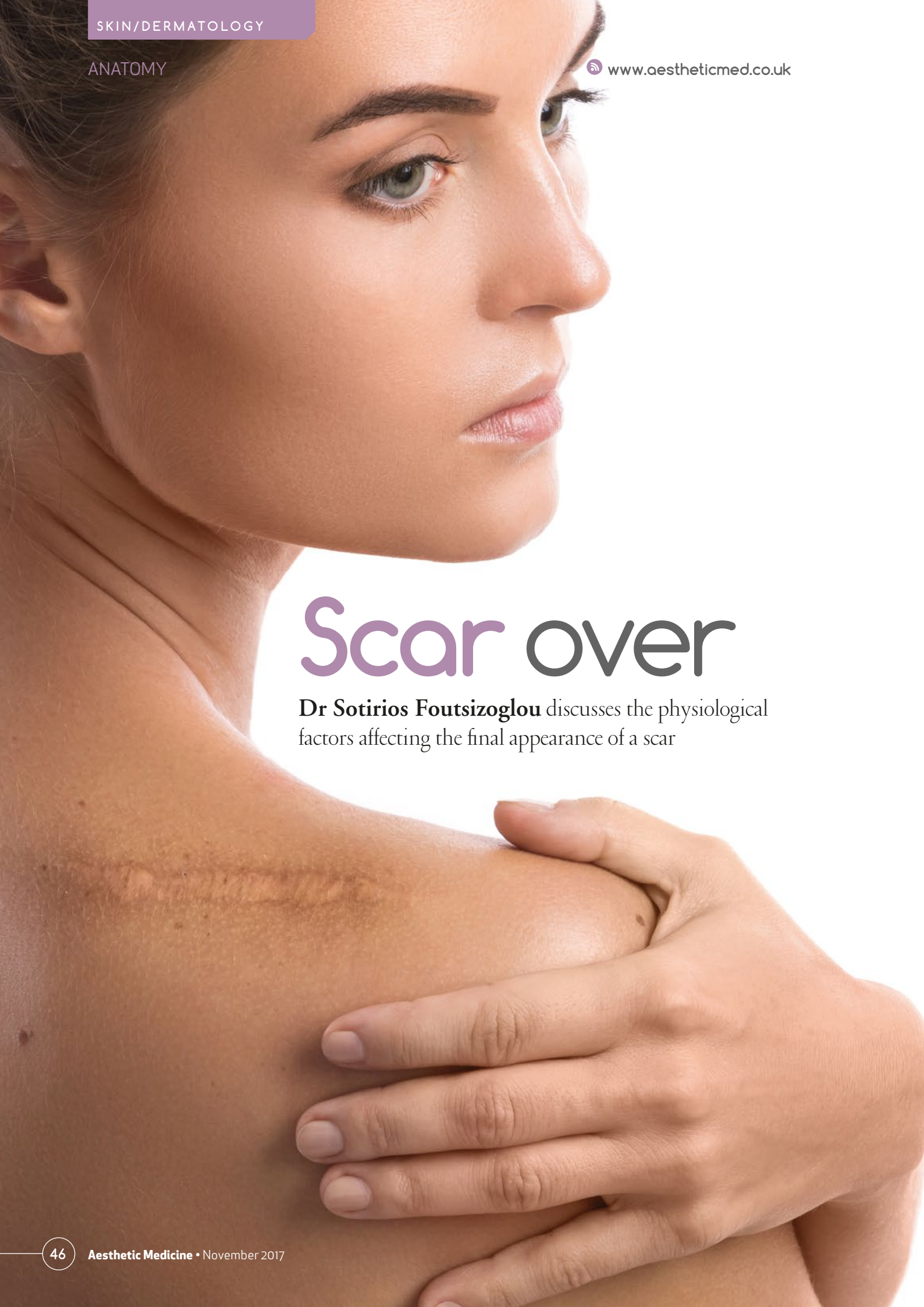

Scar over

Dr Sotirios Foutsizoglou discusses the physiological factors affecting the final appearance of a scar

Even the most intelligent patients ask this question: "will there be a scar?" When an injury occurs to the skin involving most of the dermis or an incision is made, there is always a scar. The question should be, why some people develop a fine inconspicuous scar whereas others tend to develop unsightly scars, even in favourable anatomical locations.

