The Management of Postacne Scarring

GREG J. GOODMAN, FACD,* AND JENNIFER A. BARON, MD†

BACKGROUND Therapeutic intervention for postacne scarring has historically been limited by the considerable morbidity of most treatments for only marginal disease improvement. Within the past decade, however, a greater understanding of the pathogenesis of acne scarring has led to the development of techniques that offer more favorable risk–benefit profiles.

OBJECTIVE The aims of this article are to highlight a number of newer techniques and to assign their appropriateness to particular grades of acne scarring.

MATERIALS AND METHODS Current modalities are discussed as they relate to disease process and specific acne scar types. Techniques are presented in order of most effectual therapeutic interventions for defined grades of acne scarring. Acne scarring grades have been described previously in terms of disease load, severity, and lesion morphologies.

RESULTS A comprehensive discussion of updated therapeutic techniques and their biologic rationales in the treatment of acne scarring is presented. These include targeted interventions of inflammatory and postinflammatory processes, angiogenesis, immunologic processes, dermal and subcutaneous fibrosis, hypertrophy, and keloid scarring.

DISCUSSION A requirement for developing successful treatments for postacne scarring is a greater understanding of its pathogenesis, variability among afflicted individuals, and the inflammatory mediators and immunology of the scarring process. Many innovative techniques introduced in the past decade attempt to counteract these pathologic processes while keeping the procedural and postoperative risks to a minimum.

Greg J. Goodman, FACD, and Jennifer A. Baron, MD, have indicated no significant interest with commercial supporters.

Postacne scarring may be a physically disabling and psychologically devastating disease. Unfortunately it is not particularly well treated. The more morbid resurfacing procedures are becoming less popular and are being challenged by new and innovative procedures that require less recuperation. New techniques have been added and older ones modified in attempts to improve risk benefit profiles. One should assess both the overall appearance and the morphology of each scar and treatment designed accordingly.

Various attempts have been made to describe postacne scars morphologically describing scar type and suggesting therapy appropriate for that scar type.[1–3] We will propose a new qualitative grading system (Tables 1 and 2) as a template for describing some of these new techniques.[4] This system attempts to classify patients according to the severity of their scarring and thus their burden of disease.

Macular Acne Scarring and Marking (Grade 1 Acne Scarring)

The first grade of scarring is macular changes visible irrespective of distance and represents not a problem of contour like other scar grades but of color. This color may be red, white, or various shades of brown to black.

**Skin and Cancer Foundation of Victoria and Monash University Department of Community Medicine, Victoria, Australia; †Oregon Health & Science University, Portland, Oregon*

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TABLE 1. Grades of Postacne Scarring

Grade	Level of disease	Characteristics
1	Macular	Erythematous, hyper- or hypopigmented flat marks visible to patient or observer at any distance.
2	Mild	Mild atrophy or hypertrophy that may not be obvious at social distances of 50 cm or greater and may be covered adequately by makeup or the normal shadow of shaved beard hair in men or normal body hair if extrafacial.
3	Moderate	Moderate atrophic or hypertrophic scarring that is obvious at social distances of 50 cm or greater and is not covered easily by makeup or the normal shadow of shaved beard hair in men or body hair if extrafacial, but is still able to be flattened by manual stretching of the skin (if atrophic).
4	Severe	Severe atrophic or hypertrophic scarring that is obvious at social distances greater than 50 cm and is not covered easily by makeup or the normal shadow of shaved beard hair in men or body hair if extrafacial and is not able to be flattened by manual stretching of the skin.

Erythematous Macules

If only the epidermis and superficial dermis are involved, the scars may appear as macules that may be red if inflamed and comparatively early or young scars (under 1 year) or with altered pigmentation (Figures 1 and 2).

Hyperpigmented Macules

Pigmentation of scars may be increased in more olive-skinned patients and represents mostly a post-inflammatory response that will fade in 3 to 18 months (Figure 2). This requires strict sun protection in patients who do not readily burn and who may not perceive sun avoidance as usually necessary.

Hypopigmented Macules

The white macules seen in the postacne environment usually represent true scars or postinflammatory leukoderma (Figure 3).

Mild Atrophy or Hypertrophy (Grade 2 Acne Scarring)

This grade of scarring includes those scars that may not be obvious at social distances of 50 cm or greater and may be covered adequately by makeup or the normal shadow of shaved beard hair in men or normal body hair if extrafacial (Figure 4). This equates to a superficial type of atrophic scar or rolling scar. This is the group probably most at risk from the practitioner who suffers from the adage "If the only tool you have is a hammer, you tend to see every problem as a nail" (Abraham Maslow, American philosopher and psychologist, 1908–1970).[5] In other words, this group is in danger of both exaggerating their problem and being overtreated by physicians with traditional resurfacing procedures, especially if the treating physician is not skilled with a wide armamentarium of procedures.

