

Management of Post-Acne Scarring: What are the Options for Treatment?
[Review Article]

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Full Text LinkBrowse Search
ResultsFull Text Link

Titles
Display

Main Search
Page

Not AvailableBrowse Table of
ContentsFull Text Link

TOC

Outline

- [Abstract](#)
- [What is the Nature of Post-Acne Scarring?](#)
- [How Does Post-Acne Scarring Occur?](#)
 - [The Evolution of Acne](#)
 - [The Evolution of Acne to Post-Acne Scar](#)
- [What Types of Scars Does Acne Cause?](#)
 - [Atrophic Acne Scarring](#)
 - [Hypertrophic or Keloidal Scarring](#)
- [What Types of Atrophic Scars Are Seen?](#)
 - [Superficial Macular Scars](#)
 - [Deeper Dermal Scarring](#)
 - [Multichanneled Dermal Scars](#)
 - [Fat Atrophy](#)
- [Treatment of Acne Scarring](#)
 - [Dermabrasion](#)
 - [Chemical Peeling](#)
 - [Infrared Laser Resurfacing](#)
 - [Rhytidectomy](#)
 - [Undermining of Scars](#)
 - [Deeper Autologous Filler Agents](#)
 - [Punch Techniques](#)
 - [Dermal Grafting](#)
 - [Fat Transplantation](#)
 - [Other Tissue Augmenting Agents](#)
 - [Collagen-Based Products](#)
 - [Bovine Collagen](#)
 - [Autologen®](#)
 - [Dermalogen™](#)
 - [Isolagen®](#)

Output...

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Email Article
Save Article
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Links...

Full Text
Abstract
Complete Reference

[Help](#)
[Logoff](#)

History...

Management of Post-Acne S... Go
[Previous Page](#)

- [Alloderm®](#)
- [Noncollagen Augmentation Agents](#)
- [Hyaluronic Acid](#)
- [Fibrel®](#)
- [Artecoll®](#)
- [Silicon](#)
- [Treatment of Hypertrophic Scarring](#)
- [Conclusion](#)
- [References](#)

Graphics

- [Fig. 1](#)
- [Fig. 2](#)
- [Fig. 3](#)
- [Fig. 4](#)
- [Fig. 5](#)
- [Fig. 6](#)
- [Fig. 7](#)
- [Fig. 8](#)
- [Fig 9](#)
- [Fig. 10](#)
- [Fig. 11](#)

Abstract



Post-acne scarring is a very distressing and difficult problem for physician and patient alike. Recently, newer techniques and modifications to older ones may make this hitherto refractory problem more manageable. Options for dealing with post-acne scarring are explored. The patient, his or her overall appearance and the morphology of each scar must be assessed and treatment designed accordingly. To adequately address the patient with scarring, a thorough knowledge of the pathophysiology and anatomy of the different types of scars should be sought. Once an understanding of what the pathology is and where it is occurring is attained, the most pertinent treatment for that scar may be devised. A variety of post-acne scars is produced including superficial macules, dermal troughs, ice picks, multi-channelled fistulous tracts and subcutaneous atrophy.

The wide variety of new treatment methods for post-acne scarring includes newer resurfacing tools such as CO₂ and erbium infrared lasers, dermasanding and possibly some future techniques such as non-ablative and radiofrequency resurfacing. Dermal and subcutaneous augmentation with autologous and nonautologous tissue augmentation and the advent of tissue undermining have greatly improved treatment of atrophic scars. Use of punch techniques for sharply margined scars (such as ice picks) is necessary if this scar morphology is to be treated well. One should attempt to match each scar against an available treatment as far as possible. Many of these techniques may be performed in a single treatment session but repeat treatments are often necessary.

The treatment of hypertrophic acne scarring remains difficult, but silastic sheeting, vascular laser, and intralesional cytotoxics are interesting developments. Most often occurring extra-facially and in males these distressing scars often require multiple treatments and modalities before adequate improvement is achieved.

Acne affects 95 to 100% of 16- to 17-year-old boys and 83 to 85% of 16- to 17-year-old girls.[1-4] In the majority of those who develop acne, the condition remits spontaneously by age 23 to 25 years.

Adolescents have trouble dealing with the cosmetic embarrassment that acne produces. Poor self image, pain, recurrent bleeding, and purulent discharge are just some of the problems that these young patients have to deal with. In almost all cases acne affects the face [5] and may predispose patients to difficulties in interpersonal relationships.[6] Acne has been shown to persist into adult life and a recent study of adults over 25 years of age revealed at least mild disease (clinical facial acne; Leeds acne grade >0.75) in 3% of men and 12% of women.[7] Only 1% of the individuals with clinical facial acne had sought treatment; the majority had not done so because they thought that there were no effective therapies for acne. Acne scarring was recorded in 14% of women and 11% of men. Scarring occurs early in all types of acne, not just in nodulocystic disease, but does vary with the severity and delay until effective treatment is organized. Some degree of post-acne scarring is an outcome in 95% of patients with acne.[8] Post-acne scarring is particularly devastating and may be an 'at risk' factor for suicide.[9]

What is the Nature of Post-Acne Scarring?

Acne scarring is most commonly a problem of contour and color. Contour abnormality is not well tolerated visually, neither excess (hypertrophy) nor loss (atrophy) of tissues. The color of post-acne scars may stand out if they are red, white or brown. Most often color may be expected to settle somewhat with time and natural healing, but not always. Rarely, acne scars may offend if they are long, cross cosmetic unit boundaries, or distort free margins such as the lip or eyelid.

