

TITLE: Facial Chemical Peels

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Introduction

Conventionally aging is defined as the process of becoming older. This traditional definition was recently challenged in the new *Handbook of the Biology of Aging* (Academic Press, 2006), as the process of system's deterioration with time. This definition allows for existence of non-ageing systems (when "old is as good as new"), and anti-ageing interventions (when accumulated damage is repaired). Certainly, facial skin deterioration is one of the most apparent examples of aging. For this reason combined with the social perception of youthfulness as a measure of outer beauty, it is not a surprise that people are seeking more avenues for anti-ageing interventions. This demand has prompted tremendous growth in the cosmetic industry, with numerous over-the-counter products available to "reverse aging changes."

Whether to reverse the signs of aging or to treat cutaneous lesions, ablative skin resurfacing is an integral part of the practice of facial plastic surgery and dermatology. Chemical peeling is a technique that removes superficial lesions and improves the texture of skin by the application of a chemical exfoliant. Peeling produces a controlled, partial-thickness chemical burn of the epidermis and the outer dermis. Regeneration of peeled skin from follicular and eccrine duct epithelium results in a fresh, orderly, organized epidermis. In the dermis, a new 2- to 3-mm band of dense, compact, orderly collagen is formed between the epidermis and the underlying damaged dermis, which results in effective ablation of the fine wrinkles in the skin and a reduction of pigmentation. These clinical and histological changes are long-lasting (15 to 20 years) and may be permanent for some patients.

Histology

The histological changes of the aging skin are typical of actinic changes which are the photochemical effects of solar radiation exposure. These changes include a loss of orderly differentiation in the epidermis and degeneration of the elastic network, along with some mottled pigmentation and lymphocytic infiltration. There is a decrease in collagen as well as disordered degeneration of the dermal fibers, a flattening of the dermal-epidermal junction, and multiple

actinic keratoses with atypia seen. The number of melanocytes was increased in this actinic skin, but they were unevenly distributed and contained variable amounts of melanin.

After a chemical peel, the skin regenerates, from follicular and eccrine duct epithelium results in a fresh, orderly, organized epidermis. There is a formation of new bands 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. The melanocytes present contain fine, evenly distributed melanin granules, there appeared to be impaired melanin synthesis with a generalized bleaching effect, or hypopigmentation. Lentigines are not seen. Thin, compact, parallel collagen bundles and elastic fibers are arranged horizontally along the epidermal-dermal matrix. There is decrease lymphocytic infiltration compared with that of untreated skin. During the first 2 to 5 days of a chemical peel there is epidermal necrosis, edema, and homogenization with the lymphocytic infiltration all the way into the reticular dermis. At 2 weeks, new collagen formation had begun. Histologically, the atypical clones of keratinocytes are removed and replaced by normal epidermal cells.

Kligman concluded that chemical peel reduced the development of new neoplasms. This observation has been confirmed on further research by Litton concluding that the rate of appearance with precancerous and early cancerous lesions of photoaged skin was decreased after a phenol chemical peel.

PATIENT EVALUATION

Not every patient is a good candidate for chemical peels. There are two classification systems that help the physician assess whether the individual patient as a good candidate for a chemical peel and strength of peel achieve the desire results with the least risk and complications. The clinician must take into account the patient's underlying skin type and the amount of photodamage present. Fitzpatrick classified the skin types and acute solar radiation response from type I to type VI. The Glogau classification system provides an objective assessment of the degree of photoaging, categorizing the patient's skin damage into mild, moderate, advanced, or severe (groups I–IV). "The ideal patient is a thin-skinned female with fair complexion and fine rhytids."

Patients with Fitzpatrick skin type I and type II are generally good candidates for chemical peels. Patients with Fitzpatrick skin type III and greater are at an increased risk for postprocedure pigmentary complications.

FITZPATRICK SUN-REACTIVE SKIN TYPES I TO VI

Type	Color	Tanning response
Type I	White	Always burns, never tans
Type II	White	Usually burns, tans less than average
Type III	White	Sometimes burns mildly, tans about average
Type IV	Brown	Rarely burns, tans more than average and with ease
Type V	Dark brown	Very rarely burns, tans very easily

Type VI	Black	Never burns, tans very easily
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In the Glogau classification, patients in category I are often young with minimal photoaging and are best managed with a superficial peel in conjunction with a good medical skin care program. Patients in category II or III are candidates for medium-depth peels in addition to long-term medical therapy as with retinoids or alpha-hydroxy acids. Category IV photoaging patients are best treated with medium or deep chemical peels, ablative lasers, or dermabrasion, in conjunction with long-term medical skin care regimens.