TABLE 2. Global Acne Scarring Classification: Types of Scars Making Up the Classification Grades

Grade	Level of disease	Examples of scars
1	Macular	Erythematous, hyper- or hypopigmented flat marks
2	Mild	Mild rolling, small soft papular
3	Moderate	More significant rolling, shallow boxcar, mild to moderate hypertrophic or papular scars
4	Severe	Punched out atrophic (deep boxcar), ice pick, bridges and tunnels, marked atrophy, dystrophic significant hypertrophy or keloid

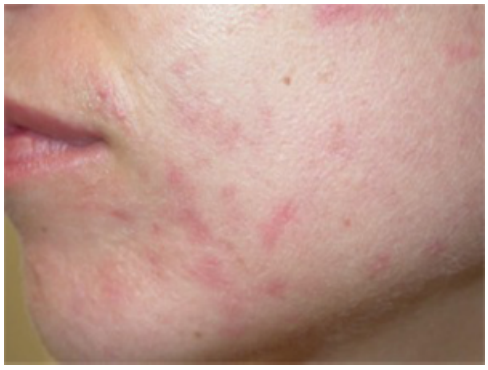

Figure 1. A patient whose acne is coming under control with erythematous healing lesions but whose lesions may go on to scar.

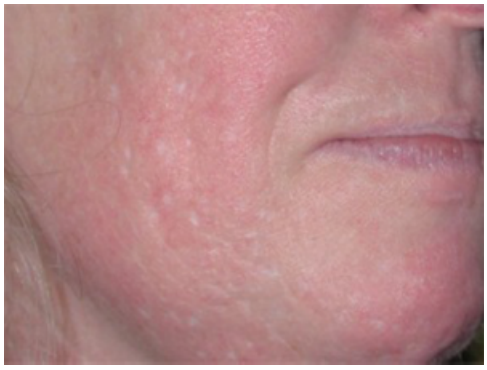

Figure 3. Hypopigmented macular scarring.

Moderate Atrophy or Hypertrophy (Grade 3 Acne Scarring)

This level of scarring is obvious at social distances of 50 cm or greater and is not covered easily by makeup or the normal shadow of shaved beard hair in men or body hair if extrafacial, but is still able to be flattened by manual stretching of the skin (Table 1). It equates to the rolling and shallow boxcar atrophic type scars and the moderate hypertrophic and keloidal scars (Table 2).

Severe Atrophy or Hypertrophy (Grade 4 Acne Scarring)

This is represented by severe atrophic or hypertrophic scarring that is obvious at social distances of 50 cm or greater and is not covered easily by makeup or the normal shadow of shaved beard hair in men or body hair if extrafacial and is not able to be flattened by manual stretching of the skin (Table 1).

Scar Types

This comprises punched out atrophic (deep "boxcar"), "ice-pick" scars, bridges and tunnels, marked atrophy, dystrophic scars, and significant hypertrophy or keloid (Table 2). The treatments for this group arguably have advanced faster than those for the less severe end of the spectrum of disease. There have been reports of success in this type of scarring with resurfacing techniques[6,7] only but generally the mainstay of treatment for punched out scars has been punch techniques as was first suggested almost 20 years ago with or without resurfacing.

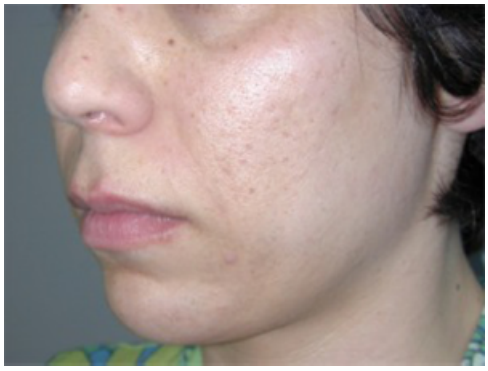

Figure 2. Hyperpigmented acne scars.

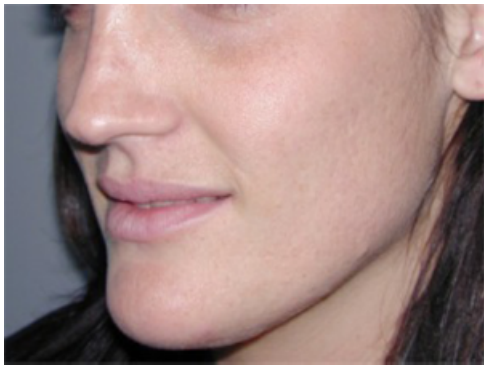

Figure 4. Grade 2 acne scarring.

TABLE 3. Global Acne Scarring Classification and Likely Treatment Options

Grade	*Level of disease*	*Likely treatment options*
1	Macular	Time, optimized home skin care, light strength peels, microdermabrasion, vascular or pigmented lasers, or intense pulsed light.
2	Mild	Nonablative lasers, blood transfer, skin needling or rolling, microdermabrasion, dermal fillers.
3	Moderate	Ablative lasers, dermabrasion, medical skin rolling, fractionated resurfacing, dermal fillers if focal, subcision and blood transfer. Intralesional corticosteroids steroids or fluorouracil and/or vascular laser if hypertrophic.
4	Severe	Punch techniques (float, excision grafting), focal trichloroacetic acid (CROSS technique) with or without resurfacing techniques (including fractionated resurfacing). Fat transfer, occasionally rhytidectomy if grossly atrophic. Intralesional corticosteroids steroids or fluorouracil and/or vascular laser if hypertrophic.

