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The Value of Medical Needling in Burn Scars

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Abstract

We aim to show that deeper needling of skin (1 mm to 3 mm depth) is a valuable tool for treating people with burn scars. Currently numerous ablative cutaneous treatments and surgical procedures are the mainstay of treating burn scars. Needling takes us to a new paradigm of instigating a regenerative phase to remove scar collagen and replace it with normal lattice patterned collagen and thereby reducing the appearance of nodular hypertrophic scars and contractions. This occurs because skin needling causes perforation of blood vessels and an automatic cascade of platelet derived growth factors etc. The dominant growth factor is TGF-β-3 which surges after needling in a way not seen in normal adult tissues but which the usual pattern is found in foetal regenerative wound repair. By repeating needling treatments one can flatten hypertrophic/ burn keloid scars and softens contractures and get closer to the ideal treatment of scars. The authors believe skin needling should be done reasonably soon after the initial injury.

Introduction

Patients with burn scar deformities frequently request help to improve both the aesthetic and functional complications of their scars. There are numerous methods available for surgeons to treat burn scars but nowadays, there is a demand for less invasive and more cost-effective procedures to give the desired benefits.

Non-invasive treatments such as silicone patches, pressure garments etc. are important ways to control scars. Minimally invasive techniques such as cortisone injections also have a place. Surgeons have generally depended on surgical interventions such as scar excision, W- or Z-pasties, and pedicled or free skin flaps to treat contractures or severe irregular scars. The quest for better results has also led to the application of many different topical therapies such as laser resurfacing, dermabrasion and deep chemical peels.

The last-mentioned methods all follow the same principal: they are ablative; they change the scar by destroying the epidermis partially or completely and scarring the dermis. The death of tissue leads to an inflammatory response. In the process of trying to treat dermal scarring the epidermis may be completely destroyed and replaced by a thinner epidermis with flatter rete ridges covering parallel-orientated scar collagen which is distinctive for scarred skin [1-3]. Furthermore, the skin becomes more vulnerable to infections [4].

The ideal scar treatment method would avoid ablation of the epidermis, and rather promote the formation of physiological dermal collagen in a lattice pattern by initiating the expression of growth factors which are relevant for scarless wound healing and regeneration of the skin. In other words, the perfect remedy would be to remove the visible defective scarring and regenerate healthier anatomically more normal skin.

In recent years, it has been shown that it is possible to a significant degree to achieve the ideal treatment by using percutaneous collagen induction or "Medical Needling" [5-7], Medical Needling is a minimal-invasive non-ablative procedure capable of improving scar quality and functionality by dermal reorganization with a decrease in scar collagen accompanied by an increase of physiological collagen and fibronectinas well as an increase of glycosaminoglycans. There is a decrease of trans-epidermal water loss because the epidermis is thicker and the stratum corneum becomes a fully functional water barrier.

Approximately 20 years ago Camirand and Doucet [8] demonstrated that by simple "needle abrasion" one could get significant clinical improvement in treating white surgical scars with a tattoo artists' device. Orentreich also reported about "dermal needling or subcision" as an alternative for treating scars and wrinkles [9]. Based on these concepts, Fernandes developed the percutaneous

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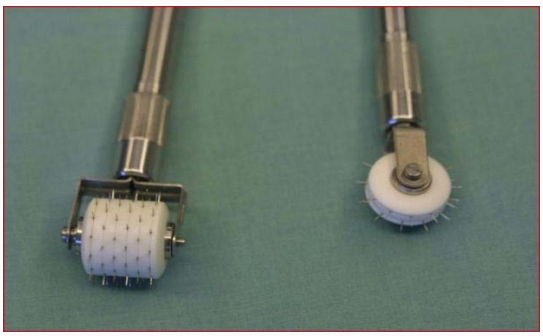

Figure 1: Needling device.

