody and Hailey, 1989).

One must consider a chemical peel at the superficial level. Histologically this extends down only into the stratum granulosum and papillary dermis. Stagnone (1989) calls this a "freshening of the skin". He advocates a very light peel with resorcin or Jessner's solution (resorcinol, 14 g; salicylic acid, 14 g; lactic acid, 14 mL; and ethanol, 100 mL). Stagnone notes that this has been widely used by lay peelers because there is little danger despite a change in concentration. Other agents that produce a light chemical peel include carbon dioxide; retinoic acid (Retin-A); 5-FU; alpha-hydroxy acids; TCA, 10% to 20%; and modified Unna paste. Retin-A used for 6 months or as long as 2 to 3 years reduces wrinkling, actinic keratoses, and pigmentary problems (Stagnone, 1989; Weiss et al, 1988). Brody and Alt (1991) and Mandy (1986) also advocated Retin-A for the prechemical peel or predermabrasion period and for 2 weeks after peel or dermabrasion. This appears to enhance the results and speed the healing process.

The depth of the peel achieved with each of the above agents will vary depending on the concentration of agent used, the duration of the application, and the number of times the agent is applied. A light peel can be characterized as one produced by trichloroacetic acid 35%, unoccluded, in single or multiple frosts. A medium-depth peel is one in which the injury extends to the upper reticular dermis, such as is produced by a combination of carbon dioxide and TCA 35% unoccluded (Brody, 1989a, 1989c; Brody and Hailey, 1989). An alternative to this would be a combination of Jessner's solution with TCA 35%, unoccluded, or TCA 50% by itself, unoccluded. Phenol 88% by itself will give a medium-depth peel.

The classic deeper-depth or deep wounding to the midreticular dermis is accomplished with Baker's solution, which consists of the following (Rees, 1980):

Phenol 88% USP	3 mL
Septisol	8 drops
Croton oil	3 drops
Distilled water	2 mL.

This can also be achieved with a 50% or greater concentration of TCA. Baker's solution can be used occluded or unoccluded.

Baker's solution is an emulsion that can be prepared before each procedure or stored for short periods of time in an amber bottle. Litton et al (1986) vary the formula from the standard Baker's peel solution by using a different detergent, glycerol, instead of Septisol. Litton's formula is made by liquefying 1 pound of phenol crystals in 8 mL of distilled water and then adding 8 mL of glycerin. Following this, Litton adds 4 ounces of liquefied phenol and 1 mL of croton oil. All of this then is put into an 8-ounce bottle, and another 4 ounces of distilled water is added. Heat is required to mix this combination together, but, according to Litton et al (1986), it does remain potent for 3 to 6 months in an amber bottle at room temperature.

Phenol itself is carbolic acid (C_6H_5OH), an aromatic benzene ring hydrocarbon formed from coal tar. Phenol is an antiseptic as well as an anesthetic (Koopman, 1982; Litton et al, 1986; McCollough and Hillman, 1980). At greater than 80% concentration, carbolic acid is a keratocoagulant precipitating the surface protein, thus preventing further penetration of the peel solution. According to Litton (1962), it produces an extremely rapid denaturization and coagulation that is irreversible (Litton et al, 1986). Further penetration of the phenol is prevented when the keratin protein binds to the phenol, creating large molecules that cannot penetrate further. McCollough and Hillman (1980) state that if the concentration of phenol is less than 50%, it becomes keratolytic interrupting sulfur bridges in the keratin layer and can then produce deeper penetration and more destruction than desired.

The croton oil included in the formula is expressed from the seed of "croton tiglium", composed of glycerides of several acids. It is toxic and very irritative to the skin. By itself, it causes pustular eruptions and skin destruction. Because of this inflammatory characteristic, it induces more collagen formation (Litton et al, 1986; McCollough and Hillman, 1980).

Soap in the solution acts as a surfactant, reducing the interface surface tension and enhancing the penetration of the waxes and cholesterol esters of phenol. Septisol (hexachlorophene and alcohol) is a partial astringent that helps remove the stratum corneum and plays the role of a surfactant. The addition of distilled water produces the desired concentration of phenol at between 50% and 60%.