Therapy of Scars (Table 3)

Macular Acne Scarring and Marking

Erythematous Macules Red macular changes are quite well targeted by vascular lasers and light sources.[8] Theoretically there may be an important role for lasers and light sources in the prevention of progression to scarring of inflamed healing acne lesions. This modality has also been used for moderately atrophic scars[9] as well as hypertrophic scarring.[10,11] Acne excoriée with erythematous scars has been treated with vascular laser and psychotherapy with success.[12]

Hyperpigmented Macules Reparative treatment may not always be required. If treatment is sought, medical therapy may suffice with topical reparative creams such as retinoic acid, hydroquinone, kojic acid, and azelaic acid used in other examples of postinflammatory pigmentation.[13,14] Alternatively or additionally, light skin peels with glycolic acids or Jessner's solution or variants or retinoic acid peels[15,16] may be utilized, although their efficacy in the treatment of postinflammatory acne marking is not established. Occasionally pigmented lesion lasers[17] or light sources may be useful for resistant cases, although there is a risk of inducing postinflammatory hyperpigmentation with these devices.[18]

Hypopigmented Macules Traditionally these lesions have been reasonably refractory to treatment. There have been scattered reports of repigmentation after manual dermabrasion[19] and needle dermabrasion[20] (utilizing a tattoo gun without pigment). Some pigment transfer procedures have been attempted. A particularly difficult type of hypopigmented macular scarring is termed perifollicular scarring. Perifollicular acne inflammation may produce small hypopigmented macular or papular scars from destruction and attenuation of collagen and elastin fibers in the surrounding tissues around the hair follicles. This is most common on the trunk[21] and is largely untreatable at the present time. If papular rather than macular and particularly if facial, they may be treated by fine needle diathermy.[2] There are a number of techniques utilized for treatment of vitiligo that may be useful in the treatment of postacne scarring. Minigrafting holds some promise,[22,23] but there appears to be little spread of pigment from the grafts into surrounding skin in patients with scarring. Epidermal suspensions both cultured and immediate noncultured may also be somewhat useful.[24] Recently in Australia, an automated commercial kit for trypsin dermal epidermal separation has become available, allowing immediately available autologous noncultured epidermal suspension (ReCell, Clinical Cell Culture Americas, Coral Springs, FL). This may improve the ease of the technique considerably over current methods[25,26] (Figure 5).

Mild Atrophy or Hypertrophy (Grade 2 Acne Scarring)

This group may have only few scars and benefit from fairly simple treatments or many scars and need more substantial treatment.

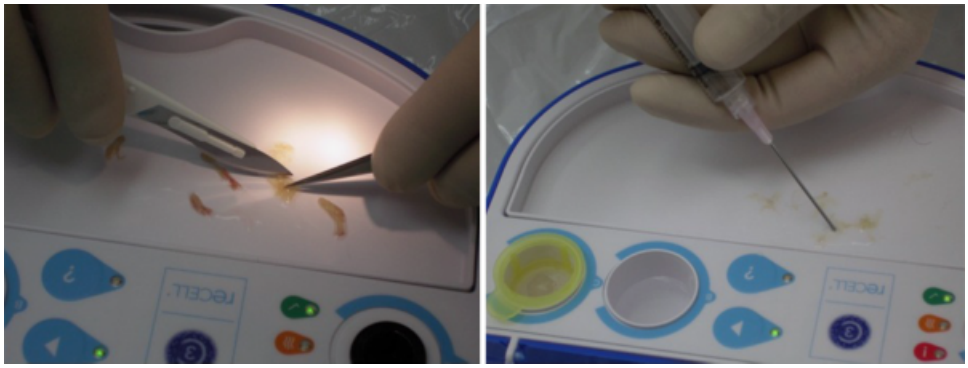

Figure 5. Trypsin dermal epidermal separation kit (ReCell, Clinical Cell Culture Ltd.).

Patients with Few Scars

Tissue Augmentation For the group with few scars, the past decade has seen the advent of a bewildering array of injectable fillers including human collagen, polylactic acid. and hyaluronic acid among the short-term agents and many agents of a longer term nature with the reintroduction of silicon and variations of polyacrylamides for longer correction. There are also the solid agents such as some of the cadaveric and expanded polytetrafluoroethylene, although they probably have limited relevance for this type of scar correction. For those with few scars, simple dermal augmentation with bovine or human collagen (Zyderm and Cosmoderm, Inamed Aesthetics, Santa Barbara, CA) or hyaluronic acid (Restylane, Medicis, Scottsdale, AZ; and Juvederm and Hylaform, Inamed Aesthetics) would be most appropriate. This category of acne-scarred patients deals with those with limited disease and overcorrection should be avoided. Thicker forms of collagen or hyaluronic acid would thus not be appropriate. A word of caution regarding the myriad of permanent agents is probably required here. All dermal augmentation agents will cause problems sometimes or not be perfectly placed, and a temporary problem allowing further refinement may be better for all than a more permanent one. This is certainly true with some of the more recent long-term agents (Figure 6). Agents with less predictable individual session outcomes should also be avoided. Agents such as silicon and polylactic acid requiring a contribution in volume from local immunologic events would fall into this category and not be sufficiently accurate for this purpose in those with mild scarring. Among autologous agents blood transfer is a reasonable option for this group of patients with stimulation of the implanted chromophore by relatively low-level vascular laser or intense pulsed light. Blood is injected immediately after drawing by simple injection with a 1-mL syringe with attached 30-gauge needle high up in the dermis distending the scar giving a bleb with a bruised appearance. This bruise is then targeted as any blood vessel would be, but with approximately 50% to 75% of the usual fluence. This treatment may be repeated at monthly intervals until adequate correction is attained. Excessive fluence may further injure the area and be counterproductive, the idea

