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Aesthetic Plastic Surgery

New Innovations in Scar Management

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Abstract. As current aesthetic surgical techniques become more standardized and results more predictable, a fine scar may be the demarcating line between acceptable and unacceptable aesthetic results. With this in mind, a scar management program has been adopted based on the modalities of wound support, hydration, and hastened maturity, all factors gleaned from scientific evidence published over the past 25 years. Tension on a scar in one axis will result in a stretched scar, probably initiated by neutrophils and their neutral proteases [18,26]. Tension on a scar from many directions or intermittently will result in a hypertrophic scar, possibly initiated by lymphocytes but definitely related to a prolongation of the inflammatory process, with increased fibroblast activity and overabundant extracellular matrix secretion [24,26]. The common initiating factor is the tension on the scar, and the critical element needed to counteract this tension is scar support. Clinical experience has shown us that the most reliable way to support a scar is by using microporous tape. Hydration is a second beneficial influence on scar control and is the basis of the use of silicone sheeting and gel [7,29,36]. Alpha Centella cream has two main components. The first is an extract from the plant *Bulbine frutescens.* This increases hydration under the tape by leaving a layer of fatty vesicles of glycoprotein on the skin surface. This also has antibacterial properties. The second component is the principal terpenoids extracted from the *Centella asiatica* plant. These include asiatic acid, madecassic acid, and asiaticoside. *Centella asiatica* has been documented to aid wound healing in a large number of scientific reports [5,12,21,22,33,34,40]. The most beneficial effect appears to be the stimulation of maturation of the scar by the production of type I collagen [4,19] and the resulting decrease in the inflammatory reaction and myofibroblast production. Thus these components have been incorporated into the formulation of a scar management program. This publication reviews much of the available literature relating to scar management and describes the formulation and use of a scar management program based on this information.

Key words: Scar management review—*Centella asiatica*

In our constant quest for superior results in cosmetic surgery, we have entered an era in which multiple attempts at "scarless" surgery have been presented. Unfortunately, in many cases, although technical breakthroughs have been made, the aesthetic objective has not been achieved and, indeed, has even been compromised. In most cases, had more attention been paid to scar management rather than to elimination of part of the scar, the overall cosmetic result would have been superior. It is surprising that when it comes to closing a wound we use the most sophisticated surgical techniques and the most advanced suture materials and choose the most comfortable appropriate dressings, but as soon as we have removed the sutures many surgeons abandon any further management of the scar and leave its ultimate result to Mother Nature.

After surgery or an injury the scarring is, in many cases, what concerns the patients most, and it may continue to cause discomfort and may be unsightly long after the reasons for the initial surgery have been forgotten. Scar outcome can be controlled to a certain extent, but its management really begins only once the stitches have been removed. This continues over a critical period for the following 8 weeks.

The literature abounds with articles which focus on the complex sequence of events that takes place with the body's effort to close open wounds. A wound is defined as a pathological state in which tissue becomes separated. The sequence of events is now reasonably well