Following my previous article on collagen synthesis in wound healing, this issue's article will address the factors affecting the final appearance of a scar. Remember that skin scars are the sequelae of the wound healing process following a breach in the epidermis and dermis. Interestingly, the same incision or wound in two different patients will produce scars that differ in quality and aesthetics. For example, oily and dark skin produces less attractive scars than thin, pale (Fitzpatrick I and II), older, and dry skin. Rules are made to be broken, however, and an occasional patient will develop a scar that is not characteristic of his or her skin type.[1]

OBTAINING A FINE-LINE SCAR

The final appearance of a scar is dependent on many factors, including the following: a) differences between individual patients that we do not. yet understand and, therefore, cannot predict; b) the type of skin and location of the body, such as chest or shoulder scars tend to widen or become hypertrophic. Conversely, eyelid incisions almost always heal with a fine-line scar; c) the tension on the closure; d) the direction and size of the wound and its anatomical boundaries; e) other local and systemic conditions, such as infection or malnutrition; and lastly, f) the suturing or surgical technique.

Descriptive Classification of Scars

Scars may be flat, thin, wide or stretched-out, depressed, trap-door, contracted, hyper- or hypopigmented, raised, hypertrophic, or keloid

Skin loses its elasticity with age. Stretched-out skin, combined with changes in the subcutaneous tissue, produces wrinkling, which makes scars less obvious and less prone to widening in older individuals. Children, on the other hand, may heal faster but their elastic skin and abundance of collagen may lead to a thicker and wider scar. In addition, as body parts grow, the scars become proportionately larger.

Fig.1. RSTLs are perpendicular to the underlying muscles of the face. Ageing tends to accentuate the appearance of RSTLs.

The direction of a laceration or excision also determines the eventual appearance of the scar. Elective incisions or the excision of lesions are usually planned to be parallel to the relaxed skin tension lines. Maximal contraction occurs when a scar crosses the lines of minimal tension at a right angle. Wrinkle lines are generally the same as the relaxed skin tension lines (RSTLs) and lie perpendicular to the long axis of the underlying muscles (Fig. 1).

The shape of the wound also affects the ultimate appearance. The "trapdoor" scar results from a curvilinear incision or laceration that, after healing and contracture, appears as a depressed groove with bulging skin on the inside of the curve.

Local conditions, such as crush injury, or systemic conditions such as vascular disease, also affect the scar. Extreme malnutrition of vitamin deficiency may be a factor in scar formation[1]

Atraumatic technique of approximation of the skin edges, debridement of necrotic tissue or foreign material, and a tension-free closure are the first steps in obtaining a fine-line scar. When sutures are used, consideration should be given to choosing the appropriate material. The skin edges should be approximated in an everted fashion as everted wound closures are always associated with a better looking scar. In addition, placement of sutures that are not excessively tight and are removed promptly are two technical factors of definite importance in increasing the likelihood of a "good" scar. Ultimately, however, scar formation is unpredictable even with meticulous technique.[1]

SYMPTOMATIC SCARS

At the end of the proliferative phase of wound healing, the collagen deposition and resorption is at equilibrium and the wound strength is approximately 50% of normal. From this point on, as the scar remodels, it should gradually soften, becoming pale and asymptomatic, leaving a fine scar at 12-18 months after injury.

In excessive cutaneous scar formation, wounds heal with an over-proliferative response, producing excessive cells and extracellular matrix components including collagen and ground substance. The collagen is predominantly immature type III and disorganised. There are higher levels of soluble collagen and collagenase indicating a greater wound turnover.[2] Excessive scar formation can be classified as either hypertrophic scarring or keloid formation. Both are manifestations of overexuberant scarring, although the upstream aetiology is probably different.[3]

KELOID SCARS

Keloids are less common and have a genetic component that limits them to < 6% of the population, primarily the young black and Asian populations < 30 years of age.

Macroscopically, these scars are thickened and elevated, extending beyond the borders of the original wound. Histologically, keloids are characterised by the overgrowth of dense fibrous tissue with large, thick collagen fibrils laid down in an irregular pattern with less evidence of cross-linking than in normal scars. There is also an increase in vascularity.

These scars can occur up to a year after trauma or with no defined injury. They reach a specific size and remain at that size for many years without regression. Keloids most commonly occur on the face, earlobes, and anterior chest (Fig. 2). >

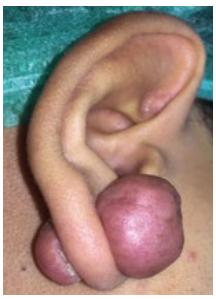

Fig. 2. Keloid scar on earlobe

HYPERTROPHIC SCARS

Hypertrophic scars are also characterised by the formation of dense collagen fibres following injury but, in contrast to keloids, do not extend beyond the original wound margins. They are more prone to forming disabling contractures and are a near-universal outcome following extensive deep burn injury.

The scars usually become more evident one to two weeks after epithelial closure. They are more responsive to treatment than keloid scars.

Hypertrophic scarring can occur at any age but mainly < 20 years, and there is a familial inheritance. Males and females are equally affected.