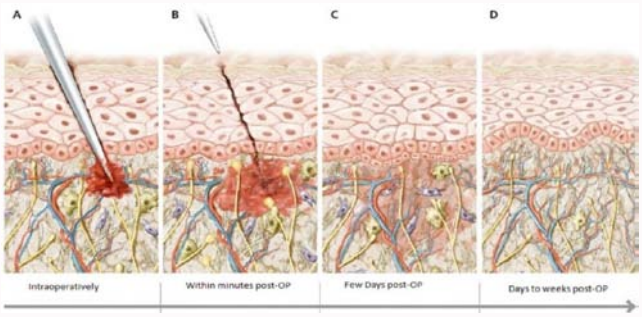

Figure 2: Schematic illustration of Medical Needling and the effects on the wound. The needle pieces the epidermis and the blood vessels of the dermis and when the needle is withdrawn the needle tract closes and by the next day it cannot be detected histologically. Within days' collagen I and elastin are generated.

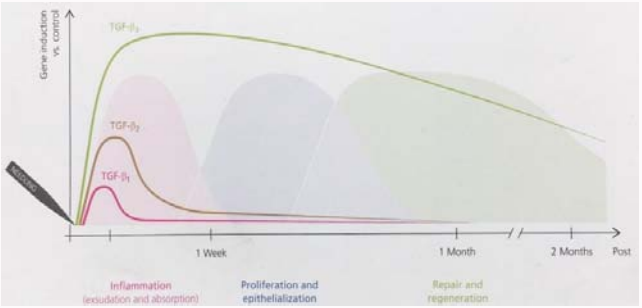

Figure 3: Microarray analyses of TGF-ß1, -2 and -3 expression level in treated animals. The induction of TGF-ß3 gene expression continues even beyond the initial wound healing phase whereas TGF-ß1 and -2 are down-regulated during the second week post-treatment**.**

collagen induction technique [10]. Thanks to targeted research within the last 15 years, impressive scientific data is now available which underlines the efficacy and safety of Medical Needling and why it works [5,7,11-15].

Science

How it works

Medical Needling is repetitive puncturing of burn scars with a roller equipped with 3.0 mm long needles that penetrate into the dermal scars and cause intra dermal bleeding (Figure 1).

The needling device is repeatedly rolled over the scar in three main directions: longitudinally, diagonally and horizontally to get the best distribution of puncture holes. According to the extent of the scar, this procedure can last 30 minutes or longer. It is important to use the device with constant pressure and do the rolling in one direction at a time to prevent shear forces. The needles are solid and do not have a lumen. Hence, they pierce the skin and mainly separate the skin cells rather than destroying them (Figure 2). They penetrate the dermis 2, 0 to a maximum of 3 mm and produce thousands of micro puncture wounds and intradermal bleeding. Some blood comes up through the channels to cause bleeding on the surface. The most important bleeding occurs in the dermis but bleeding through the skin gives us a good idea of what is happening down below in the dermis.

Induction of the wound healing cascade

This trauma initiates the activation of the physiological wound healing cascade but with a significant difference. Normally trauma causes the temporary presence of TGF-β-3 and the wound heals predominantly under the influence of TGF-β-1 and -β-2 which results in scar tissue. After needling TGF-β-1 and -β-2 rapidly disappear from the scene and TGF-β-3 dominates and that results in scarless healing and regeneration [16] Skin needling induces a new (and as yet unrecognized) phase of regenerative healing which should not be confused with the post traumatic inflammatory cascade. Platelets, keratinocytes and neutrophils secrete growth factors such as platelet derived growth factor (PDGF), fibroblast growth factor (FGF), vascular endothelial growth factor (VEGF), tissue growth factor and transforming growth factor-α and -ß (TGF-α, TGF-ß). These initiate the synthesis of dermal structures such as collagen, elastin and fibronectin and also stimulate the migration of fibroblasts and keratinocytes [13,17]. The modulation of these growth factors right at the beginning signal the differences between this new paradigm of scarless healing versus the archetypal healing with scar formation.