How Does Post-Acne Scarring Occur?

The Evolution of Acne

The first element in the evolution of an acne scar is evolution of acne itself. There is interplay between the onset of puberty, with its rising androgen level stimulating the sebaceous glands, and the inhibited outflow of sebum from the glands to the skin surface. This inhibited outflow occurs from an abnormal hyperkeratinization of the follicular epithelium in the pilosebaceous follicle. A pilosebaceous follicle is one where a sebaceous gland opens on to the hair follicle canal. Acne develops in follicles with large sebaceous glands and almost inconsequential, hair structures.[10]

The earliest event in acne lesion formation appears to be a change in the lower part of the follicular wall with the horny cells becoming stickier, causing impaction of these horny cells and dilatation of the sebaceous follicle. With this a noninflammatory microcomedo is born. This may stay as it is, it may enlarge into a closed or open comedo (blackhead), or it may go on to an inflamed acne lesion such as a papule, pustule, nodule or cyst from which scarring is likely.

Open comedones are quiescent lesions that rarely transform into inflammatory acne. However, closed comedones are the site of future inflammatory acne lesions.[11] In the largely anaerobic conditions existing in the impacted milieu of the closed comedo, *Propionibacterium acnes* (also known as *Corynebacterium*), an obligate anaerobe, flourishes.[12] *P. acnes* is instrumental in the production of the inflammatory stage of acne, the stage that brings with it the risk of scarring.

Inflammation in acne eventually leads to a thinning of the follicular wall, and once this wall is breached, the extravasation of irritating follicular contents into the dermis leads to a variety of lesions.

With this follicular explosion, there is the onset of dermal inflammatory disease. Hairs, lipids, keratin, and dead keratinocytes excite foreign body inflammation and released *P. acnes* activate both classic and alternative complement pathways, amplifying the inflammation.[13] The free fatty acids initially produced by

the intrafollicular lipase activity of *P. acnes* are also now in the dermis and are irritating to their new environment.[14]

The Evolution of Acne to Post-Acne Scarring

The rupture of the hair follicle results in a perifollicular abscess. Small abscesses discharge through the skin and heal uneventfully without scarring in about 7 to 10 days. The epidermis attempts to grow out from the side wall of the follicle to encapsulate the inflammatory reaction and thus to resolve the lesion without incident. If this encapsulation is incomplete and further rupture occurs, multichanneled fistulous tracts will result,[15] clinically expressed as a series of grouped open comedones or ice pick scars.

Other scars owe their appearance to the extent and the depth of the inflammation. If the dermal inflammation is severe, sloughing is significant with significant dermal scarring as its sequela. With increasing severity of inflammation or if rupture occurs deeply in the follicle, destruction continues to spread into the subcutis, causing deep tissue destruction. When such deep inflammation occurs, nodules and cysts are the outcome and often result in scarring that is either atrophic or hypertrophic (hypertrophic or keloidal).

What Types of Scars Does Acne Cause?

Atrophic Acne Scarring

Scars that result in a loss of tissue are the most common types of scarring seen after acne. Acne lesions are unusual in that the inflammation is initiated deeply.[16] The subsequent scarring thus affects deeper structures. As the scars mature and contract they draw in the surface layers and cause indentation or atrophy. There is also loss of structure and volume by the inflammatory mediators that add to the appearance of atrophic scarring.

Hypertrophic or Keloidal Scarring

Much less commonly, acne scarring may become thickened (hypertrophic or keloidal) rather than atrophic. Certain individual characteristics seem to predispose patients to this type of acne scarring: these include family history [keloids demonstrated an increased incidence of human leucocyte antigens (HLA) B14 and HLA BW16 in one study [17] and an increased incidence of HLA BW35 and HLA BW21 in another [18]], racial grouping, age between 10 and 30 years, and severity and site of inflammation. Keloids spread outside the confines of the original wound, have little or no maturation, are very symptomatic, and have certain histologic characteristics including thickened, tightly packed glassy collagen bundles. Hypertrophic scars are also thickened but remain within the confines of the original acne lesion with the scar progressing for a few months and then slowing before regressing after some years. Histologically, hypertrophic scars show numerous fibroblasts but relatively few collagen bundles, but with some myofibroblasts, and this may explain the scar contracture that is seen.[19]

What Types of Atrophic Scars Are Seen?

The depth and extent of the inflammation determine the amount, type, and depth of scarring.

Superficial Macular Scars

If the scarring process is relatively superficial, erythematous or pigmented macules may result. This is particularly true of comparatively early scars (under 1 year) where erythematous scars are present or in olive-skinned patients with postinflammatory hyperpigmentation. Both these problems improve over ensuing

months and, besides sun protection (to guard against aggravating the hyperpigmentation), further reparative treatment is not usually required. Medical therapy with retinol (vitamin A), tretinoin (retinoic acid) or [alpha]-hydroxy acids in conjunction with topical corticosteroids may be useful if the patient seeks treatment ([fig. 1](#)).^[20,21] Light skin peels with glycolic acids; Jessner's solution or variants may also be utilized effectively. Vascular laser therapy is another useful method of maturing these scars more rapidly ([fig. 2](#)).

[Graphic](#)

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Fig. 1. Macular scarring for which medical therapy alone would suffice.