Procedure

Preparing a patient for a chemical peel begins with preoperative prescriptions given at the time of scheduling. An antibiotic for prophylaxis of infection is begun 1 day before surgery. Commonly this would be a cephalosporin such as Keflex, 500 mg BID. The night before the procedure, the patient should receive a sedative-hypnotic for sleep. The patient is instructed to take 5 mg of diazepam (Valium) PO 2 hours before arriving at the surgery center. Skin preparation is important. Before receiving a preoperative sedative, the patient is instructed to remove all makeup, including mascara, eyeliner, eye shadow, and lipstick. The face is washed twice with Septisol and rinsed thoroughly after each washing. PHisoHex or pHisoDerm should not be used since it leaves a residue on the skin (McCollough and Hillman, 1989; McCollough and Langsdon, 1988). This residue may interfere with the penetration of the phenol. Collins (1989) feels that preparation is of utmost importance

because the cutaneous surface is cluttered with sebum, oils, dirt, and cosmetics that are often heaviest at the bottom of the rhytids and pores. Brody and Alt (1991) believe in treating the skin with retinoic acids (Retin-A) immediately before the peel to stimulate skin healing and to remove more of the stratum corneum.

Once the face has been prepared, preoperative sedatives are given. Generally, the patient is given 15 to 20 mg of diazepam (Valium) PO as well as an antinausea medicine such as 50 mg of promethazine and phenylephrine (Phenergan) and 10 mg of metoclopramide (Reglan). Buffered aspirin is given to help in reducing the burning sensation along with a Tylox (oxycodone and acetaminophen) capsule to provide some analgesia. An intravenous line is started, and approximately 500 mL of an intravenous fluid such as Ringer's lactate is given. Normal saline or 5% dextrose is satisfactory. The patient is then moved into an operative suite or room where complete monitoring equipment is available. The patient is monitored continuously with cardiac, blood pressure, and oxygen saturation equipment (Fig. 32-3). Typically the patient is given a narcotic of hydromorphone (Dilaudid), 1 or 2 mg, fentanyl citrate (Sublimaze), or both titrated during the case. I prefer the patient to be given a longer-acting narcotic that will last into the postoperative period. Following this, midazolam (Versed) is given for amnesia as well as sedation. Other authors advocate the use of lorazepam (Ativan) for strong amnesia, or either methohexital (Brevital) or diprovan (Propofol) may be used for short-acting complete amnesia to provide an opportunity to inject bupivacaine (Marcaine) 0.05% with 1:100.000 epinephrine to regional sensory nerves. This long-acting regional block anesthesia provides a reduction in postoperative discomfort for the patient. The bupivacaine is used to block the supraorbital, infraorbital, incisive foramen, and mental nerves, as well as infiltrating the lower eyelids and the preauricular area. With this method the patient is essentially spared the typical 4- to 6-hour postoperative burning pain. Throughout the procedure, sedation is titrated to keep the patient completely comfortable.

Before applying any phenol solution, each region of the face is scrubbed with acetone to provide desquamation (McCollough and Hillman, 1980). The epidermabrasives and defatting preparations alter the skin permeability to the wounding agents, and it is the skillful management of the defatting agent that allows one to obtain optimum results from one area to the next (Fig. 32-3) (Brody and Hailey, 1989).

Factors that affect the depth of penetration of wounding agents, according to Brody (1989c) and Collins (1989) include the amount of solution applied (wet versus dry or semidry applicators), the degree of rubbing or cleansing of the defatting agent, and the degree of superficial abrasion caused by the gauze sponge (McCollough and Hillman, 1980; McCollough and Langsdon, 1988). Brody referred to McCollough's technique of superficially abrading the skin with a gauze sponge while cleaning the skin with acetone as an important factor in allowing the unoccluded phenol to penetrate as deeply as when it is occluded with tape (Brody, 1989a). The duration of wound agent contact with the skin, the inherent skin sebaceous gland activity, the prior use of retinoic acid (Retin-A), and finally taping versus nontaping after applying the agent all affect the depth of penetration achieved. Collins (1989) points out that the sebum, oils, and cosmetics are not adequately removed with soap alone, and acetone is required.