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defined, involving the initial vascular phase with the release of mediators, platelet factors, and coagulation cascade. This is followed by the inflammatory phase, involving neutrophils, macrophages, cytokines, lymphocytes, and monocytes. Too little inflammation delays healing; too much leads to excessive scarring—products of the inflammatory response regulate collagen synthesis in scar formation. Interleukin-1, produced by monocytes, increases collagen and fibronectin synthesis by fibroblasts. TGF-B is released by platelets at the site of injury and is highly chemotactic for macrophages and monocytes. It also stimulates production of collagen and fibronectin by fibroblasts [1] Scientists today are striving to find natural agents to attenuate or regulate the excessive cell proliferation and synthesis and contraction of the extracellular matrix during repair by scar fibroblasts. TGF-b has received the most attention due to its action in promoting fibrosis [41] and agents such as mannose-6-phosphate and decorin (dermatan sulfate proteogycan II) have been proposed as antagonists [41]. TGF-B1 protein is localized in the nodules of hypertrophic scars and stimulates the expression and formation of myofibroblasts [9]. Thus a balanced inflammatory phase is critical. After a 5-day lag, which corresponds to matrix accumulation composed largely of fibronectin and hyaluronic acid, an increase in wound breaking strength begins that coincides with a high rate of type 1 collagen synthesis. The rate at which wounds gain tensile strength thereafter, however, is slow, reflecting a much slower rate of collagen accumulation. For example, wounds have gained only about 20% of their final strength by the third week. In addition, wounded tissue fails to attain the same breaking strength as uninjured skin. At maximum strength a scar is only 70% as strong as intact skin [8]. The gradual gain in tensile strength is secondary not only to new collagen deposition, but also to collagen remodeling, with formation of larger collagen bundles and an alteration of inter molecular cross-links [8]. The wound enters the fibroblastic phase within 5 days and this phase lasts up to 4 weeks. Elements involved here include fibroblasts, growth factors, fibronectin, collagen (mainly type III, then type I), angiogenesis, and epithelialization. The price of wound repair is a scar, which appears reddish at first but, as the connective tissue tightens and vascularization slows, gradually loses its color.

The vast majority of wounds that we deal with in elective surgery are closed and past the major wound healing phases before we even consider the scars. Clean approximated wounds are clinically resurfaced within 48 hours. A preferred scar is one that has undergone rapid maturity with little contraction or increase in width. It is one that has not formed more collagen than is necessary for its strength.

All wounds heal by scar formation and this scar will never disappear. However, a good scar is one that is thin and flat and approximates the color of the surrounding skin rather than a scar that is stretched, thickened, and irritable and is a different color than the surrounding skin. In normal wounds that scar, collagen synthesis peaks at day 21 after wounding and reaches a level similar to that of normal fibroblasts by day 26 postwounding. Although the conversion of normal scarring to hypertrophic scarring or the apparent overgrowth of scar beyond normal healing usually occurs 6 to 8 weeks after injury in humans, those scars that will become hypertrophic usually are erythematous and raised much earlier. Thus there is often a clinical indication that a scar has a propensity to become hypertrophic [27].

Three components have been identified as critical to scar control.

Scar Support—Investigative Evidence

Sommerland and Creasy [37] compared the effect on stretching of four techniques of wound closure and studied the microarchitecture of stretched scars. They concluded that if there is little tension across a scar itself, the mechanically weak collagen bond withstand the forces on it—a narrow scar results and the scar collagen remains aligned along the scar. If, however, the tension across the scar is sufficient to overcome the bond when sutures are removed, the longitudinal fibers separate, and new collagen is laid down haphazardly across the scar. They observed that scars that stretch do so at a constant rate, almost doubling their width between 3 weeks and 3 months and increasing by nearly 50% between 3 and 6 months but stretching very little thereafter. Thus any technique which produces a narrower scar at 3 weeks would produce a proportionately narrower scar ultimately and this could make a significant difference in potentially wide scars [37]. The strength of the scar after 3 weeks is only 20% that of intact tissue [8]. Thus scar support is critical in this 2- to 3-week period and for longer periods when increased tension across the scar would result in exaggerated scarring.

Observations made by Elliot et al. [13] regarding presternal scars showed that not only was hypertrophy common, but there were significant regional differences, with a particular tendency to scar hypertrophy overlying the body of the sternum, especially in females. The lowest part of the scar showed the most stretching. How do we explain this? In 1861 Karl Langer measured the degree and direction of tension in various parts of the body by observing the distortion of a cut disk of skin measured from a wooden template [20]. In their paper in 1991 Meyer and Mcgrouther used a 5.5-mm skin biopsy punch as the template and measured distortion in different areas [26]. It was concluded that hypertrophic scarring occurs in areas of high tension with “pull” in many directions; a stretched scar results from increased tension in one axis only. Thus, over the sternum, the lines act in all directions, from movement of the arms, shoulders, neck, breasts, etc., whereas over the upper abdomen, the overwhelming tension is predominantly in the horizontal plane from repeated constant respiration. “The multidirectional tension may over stimulate the fibroblast causing it to produce excess collagen which is the main constituent of the scar” [26]. According to these authors the

midline abdominal scar will tend to be stretched if under tension—What happens if a laparotomy scar is put under tension?