TGF and the induction of collagen I

This new repair and regeneration mechanism is relevant for the formation of collagen I which is the physiological type collagen in a lattice pattern in healthy skin whereas collagen III is more prevalent in parallel orientated scar collagen. TGF-β-3 makes a transient presence in standard surgical wounds and has largely disappeared within 24 hours of injury. Typical scars, we now know, are the result of dominant activity of TGF-ß1 and -2.In contrast, TGF- ß1 and -2levels are extremely low in scarless embryonal wound healing while the levels of TGF-ß3 are remarkably high [1,5,16,18].

Medical Needling particularly influences the liberation of TGF-ß1, -2 and -3. Within days' post treatment the levels of TGF-ß1 and -2 are significantly down regulated whereas TGF-ß3 reaches high expression levels even beyond the initial wound healing phase [7,13]. In support of this, the production of type I collagen was found to be increased after Medical Needling (Figure 3,4 and 5).The changes seen in skin needling indicate a lower level of TGF-beta-3 by 2

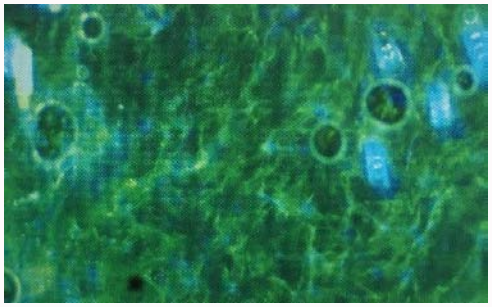

Figure 4: Immunofluorescence visualization of collagen I: Staining with antibodies directed against Collagen I (Alexa488) and DAPI. Un-needled animal of the dermis failed to react with the antibodies.

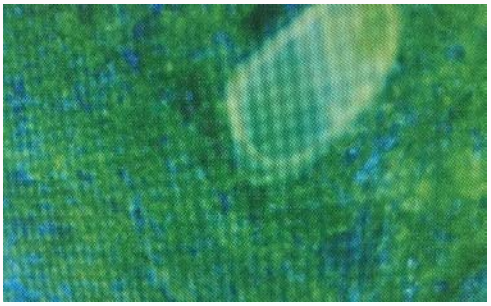

Figure 5: Immunhistochemical staining, anti-collagen I. Needled animal with 8 weeks of skincare stained without primary antibody. The amount of type I collagen was qualitatively increased in treated group compared to their controls judged by the brighter fluorescence.

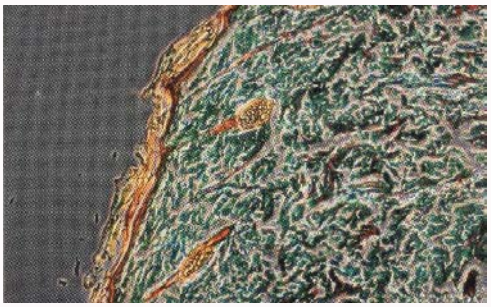

Figure 6: Masson's Trichrome staining. (a) Untreated animal (control).

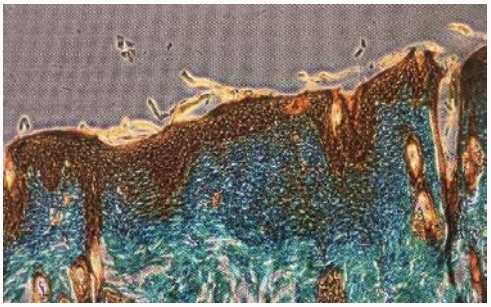

Figure 7: Needled animal with 8 weeks of skincare Collagen fiber bundles were increased, thickened, and more loosely woven in both the papillary and reticular dermis most prominently in the needled plus skincare group (D). Elastin fibers in the dermis highly linear and the epidermal dermal interface showed regular dermal papillae; cellular polarity and normal epidermal differentiation appeared to be maintained; and the elastin network within the reticular dermis was regularly thickened and organized in all groups.