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Fig. 2. Macular scarring for which vascular laser therapy or low strength chemical peeling with Jessner's solution [14% each of lactic acid, salicylic acid and resorcinol in alcohol (ethanol)] or glycolic acid would be applicable in addition to medical therapy.

Deeper Dermal Scarring



Multichanneled Dermal Scars



The attempts by the epidermal outgrowths from the hair follicle to encapsulate the inflamed contents of the exploded follicle may be only partially effective, resulting in multichanneled tracts.^[15] Wide excision of the entire multichanneled apparatus may be required.

Sharp punched out focal 'ice pick' scars are the result of the deeper dermis being affected in a punctate fashion. These are distressing scars that are resistant to many corrective techniques and usually require removal of the scar by one of the punch revision techniques. More extensive dermal damage causes linear or broader sharply margined and troughed scars. These benefit from dermal augmentation, either autologous or nonautologous.

Fat Atrophy



At least 2 mechanisms are at work to produce the significant subcutaneous deficit seen in many patients with acne. Disrupted acne follicles and cysts release inflammatory mediators that destroy facial fat. Cysts are also space-occupying lesions that leave a void after their resolution that the atrophied subcutaneous tissues cannot fill. Instead, the tissues are drawn in from surface layers and this effect is amplified as the scarring around the vicinity of the cysts matures and contracts.

Aging exaggerates this lipoatrophy. The 'coat hangers' represented by the facial convexities of the forehead malar and cheek regions diminish, allowing the skin to sag. The concavities of the preauricular, temples, inframalar, and perioral tissues become exaggerated and scarring in these regions appears worse. The hanging skin is also suspended in an odd manner by the strands of scarring, causing a cascading appearance of troughs and ridges that becomes more noticeable with advancing years ([fig. 3](#)).

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Fig. 3. (a) Preoperative view illustrating the irregular cutaneous cascading seen at this age group of patients with acne scarring, where the scarring interrupts the downward movement of the aging skin; (b) postoperative view (at 6 weeks) of patient after fat transfer and subcision alone. There is usually little morbidity associated with this procedure.

Treatment of Acne Scarring

Dermabrasion

It is appropriate to start discussion on the treatment of post-acne scarring with the first technique that substantially aided patients with this disorder. The work of the dermatologist Kromayer at the beginning of the 20th century [9,22-27] was instrumental in the development of this treatment. Advances in equipment, techniques, and anesthesia have steadily occurred over the last 3 decades with wire brushes, diamond embedded fraises, and serrated wheels being utilized.[28-35]

Dermabrasion using wet and dry sandpaper has been largely superseded by machine driven techniques. These machines were capable of more even dermabrasion with graded end pieces from fine through to coarse. The choice of hand piece is directed both by the depth and type of scars being treated and by operator preference.

Dermabrasion is a highly skilled procedure and variation between operators in clinical outcomes is substantial. Thus, complications such as patchy hypopigmentation and scarring do occur. Manual dermabrasion has been reinstated recently utilizing drywall/plaster sanding screen [36] or moistened silicone carbide sandpaper to manually dermabrade uniformly.[37] In this latter study, low strength trichloroacetic acid was added to the abraded wound.

Microdermabrasion using aluminum oxide crystals has attracted some considerable attention for various conditions including acne scarring.[38] In this treatment, small crystals of aluminum oxide are fired against the skin from a nozzle housing a compression and aspiration system; this causes multiple small lacerations, and used crystals are aspirated back from the skin surface and discarded. Multiple treatments are required and efficacy for the treatment of acne scarring remains somewhat unclear.

In contrast, laser resurfacing is an easier procedure to perform and therefore is more reproducible between operators. As such it has gradually replaced dermabrasion, and has excited the interest of the press and public in a way that dermabrasion never did. Dermabrasion, when well performed, remains an important part of the armamentarium of the practitioner involved in the treatment of acne scarring and is a low cost alternative to laser therapy.

Chemical Peeling

Recurrent light peeling with Jessner's solution, low strength trichloroacetic acid (20 to 35%), or stronger glycolic acid peels do help mild scarring and may be the best choice for this early superficial scarring group. Home regimens, incorporating [alpha]-hydroxy acids, antioxidants and tretinoin, may also be useful.

Despite the advances in stronger peeling techniques with the standardization of peel concentrations,[39] augmentation of peeling agents,[40] and the introduction of peeling pastes,[41] the degree of long term improvement is insufficient to satisfy most patients with acne scarring. It appears that once the swelling from the procedure resolves, the apparent improvement largely disappears.

Infrared Laser Resurfacing

Pulsed and scanned CO₂ laser resurfacing,[42-46] a technique largely introduced by dermatologists, is now performed by practitioners of many other disciplines. Lasers have become major resurfacing tools for acne scarring (fig. 4).

[\[Help with image viewing\]](#) Fig. 4. (a) Patient with macular erythematous scarring before CO₂ laser resurfacing; (b) patient with macular scarring after resurfacing with significant clearance.

More hyperbole than reality has led to an overselling of the virtues of CO₂ laser resurfacing and an understating of risks. Complications such as late hypopigmentation and scarring with CO₂ laser do occur. Scarring is an unusual complication, but may be severe. Hypopigmentation is subtle, occurs late and is now being more commonly reported with longer term follow-up of patients.[\[47,48\]](#)

Healing is considerably delayed with the CO₂ laser system compared with dermabrasion, as there is heat-induced tissue damage left in the skin that must be cleared before healing can begin. This prolonged healing phase is associated with an equally prolonged erythema as new blood vessels are formed to aid in healing. The search for a less thermally destructive laser, one that is able to more cleanly ablate tissue rather than slowly cook it, initially led to further shortening the pulse of CO₂ lasers and eventually to the introduction of the erbium laser.