Hogstrom et al. [18] measured wound margin strength after median laparotomy closed with or without tension. Tension on the wound resulted in a marked decrease in strength. This observed decrease in strength is critical to the process of wound management, where agents should be used that increase wound breaking strength as opposed to those that decrease this strength (vitamin E). Neutrophils were found to be present in large numbers in the areas of tension coinciding with the decrease in breaking strength. The neutrophils release neutral proteases (elastase, cathepsin G, and true collagenase) thought to be responsible for the degradation of collagen and the decrease in breaking strength [18]. This decreasing wound strength with repeated rhythmical tension from respiration resulted in the commonly observed stretched laparotomy scar.

What other cells play a regulatory role in wound healing?

Martin et al. [24] and Muir [28] showed that lymphocytes were present in the wound much earlier than previously described, namely, day 1, with peak numbers between day 8 and day 14, and remain present for as long as 4 months. The probability exists that in normal wound healing, lymphocytes are involved in the regulation of fibroblast function and the balance of lymphokines is well controlled. In hypertrophic and keloid scars, where all these cells persist much longer than in normal wounds, perhaps the imbalance of lymphokines is the cause of excessive fibrosis. As the inflammation subsides, there is decreased lymphocyte production, decreased fibroblast activity, and regression of the scar. Although not yet proven, it is becoming more likely that aberrations of lymphocyte function may be responsible for abnormal healing.

Thus tension on a scar in one axis will result in a stretched scar, probably initiated by neutrophils and their neutral proteases. Tension on a scar from many directions or intermittently will result in a hypertrophic scar, possibly initiated by lymphocytes but definitely related to a prolongation of the inflammatory process, with increased fibroblast activity and an overabundant extracellular matrix secretion. The common initiating factor is the tension on the scar, and the critical element needed to counteract this tension is scar support.

Clinical experience has shown us that the most reliable way to support a scar is using tape. Microporous tape is the most common form used, as it seems to accelerate healing without the accompanying bacterial growth seen with completely occlusive dressings. Thus, resistance to infection seems to be secure, but what about the efficacy of microporous tape? Those of us using prolonged taping on scars regard it as the most reliable of the modalities available for the prevention of thickened scars. This not only is a clinical impression but has been confirmed in various publications.

On observing scars across the wrist joint, Reiffel [31] noted that the portion of scar overlying the immobile palmar fascia healed well, while that portion that stretched and relaxed as the wrist extended and flexed became hypertrophic. He concluded that it was longitudinal stretching parallel to the long axis of the wound that stimulates the process of hypertrophy. Taping was therefore recommended longitudinally in the direction of the scar rather than at right angles to it. This theory is probably in keeping with the multidirectional tension forces described earlier. The author concludes that the control or elimination of such stretching forces by the long-term use of paper tape, beginning at 2 weeks, has been effective in preventing hypertrophic scarring. Patient compliance was found to be a problem—these patients were instructed to change the tape on a daily basis for periods of a few months. This is a critical point, as patient compliance is an essential to any scar management program. We have found that it is unnecessary and counterproductive for the patient to remove the tape on a daily basis—the tape should be left in place until spontaneous separation occurs (usually 7–10 days). This technique has been used over the past 8 years with proven efficacy (Widgerow and Chait, personal experience).