Glogau classification of photoaging groups

Group	Classification	Typical age (y)	Skin characteristics
I	Mild	28–35	Little wrinkling or scarring and no keratoses
II	Moderate	35–50	Early wrinkling, mild scarring, and sallow color with early actinic keratoses
III	Advanced	50–65	Persistent wrinkling, discoloration with telangiectasias and actinic keratoses
IV	Severe	60–75	Wrinkling—superficial to deep actinic keratoses ± skin cancer

The indications for facial resurfacing are divided into aesthetic indications and therapeutic indications. The aesthetic indications include 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 wrinkles skin and after blepharoplasty or face-lift. The therapeutic indications include multiple actinic, seborrheic, and solar pigmented keratoses, Superficial basal cell carcinomas, lentigo maligna lentiginis and Melasma (discoloration of skin caused by pregnancy).

CONTRAINDICATIONS

Telangiectasias are relative contraindications in that they become more apparent after chemical peels. Confirmed malignant lesions should not be treated with chemical peels 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 peels. Contraindications include the presence of hepatorenal disease or cardiac disease (for phenol peels), unless approved by an appropriate specialist. Patients who are unstable psychologically should not be treated with any resurfacing modality, particularly because the postoperative care may require intense patient involvement, education, and understanding (Cummings 2005).

Relative Contraindications

- Darker skin type (Fitzpatrick IV, V, and VI)
- Keloid formation by history
- History of herpes infections
- Cardiac abnormalities
- A history of diabetes mellitus or previous facial irradiation
- Marked quantity of vellus hair present
- Unrealistic patient expectations
- Physical inability to perform quality postoperative care

- Telangiectasias
- Anticipation of inadequate photo protection because of job, vocation, or recreation

Absolute Contraindications

- Significant hepatorenal disease
- HIV-positive patient
- Significant immunosuppression (i.e., hypogammaglobulinemia)
- Emotional instability or mental illness
- Ehlers-Danlos syndrome
- Scleroderma or collagen vascular diseases
- Recent isotretinoin (Accutane) treatment (within 6–12 months before)

Patients who are unwilling to wear makeup to cover postoperative hypopigmentation are not good candidates for this procedure. Those patients who are unwilling to decrease their sun exposure are likewise poor candidates for chemical peel, because the procedure does in fact reduce their melanin protection.

If a phenol chemical peel is to be used, special attention must be given to cardiac, liver, and kidney function in the preoperative medical workup. Any impairment of liver or kidney function could slow the excretion of phenol, potentially increasing the bloodstream concentration and leading to cardiac irregularities or even death.

Patient Preparation

Positive history of herpetic outbreaks should warrant appropriate prophylaxis. In patients with no preceding history of fever blisters who are undergoing a medium or deep peel, Valtrex is an appropriate prophylaxis at 500 mg PO bid starting the day before the peel and continuing through the 10th to the 14th day. For patients with a known history of herpetic outbreaks, we have found that prophylaxis with acyclovir at 800 mg PO bid over the same period, until re-epithelialization is complete, is more effective at preventing outbreaks.

Before undergoing any ablative resurfacing procedure, the patient's skin needs to be prepared properly. Vitamin A derivative therapy (a retinoid or tazarotene) are given for at least 4 weeks before the procedure. These product speeds epidermal healing and enhances the effects of the procedure and increases the depth of a chemical peel by decreasing the thickness of the stratum corneum. Patients are warned to avoid unprotected sun exposure for at least 2 months before and after the procedure.

Superficial Peels

Destruction of the epidermis defines a full superficial chemical peel inducing the regeneration of the epidermis. Superficial peels with TCA (10% to 25%) and many other agents improve pigmentary irregularities and may improve some minor surface changes and impart a fresher appearance to facial skin.

Superficial peels usually cause mild erythema and desquamation, with a healing time varying from 1 to 4 days, depending on the strength of the chemical agent. These agents include low concentrations of glycolic acid; 10% to 20% TCA; Jessner's solution (resorcinol, 14 g;

salicylic acid, 14 g; lactic acid, 14 mL; ethanol, 100 mL); tretinoin; 5-fluorouracil (5-FU); and salicylic acid, a beta-hydroxy acid. Glycolic acid (50% to 70%) produces superficial peels that remove actinic keratoses, fine wrinkles, lentigines, melasma, and seborrheic keratoses. As with other peels, the depth of penetration can be titrated by the timed duration of the application of the acid. Peels are left on the skin for 3 to 7 minutes and can be repeated 3 to 4 times. During application, there may be a mild stinging followed by a level I frosting, defined as the appearance of erythema and streaky whitening on the surface. Regular washing with a mild cleanser and the use of routine moisturizers and sunscreens is all that is needed after the procedure. Glycolic acid can be used to peel skin of all skin types with minimal risk.