The erbium laser beam is more avidly taken up by water than the CO₂ laser beam. The energy is extinguished rapidly in skin tissues, rapidly boiling the available water in its path, causing a clean ablation of tissue and leaving only minimal thermal damage. For every joule of fluence used, only 4 to 5µm (less than the thickness of most blood cells) is ablated. To increase the total depth of laser beam-induced tissue destruction, one needs to increase the number of joules impacting on the skin. This may be achieved by using high energy single pulses or by stacking a number of passes with a low power laser or overlapping the pulses (either by freehand methods or with a computerized scanner device).

Physics aside, what has been the real benefit of these lasers on post-acne scarring? Resurfacing procedures produce both horizontal and vertical effects on tissues - by a vertical ablation of the shoulders of scars and by a horizontal 'tightening' induced by a reorganization of collagen bundles parallel to the skin surface. How much improvement comes from each of these vectors is debatable.[\[49,50\]](#) Some resurfacing techniques, such as chemical peeling and CO₂ lasers, appear to rely on a 'horizontal' vector, and some, such as dermabrasion and erbium lasers, the more 'vertical' component.

In an effort to unite these theoretical vectors for the treatment of post-acne scarring, there appears to be some merit in the use of both CO₂ and erbium lasers in a combined fashion.[\[51,52\]](#) CO₂ laser may be delivered during part of the erbium pulse or it may be delivered first, followed by the erbium laser to ablate the layer of thermally denatured tissue left behind by the CO₂ laser. This promotes faster healing than using the CO₂ laser alone.

One cannot, however, attain uniformly good improvement in acne-scarred skin by stretching out or ablating tissues if there is deep dermal and subcutaneous structural loss. Overuse of these lasers in an effort to make them do what they cannot will only succeed in producing scarring and hypopigmentation.

In an effort to find a way of treating dermal disease without invoking an epidermal wound, non-ablative resurfacing has been described. Both Q-switched 1064nm Nd:YAG and 1320nm Nd:YAG lasers have been used for this technique. The target is water in both the epidermis and dermis. However, the epidermis in this treatment is protected by spraying a short burst of cryogen at the time of the laser pulse and again as the heat from the target is scattered back to the skin surface. The dermal water acts as the target, absorbing the infrared energy and being denatured somewhat by it. Visibly this impact produces only edema and erythema which rapidly resolve over the next few days. Although studies thus far have been limited to rhytides,[\[53-55\]](#) this technology is theoretically attractive for patients with post-acne scarring mainly affecting the dermis. Safety and efficacy still need to be firmly established for this interesting treatment.

Ablation of the skin by radiofrequency devices such as a bipolar multielectrode device originally introduced

for arthroscopic surgery has recently been added to the list of possible resurfacing tools. A similar effect to laser resurfacing is claimed for this device, with substantially fewer costs, reasonable safety and a relatively short recovery time, although it is early days for this technology.[56-58]

Rhytidectomy

Stretching of the skin by patients is a favorite technique used to illustrate to the practitioner how they feel they would ideally like to look. Unfortunately the surgical equivalent of what the patient is doing, the face-lift or rhytidectomy, is usually disappointing, with scars returning to where they were within a few months.[59] If true tissue laxity exists, this should be treated on its merits, but for patients with acne scarring it produces little to no benefit and if they are gaunt from their acne scarring it will make them more so.

Undermining of Scars

Undermining of acne scars has been practiced for many years as a way of forming a pocket or as an aid in the implantation of 'Fibrin' foam,[60] Fibrel[®], [61-63] dermal grafting [64-66] and microlipoinjection. It has recently been described as an isolated corrective technique,[67,68] relying on breaking up and detaching the scar, and releasing the skin surface from deeper tissues. This process leads to a pooling of blood under the defect that acts as a space-occupying, temporary augmentation agent keeping the scar base from immediately reattaching to the surface layers. Subsequent organization of this blood clot induces longer term partial correction of the defect by the formation of connective tissue. Successive treatments are required for further improvement (fig. 5).

[Graphic](#)

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Fig. 5. (a) Patient with acne scarring before treatment with subcision; (b) patient 3 months after single subcision treatment.

Most atrophic acne scars improve well with 1 to 3 treatments. This technique may be readily combined with resurfacing, allowing patients with mild to moderate acne scarring to undergo resurfacing in a less aggressive manner.

The technique involves a probe, either a sharp 18 to 21 gauge hypodermic needle or a blunt cannula that is inserted under the skin and the scar undermined in an intradermal or subdermal plane. Once resistance to an initial backwards and forwards motion declines and the scar is almost freed from the surface, the direction is changed to a side-to-side sweeping action. This completes the freeing up of the skin from its base. The depression usually visibly lifts at the completion of the procedure. Some degree of bleeding is usual and probably important to attain satisfactory improvement.

Predictable sequelae include temporary bruising and swelling, but complications are rare. These complications include acneiform cystic lesions from disruption of acne sinus tracts and variable responses ranging from partial to excessive. Partial response is usual; an excess response is seen in 5 to 10% of patients.