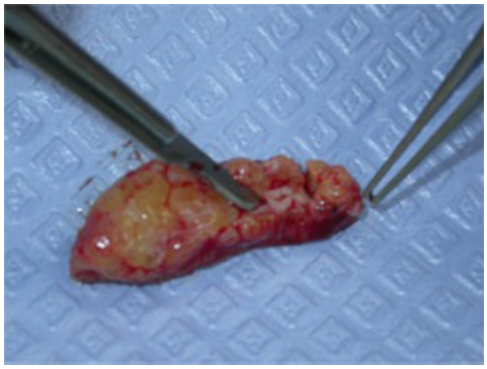

Figure 6. Excision of permanent dermal implant material that had produced granulomas where injected for postacne scarring (Dermalive, Dermatech, Paris, France).

being to induce a low-level heat injury inducing further collagen deposition.[27,28]

Patients with Many Scars

Microdermabrasion. Microdermabrasion utilizing aluminum oxide crystals has become a very popular technique and has been suggested to be useful in the treatment of facial scarring.[29] In this treatment small crystals of aluminum oxide are expelled from one nozzle toward the skin abrading it by a series of small lacerations, and then used crystals are aspirated back from the skin surface and discarded. Multiple treatments are required and efficacy for the treatment of acne scarring remains uncertain.

Skin Needling. Skin needling or skin rolling, also termed collagen induction therapy, in its simplest form employs a 30-gauge needle introduced into the skin to a controlled depth with the aid of an ADG needle or gripping the needle with a small artery forcep at approximately 2 to 3 mm and stabbing the skin repeatedly, but this is only appropriate for small areas of scarring. For larger areas, a tattoo gun without pigment may be used[20] or therapy delivered via a needle-studded rolling pin[30] that is rolled over the face or extrafacially. With this technique the rolling is usually continued until some bruising is noted. These methods induce a nonspecific trauma that is both epidermal and dermal. The epidermal trauma heals rapidly by transepidermal migration but the dermal trauma heals with collagen remodeling because that is the final wound healing mechanism no matter what the insult may be. The treatment may be used extrafacially and is especially useful for treating the neck when the face is also treated. Most body areas are accessible to this technique. There are some areas that are not treatable with this technique, however, such as the nose and the periorbital zones. This treatment appears to be repeatable and, probably more pertinent, appears to be synergistic with other methods such as nonablative lasers, blood transfer, and vascular lasers (Figure 7). It is the author's practice to always supplement the procedure of skin rolling or needling with other procedures, both simultaneously (blood transfer, vascular laser, and subcision for bigger scars) and sequentially starting 1 month after the procedure and continuing monthly for three treatments (nonablative 1,450-nm diode laser). A recently introduced laser employing the concept of "fractionated photothermolysis" produces small vertical zones of full-thickness thermal damage by a midinfrared laser.[31] This is akin to sinking posts or drilling holes of thermal damage with areas surrounding these posts left free of damage. This is a method of ablative resurfacing without the patient having to experience a pronounced healing phase. It has a different set of possibilities to other types of nonablative resurfacing with epidermal disease being also targets for this system. Conceptually it may be the laser equivalent of skin needling and would be expected to have a significant role in the treatment of scars.

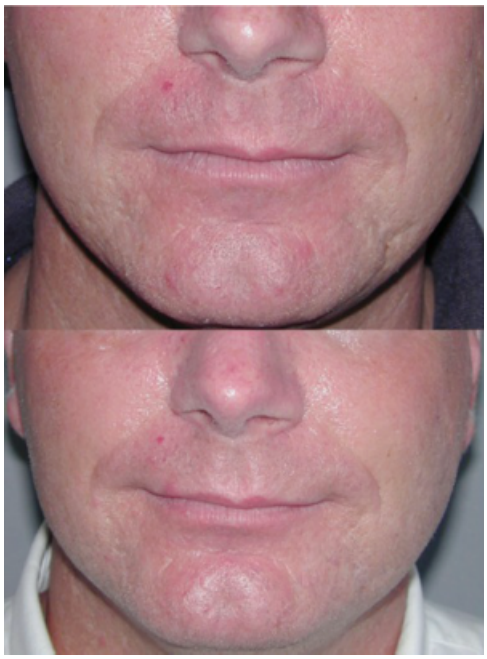

Figure 7. The combined use of skin rolling and blood transfer in the treatment of localized scarring of the chin.