Medium Peel

Medium-depth peels offer results that even repeated superficial peels cannot match. Necrosis of the epidermis and induction of inflammation within the papillary dermis constitutes a medium-depth peel. It is most useful for the removal of epidermal or superficial lesions and the improvement of skin texture in moderate photodamaged skin (grade II Glogau photoaging skin). The procedure is performed to remove actinic keratoses, repair mild photoaging of the skin including rhytides, treat pigmentary dyschromias, and improve depressed scars.

Trichloroacetic acid (TCA) has been the gold standard in quantitating chemical peel strength and depth. TCA is naturally found in crystalline form and is mixed weight-by-volume with distilled water. It is not light sensitive, does not need refrigeration, and is stable on the shelf for more than 6 months. In the past concentrations of TCA approaching 50% or higher were used to achieve injury to the superficial dermis. This concentration of TCA, though, has been found unreliable and associated with a higher incidence of pigmentary dyschromia, textural change, and even scarring.

In an attempt to reduce the morbidity of higher-concentration TCA, a combination of products has been devised that improves the absorption of the lower concentration of trichloroacetic acid without the associated complications. The most common agents include a combination of 35% TCA with Jessner's solution, 70% glycolic acid, or carbon dioxide (CO₂) laser. Phenol 88% by itself will give a medium-depth peel.

Brody first developed the use of solid CO₂ applied with acetone to the skin as a freezing technique before the application of 35% TCA. The preliminary freezing appears to break the epidermal barrier for a more even and complete penetration of the 35% TCA. Monheit then demonstrated the use of Jessner's solution before the application of 35% TCA. The Jessner's solution was found effective in destroying the epidermal barrier by breaking up individual epidermal cells. This also allows a deeper penetration of the 35% TCA and a more even application of the peeling solution. Similarly, Coleman has demonstrated the use of 70% glycolic acid before the application of 35% TCA. Its effect has been very similar to that of Jessner's solution.

The procedure is usually performed with mild preoperative sedation and nonsteroidal anti-inflammatory agents. The patient is told that the peeling agent will sting and burn temporarily, and aspirin is given before the peel and continued through the first 24 hours if the patient can tolerate the medication. Its inflammatory effect is especially helpful in reducing

swelling and relieving pain. For full-face peels, though, it is useful to give preoperative sedation (diazepam 5–10 mg orally) and mild analgesia, meperidine 25–50 mg and hydroxyzine hydrochloride 25 mg intramuscularly. The discomfort from this peel is not long lasting, so short-acting sedatives and analgesics are all that are necessary. A fan to cool the patient is also helpful.

Vigorous cleaning and degreasing are necessary for even penetration of the solution. The face is scrubbed gently with Septisol soaked gauze pads and water, then rinsed and dried. Next, an acetone preparation is applied to remove residual oils and debris. The skin is essentially debrided of stratum corneum and excessive scale. A thorough degreasing is necessary for an even penetrant peel. The physician should feel the dry, clean skin to check the thoroughness of degreasing. If oil is felt, degreasing should be repeated. A splotchy peel is usually the result of uneven penetration of peel solution because of residual oil or stratum corneum and a result of inadequate degreasing.

Jessner's solution is applied with a painless white fine frosting resulting. On application, a faint frosting will appear within 1 minute within a background of mild erythema. Even strokes are used to apply the solution to the unit area, covering the forehead to the cheeks to the nose and chin. The eyelids are treated last, creating the same erythema with blotchy frosting. After the Jessner's solution has dried, the TCA is applied. The TCA is painted evenly with one to four cotton-tipped applicators that can be applied over different areas with light or heavier doses of the acid. The perioral area and eyelids are treated, coming within 1 to 2 mm of the lower eyelid margin. An assistant should always be on standby with sterile eye wash for irrigation in case the surgeon inadvertently spills any peel solution into the eye. It is important to note that the amount of TCA delivered to the skin surface is dependent on the number of applications, the degree of saturation, the amount of pressure applied to the skin, and contact time with the peel solution. The cotton-tipped applicator is useful in quantitating the amount of peel solution to be applied. The white frost from the TCA application appears complete on the treated area within 30 seconds to 2 minutes. Even application should eliminate the need to go over areas a second or a third time, but if frosting is incomplete or uneven, the solution should be reapplied. Before re-treating an area, however, one should wait at least 3 to 4 minutes to ensure that the frosting has reached its peak before determining for asymmetry.