The face may be divided into six aesthetic units (see discussion of phenol toxicity). After thorough scrubbing with acetone-soaked 2-inch x 2-inch gauze squares until the motion

sounds like sandpaper across the skin, each region is treated with Baker's solution applied with a cotton-tipped applicator. Areas such as the forehead, cheek, and perioral region are treated with a fairly wet applicator. The applicator is semidry when used on the eyelid or periorbital regions. Feathering is done at the periphery of the peeled areas. It is important to "feather" into the hairline, all the way into the brows, under the jawline, and across the junction from region to region as the peeling proceeds (Fig. 32-4). Brody (1989b) believes that feathering is very important and always peels 5 mm beyond the natural boundary of an aesthetic unit. Litton et al (1986), however, feel that because the phenol works as an all-or-none phenomenon, feathering is difficult to achieve. McCollough and Langsdon (1988) and I believe that feathering is possible by applying a lesser volume of solution at the edge. Less rubbing with the acetone at the edge also allows a lesser penetration of the phenol. It is important to feather down underneath the jawline to hide the transition zone of peeled to nonpeeled skin within the shadow of the mandible (Fig. 32-5). Feathering is certainly important when treating the lower eyelid-infraorbital transition or the perioral region across the cheek-lip groove onto the cheek.

When treating the perioral area, one must apply the phenol solution initially with a slanted cut wooden applicator to place the phenol solution down into the bottom of each individual rhytid (Fig. 32-6). The lip is then stretched to spread out the rhytids while applying the solution with a very wet cotton-tipped applicator. One needs to obtain a distinct white frost appearance that is carried 2 to 3 mm across the vermilion border. When treating the lower eyelid, it is important to use a semidry applicator rolled once across the skin. The lower eyelids need to be treated to within 1 to 2 mm of the ciliary margin. On the upper eyelid, one must be very judicious about treating below the supratarsal fold. Most surgeons stop at the supratarsal fold.

Each of the five aesthetic units is treated, 15 to 20 minutes apart: the forehead, the right cheek, the left cheek, the perioral region, and finally the nose and periorbital areas together (Fig. 32-7). If one considers the nose as a unit separate from the periorbital area, six units are designated. When applying the solution to the cheek, it is important to use a wet applicator and to overlap each application so as to avoid streaking. The solution should be applied in the direction of relaxed skin tension lines. A prominent white frost should be obtained immediately on application of the solution in each area. This rapidly changes to an intense pink with a band or zone of erythema extending 1 to 2 cm beyond the area treated with solution. Therefore, when treating the next region adjacent to the previously peeled region, it is important to peel across this band of erythema so as not to have skip areas. A burning sensation for the patient lasts between 15 and 20 seconds. However, the pain is likely to return within 20 minutes to 2 hours after the peel and will last for 6 to 8 hours. The chemical peel is generally painless to the patient after this period of time.

During the procedure, 500 mL of fluid is given intravenously. During the recovery period another 500 mL of solution is given. Thus a total of 1500 mL of solution is administered intravenously during the perioperative period.

Taped Versus Nontaped Phenol Chemical Peel

Baker and Gordon (1961, 1966, 1986) believe it is necessary to mask tape immediately after the application of the phenol solution to obtain the desired result. They use a waterproof

nonporous tape and leave it on for 48 hours. The action of the phenol is prolonged by the use of such a vapor barrier. Stegman (1982) showed histologically that there was a deeper penetration with the occluded phenol than with the nonoccluded. Baker and Gordon and Stegman felt that this method produces a more desired result. However, others (McCollough and Hillman, 1980; McCollough and Langsdon, 1988) feel that one can achieve preferable results without taping. It is not necessarily preferable to get deeper penetration of the solution, since this may increase the chance of complications such as hypopigmentation, depigmentation, scarring, and prolonged erythema.

McCollough and Hillman (1980) presented a 4-year experience comparing nontaping versus taping, which suggested a reduced incidence of postoperative scarring in the untaped patient. The total cost to the patient is reduced by eliminating the need for a second anesthetic to removed the tape. There is increased convenience and comfort for the untaped patient, and it is possible to monitor the early healing when no tape is used.

It is interesting that in the their book (1986) and article (1989), Baker and Gordon have changed their routine to avoid taping. However, they still believe in the importance of a vapor occlusion barrier, which is accomplished by applying petroleum jelly (Vaseline) immediately after application of the peel solution. They feel there is greater comfort, improved ability to evaluate the wound, less chance of streaking, and no eschar or crusting formation with this nontaped moist occlusive technique. They also note that it is important that the patient avoids removal at 48 hours.