Deeper Autologous Filler Agents

Acne scarring may present with different shapes and sizes and at different levels within the skin. Acne scars are cosmetically concerning to a patient if they are abnormally colored or shaped, have altered contour or texture or are longer than about 1cm. The color and texture of an acne scar are often best dealt with by resurfacing techniques and visible light laser therapy.[69] Resurfacing procedures such as dermabrasion, [70,33] CO₂ laser,[71,72] or erbium laser resurfacing [73,74] may improve small or superficial contour

defects.

As an aid to resurfacing, or sometimes as an alternative for slightly deeper lesions, augmentation with a variety of agents is possible. These include the use of nonautologous human cadaveric agents, nonhuman biologic tissue implants with bovine collagen or hyaluronic acid, or nonbiologic augmentation with silicon, polymethylmethacrolate microspheres, and Gore-Tex™. Lastly, a variety of autologous (patient derived) filling agents is also available for these superficial defects, such as punch grafting,[75,76] autologous lipocollagen,[77] autologous collagen,[78] and cultured fibroblasts.[79]

When deeper defects are the concern, dermal grafts and autologous fat transfer techniques are the treatment of choice.[80] With deep atrophic scarring, treating only the surface will give a very temporary and disappointing result as the initial swelling subsides, revealing continuing tissue deficiency. Dermal and fat transfer techniques may be used at the same time as resurfacing techniques, allowing optimal treatment of surface and deep scarring. By treating the patient this way, excessive surface treatment may be avoided and the consequent risk of complications is diminished.

Fat transfer was initially not thought useful in the treatment of scarring,[81] but refinements in understanding and technique have changed our views on fat transfer and it is now credited with being a potential long term grafting material. The role of fat is not to fill dermis but as a deep volume wadding, a replacement for tissue lost, and a foundation for the more superficial tissue. This completed, a more superficial assault by dermal augmenting agents and resurfacing techniques is enhanced (figs 6 and 7).

[Graphic](#)

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Fig. 6. (a) Patient with severe acne scarring of the cheeks before operation; (b) patient 5 months after fat transfer, subcision and resurfacing with CO₂ and erbium

lasers.

[Graphic](#)

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Fig. 7. (a) Patient with deep troughed scarring and fat atrophy accentuating this problem; (b) patient 2 months after fat transfer, dermal grafting, subcision, and

resurfacing with erbium and CO₂ lasers.

Punch Techniques



There are some scars that have a poor surface and are small and punched out in appearance. Examples of such scars include acne scars with white atrophic bases or sharply punched out 'ice pick' scars and some chicken pox or postherpetic scars. These may be amenable to punch removal techniques such as punch excision, punch elevation, and punch replacement.

One needs to match the size of the punch removal closely to the scar concerned, so as to remove the scar with only a very small margin of normal tissue. The punch instruments must be sharp, straight walled, seamless, and available in a multitude of sizes from 1 to 4mm.

All punch techniques remove a pitted scar with a small rim of normal tissue. With punch excision, the site may be allowed to heal by secondary intention or the wound sutured.

The first inkling that scar removal may be optimal after or at the time of resurfacing was gleaned when biopsies performed at the time of dermabrasion were extremely hard to detect once the dermabrasion had healed.[82] It was then noted that dermabrasion after excision, but before closure, improved the subsequent

scar appearance,[83] and recently, simultaneous resurfacing with a CO₂ laser and punch excision [84] has been advocated. If a scar is excised and resurfacing follows 4 to 8 weeks later, the scar may be blurred or even totally eradicated.[70,85,86] Several studies have suggested that if resurfacing is performed in this postoperative period, the healing takes on almost a fetal type,[87] with a generalization of the extracellular matrix glycoprotein, integrin subunits in the epidermis,[88] and dermal collagen fibers becoming unidirectional parallel to the epidermal surface after dermabrasion associated with an up-regulation of tenascin.[89] It appears that dermabrasion (and by inference possibly other resurfacing procedures such as lasers) alters cell to cell and cell to matrix interaction within the epidermis and the dermis and between these layers, inducing a decreased potential for scar formation.

One must be reticent about excising acne scars if the acne is active, for if there are cystic remnants, this may incite postoperative cyst formation with apparent (though not real) wound infection complete with pointing and wound dehiscence, with consequent scar widening.

With punch elevation the scar is punched but not discarded. The tissue cylinder may be freed up at the level of the subcutaneous fat or transected from its fat base. The scar floats up and is held in place with forceps so the scar sits at the same level as the surrounding skin. The cylinder of tissue will become fixed in place by the patient's serum and sit as a flap attached at its subcutaneous base or as a free graft. It is then immobilized with surgical tape for several days. Resurfacing can be performed 4 to 8 weeks later if required. However, this is quite a limited technique as it relies on the floor of the scar displaying good texture and color, an uncommon finding.

Punch replacement grafting [75,90] begins with the scar being punch excised and the scar discarded. This scar is then replaced with a slightly larger full thickness skin graft usually from the postauricular area. Virtually all of the grafts will survive unless traumatized. Some grafts will heal level with the skin surface and some will elevate or pin cushion. Resurfacing 4 to 8 weeks later may be used to flatten and blur the grafts. After 6 to 12 months the grafts become very difficult to locate (fig. 8). This technique is quite painstaking, as often 20 or more replacement grafts are required in a single session. Occasionally, several sessions of punch replacement grafts are performed several weeks apart.

[Graphic](#)

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Fig. 8. (a) Patient with punch grafts in situ; (b) another patient 2 years later with previous punch grafts that are difficult to detect.