The physician should achieve a level II to level III frosting. Level I frosting is erythema with a stringy or blotchy frosting, seen with light chemical peels. Level II frosting is defined as white-coated frosting with erythema showing through. A level III frosting, which is associated with penetration through the papillary dermis, is a solid white enamel frosting with little or no background of erythema. A deeper level III frosting should be restricted only to areas of heavy actinic damage and thicker skin. Sensitive areas such as thin eyelid skin and bony prominences (which have a high propensity for scarring) should be limited to a level II frosting. A solid white enamel frosting without an erythematous background indicates injury to the reticular dermis and is too deep for a medium peel.

Anatomic areas of the face are peeled sequentially from forehead to temple to cheeks and finally to the lips and eyelids. The white frosting indicates keratocoagulation or protein denaturation of keratin, and at that point the reaction is complete.

Eyelid skin must be treated delicately and carefully. A semidry applicator should be used to carry the solution to the level of the superior aspect of the tarsus on the upper lid and to 2-3 mm from the lash line on the lower lid. The patient should be positioned with the head elevated at 30 degrees and the eyelids closed. Excess peel solution on the cotton tip should be drained gently on the bottom before application.

An immediate burning sensation is felt with the application of the TCA peel, but this begins to dissipate with the onset of frosting and is typically resolved by the time of discharge. Cool saline compresses are placed over the face for 5 to 6 minutes after the peel until the patient is comfortable. On completion of the peel, a brawny, dusky erythema will progress over the first 12 hours. For the first 24 hours, the patient is instructed to soak four times a day with a 0.25% acetic acid compress made of 1 tablespoon white vinegar in 1 pint of warm water. A bland emollient is applied to the desquamating areas after soaks. After 24 hours, the patient can shower and clean gently with a mild nondetergent cleanser. Mild to moderate edema soon follows and can be severe over the thin eyelid skin and forehead regions. As the edema begins to resolve, dark crusts appear that peel off during the subsequent 5 to 7 days to reveal a new, erythematous epithelium. The redness will soon fade to a pink color that resembles a sunburn and can typically be camouflaged with makeup by the 10th day after the peel. The patient can begin using sunscreens as tolerated. He or she should wait at least 3 months before resuming regular aesthetic skin care services such as superficial chemical peels or microdermabrasion. Cleansing facials can begin as early as 4 to 6 weeks after the peel. Repeat medium-depth chemical peel should not be performed for at least 1 year. Several studies have demonstrated microscopic improvement of collagen thickness progressing over a 6- to 13-month period.

Deep Chemical Peel

The more advanced changes seen with deeper grooves and wrinkles, pebbly appearance of the skin, and more pronounced gravitational changes of Glogau III and IV photoaging skin require either deep chemical peeling or laser resurfacing. Deep peels are usually performed using the Baker-Gordon solution. This preparation includes phenol, water, Septisol, and croton oil. Septisol acts as a surfactant which results in more even penetration. Croton oil is a vesicant epidermolytic agent that enhances the absorption of phenol (phenol applied alone results in only a medium-depth injury). This depth can also be achieved with a 50% or greater TCA peel; however, the high risk of scarring and pigmentation problems have resulted in a trend away from these concentrations.

Phenol itself at concentrations greater than 80%, carbolic acid is a keratocoagulant precipitating the surface protein, thus preventing further penetration of the peel solution. Phenol produces an extremely rapid denaturization and irreversible coagulation. Further penetration of the phenol is prevented when the keratin protein binds to the phenol, creating large molecules that cannot penetrate further. Concentrations of phenol less than 50%, it becomes keratolytic, interrupting sulfur bridges in the keratin layer, and can then produce deeper penetration and more destruction than desired. Therefore, as one decreases the concentration of phenol, the depth and therefore wounding of tissue becomes more severe. Occlusion of the peeling solution with tape increases its penetration, creating injury to the mid-reticular dermis. The unoccluded technique involves more cleansing of the skin and the application of more peel solution.

