The Relationship Between Keloid Growth Pattern and Stretching Tension

Visual Analysis Using the Finite Element Method

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Background: Keloids grow and spread horizontally, like malignant tumors, for reasons that remain unknown. Yet, stretching tension is clearly associated with keloid generation, as keloids tend to occur on high tension sites such as the anterior chest and scapular region. Thus, we analyzed the relationship between keloid growth patterns and stretching tension using a visualized finite element study.

Materials and Methods: Keloids, normal skin, and fat structures were reproduced using DISCUS software. The contours were transferred to ADINA analytical software to rebuild and mesh volumes.

Results: (1) High tension was observed at the edges, and not in the entire region, of stretched keloids. (2) Keloid centers were regions of low tension, which helps to explain the healing that generally occurs in the central regions of keloids. (3) Expansion of a keloid occurred in the direction in which it was pulled. (4) The "crab's claw"-shaped invasion occurred in response to increased stretching tension. (5) Skin stiffness in the circumference of a keloid was associated with greatly increased tension. (6) Fat hardness and thickness did not influence the amount of tension. (7) Adhesion with subcutaneous hard tissue greatly increased the tension in the keloid.

Conclusion: These stretching results have advanced understanding of keloid formation under various conditions. Our results suggest that stretching tension is an important condition associated with keloid growth.

Key Words: keloid, growth, invasion, finite element method, analysis, tension

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Keloids grow and spread, ie, as a "crab's claw."[1] Keloid growth resembles that of malignant tumors, except keloids do not invade other organs. Although the physiological mechanism of this growth pattern remains unknown, stretching tension is clearly associated with keloid generation, as keloids tend to occur on high tension sites such as the anterior chest and the scapular region.[2] Because of the lack of objective evidence, there has been disagreement about whether a site of frequent keloid formation, such as the earlobe, is under tension or not. As for earlobe keloids, we suspected that the earlobe would be an intermittent site of high tension caused by pillow stretching in the lateral or prone sleeping position.[3] Thus, we thought that stretching tension would play an important role in keloid development or hypertrophic scaring.

We used a visualized finite element method (FEM) to clarify the relationship between the keloid growth pattern and stretching tension as this method has been used to obtain approximate solutions for stress and strains in the field of engineering. It is an appropriate method for representing the irregular geometry of skin. The most important advantage of this model is its ability to test an unlimited number of force application systems once an adequate FEM has been created.[4]

METHODS

Obtaining Anatomic Shapes in 3D

The analysis was performed using a personal computer (Pentium 4: 2.4 GHz with 1 GB memory, Intel Corp, Santa Clara, CA) equipped with DISCUS drawing software (version 3.01, rexsoftware, Osaka, Japan) and ADINA analytical software (version 8.08, ADINA R&D, Inc., Watertown, MA). Photographs of typical keloids, including those of normal skin, were taken, and their shapes were traced on a computer. DISCUS drawing software was used to acquire the external contours of the different elements of the vertical plane model (V-model) and the horizontal plane model (H-model). These models contained keloid, normal skin above the keloid, and fat under the tumor. Then, each condition was changed and analyzed.

Setting Mechanical Characteristics

All the rebuilt structures were considered individually as isotropes. Hardness values for each element of Young

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modulus were as follows: keloid 25 kPa, skin 20 kPa, and fat 15 kPa. These modulus elements were based on the work of Chow.[5] Poisson ratio was set at 0.49.

Setting Loading Conditions

Vertically and horizontally directed stress was induced to represent the stretching tension. This stress was expressed as right of or above displacement of the edges of each model. We changed the keloid, skin, and fat conditions as described below. Moreover, we analyzed the adherence of keloid with bone as:

a. Simple stretching,
b. Changing the shape,
c. Shrinking and stretching,
d. Changing the condition of normal skin,
e. Changing the condition of fat,
f. Adhering keloid with bone.

Meshing and Discretization of Elements

The contours obtained were transferred to ADINA analytical software (version 8.08, ADINA R&D, Inc.) for individual assembly, and thus, the volume of each element was reconstructed before meshing. Meshing or discretizing all the elements was carried out with 4 noded isoparametric 2-dimensional elements.

RESULTS

Simple Stretching

The simple stretching force applied in the V-model produced 2 useful values (Fig. 1). First, high stretching tension, indicated in red, was observed around the border between the keloid and normal skin. Second, low stretching tension, indicated in blue, was observed around the center surface of the keloid. In this configuration, the maximum tension was noted on the border of the keloid (4447 Pa), whereas the minimum tension was observed in the center of the keloid (232.5 Pa) (Fig. 1).

The H-model study was verified by performing stretching displacement both horizontally and vertically on a longitudinal keloid (Fig. 2). Results from horizontal stretching were the same as those from the V-model: the keloid edges had the highest tension and the central region of the keloid had the lowest tension. The maximum tension was 1756 Pa around the edge of the keloid, and the minimum tension was 1110 Pa on the normal skin in the central side of the keloid. In addition, vertical stretching dispersed the tension on the edge of the keloid, with no evident concentration of tension (Fig. 2).

Changing the Shape

Idiopathic keloids usually begin as a circular rash; acne is sometimes the suspected cause. Circular keloids gradually grow to elliptical keloids, and finally to stick-shaped keloids that spread with a central healing tendency (Fig. 3).