Importantly, punch techniques allow deeper scars to be treated so that less aggressive resurfacing may be practiced.

Dermal Grafting

When dermis has been lost, strips or pieces of dermis may be harvested and transplanted to replace this loss. Essentially a permanent autologous collagen, dermal grafting may be used to correct linear or other deep dermal scars. The donor site is often the retroauricular fold but may be wherever it is possible to leave an unobtrusive scar. After de-epithelialization with laser resurfacing or dermabrasion, the remaining dermal strip is excised and the area sutured. A 14 or 16 gauge sharp trocar such as an intravenous cannula is useful for preparing the recipient site pocket with the cannula entering at one end of the trough and exiting at the opposite end of the linear defect. The cannula is withdrawn but the plastic sleeve may be left behind extruding from both ends of the scarred area. The dermal graft is then attached to a suture on a straight needle that is fed through the plastic sleeve until it exits the other end. The trailing graft abuts the proximal end of the plastic sleeve and the sleeve and graft are pulled together until the graft comes to lie in the formed dermal pocket. The procedure may be performed with or without a simultaneous resurfacing procedure (fig. 9). The

technique most often offers permanent correction with the only common complication being cyst formation if all epidermal and appendageal remnants have not been removed. Graft resorption may also occur in time with some grafts.

Graphic

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Fig 9. (a) Patient before laser resurfacing and dermal grafting; (b) patient 6 weeks after resurfacing and dermal grafting.

Fat Transplantation



Fat comes close to being an ideal augmentation material as it is cheap, readily available, and incapable of being rejected or causing allergic or other adverse tissue reactions. It is easy to work with and is without risk of communicable disease. However, the issue of permanence is still unresolved. In the past it was deemed to be temporary [\[91,92\]](#) but recent improvements in technique suggest that fat transfer may produce longstanding correction.[\[93-96\]](#)

The technique consists of 2 phases - procurement of the fat graft and placement of this graft. Both phases are critical and have changed considerably. The procurement of fat [\[97\]](#) is no longer accomplished by high pressure machine aspiration but now is performed with gentle hand aspiration. Tumescence anesthesia is utilized for patient comfort and to limit blood contamination. A closed technique with less handling of the extracted fat before re-injection is now often used.

The debate about whether to treat fat after removal and before re-injection has been richly contested. [\[98-110\]](#) Perhaps Lexer [\[111\]](#) was right almost 80 years ago, when he stated that 'it is necessary that the fat tissue is not damaged at the moment of its removal nor at the moment of its implantation'. He may have added if writing today that it should not be damaged between these 2 stages as well.

The injection phase with small parcels of fat implanted in multiple tunnels allows the fat graft maximal access to its available blood supply. With the likelihood of increased graft survival, over-correction is not required. If further fat transfer is needed, additional fat may be taken at the time of original suction and stored frozen for further postoperative refinements as may be required.

Fat is best injected using blunt cannulae via a small nick in the skin. When injecting into acne scarred skin, undermining or subcision [\[26\]](#) may be used to break up the scar tissue and release it from its attachments to deeper tissues where these residual scar attachments are seen to impede an even improvement in skin contour. Undermining is continued until this normal contour is achieved.

Good implantation requires the fat being injected at many levels and developing a 3-dimensional lattice structure to support the more superficial skin layers. Use of a 1ml tuberculin type syringe, 18 gauge cannulae, and injection aliquots of 0.1 to 0.2ml per site affords the finest control of the injection volume. This procedure may be used with other procedures such as laser resurfacing, subcision, and dermal grafts at the one procedure.

Some fat will not survive the transfer, with losses occurring during the suction or the injection or presumably by failing to develop a sustaining blood supply. Approximately 50% of patients with acne scarring treated with this technique require further top-up procedures. Overcorrection should be avoided. It is better to freeze residual fat and use this as required during the ensuing 6 and possibly 12 months. Blindness has been reported with fat transfer [\[112\]](#) and trauma is possible to important structures, so one should use blunt cannulae, low pressure injections, and small aliquots of fat and inject only on withdrawal of the cannula.

Aging adversely affects the acne-scarred face in a number of ways. Deep acne scarring, as discussed, is a

cause of significant and sometimes severe facial fat atrophy. As most patients age they lose facial structure, including fat, which amplifies the loss in acne-scarred areas. This combination often influences the patient to seek corrective surgery for longstanding acne scarring when they reach middle age. Facial skin starts to sag with aging and starts to literally hang on the scars. The inelastic scars hold focal areas of the skin in place not allowing its even descent, engendering an uneven cascading appearance.

Fat transfer in these patients is a procedure that is vital both in its reconstructive ability for deep acne scarring and also for its effect to bring back the youthful qualities of a fuller facial appearance. One must be careful to balance the face if large amounts of facial fat are required for acne-scarred areas. It may be necessary to implant fat in older patients in malar, chin, forehead or other areas if concavity augmentation such as cheeks and temples is to be kept in balance. The best place to look for guidance in this accessory augmentation is to study earlier photographs of when the patient was younger, where areas of tissue resorption may be noted and addressed.

Other Tissue Augmenting Agents

As well as autologous augmentation there are many nonautologous biologic, and nonbiologic tissue augmentation agents that may be used for atrophic scar contour correction. Achievement of safe, long term or permanent correction using a tissue augmentation agent is an attractive option. At present such a product is not at hand, but this is a burgeoning area of interest.