Postoperative Care Routine

Postoperative care routine includes an analgesic such as Percocet or Tylox (oxycodone and acetaminophen). Patients are instructed to do nothing but rest until the following morning's office visit. If they are experiencing some burning pain despite the analgesic, they may apply cold compresses to the face and begin a thin application of vegetable shortening (Crisco), petroleum jelly (Vaseline), or Aquaphor ointment. The patient is discharged from the surgery center to home or a nearby hotel with a friend, family member, or nurse staying with the patient. The patient will need assistance and should not be left to drive or take care of himself or herself. The patient is instructed to return to the office or clinic the first postoperative morning and is given a postoperative instruction booklet that discusses the expectations of marked edema, moderate temperature elevation to 99° to 100°F, occasional nausea, and moderate discomfort. The day after surgery, a thick layer of vegetable shortening (Crisco) is applied over the entire area that was treated (Fig. 32-8). Crisco is really a vitamin A and vitamin E oil. The patient is shown how to apply the Crisco in "icing on a cake" technique thick enough to keep the area moisturized adequately (Fig. 32-9). An alternative, depending on the patient's preference, is Eucerin cream. Bacitracin ointment may be applied, but there is some risk of developing a sensitivity to the antibiotic. McCollough and Hillman (1980), McCollough and Langsdon (1988), and Baker et al (1989) believe that A & D ointment or Aloe Vera cream should be used. The patient is instructed to wash or rinse the face without soap using tepid tap water five or six times per day. The patient can do this in the shower or splash water from the sink while massaging the skin in the peeled area. The massaging action is critical to remove the old cream or oil as well as the desquamating layer of peeling skin and crust. The patient may use either the tips of the fingers or wet cotton balls to gently remove the crust. The patient is given 10 mg of dexamethasone (Decadron) IV

intraoperatively and is given methylprednisolone (Medrol Dose-Pak) postoperatively to reduce swelling. The patient is encouraged to take 1000 to 2000 mg per day of vitamin C and a multivitamin as well and to continue the antibiotic prophylaxis for 4 to 5 days postoperatively.

The patient is asked to return to the office on the fourth postoperative day to assure the physician that the wound is being cleaned as instructed. This is the time when, if the wound has been neglected, superficial infection may become a problem. A *Pseudomonas* infection can ensue and create a deeper injury (Fig. 32-10). Often the patient does not understand the degree to which his or her involvement is required to remove the desquamating skin and crust. The patient is again evaluated about 4 days later to observe the amount of crusting. At this time, he or she is allowed to reduce the amount of moisturizing cream that is being applied. After 7 to 10 days, the patient can begin to apply makeup. A makeup artist is available, and instructions are given about how to cover the intense erythema (Fig. 32-11). A good, brand-name hypoallergenic moisturizer is recommended. Dermage and Estee Lauder concealer are makeups that are effective in covering the red areas. Generally a ming green base is used before the foundation is applied to neutralize the red.

It is also very important that the patient be instructed early in the use of sunscreens. An SPF (sun protection factor) of 15 or greater is advised. The patient is not allowed any direct sun exposure for 6 weeks and is told to minimize sun exposure for up to 6 months.

To reduce the possibility of hyperpigmentation, estrogens should be withheld before the peel and for at least 6 to 8 weeks postoperatively. As the erythema is fading, pigmentation abnormalities are possible, and estrogen may increase the risk of this abnormality.

Commonly, Hytone cream 2.5% (a nonfluorinated steroid) is recommended for 2 or 3 weeks to decrease the erythema and the hypersensitivity that is common. The patient may complain of intense pruritus during this period, and Pramosane 2.5% steroid cream with an antipruritic agent can be helpful. The patient also may require hydroxyzine (Atarax), 10 to 20 mg PO, TID and at night to decrease the sensation of itching. Hypnotics may be necessary for sleep.

Alt (1989) recommended that as soon as epithelialization has occurred, the patient should be given retinoic acid (Retin-A) to enhance or prolong the chemical peel effect. I prefer waiting at least 2 months after the peel before starting Retin-A and then using it only if the patient desires it.

The patient is observed at 2 weeks and again 6 weeks later to look for the early development of splotchy hyperpigmentation. Collins (1989) recommends the use of hydroquinone gel almost routinely for 2 to 4 months as a prophylactic regimen against this, particularly in darker-skinned individuals. If splotchy pigmentation develops, a combination of Retin-A, Eldoquin Forte, and Aristocort may provide an improvement (see discussion of complications and hyperpigmentation). The patient is observed at 3 months, 6 months, and 1 year. Photographs are taken at each session (Figs. 32-12 to 32-16).