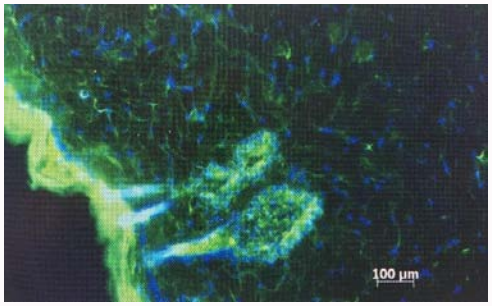

Figure 8: Immunhistochemical staining, anti-elastin. Untreated animal Immunofluorescence visualization of elastin: Staining with antibodies directed against elastin (Alexa488) and DAPI. Un-needled animal of the dermis failed to react with the antibodies.

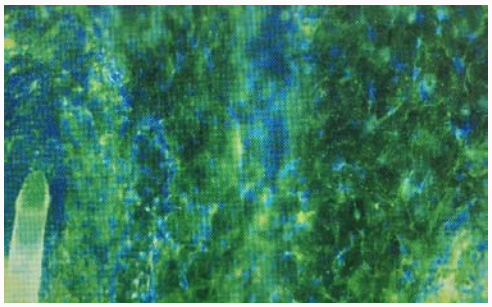

Figure 9: Immunhistochemical staining, anti-elastin. Needled animal with 8 weeks of skincare stained without primary antibody. The amount of elastin was qualitatively increased in treated group compared to their controls judged by the brighter fluorescence.

months. Ferguson's team argue that it is the initial height of the rise in concentration of TGF-beta-3 that is probably the most important influence, not so much the duration of the raised levels because as he points out the TGF-beta-3 does not stay raised for the extended healing period [19]. Studies in humans that are as yet unpublished show that TGF-beta-3 is raised after needling and become higher when needling is done at short intervals (Fernandes personal communication).

Dermal remodeling

Dermal reorganization after Medical Needling not only depends on the formation of physiological orientated collagen I but also on the inclusion of glycosaminoglycan molecules and fibronectin. This was shown in the animal model through the quantitative analysis of gene expression as well as through immunohistological analyses [13,20,21]. As seen in (Figure 6 and 7), the entire connective tissue framework appears thicker and denser post treatment.

Increase in skin elasticity

Moreover, the stimulation of the endogenous FGF contributes towards improved skin elasticity. As seen in (Figure 8 and 9), the amount of elastin is significantly higher after Medical Needling

Normalized perfusion

The secretion of VEGF during the healing phase stimulates angiogenesis and leads to the formation of tiny blood vessels in the corium. This helps to normalize the characteristic pathological erythema of scars after burn injuries. As seen in Figures 8 and 9, the amount of VEGF significantly rises after Medical Needling.

Increase in skin moisture content

Scars often appear dry and loose due to a decrease of glycosaminoglycans with a result of reduced water retention in the skin and due to thinner epidermis with increased trans-epidermal water loss. Medical Needling is associated with a higher inclusion of glycosaminoglycans (Figure 10 and 11) and with a thicker epidermis post treatment (Figure 12 and 13). Both help to maximize the moisture of the skin back to the reference of healthy skin.

Increase of epidermal thickness

In contrast to ablative treatments, the skin structures are not injured after Medical Needling. The epidermis remains physiologically intact which means that potentially side effects such as inflammation, new scarring or dyspigmentation are reduced to a minimum. Furthermore, it has been shown in the animal model that the thickness of the epidermis increases up to 140 % after treatment

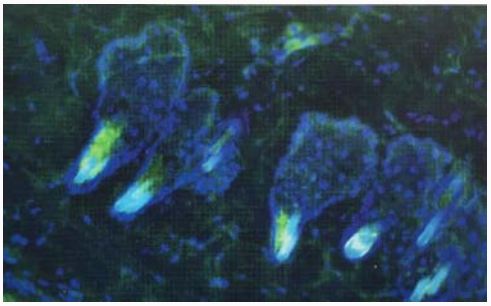

Figure 10: Immunofluorescence visualization of GAGs (Alexa488-conjugated) and DAPI (representative example). Un-needled animal with 8 weeks of skincare. GAGs showed dense deposits occupying much of the dermis, leaving only isolated collagen bundles visible (white arrows).