Orentreich et al. [30] showed that tape occlusion produces anhydrosis, with 6 days of occlusion causing anhydrosis that requires 3 weeks for reversal. Gordon and Maibach [14] showed this to result from the effect of occlusion on the eccrine duct or secretory coil of the sweat gland. Marples and Kligman [23] showed that bacterial growth under tape is proportional to the degree of occlusion. As a result, microporous tape has been recommended as best over skin wounds because accelerated healing seems not to be accompanied by bacterial growth, as is seen under completely occlusive tape [32]. Hofman and Maibeck [17] showed that occlusion that produces an underlying dermatitis causes transepidermal water losses of up to 40 times the basal value. Neither dermatitis nor transepidermal water loss has been encountered beneath microporous tapes. Closure with microporous tape produces far more resistance to infection than other closure techniques [6,11]. It is therefore critical, when choosing an agent to interface with the tape, to avoid agents prone to skin irritation and dermatitis (vitamins A and E) or those with thick consistencies that may result in fully occlusive dressings rather than the semiocclusive nature of microporous tape. Sawada et al. [36] confirmed the efficacy of microporous tape on sutured wounds. They reported better results with Blenderm than with Micropore, thought to be due to the extra hydrative effect of the former. We believe that by combining a hydrative agent on the surface of the tape, this advantage is negated, and Micropore is still felt to be superior in its adhesion and skin interaction.

Taping is thus recognized as an effective method of wound support and controlled scar formation.

Hydration—Investigative Evidence

Topical silicone gel or silicone cream with occlusive dressing has proved to be an efficacious method for the

treatment and prevention of hypertrophic scars and keloids, but how this action is triggered remains unknown. There is little possibility that topically applied silicone passes through the intact epidermis or gains access to the dermis. Hydration rather than silicone would appear to be the primary modulator of improved wound healing.

A keratinocyte–fibroblast coculture was used by Chang et al. [7] to mimic the use of topical silicone gel or cream. When testing the effects of various agents on fibroblast proliferation, Hanks balanced salt solution had more of an inhibitory effect on fibroblasts and their production of collagen and glycosaminoglycans than silicone or liquid paraffin. This work supports the conclusions of other authors [29,35] that the hydration effect of silicone gel is probably responsible for the enhanced healing effects and that silicone is not essential to the process. When silicone gel was substituted with water, the results on scars were equally as good. Compared with microporous taping, the taping proved to be more effective in preventing hypertrophic scars [29]. Davey et al. [10] believe that the relatively impermeable silicone gel acts in the same way as the stratum corneum. It reduces water loss and restores homeostasis to the scar, thereby reducing capillary hyperemia, collagen deposition, and hypertrophic scar formation. The water vapor transmission rate of silicone gel was found to be about half that of skin (4.5 vs 8.5 $g/m^2/h$). Since the scar surface does not appear to be wet when the sheeting is used, it appears that the sheet may promote hydration of the scar. Hirshowitz et al. [16] have described the mechanism of action of silicone sheets and their newly designed cushion to be on the basis of a negatively charged silicone surface affecting the scar with static electricity, causing involution of hypertrophic and keloid scars. It may well be that the positive effects seen in these cases may once again be attributed in large degree to the hydration effects elucidated in so many previous publications. Silicone sheeting and gel are used in most cases in the treatment of established exaggerated scarring. Unfortunately it has proved to be awkward to keep in place and has been associated with discomfort, maceration, and even fungal infection [45], making patient compliance difficult. Although silicone gel proved effective as an hydrating agent, a natural substitute would be preferable. Leaf sap from *Bulbine frutescens* is widely used for the treatment of wounds, burns, itches, rashes, cracked lips, herpes, etc. [42]—they have antibacterial properties, but the healing effect is likely due mainly to the glycoprotein in the leaf gel. More importantly these fatty vesicles of glycoprotein are not absorbed but remain on the skin surface together with the tape, ensuring ideal hydration and occlusion of the wound [25].