Chemical Peel Combined With Other Surgical Procedures

Regional chemical peeling can be an excellent adjunct to face-lift and blepharoplasty

(Baker, 1962). McCollough and Hillman (1980) feel that regional peel in combination with facial surgery works out well, particularly in the perioral and periorbital areas. Becker (1983) reviewed his face-lift population and noted that in 80% of the patients he performed a perioral or circumoral chemical peel with no complications of flap necrosis or scarring.

Most surgeons feel that simultaneously peeling the undermined skin of a face-lift flap should be avoided. In fact, Hayes et al (1990) demonstrated a significant increase in flap necrosis in a study of patients undergoing a local flap and simultaneous chemical peel. They showed that lifting the flap caused changes in the subcutaneous tissue and reticular dermis that make the flap more vulnerable to secondary injury. The upper reticular dermis heals be reorganization, and the deep reticular dermis heals by scar formation. Hayes et al compared peeled and unpeeled skin flaps at 21 days and demonstrated that there was a statistically significant increase in tissue loss in the flaps that were simultaneously peeled. Microscopically, there was edema and an intense, acute-phase neutrophilic infiltrate found in the upper reticular dermis with the flap elevation alone. The infiltrates would extend down into the deep reticular dermis, destroying much of the skin when simultaneously peeled. Dermal injury that is great enough to cause destruction of collagen in the reticular dermis is likely to cause damage to the vascular supply to the skin, thus compromising the viability of the skin flap. McCollough and Hillman (1980) recommended a 3- to 6-month wait before peeling areas that had been undermined. Litton et al (1986) wait 6 months after surgery for chemical peel. Spira et al (1974) and Brody (1989b) feel that you should wait 2 to 3 months before performing the surgery after a peel or wait only 1 to 3 months after surgery to perform a chemical peel. I feel that caution is wise with the lower eyelids as well and recommend at least a 2-month wait after blepharoplasty. However, if one is not undermining a skin flap, as, for instance, in a transconjunctival blepharoplasty, simultaneous chemical peel is possible without untoward complications.

Combining chemical peel with dermabrasion is common. Horton and Sadove (1987) commonly perform chemical peel followed by dermabrasion of the same area for rhytids in the perioral area. These rhytids have often been resistant to removal with one chemical peel or even with deep phenol peeling. One can perform dermabrasion for acne scarring of the cheek and peel the lower eyelids at the same time. Regions can be divided, being sure to feather the peel into the areas lightly dermabraded (Stagnone, 1987). Others have recommended routinely doing trichloroacetic acid chemical peeling followed by dermabrasion in the same area (Stagnone, 1977).

Phenol Toxicity

Phenol is essentially toxic to all cells. Because it is absorbed through the skin into the bloodstream, it must be excreted rapidly. Beeson and McCollough (1985) noted that phenol is excreted 80% unchanged in the urine or conjugated with glucuronic acid and sulfuric acid. Litton et al (1986) note that the body has a preexisting mechanism for removal of phenol by conjugation, oxidation, and direct excretion. Any toxic reaction to phenol would depend on the free or unconjugated portion in the blood or tissues, with a toxic dose between 8 and 15 g for adults. Stagnone et al (1987) noted fatal blood levels of phenol at 7.5 mg/100 mL. The liver, kidneys, gastrointestinal tract, lungs, and red cells all have some capacity to detoxify phenol, with 60% to 80% of any given dose being conjugated. The remainder is oxidized to CO_2 and water. A very small quantity is oxidized to pyrocatechol and hydroquinone. Wixler

et al (1984) demonstrated that the excretion of phenol during a chemical peel can be facilitated by forced diuresis (a fluid infusion combined with a diuretic). Volume preloading before, during, and after chemical peel with intravenous fluids is adequate to dilute the phenol and enhance excretion through the kidneys in patients with normal liver and renal function. Botta et al (1988) recommend maintaining a fluid load to force diuresis with furosemide, 20 mg given 10 minutes before the application of phenol. Farber et al (1984) recommend IV infusion of a plasma expander, but other investigators do not feel expanders are necessary.