Collagen-Based Products

Bovine Collagen

This very successful material has treated well over 1 million patients since 1981, although acne scarring is not commonly the indication for its use. Most major antigenic telopeptides in bovine collagen are removed in processing this material; however, minor antigenic determinants remain and double skin testing is advisable to exclude potential adverse reactors.[113] Acne scars retain bovine collagen comparatively well,[114] with Zyderm 11[®] used for smaller distensible acne atrophic scars and Zyplast[®] for deeper acne scars (fig. 10). Two or 3 treatment sessions are usually required for the best correction that may allow 1 to 2 years of correction to be attained.

[Graphic](#)

[\[Help with image viewing\]](#)

Fig. 10. (a) Patient with acne scarring before bovine collagen injections; (b) patient after bovine collagen injections showing good improvement.

Autologen[®]

Because of the inherent allergenicity of bovine collagen and to produce possibly a longer lasting result, autologous collagen, processed from the patient's own tissues, is now available (Autologen[®]).[115] Unfortunately, a substantial volume of donor tissues is currently required, but growing autologous collagen from small punch biopsies may be possible in the future. The longevity of correction, very important in scar correction, has been quite good with correction maintained for 1 to 2 years with 1 to 4 injection sessions.[115]

Dermalogen[™]

Human cadaveric donor tissue processed into collagen from tissue banks (Dermalogen[™]) should be potentially less allergenic than bovine collagen, but very stringent requirements for control of potential

communicable disease are mandatory. This readily available source of human collagen may allow correction of many scars without requiring the patient to undergo a number of procedures.

Isolagen®

Isolagen® is an injectable in vitro culture of autologous fibroblasts in an extracellular matrix. A punch biopsy is taken and the fibroblasts are cultured. With re-implantation of the material it may continue to expand, presumably by new collagen production from the fibroblasts, for many months after augmentation. It has been used for quite dramatic acne scars and may be used in conjunction with resurfacing techniques.[79] As it is autologous there is little risk of reactions to this agent and it seems to show little tendency for resorption. One must have patience with this agent as results are subtle and improvement may be a gradual and somewhat delayed process.

Alloderm®

This is acellular human (cadaver) allograft dermis that may be used in multiple sheets to augment deeper defects of structure and possibly in single sheets for smaller acne scars. Rejection is minimized by removing all cellular material, and longevity and nonresorption are among its best characteristics.[116]

Noncollagen Augmentation Agents

Hyaluronic Acid

Hyaluronic acid has the same chemical composition in all sources of this material. Besides human dermis, this material is present in high amounts in synovial fluid, vitreous body, umbilical cord, rooster combs, and certain streptococcal species. The last 2 sources are those that commercially have offered us this dermal augmentation agent. Intrinsically, it contains no protein and thus has low allergenicity for all species and the material is viscoelastic, transparent, nontoxic, stabilizable to allow longer term correction, and holds 1000 times its molecular weight in water, acting as a sponge material when injected. The 2 agents available are Hylaform® and Restylane®. Hylaform® is derived from rooster combs and has a hyaluronic acid concentration of 6 mg/ml; Restylane® is derived from the coating of fermented streptococci. Both agents may be used for acne scars, with 1 to 3 treatments allowing long term but temporary correction.[117-120]

Fibrel®

Fibrin foam [60] or Fibrel® implantation [61-63] has shown promise in the treatment of scars for many years. Fibrel® uses the mechanisms of wound healing to produce collagen under areas of scarring. A mixture of a porcine gelatin matrix providing the scaffold for the clot to form, plasma for the necessary ingredients for collagen synthesis, and E-aminocaproic acid to inhibit fibrinolysis allows an excess collagen reaction to form under the scar. Probably xylocaine may substitute for the patient's plasma. Excellent persistence is attainable although the awkwardness, the requirement for allergy testing, tissue infarction if injected inadvertently into deeper subcutaneous vessels, and prolonged inflammation in a few patients have caused the demise of this agent.

Artecoll®

Artecoll® is a suspension of polymethylmethacrylate microspheres in a 3.5% collagen solution with 0.3% lidocaine (lignocaine). It is a permanent augmentation agent implanted at the dermo-subcutaneous junction. It has only limited use with acne scars because of this placement requirement.

This unfortunately maligned product was an excellent augmentation material for post-acne scars. Correctly used microdroplet injection of medical grade 360 centistokes silicon built up gradually over a number of injection sessions was without peer for isolated scars.[\[121,122\]](#) It may be that this material will surface again if the hysteria engendered over its dubious problems in other areas is resolved.[\[123,124\]](#)

Treatment of Hypertrophic Scarring

One must first decide whether treatment is indicated. If the scar is relatively asymptomatic and mildly hypertrophic one may reasonably delay treatment pending natural remission of the excess scar. However, treatment is indicated if the scar is a true keloid, symptomatic, progressively enlarging, or cosmetically unacceptable.

Available therapies are not overly effective, but corticosteroids topically applied or injected intralesionally remain the mainstay of treatment.[\[125,126\]](#) Potent preparations such as betamethasone dipropionate 0.05% or betamethasone valerate 0.05 or 0.1% are applied with or without plastic occlusion. Caution must be exercised with these preparations under occlusion because of their marked atrophogenic effects. It is unclear whether the less atrophogenic agents such as mometasone furoate or methylprednisolone aceponate are able to deliver the efficacy of the other agents.