Accelerated Scar Maturity—Investigate Evidence

A review of the literature on scar creams [43] revealed the following information: **vitamin E** systematically seemed to inhibit the inflammatory response, wound healing, and tensile strength but topically did not display any beneficial effect. Clinically, however, in large doses topically, one often observes effects on the scar similar to those of steroids—that is, delayed wound healing and stretched scars probably related to the reduced tensile strength. Recent publications [15] have highlighted the skin irritation and reduced breaking strength caused by vitamin E and, at last, have put to rest the long-standing myth that vitamin E has a part to play in early scar control. Once again, hydration appears to be the only beneficial effect, far outweighed by the potential negative effects. Using vitamin E later on in the scar's maturity (4–6 weeks and later) may well flatten the scar due to its hydrative capabilities but, due to its decreased breaking strength effect on the scar, may result in a stretched weakened scar at best, and at worst, if used too early, can result in wound separation.

Vitamin A (0.05% retinoic acid) had some beneficial effect, but side effects of hypervitaminosis, skin irritation, etc., were reported. Cyclosporin could possibly be useful in prevention if T lymphocytes are proven to play a role (still experimental). Collagen inhibitors such as BAPN prevent cross-linking of collagen. Results are still being awaited but these preparations would not be used routinely in normal scar management. Limited use of corticosteroids topically fails to reduce scar formation, but with intralesional administration in established keloids, they do retard excessive collagen deposition.

Madecassol is the brand name given to a group of chemicals related to asiatic acid, which is extracted from the *Centella asiatica* plant. *Centella asiatica* is a perennial creeper grown mainly in tropical areas of Madagascar, Asia, and Africa. The plant has been used as a healing agent in Madagascar and the West Indies for hundreds of years. It was imported into France around 1850 and was included in the French pharmacopoeia in 1884 for its healing properties. In 1941 the chemical formula was isolated but it has been found to be too complicated to synthesize [5]. Madecassol itself has been used mostly as an injectable or oral form in the treatment of keloids. In 1965, El Hefnawi [12], an Egyptian dermatologist observed the tapering effect of a large keloid following intralesional injection of Madecassol. He subsequently reported its use in 10 other keloids. In 1967, Bosse et al. [5] reported the use of Madecassol in extensive keloids covering approximately 40% of the body surface area—the intramuscular form showed relief of symptoms, and over a period of months the scars "melted away." They reported successful use in over 800 cases. They also observed that the oral form was as effective as the injectable form.

Asiatic acid, madecassic acid, and asiaticoside are the principal terpenoids found in *Centella asiatica. Centella asiatica* has been documented to aid wound healing in a large number of scientific reports [5,12,21,22,33,34,40]. The most beneficial effect appears to be the stimulation of maturation of the scar by the production of type I collagen [4,19] and the resulting decrease in the inflammatory reaction and myofibroblast production. Collagen 1 is involved in wound healing and decreases in the skin

Fig. 1. Alpha Centella scar management program.

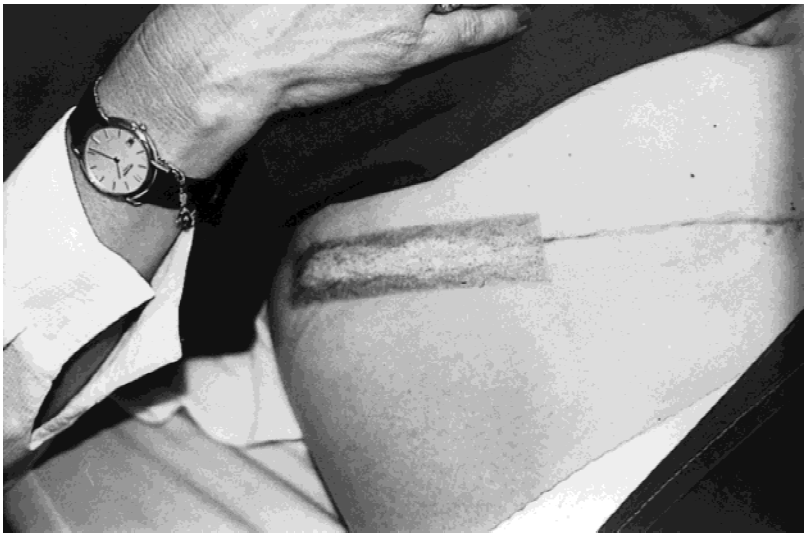

Fig. 2. White sheen evident on surface of tape following application.