Nonablative Lasers. Nonablative lasers appear to have a role in this type of scarring. The major lasers for this purpose have been the midinfrared lasers at wavelengths of 1,320, 1,450, and 1,540 nm appropriately cooled to protect the epidermis whilst targeting dermal water.[32–36] These lasers used

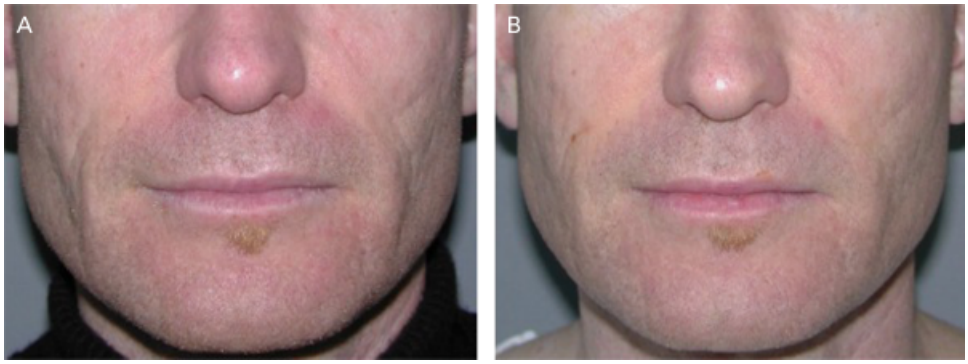

Figure 8. The combined use of skin rolling and nonablative resurfacing laser (1,450-nm wavelength).

conducted heat from the chromophore to produce a diffuse dermal injury heating to above 50°C. Repeated treatments are required and longevity of result is still largely unknown. The concept would be that dermal insults are reasonably consistent in their healing pattern no matter what the wounding agent, with collagen remodeling occurring late in the healing phase after appropriate coagulation/inflammation, reepithelialization, granulation tissue formation, and angiogenesis phases. The more significant the dermal insult the more eventual collagen deposition and improvement one seems to note.[37] This has up to now come with the issue of increasing morbidity and risk, however. With the advent of nonablative resurfacing and fractionated photothermolysis, maybe this nexus of significant results and significant risk can be broken. This would be particularly useful in a patient with milder disease who would be particularly suited to techniques with a suitably higher benefit to risk ratio. Occasionally, subcision techniques may be added to this subgroup in conjunction with nonablative lasers or skin needling for slightly more severe scarring. Small papular scars that may appear on the nose and chin respond well to fine wire diathermy, not a new technique but recently described for this subgroup.[2]

Moderate Atrophy or Hypertrophy (Grade 3 Disease)

Those with Few Scars

If there are few scars then their augmentation by temporary or longer-term autologous or external agents may be appropriate. Combinations of techniques such as subcision, blood transfer, nonablative or vascular laser, and skin needling may be useful for more significant scarring (Figure 8).

Subcision. Subcision of scars appears to work by breaking up the attachments of these contour abnormalities and releasing the surface from the deeper structures. Blood accumulates under the defect, and its subsequent organization is thought to result in connective tissue formation. This leads to long-term correction of the defect, although this is usually not complete after the first treatment. Successive treatments appear to produce further improvement. The technique of undermining scars has been widely practiced over many years as an adjunct to fibrin foam[38] or animal-based collagen implantation (Fibrel),[39–41] dermal grafting,[42–44] and microlipoinjection. As a stand-alone corrective technique, it was described just 10 years ago.[45,46] The technique involves the insertion of a probe that may be a sharp hypodermic needle (18–26 gauge depending on scar size and depth), a filter needle (Nokor, Becton Dickinson, Franklin Lakes, NJ) or even a blunt cannula. The depth of the probe insertion depends on the type of scar being treated with intradermal insertion for small superficial scars whereas deeper dermal undermining is performed for more severely bound down scars. Larger scars are often undermined at the deeper subdermal level. The initial actions are a backward and forward motion much like the tunneling of a liposuction procedure. After this tunneling is performed sufficiently to almost free the

attachments, the instrument is passed sideways in a sweeping action to complete the freeing up of the skin from its base. The depression will be seen to visibly lift and the procedure is complete. Some bleeding appears to be useful to establish a short-term spacer to keep the tissues from early reattachment but the later organization of the ecchymosis may be responsible for the laying down of new host collagen. Predictable sequelae such as bruising and swelling are often present for 1 to 2 weeks. Acneiform cystic lesions may follow disruption of the pilosebaceous apparatus or subcutaneous sinus tracts and settle with very-low-dose intralesional steroid injection with or without antibiotics. A range of responses ranging from partial to excessive is seen. Partial response is usual whereas excess response is seen in 5% to 10% and may require patience because natural resolution usually occurs, albeit slowly, over months. As a simple technique that appears to produce long-term correction of contour defects, it deserves to be a first-line treatment for many isolated moderate atrophic scars.