The aetiology can be idiopathic or secondary to wound infection, dehiscence, or tension. Wounds with a higher risk of becoming hypertrophic include chronic wounds or those with an increased inflammatory phase, wounds not in RSTLs, wounds in areas of high tension such as the anterior chest, shoulders, and anterior neck, and wounds that heal by secondary intention (Fig. 3).

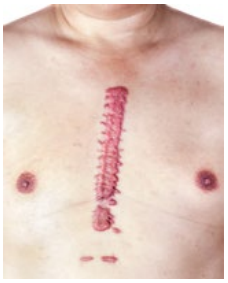

Fig. 3. Hypertrophic scars of the anterior chest

MANAGEMENT OF ABNORMAL SCARS

The exact aetiology and pathophysiology of excessive scar formation remains unknown. Many theories have been proposed to account for the fibroproliferation observed in hypertrophic scar and keloid formation, including mechanical strain, inflammation, bacterial colonisation, and foreign body reaction. Unfortunately, investigation of the mechanisms underlying these diseases has been hindered by the absence of animal models that reproduce the characteristics of human overscarring.

> Scar formation and the response to injury are still poorly understood by scientists and clinicians alike

Modalities employed to reduce the unsightly appearance of excessive scarring include:

- Pressure therapy (e.g. silicone sheeting, elasticated compression garments) may have a mechanomodulatory action increasing the collagenase activity and decreasing the wound metabolism, leading to an early maturation of the scar. Ideally, the pressure should exceed normal capillary pressure (24 mmHg). Pressure applications are applied when the wound is fully healed and need to be worn for 18-24 hours a day to show some effect.[2]
- Silicone gel may exert its therapeutic action either by a direct chemical effect or by causing wound hypoxia and increased hydration with success rates of more than 80% for hypertrophic and 35% for keloid scars. Other occlusive materials such as hydrogels have been trialled with similar success rates to silicone.
- Corticosteroids (intralesional triamcinolone) reduce collagen levels either by increased activity of collagenase due to reduction in wound α2-macroglobulin content or reduced collagen deposition due to a reduction in fibroblast activity. Steroids also reduce inflammation.
- Topical retinoic acid and vitamin E inhibit fibroblast production and reduce fibroblast numbers respectively.
- 5-Fluorouracil (5-FU), a thymidylate synthase (TS) inhibitor, interferes with DNA replication and cell division. Other drugs such as penicillamine, colchicine, interferon and cyclosporin A have also been used with variable results.
- Laser can reduce pigmentation and shortens the inflammatory phase of wound healing. The prolonged secretion of inflammatory cytokines has been shown to induce fibrosis.
- Surgery (e.g. serial excision, realignment of the scar along RSTLs to reduce tension, flaps, or grafts) should be reserved until the scar has matured, unless it is causing a contracture. The patient should be warned that surgery may result in a worse scar. Recurrence rates and outcome can be significantly improved by combining surgery with adjunctive treatments such as external beam irradiation and steroids.

CONCLUSION

Wound healing is one of the most complex physiological processes. During the progression from an injury to a stable scar, the intrinsic and extrinsic clotting system is activated; there is an acute inflammatory response; neovascularisation proceeds through angiogenesis and vasculogenesis; cells proliferate, divide, and undergo apoptosis; and extracellular matrix (ECM) is deposited and remodelled. These occur simultaneously and also interact and influence each other at the level of gene transcription and protein translation in a dynamic and continuous fashion. Thus, it is not surprising that scar formation and the response to injury are still poorly understood by scientists and clinicians alike, except at a purely descriptive or empirical level. The sheer number of commercially available products of unproven efficacy is a testament to the lack of mechanistic understanding of wound healing and scar formation. Several stem cell populations have been identified in the skin and are increasingly studied as potential therapies for wound repair without scarring. These progenitor populations include epidermal stem cells, hair follicle stem cells, and adipose-derived stem cells that have the capacity to restore almost all skin compartments.[4,5] The question for the researchers is how to exploit these powerful cell populations to promote cutaneous repair in disease states or following injury. **AM**

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Dr Sotirios Foutsizoglou developed a particular interest in anatomy during his time working in plastic and reconstructive surgery in the NHS. He became heavily involved in teaching anatomy and physiology to medical students and junior doctors and has worked as an anatomy demonstrator for Imperial College. He is currently completing his last year of training in Plastic and Reconstructive Surgery at Evangelismos General Hospital of Athens. Since 2012, in his role as the lead trainer of KT Medical Aesthetics Group, he has been training practitioners in facial anatomy and advanced non-surgical treatments and procedures. He has written and lectured on facial anatomy and complications associated with injectables both nationally and internationally.