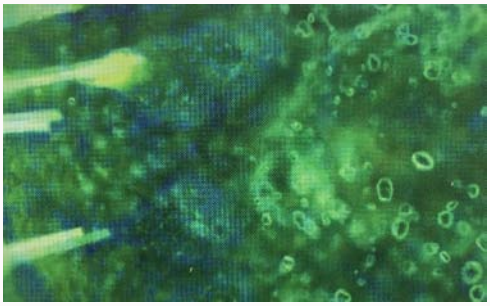

Figure 11: Immunhistochemical staining, anti-GAG Needled animal. Needled animal with 8 weeks of skincare stained without primary antibody. As observed in the PAS staining a marked increase in the amount of GAGs was observed throughout the different needled groups in comparison to the un-needled groups.

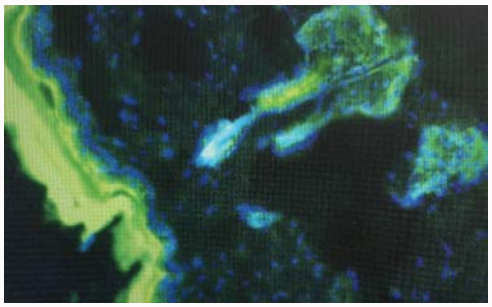

Figure 12: Immunhistochemical staining, anti-VEGF. Untreated animal Immunofluorescence visualization of elastin: Staining with antibodies directed against elastin (Alexa488) and DAPI. Un-needled animal of the dermis failed to react with the antibodies.

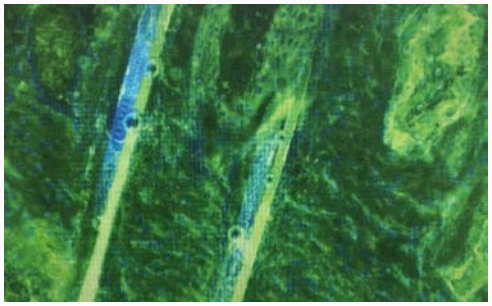

Figure 13: Immunhistochemical staining, anti-VEGF Needled animal. VEGF showed a membranous staining pattern along the intercellular junctions in the basal and suprabasal layers of the epidermis. A brighter fluorescence indicates that the amount of VEGF in the dermis is augmented in the needled groups compared to the un-needled groups.

versus untreated ones [22] (Figure 6 and 7).

Role of vitamins in wound healing

Maximal post Needling improvement was seen in combination with pre- and post-treatment of the skin with vitamin A and oxidants Vitamin C and E (Figure 6 and 7).

No dyspigmentation after Medical Needling

A disadvantage of ablative scar treatments is that there is an increased risk of dyspigmentation especially in darker skin types [23-25]. On 480 patients, it has been shown that there is no risk of dyspigmentation after Medical Needling.(26)Furthermore, Medical Needling does not change the number of melanocytes but the expression levels of Melanocyte-stimulating hormone (MSH) and Interleukin-10 (Il-10) are modified [26]. MSH which influences the proliferation and activity of melanocytes is significantly down regulated within days after treatment. Il-10 as an anti-inflammatory cytokine is upregulated post operatively [20]. In a subsequent study, it has been shown that it is not possible to re-pigment larger areas of hypopigmented skin with Medical Needling alone.