Those with Many Scars

Technique-sensitive Resurfacing (lasers, radiofrequency, medium-strength peels, plasma and abrasion). The atrophic forms in this group are improved by ablative laser resurfacing if widespread atrophy is seen. Carbon dioxide laser was the laser system first utilized for postacne scarring[47–49] commonly replacing dermabrasion and strong chemical peeling. Recently in a comeback for dermabrasion, various methods of dermasanding have been added, utilizing various carpentry tools such as drywall/plaster sanding screen[50] or moistened silicone carbide sandpaper to manually dermabrade the skin.[51] Newer standardized trichloroacetic acid peeling lotions,[52] augmentation of peeling agents,[53] and the introduction of peeling pastes[54] have been useful for photodamage but have only been found to be variably useful for postacne scarring. These newer adaptations of older techniques still suffer from a prolonged healing phase and morbidity.[55]

Although erbium:YAG laser has been added to the armamentarium, it is uncertain that this laser has added much to the efficacy.[56,57] Modulating the erbium laser has meant that it behaves more like a carbon dioxide laser with arguably a better safety profile.[58] Another popular method has been to combine erbium and CO_2 lasers either simultaneously or sequentially.[59,60] The statements made above regarding the excessive morbidity of resurfacing techniques, however, are almost as true for these newer lasers. It is also true of ablative radiofrequency devices introduced over the past decade.[61–63] The greatest fear for those performing laser resurfacing is the incidence of hypopigmentation[64,65] seen often as a later event, well after initial healing. This has been treated by intensive topical photochemotherapy but remains a difficult issue. Applying trypsin-digested donor epidermal cells as described above immediately after the resurfacing operation may theoretically provide some protection from this complication. This technique may also provide zones of viable epidermal cells that may allow more rapid epithelialization hence decreasing morbidity. This moderate and atrophic subgroup may also benefit from the treatments described in the milder group especially medical skin rolling combined simultaneously with subcision and later by nonablative laser.

Moderate Hypertrophic Disease.
If moderate hypertrophic disease is evident, the treatments that have been described over the past decade have included vascular laser and fluorouracil (and other cytotoxics) injections.

Intralesional Cytotoxic Therapy Traditionally high-strength corticosteroids have been used for the intralesional drugs of choice in the treatment of hypertrophic and keloidal acne scars. There has been recent interest in the intralesional use of the cytotoxics fluorouracil[66–68] and bleomycin,[69,70] however, as treatments of hypertrophic and keloidal scars. Fluorouracil is usually utilized at a concentration of 50 mg/mL and has been mixed 80:20 with low-strength intralesional steroid (Figure 9). It may be

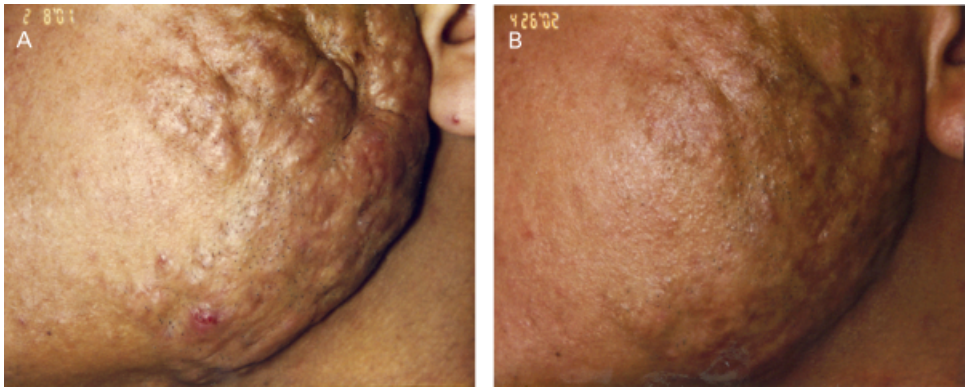

Figure 9. Grade 4 keloidal acne scarring treated with intralesional fluorouracil with superadded steroids. (A) Before fluorouracil injections. (B) After fluorouracil injections.

used alone, however.[71] Usually approximately 1 mL is utilized in each session and often 0.1 to 0.3 mL is all that is required for an individual scar. Recently the molecular basis of the action of fluorouracil has been elucidated.[72] Fluorouracil appears to be a potent inhibitor of TGF-β/SMAD signaling, capable of blocking TGF-β-induced, SMAD-driven upregulation of COL1A2 gene expression in a JNK-dependent manner.

Vascular Lasers In 1995, it was reported that flashlamp-pumped pulsed dye tunable laser was useful in the treatment of keloid sternotomy scars with improvement in scar height, skin texture, erythema, and pruritus in the laser-treated scars.[73] This initial work has been borne out by more recent studies.[74,75]

Other Therapies More recently intralesional verapamil at a concentration of 2.5 mg/mL (0.5–2 mL injected volume depending on the size of the scar)[76] or topical imiquimod[77] have been suggested as postoperative adjunctive treatment to surgical excision of keloidal.