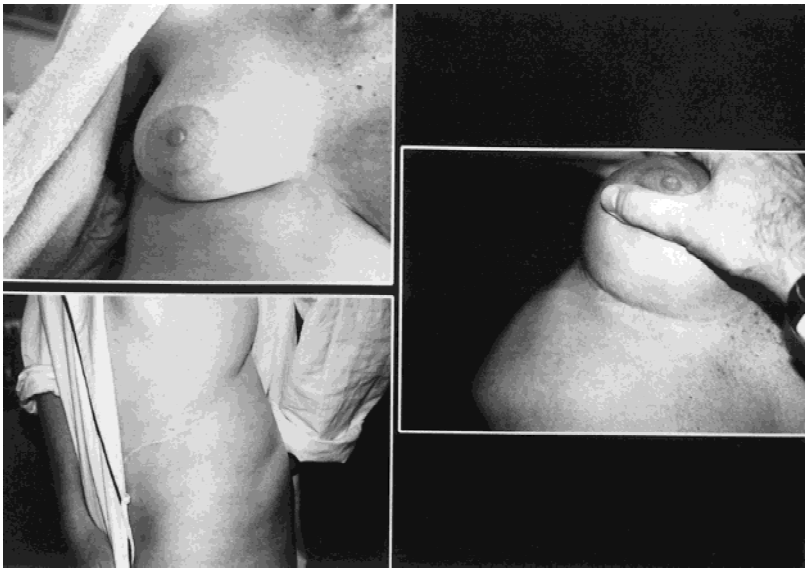

Fig. 3. Typical satisfactory scarring evident following use of the scar management program in patients following breast reduction, abdominoplasty, and breast augmentation procedures.

with age. The amount of type III collagen present in hypertrophic scars is higher than in normal scars [9]. It appears that new collagen is being laid down continually in old hypertrophic scars. The normal dermal ratio of type I to type III is 3.5:1; hypertrophic scars, 2:1; embryonic scars, 1:1 [2]. In early hypertrophic scar tissue, the presence of increased type III collagen indicates the synthesis of "embryonic" collagen. By increasing the amount of type I collagen and thus increasing the I:III ratio, *Centella asiatica* extracts may well be inducing more rapid maturity of the scar.

Although most studies have used *Centella asiatica* extracts in readily established exaggerated scarring, there are authors that have used it prophylactically, but probably not to its full potential [19]. The active ingredients that occur naturally in the leaves and stems of *Centella asiatica* are mainly the glycosides asiaticoside and madecassoside. Triterpenes, namely, asiatic acid and madecassic acid, also occur in the plant in the free state, that is, not linked to sugars as glycosides. The activity of *Centella asiatica* has been attributed mainly to the triterpenic fraction (TTF) of *Centella asiatica.* When applied topically one would ideally like to have a viable amount of the free triterpenoid available, as well as the natural blend of the glycosides for slower conversion to the genin fractions of the glycosides. Bonte et al. [4] suggested a total viability limit of 15–20 μg/ml of the total purified extract. This translates to the concentration of 2% total extract used in Alpha Centella®. In studies conducted in Italy on the total triterpenoid fraction extracted from *Centella asiatica,* a statistically important increase was observed in the percentage of collagen and in cell layer fibronectin. This effect on collagen and fibronectin may explain the action of the triterpenic fraction of *Centella asiatica* in promoting wound healing [39].

This promotion to more rapid maturity of the scar may have manifested itself in the experiments of Rosen et al. [33], and Velasco Romero [44], and Suguna et al. [38], where local application to a sutured wound of asiaticoside significantly increased the breaking strength of the wound (as opposed to vitamin E).