Systemic toxicity is first suggested by CNS stimulation, including tremors, hyperreflexia, and hypertension. Following this, CNS depression occurs with respiratory failure, hypotension, and cardiac arrhythmias ensuing rapidly. According to Koopman (1982) 75% of a given dose of phenol is excreted in the urine and 25% is metabolized to CO_2 and water. Brody (1989b) and Wixler et al (1984) report that 70% of phenol applied to the skin is absorbed within 30 minutes. Stagnone et al (1987) did a study in dogs in which a phenol load was given, and 100% died of cardiac myocardial toxicity. The ultimate event was electromechanical dissociation. When compared with trichloroacetic acid (TCA), there were no cardiac abnormalities noted in the TCA group. Truppman and Ellenby (1979) studied 43 patients and found that 10 (23%) developed some cardiac arrhythmias. The order in which cardiac signs of toxicity developed was as follows: (1) tachycardia, (2) premature ventricular contractions, and (3) atrial fibrillation (Gross, 1984). The duration lasted from 2 minutes to 19 minutes. Truppman and Ellenby noted an increase in arrhythmias with shorter procedures and with larger areas being covered. The average time from application of phenol to onset of arrhythmias was 17.5 minutes. All 10 patients who experienced arrhythmia had at least 50% of their face covered at one time. It is interesting to note that Price (1990) found many spontaneously occurring ectopic beats unrelated to the actual application of the agent in patients studied with a Holter monitor while sedated and awaiting a chemical peel or other surgical procedure. He found no correlation between the screening ECG findings preoperatively and the Holter monitor recording of continuous cardiac rhythm during the procedure. Patients studied preoperatively with the Holter monitor were compared with those studied intraoperatively. The preoperative patients actually had more ectopic beats than those during the surgery. Beeson and McCollough (1985) reference Hammil as suggesting that the peel solution should be applied to a limited area of no more than 150 cm^2 at a time. Hammil divides the face into five aesthetic units. Litton says that in his experience no arrhythmia has occurred in a patient whose chemical peel spanned 60 minutes or more when the areas treated constituted less than 50% of the face. Phenol should be avoided in patients with renal disease, because they may not excrete it rapidly enough to avoid direct cardiac toxicity. Waiting as much as 30 minutes between each area to be treated and not peeling greater than 50% of the face at one time minimize the risk of phenol toxicity in most patients. Litton et al (1986) recommend monitoring the patient 1 to 2 hours postoperatively, whereas McCollough and Langsdon (1988) feel that 30 minutes is adequate. Collins (1989) has suggested that there is no systemic toxicity known from TCA, and, in fact, TCA can be applied to the entire face all at one time. He suggests using TCA peel in patients with potential or known cardiac abnormalities or hepatorenal disease. TCA peel is not an all-or-nothing phenomenon. The effect observed depends on the concentration used, the duration of the application, and the number of applications. Brodland and Roenigk (1988) point out that, unlike phenol, TCA is neutralized by the serum in the superficial dermal plexus and is therefore nontoxic to the heart and kidney.

Ways to reduce risk of cardiac or renal complications with phenol chemical peel are as follows (Brody, 1989b):

1. Carefully evaluate the patient preoperatively with regard to cardiac and renal function.
2. Peel the face in five to eight sequential aesthetic units or segments.
3. Allow at least 10 to 20 minutes between each segment application.
4. Hydrate patient with at least 500 mL of lactated Ringer's solution before the peel and 1000 mL of the same solution during and after the procedure to enhance renal clearance and dilute the blood volume.
5. Allow 60 to 120 minutes for a full-face chemical peel.
6. Never peel more than 50% of the face at one time.
7. Use cardiac, blood pressure, pulse, and oxygen saturation monitoring before, during, and after the procedure.
8. Have emergency cardiopulmonary resuscitation equipment present at all times.
9. Have personnel who are trained in advance cardiac life support immediately available.