In an effort to decrease atrophy associated with corticosteroids, one may combine their use with a substance that may reverse this undesired effect such as oral retinol [\[19,20\]](#) or topical tretinoin.[\[127,128\]](#) Topical tretinoin may have an independent role in the treatment of excessive scarring.[\[129-131\]](#)

Intralesional corticosteroid injections with triamcinolone acetonide 10 or 40 mg/ml or betamethasone sodium phosphate and betamethasone acetate 5.7 mg/ml can be used therapeutically on established scars or in an interventionist manner for scars that are rapidly enlarging.

In existing scars, intralesional corticosteroids are injected in several sessions quite superficially into the lesions using a 30 gauge needle. Local anesthetic injected subcutaneously under the scar 10 to 15 minutes before the scar is infiltrated or a short spray of a skin refrigerant and adding some local anesthetic to the intralesional corticosteroid are useful techniques to decrease the pain of the corticosteroid injection. Usually it is best to start with triamcinolone acetonide 10 mg/ml or betamethasone sodium phosphate and betamethasone acetate 5.7 mg/ml, reserving triamcinolone acetonide 40 mg/ml for resistant cases.

There has been recent interest in the intralesional use of the cytotoxic agent fluorouracil, as a corticosteroid-sparing agent, in the treatment of inflamed hypertrophic scars.[\[132\]](#) This substance is utilized at a concentration of 50 mg/ml and often mixed with low strength intralesional corticosteroid. 0.1 to 0.3ml is all that is usually required for an individual scar. It is quite painful and pre-emptive local anesthetic is needed. If this agent can be shown to match the efficacy of intralesional corticosteroids its lower atrophogenic potential would be advantageous. Future trials will establish its place in the treatment of these scars.

Silicon gel sheeting is useful on its own or in combination with topical preparations. Silicon gel sheeting should be kept in place for at least 14 hours a day and worn until the silicon sheet starts to disintegrate. The mechanism of action is unknown but the occlusive effect appears to be important.[\[133,134\]](#) It is used either on established scars or after surgery once wounds have healed. Topical silicon gels are also available and are usually more tolerable cosmetically for patients with facial scars. However, there are some doubts about the efficacy of sheets, gels, and creams.[\[135,136\]](#)

Cryosurgery of established scars has been the subject of several studies.[\[137-139\]](#) The number of treatments

needed to effect significant improvement requires a stoic individual. It is best to combine cryotherapy with other modalities such as surgery or corticosteroids. It also may have a better role in the treatment of earlier more vascular lesions.[140]

Tunable flashlamp-pumped pulsed dye laser has been found to be useful in the treatment of keloid sternotomy scars, with improvement in scar height, skin texture, erythema, and pruritus in the laser-treated scars. [141-143] It is hypothesized that the increased vasculature of hypertrophic scars may be targeted selectively. Similar results may be attainable with other vascular lasers (fig. 11). Although most studies suggest improvement with the tunable flashlamp-pumped pulsed dye laser, not all authors agree that this treatment is useful.[144] Certainly other lasers such as CO₂, argon, and Nd:YAG have been used with less than encouraging results in most studies.[142,145-147]

Graphic

[\[Help with image viewing\]](#)

Fig. 11. (a) Chest keloidal scar before intralesional corticosteroids and 532nm vascular laser therapy; (b) keloidal scar after intralesional corticosteroids and 532nm

vascular laser therapy.

Despite being a high risk venture in some patients, excision of existing hypertrophic or keloidal acne scars may be contemplated. Similarly a chronic acne cyst may require excision in a patient at high risk of developing post-operative scarring. In such patients, additional techniques may be considered to decrease the chance of unacceptable scarring in the post-operative period. Intralesional corticosteroids given prophylactically at the time of preoperative local anaesthetic infiltration [125] for scar or cyst excision together with delaying suture removal by 30% may help avoid sequelae. It may be that this 1 injection is all that is required to prevent recurrence. Another option is to use intralesional corticosteroids starting 1 week after suture removal and repeated every 2 to 3 weeks on 4 occasions.[148]

Superficial x-ray therapy is an effective adjunct to excisional surgery for keloids. The treatment was introduced in 1906.[149] It appears that radiotherapy is best given within 48 hours of surgery.

Pressure bandaging is an inconvenient but effective therapy and is most commonly used in burns rather than acne to prevent keloid formation in early scars.[150,151] Pressure needs to be applied for 20 hours or more a day for a period of at least 9 months.[152]

Colchicine 0.5mg 3 times a day for 3 to 4 months after keloid surgery may prevent recurrence. It is started at the time of suture removal and is thought to increase collagenase production and inhibit histamine from mast cells.[153]

Oral methotrexate taken at a dosage of 15 to 20mg every 4 days starting 1 week before surgery and continuing for 3 to 4 months,[28] interferon [alpha]-2b,[154] interferon-[gamma],[155] pentoxifylline,[156] and tranilast [156] also have their advocates in the treatment of hypertrophic scarring.

Conclusion

An understanding of the pathophysiology of acne and its sequela of post-acne scarring is essential for anyone with a serious interest in this disease. Early and aggressive treatment of acne before scarring occurs is so important. We all have to bear a responsibility for this - doctors, the media, and parents. Despite the prevalence of acne it should not be considered a 'normal part of growing up'. Acne is not 'cute' and neither is the life-long nightmare that post-acne scarring can be for patients.

Post-acne scarring remains a challenge to all concerned. Practitioners must look upon not only each patient's