Thus, researching the literature over the past 25–30 years, we have identified three consistent scientifically documented essential components to scar control: *support, hydration,* and *accelerated scar maturity.* These components have been incorporated into the formulation of a scar management program—Alpha Centella® [Alphaplast International (Pty.) Ltd., P.O. Box 669, Gallo Manor 2052, South Africa] (Fig. 1). Support is provided by microporous tape. Enhanced healing is provided by the active ingredients of *Centella asiatica,* the extraction of which is extremely complex and accomplished in a standardized form in the Alpha Centella® range.

This scar program is aimed primarily as a preventative against the formation of excessive scarring. Although these agents have all been used individually and proven to be safe and effective, the combined product is uniquely advantageous. The formulated cream is applied to the surface of the tape. By combining all beneficial scar control modalities, synergism is achieved, with earlier maturity and a more favorable scar appearance. To avoid skin reactions and improve patient compliance, the taping remains in place until spontaneous separation occurs and is not removed on a daily basis as reported previously [31].

Clinical Experience—Practical Application

Once the sutures have been removed after surgery, the microporous tape is applied directly over the new scar. Let us look at the practical usage of the product. It should lie in the direction of the wound and overlap each side by approximately 1–1.5 cm. This tape remains in place until spontaneous separation takes place, usually at 7 to 10 days. The patient bathes or showers with the tape in place. Alpha Centella cream is applied to the surface of the tape twice a day—morning and night. The cream is applied in very small amounts onto the surface of the tape until a white sheen is evident (Fig. 2). After 2 to 3 min the cream is seen to have been completely absorbed into the tape. This is manifested by the dry surface of the tape. The impregnated tape then acts as a slow release of active ingredient throughout the day until the next application. Patients are instructed that the critical period for the use of Alpha Centella® cream is the first 6–8 weeks. Thereafter they should continue with taping alone until maturation of the scar takes place as manifested by a flat white scar.

Clinical Experience—Results

Our clinical experience relates to at least 6-month follow-up of the product in 106 patients. No allergies, hypersensitivities, or side effects related to the cream preparation were encountered. Hypersensitivity to the tape was encountered in 12 patients. Six of these patients did not need to stop use of the product—the sensitivity cleared spontaneously. Five of the 12 patients stopped the tape temporarily but continued with the cream, and all managed to continue with the tape–cream combination after a period of from 3 to 10 days. The remaining single patient abandoned further use of the product at 4 weeks. Patient compliance was excellent, with no other patients abandoning treatment. This undoubtedly relates to the ease of use of the product. All patients used the product postoperatively to manage their scars in a preventive capacity rather than using it in established scars. The overriding observation in these patients was the excellent end results seen in over 90% of patients (Fig. 3) and the hastened occurrence of scar maturity in these cases. This averaged 3 months, as opposed to the 4- to 6-month period seen prior to product use. No scar revision procedures were necessary in any of the 106 patients.

Thus, the scar management program has fulfilled all the criteria for successful scar control. Although these

results are presented in a nonformalized manner, scientific data will be published in the near-future. The purpose of this publication is to present the available research relating to scar management over the past 25 years and to describe the formulation of a scar management program based on these data.

Conclusion

A host of techniques and modalities for scar control have been presented over the years. It would appear that the only reason so many approaches are offered is that no one modality has had widespread success. Almost every new surgical technique and variation described is eventually tried as a method for reducing the extent of scarring. Laser scar therapy [25], at best, is no better than that achieved with the less expensive combination therapy suggested above.

When discussing the management of scars, it is important to differentiate between keloid and hypertrophic scars, as the pathogenesis is different in each case, a genetic predisposition playing a strong role in keloid formation [41]. This paper describes modalities aimed at producing a cosmetically acceptable scar following surgery or injury—keloid scarring demands different approaches.

Our current therapy, based on tried and tested methods, but now used synergistically in one program providing support, hydration, and hastened scar maturity, has proven in our hands to be the most effective scar management yet.

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