Complications

Brody most accurately describes chemical peel by saying, "Unfortunately there may be circumstances when this controlled wounding (that we are creating by applying a wounding agent to the skin) becomes uncontrolled, resulting in selected complications". Chemical peeling may have some sequelae that are expected rather than considered to be true complications of the procedure. Sequelae of chemical face peeling are as follows:

1. Pigmentary changes
    a. Hyperpigmentation
    b. Hypopigmentation
    c. Depigmentation
2. Persistence of rhytids
3. Prolonged erythema
4. Persistent texture change of skin
5. Hypertrophic epidermal healing
6. Atrophy of skin
7. Milia
8. Skin pore prominence
9. Increased prominence of telangiectases
10. Darkening and growth of preexisting nevi
11. Infection
    a. Herpes
    b. *Pseudomonas* organism
    c. *Staphylococcus/Streptococcus* organisms
    d. *Candida* organisms
12. Lower eyelid ectropion
13. Cardiac arrhythmias
14. Renal failure
15. Laryngeal edema (Kline and Little, 1983)
16. Toxic shock syndrome (Dmytryshyan, 1983; Loverma, 1987)
17. Poor physician/patient relationship.

Pigmentation changes are by far the most common sequelae of chemical peel. Litton and Trinidad (1981) surveyed 565 surgeons, and two thirds said they had seen pigmentary problems in their patients. The most common of these was hypopigmentation (Fig. 32-17). These pigmentary abnormalities are more apparent because there is a line of demarcation between the peeled and nonpeeled areas. This undesired sequela can be minimized by the feathering technique described earlier. Streaking can occur from an uneven application of the skin preparation or the peel solution itself. Splotchy pigmentation may be either hyperpigmentation or hypopigmentation and is increased with exposure to sun or the systemic use of estrogens; it is more commonly seen during the first 6 months of healing. Litton et al (1986) describe pigmentary abnormalities that include depigmentation, hypopigmentation, hyperpigmentation, blotchy pigmentation, streaking, and an overall phenol bleaching that he described as the "pink look". Brody (1989b) states that pigmentary changes rarely occur in Fitzpatrick type I to III patients, but in type IV patients there are frequently quite prominent lines of demarcation. Type V and VI patients, if peeled, will usually develop hyperpigmentation, which may resolve over 18 to 24 months. Brody describes the type V patient as the Oriental, Latin, or Indian patient who is more likely to have irregular coloration preoperatively and should not be peeled. Retin-A 0.1% and Eldoquin Forte 4% to 5% mixed with Aristocort 0.1% should be applied at the onset of early hyperpigmentation or used routinely in at-risk patients during the first 6 months after peel (Fig. 32-18).

According to Litton and Trinidad's survey (1981) scarring is the most commonly reported complication. He states that 21% of the 568 surgeons he polled reported scarring in lip and chin peels. Farber et al (1984) say that there is a much higher incidence of hypertrophic scarring with TCA in a greater than 50% concentration than in phenol peels.

Another very common sequela of chemical peel is prolonged erythema and hypersensitivity to makeup (Fig. 32-19). McCollough and Langsdon (1988) commonly use Hytone 2.5% nonfluorinated steroid to minimize this and reduce the duration of redness. One can even use Elacon 0.1%, a potent nonfluorinated steroid, to reverse this process of hypertrophic epithelial response. Brody (1989b) notes that scarring may not become apparent until as long as 3 months after the chemical peel. This type of scarring is described as hypertrophic subepithelial scarring and may occasionally require intralesional steroid treatments (Fig. 32-20).

Unfortunately, persistent rhytids or wrinkles are a relatively common postoperative sequela. McCollough and Brody feel that waiting 3 to 6 months is appropriate before repeating a chemical peel. I often touch up the rhytids of the vermilion and perioral area with dermabrasion at between 3 to 6 months. Repeeling rhytids at less than 2 months can result in severe hypopigmentation and subepithelial hypertrophic scarring. Prolonged erythema should be expected with early repeeling.

True infection is uncommon, but herpetic breakouts are very common, particularly in patients who have had a previous history of at least one fever blister in the area treated. Superficial infection with *Pseudomonas, Staphylococcus,* or *Streptococcus* is rare and can usually be attributed to poor postoperative wound care. *Candida* infections may occur, prolonging the epithelialization. These should be treated with nystatin cream (Fig. 32-21). Brody points out that trichloracetic acid and phenol themselves are bactericidal, preventing most infections (Rappaport and Kramer, 1984). However, he points out that after chemical

peel, ointments can cause folliculitis, which may become secondarily infected with *Staphylococcus* or *Streptococcus*. He treats *Pseudomonas* with ciprofloxacin.