Re-pigmentation of hypopigmented burn scars with Medical Needling and Non-cultured autologous skin cell suspension (NCASCS)

Currently numerous methods are available to treat hypopigmented skin, such as split skin grafting [27,28] lasers [29-31] and cultured skin cell transplantation(30-32) In recent years, research focused additionally on Non-cultured skin cell suspension. The Autologous Cell Harvesting Device is used to create a spray suspension of living autologous skin cells. These cells are harvested intraoperatively and directly applied, in suspension, to the prepared wound. In order to prepare an area for treatment with NCASCS, the wound has been first treated by using dermabrasion or lasers which are both ablative methods. By their nature, ablative treatments remove skin structures and cells, including the basement membrane, which are replaced by a thinner epidermis with flatter rete ridges [1,2,32]. This initiates an inflammatory response that stimulates fibroblasts to produce parallel oriented scar collagen instead of physiological lattice pattern collagen [1,33]. Additionally, the risk of dyspigmentation increases after these ablative treatments due to associated damage to the melanocytes [34,35].

An ideal wound preparation for the autologous cell suspension would be a treatment that does not destroy structures of the epidermis yet creates a conduit that allows ingress of melanocytes that promotes the formation of physiological collagen instead of scar collagen and initiates the expression of growth factors. As we described above, Medical Needling offers all these advantages. To combine both procedures it is at first necessary to prepare a depigmented scar with intense medical needling. Afterwards the autologous cell suspension is applied through a spray syringe on the wound. The hypothesis is that the melanocytes of the cell suspension link through the epidermal canals onto the basal membrane. In a pilot study with 20 patients it has been shown that it is possible to get marked subjective and objective improvements regarding re-pigmentation with the combination of Medical Needling and NCASCS [36].

Conclusion

Medical Needling offers anew alternative treatment for burn

scars by changing pathological scar collagen with thousands of micro punctures into a scar with normal collagen. When we prick skin, we puncture blood vessels and the release of platelets signals the release of growth factors which then improve dermal collagen, vascularization and epidermal thickening. Medical Needling liberates growth factors like VEGF and TGF-ß3 which initiate the replacement of parallel, packed scar collagen by physiological collagen I in a lattice orientation, with added elastin and glycosaminoglycans. The scars after Medical Needling tend to appear smoother, softer and less itchy and much less obvious. The skin becomes altogether more elastic and as a result contractures are also softened. And in some cases, invasive surgery to treat contractures becomes un-necessary. Skin needling is showing us that it relieves tensions in tissues and minimizes the need for Z-Plasty and major flaps. Early skin needling could help avoid scar contractures which is one of the most crippling features of burns. A significant problem arises for young girls as their breast develops, because the breast tissue is entrapped by scar tissue and does not develop properly. Skin needling is worth doing for these patients and should be done as early as possible after the burn injury. There is good reason to believe that if we can change the spectrum of tissue healing dominated by TGF-beta 1 and 2 in the acute phase and convert it into a regenerative phase promoted by TGF-beta-3 that we will have long term effects and avoid contractures. Our experience at this stage is largely on treating well established burns but the authors feel that skin needling should become a part of the early management of burn scars. For re-pigmentation, it offers the ideal pre-treatment for Non-cultured autologous skin cell transplantation. Both treatments preserve the epidermis which results in a reduced risk of new scarring or dyspigmentation.

Medical needling offers a treatment that for the first time in medical history can cause regeneration of tissue and soften burn scars, reduce contractures. When done repeatedly and intensively then burn scarred tissue can seem to be almost normal unscarred skin. However, the timing probably is of utmost importance. The authors believe skin needling should be done as soon after the burn injury as is reasonable because they have had experience treating burns within a few hours to days of the initial burn injury and it seems the earlier the needling, the greater the chance to heal with minimal scars.

Skin needling needs to be understood by clinicians treating burns so that this valuable technique can be offered to as many burn victims as possible.

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