Preoperative antibiotics such as cephalexin (Keflex) (500 mg bid) are started 24 hours before the procedure and continued for 1 week. Patients are offered a sedative to help them sleep the night before the procedure. Patients are given 5 mg of diazepam (Valium) PO 1 to 2 hours before the peel. The face is washed twice with Septisol and rinsed thoroughly after each washing. The patient is given a preoperative sedative (diazepam), anti-nausea medication (promethazine), and a prokinetic agent (metoclopramide; e.g., Reglan). An intravenous line is introduced and approximately 500 to 1000 mL of Ringer's lactate solution is administered. The patient is given a narcotic medication to offset the burning sensation, typically 1 or 2 mg of Dilaudid. Additional midazolam (Versed), usually 1 to 2 mg, is given for sedation and as an amnestic agent at this time. Sensory nerve blocks are then administered with injections of bupivacaine (Marcaine) 0.05% with 1:200,000 epinephrine solution. The regional blocks include the supraorbital, infraorbital, incisive foramen, and mental nerves, as well as infiltration of the lower eyelids and preauricular area. This spares the patient the typical 4 to 6 hours of postoperative burning discomfort. An additional liter of saline is given over the course of the procedure. (Cummings 2006)

Facial subunits are addressed one at a time with 15-minute time interval between units. This is done to avoid buildup of phenol to toxic levels in the blood. One needs to obtain a white frost 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, and most surgeons do not breach this boundary.

Erythema may take months to resolve. Pigmentary changes and scarring are also more frequently seen with deep peels. The skin continues to improve over several months as collagen remodeling takes place. A remarkable degree of improvement is to be expected.

Phenol is rapidly absorbed into the circulation and may cause cardiac arrhythmias. Phenol, the active agent, is known to have cardiac toxicity and has hepatic and renal elimination. These effects necessitate a more in-depth workup and usually include a monitored setting for the application. An anesthetist or anesthesiologist is required to administer sedation and analgesia while monitoring the patient's cardiac status, pulse-oximetry, and blood pressure. Any patient who has a history of cardiac arrhythmias or is taking medications that are potentially arrhythmia precipitating may not be a good candidate for Baker's phenol peeling. Additionally, patients with poor renal or hepatic function are poor candidates. To prevent toxicity, volume loading with intravenous fluids before, during, and after phenol peeling. Botta recommend maintaining a fluid load to force diuresis with 20 mg of furosemide given 10 minutes before the application of phenol. Waiting as much as 20 to 30 minutes between treatment of each area and not peeling more than 50% of the face at one time minimizes the risk of phenol toxicity in most patients.

Postoperative Care

The patient is given 10 mg of dexamethasone IV intraoperatively and methylprednisolone postoperatively to reduce swelling. The patient is asked to return to the office on the third postoperative day to assure the physician that the wound is being cleaned as instructed. The patient is then again evaluated in 3 to 4 days to observe the amount of wound healing and

residual crusting. After 7 to 10 days, the patient can begin to apply makeup if epithelialization is complete. The use of sunscreens and sun avoidance is critically important. Sunscreen with an SPF of 30 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 4 weeks 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. The patient returns for an office visit at 2 weeks and again 6 weeks later for evaluation of the early development of splotchy hyperpigmentation. If splotchy pigmentation develops, a combination of Retin A, hydroquinone (Eldoquin Forte), and triamcinolone (Aristocort) may provide an improvement. The patient is seen again at the 3-month, 6-month, and 1-year period postoperatively.

Complications

Pigmentation changes are by far the most common sequela seen with chemical peels. Pigmentary changes include hyperpigmentation, hypopigmentation and depigmentation. The most common is Hypopigmentation. Persistence of rhytids, prolonged erythema, persistent texture change of skin, hypertrophic subepidermal healing, milia, skin pore prominence, increased prominence of telangiectasias, and darkening and growth of preexisting nevi are other well known sequela of Chemical peel.

Complications of chemical peels include skin infection, lower eyelid ectropion, cardiac arrhythmias, renal failure, toxic shock syndrome, and facial scarring. Infections are mainly caused by Herpes simplex virus, *Pseudomonas*, *Staphylococcus*/*Streptococcus* and *Candida* organisms. Infections are uncommon, but herpetic breakouts can almost be expected if appropriate antiviral prophylaxis is not given. Superficial infection with *Pseudomonas*, *Staphylococcus*, or *Streptococcus* species is rare and can usually be attributed to poor postoperative wound care. *Candida* infections can occur, which will delay epithelialization. These should be treated with topical nystatin cream. Prolonged ointments after chemical peel may promote folliculitis and acne, especially in patients with a prior history. These conditions may become secondarily infected with *Staphylococcus* or *Streptococcus* species and should be treated with the appropriate oral antibiotic in addition to topical clindamycin.

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