Any patient with a positive history of at least one fever blister can be expected to have an outbreak of these lesions (Fig. 32-22). Uncontrolled or untreated, this infection can spread over the entire peeled area, including the entire face (Fig. 32-23). Fear of transmission into the cornea is of great concern.

Brody and Alt (1991) now treat patients prophylactically with acyclovir (Zovirax), 200 mg TID 1 day before and 4 to 5 days after the peel. They state that if the infection becomes active, the dosage should be increased to 500 mg QID. Brody and Alt report that scarring is rare after a herpetic breakout. A history of herpetic lesions is no longer considered a contraindication to chemical peel, because of our ability to prevent serious complications with acyclovir (Zovirax). I recommend beginning the patient 1 day before the peel with 2.0 g of acyclovir (Zovirax) per day and continuing treating at that level for at least 10 days after chemical peel. Patients with a strong history of herpetic breakout should be treated for at least 2 weeks after chemical peel. Patients may even break out with facial herpes lesions on the twelfth postoperative day after having been treated with acyclovir (Zovirax) for 10 days and having completely epithelialized before the breakout. If a patient actually breaks out with active herpetic lesions, the dosage should be increased to 4.0 g per day.

Spira et al (1974) described lower eyelid ectropion in a patient who underwent chemical peel of the eyelids soon after having a blepharoplasty. They recommend not peeling the lower eyelids until 6 months after blepharoplasty. One may expect some temporary mild ectropion if a patient has poor recoil to the tarsus or a slight rounding from an earlier blepharoplasty. When a mild ectropion develops, the patient should be treated with topical steroids and intralesional steroids as necessary.

The development of skin pore prominence discussed by Litton and Trinidad (1981), is disputed by other authors. Alt (1989) and McCollough and Hillman (1980) feel that pores, if changed at all, are slightly tightened and improved. This is, however, not an expectation to be given to the patient preoperatively.

Telangiectases are themselves not more prominent; but because of some of the bleaching effect of the skin or hypopigmentation, they may become more noticeable. Treatment with the Candela dye laser or electrocautery is satisfactory for the removal of noticeable telangiectases.

The occurrence of milia postoperatively requires the use of a Buf-Puff (3M Company, St paul, MN). I use retinoic acid (Retin-A) in the early peel period only for removal of milia. Brody and Alt (1991) recommend the use of electrodesiccation and extraction with an 18-gauge needle or a No 11 blade for treating milia. However, many of the milia will resolve spontaneously.

Brody (1989b) feels that there may be atrophy or loss of normal skin markings in some cases. This is not true atrophy histologically. In fact, Kligman et al (1985) dispute this finding, in that they observed normal epidermis and a thickened epidermis for up to 20 years after peel.

There can be, however, a texture change in the skin that may appear to be atrophy. It presents clinically as a prolonged roughness and dryness of the skin, particularly noted while wearing make-up. This is usually temporary but can be prolonged, although it will resolve with time.

Brody reported three cases of toxic shock syndrome that resulted in fever, syncopal hypotension, vomiting, and diarrhea, occurring at 2 to 3 days postoperatively. Within 2 or 3 more days, those patients developed a scarlatiniform rash, and *Staphylococcus aureus* was cultured from the skin.

Complications can develop as a result of errors such as incorrectly mixing the formula or accidentally allowing solution into the eye. One must have an assistant ready with normal saline or ocular flush solutions to irrigate the eye if this should occur. Some surgeons recommend the use of propylene glycol or glycerol in the eye prophylactically to reduce the chance of injury to the eye. Olive oil can be used to neutralize the effect of the solution on the skin if necessary.

Summary

In summary, chemical face peeling can be an extremely satisfactory adjunct to a facial plastic surgeon's armamentarium and can provide a great deal of patient satisfaction. Phenol chemical peeling has been shown to resurface the facial skin with new epithelium. Patient selection and carefully matching the needs of the patient with the most appropriate type of peeling agent are keys to a successful outcome. Understanding the significant variabilities in technique can provide the surgeon with an adequate degree of flexibility to meet the needs of individual patients. Learning the technique of chemical peel, however, is only one small part of what is required to achieve consistently satisfactory results. Managing the postoperative care of the patient and dealing with the sequelae of the chemical peel are vitally important. All of these factors taken into consideration will allow the surgeon and patient to achieve a mutually satisfactory result and a good long-term relationship.