Severe Atrophy or Hypertrophy (Grade 4 Acne Scarring)

Punched-Out Atrophic Scars Punch techniques such as punch excision,[78] grafting,[79] and elevation or float techniques[80] have been useful and probably remain the gold standard for larger punched-out scars (deep boxcar and larger ice-pick scars). More recently, focal trichloroacetic acid,[81] termed the CROSS technique, has excited interest in the treatment of smaller ice-pick and poral-type scars, which have always been difficult. This technique requires the use of high concentrations of trichloroacetic acid (60%–100%) used in multiple sessions until the center of the scar is seen to flatten, basically scarring the inside of the cylindrical scar making it cosmetically more appealing. A similar concept was discussed with the use of high-energy CO_2 laser.[82]

Widespread Grossly Atrophic Disease For grossly atrophic disease with destruction of the deeper tissues, fat remains the optimal replacement agent. Fat is an excellent deeper augmentation material. It is cheap and readily available and will not be rejected nor suffer allergic reactions. It is easy to work with and is without risk of communicable disease. The issue of permanence has gradually been resolved. Fat is probably not the temporary augmentation technique it was first thought[83,84] but correctly implanted it seems to produce accurate, longstanding, autologous correction.[85–88] The concept of replacement of like with like makes fat transfer very attractive. Added to this is the ability to combine this augmentation with most other surface techniques such as resurfacing or with many under-the-surface techniques such as subcision. Fat is injected through a small nick made with a vented needle (Nokor, Becton Dickinson), 11-gauge blade, or similar instrument. When injecting into acne-scarred skin, undermining or subcision[24] is used to break up the

scar tissue and release it from its attachments to deeper tissues. When subcision is employed with fat transfer it may add to the precision of correction. The fat injected will "normalize" the contour except where residual scar attachments impede this. These are then further undermined and released until this normal contour is achieved. Fat is best injected deeply as a three-dimensional lattice of 0.1- to 0.2-mL aliquots and built up to support the more superficial skin layers. The 1-mL tuberculin type syringe allows the finest control of the injection volume. Inevitably some fat will not survive the transfer process. Appropriately performed, more than 50% of transplanted fat would be expected to survive. Most acne-scarred patients, however, benefit from further top-up procedures, probably best timed for 3 months after the procedure. Overcorrection should be kept to no more than 10%. The residual fat may always be frozen and this frozen fat may be used for at least 12 months after the procedure. Aging adds to the problems of the acne-scarred face in a number of ways. Deep acne scarring may produce severe facial fat atrophy. As facial structure (including fat) is lost with age, the acne-scarred areas fare worst. The combination of fat loss selectively with acne scarring and generally with aging often influences patients to seek corrective surgery for longstanding acne scarring in their third, fourth, or even fifth decades. As facial skin starts to sag with aging, it starts to be irregularly suspended on old fibrotic acne scars. This denies the skin its ability to evenly descend, producing an irregular cascading appearance. Fat transfer is able, cosmetically, to reproduce the youthful appearance of a fuller face in acne-scarred patients as well as its reconstructive ability as a deep foundation for deep acne scarring. Balancing the cosmesis of the facial structures becomes necessary if large amounts of facial fat are utilized. Implantation of fat in malar and chin or other convexities may be necessary, if concavity augmentation of cheeks, preauricular areas, and temples are to be kept in balance (Figures 10, 11). Old patient photos should be scrutinized to improve and rejuvenate the patient's appearance rather than change the patient's countenance to something alien or foreign to them that they may find uncomfortable.[89]

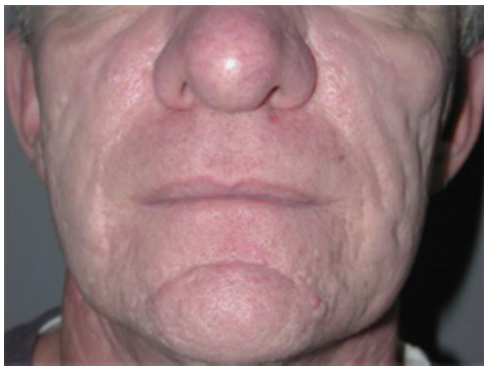

Figure 10. Grade 4 scarring showing fat atrophy amplified by aging changes.

If fat is not available, agents such as polylactic acid and hyaluronic acid may be used to augment substantially depressed acne scarring (Figure 12).

The Future of Acne Scarring Therapy

The question of why one patient is able to heal without scarring while another with apparently

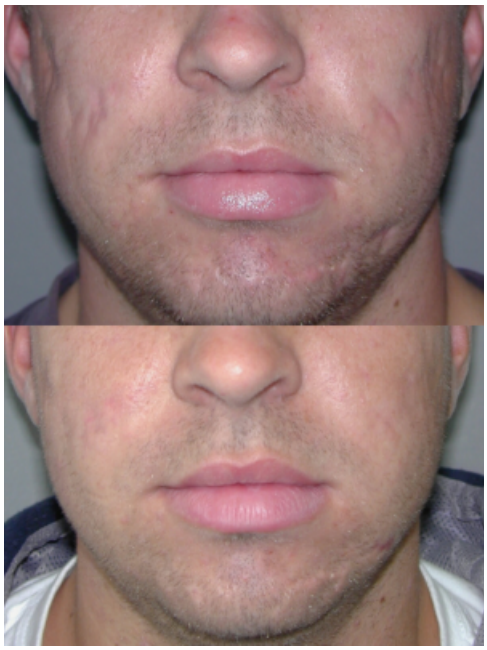

Figure 11. Grade 4 scarring showing fat atrophy treated with fat transfer, subcision, and laser resurfacing.

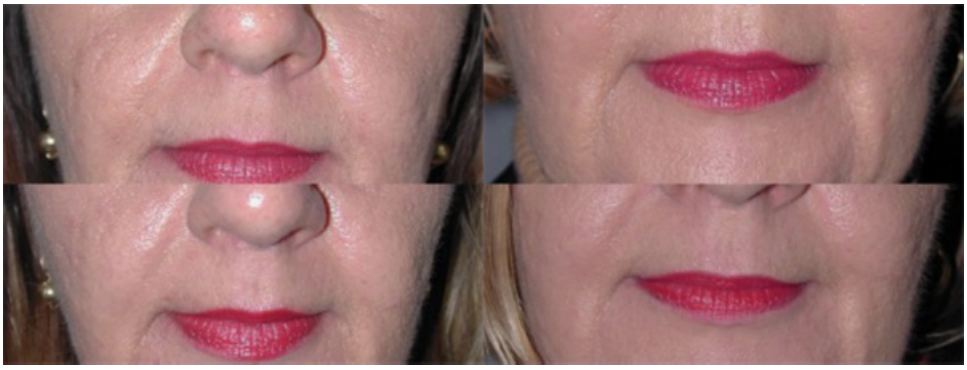

Figure 12. Polylactic acid; before and 6 weeks after a single injection session.

similar severity goes on to scar has always been vexing. Recently, one study examined this by utilizing patients known to be acne scar prone and compared them to those who do not tend to scar. They found that there were noticeable differences in their inflammatory profile while healing.[90] In particular, they found that there was a healthy early, relatively nonspecific, and robust inflammatory infiltrate typical of a Type 4 hypersensitivity response with significant early angiogenesis in patients not prone to scarring, all in keeping with effective and rapid clearing of the offending antigen. In contrast, patients prone to scarring tended to show a relatively more specific but ineffectual early inflammatory response. Angiogenesis remained high in resolving lesions with a further stage of inflammation comprising macrophages and skin-honing memory cells. They suggest that based on the poorly resolving inflammation, scarring would be a more likely outcome and suggest a role for anti-inflammatory medications. If prolonged angiogenesis is seen in those who go on to scar, then how may this relate to the atrophic scarring seen most commonly in the postacne scenario? One scenario could be as follows. For blood vessels to flourish and invade into an injured area, there is a required increase in metalloproteinases to cut a path for this vascular advance. An endothelial cell forming the wall of an existing small blood vessel is activated, makes matrix metalloproteinase (MMP) enzymes that break down the extracellular matrix, invades the matrix, and then begins to proliferate.[91] Angiogenesis in vivo is distinguished by four stages: Stage 1 is defined as an altered proteolytic balance of the endothelial cell allowing it to digest through the surrounding matrix. These committed cells then proliferate (Stage 2) and migrate (Stage 3) to form aligned cords of cells. The final stage is the development of vessel patency (Stage 4), generated by a coalescing of intracellular vacuoles.[92] Metalloproteinases are important as enzymes able to remodel the extracellular matrix.[93,94] Three main types in the dermis that appear to be particularly important are MMP 1—Type 1 collagenase, MMP 2—Type 4 collagenase (72-kDa gelatinase), and MMP 3—stromelysin-1 (transin).

Four inhibitors of the excessive activity of these enzymes have been described, so-called tissue inhibitors of metalloproteinases (TIMP). If collagenases and other metalloproteinases are overactive or active for a longer time than required to support prolonged angiogenesis, the dissolution of dermal support may occur. It may be that the interplay of metalloproteinases and their inhibitors may be involved in the eventual scarring whether atrophic or hypertrophic, both appearing to be a breakdown of the normal balance of collagen production.

So if we are to intervene with physical therapies, it may seem reasonable to target either the poorly resolved inflammatory response or the equally poorly resolving angiogenesis. Vascular lasers and light sources could be examined with this in mind.

Common agents such as retinoic acid, imiquimod, calcipotriol, corticosteroids, low-dose fluorouracil, diclofenac, tetracyclines, hyaluronic acid, estrogen metabolites, genestein, heparin, cyclosporine A, steroids, and COX2 inhibitors have all been suggested to be antiangiogenesis substances. Many of these agents have both anti-inflammatory and antiangiogenesis characteristics and may deserve investigation to help avert early acne scarring. It is also possible that research activities surrounding new antiangiogenesis agents as anticancer treatments may yield further useful agents for this approach.[95]

Summary

Postacne scarring remains a very difficult task to correct. Great strides may be made in this condition only with attention to a greater understanding of its pathogenesis, the reasons why certain people scar and others do not, the inflammatory mediators, and the immunology of the scarring process. The variety of scars mirroring the variety of acne lesions means that no simple single process is likely to provide an answer to all acne scars. There have been some imaginative additions to treatment of both hypertrophic and atrophic disease but we still seem to be far away from successful resolution for this distressing disease.

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Address correspondence and reprint requests to: Greg J Goodman, FACD, Skin and Cancer Foundation of Victoria and Monash University Department of Community Medicine, 8th Floor 443 Toorak Road, Toorak 3142, Victoria, Australia, or e-mail: greggoodman@greggoodman.com.au