Shrinking and Stretching

Even when purely circular, elliptical, or stick-shaped keloids were pulled, the "crab's claw" deformity in the tension was not observed in the circumference. However, the crab's claw-shaped tension was increased in a keloid which was pulled in all directions after it had shrunk (Fig. 4).

Changing the Condition of Normal Skin

By changing the hardness (ie, Young module) of the normal skin from 20 kPa to 25 kPa, the tension rose remarkably from 2977 Pa to 5081 Pa in the H-model and from 4447 Pa to 5954 Pa in the V-model (Fig. 5). When the skin in the circumference of the keloid was stiff, the tension applied to the keloid increased greatly. And the tension decreased from 4447 Pa to 3966 Pa in the V-model when the thickness of the skin was doubled (Fig. 6).

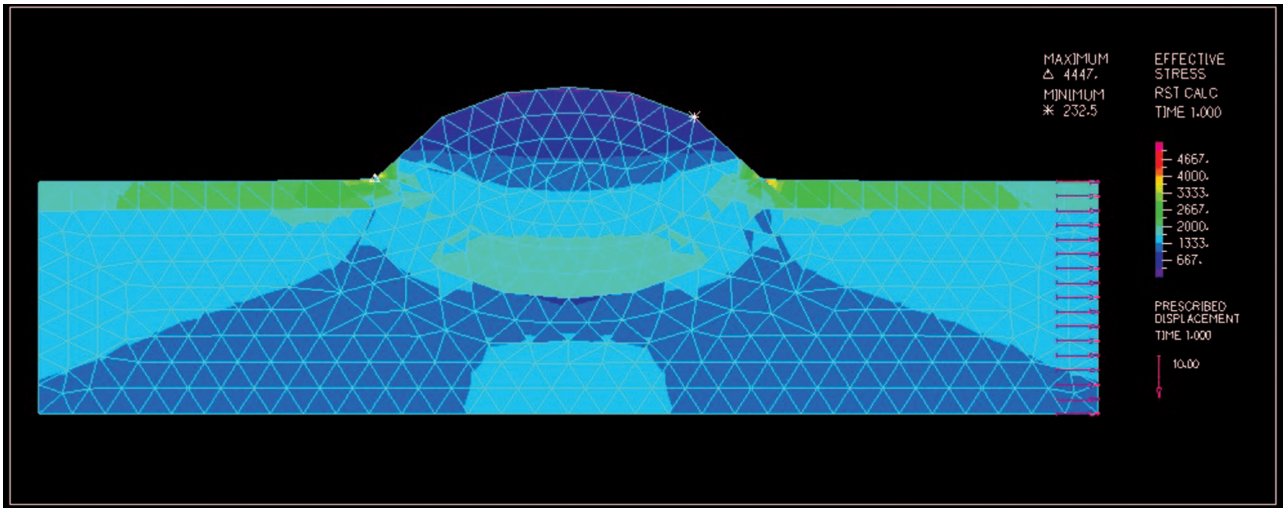

FIGURE 1. The model of simple stretching (the V-model). The tension around the border of the keloid was 4447 Pa, whereas the tension around the center of the keloid was 232.5 Pa.

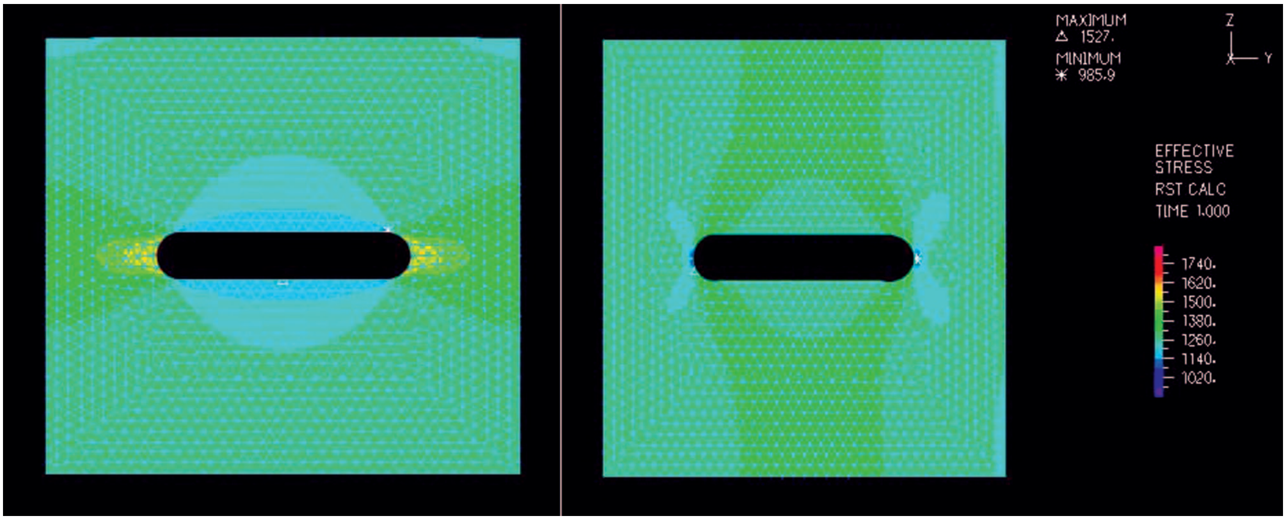

FIGURE 2. The model of simple stretching (the H-model). The stretching tension was forced horizontally (left) and vertically (right) with a longitudinal keloid.

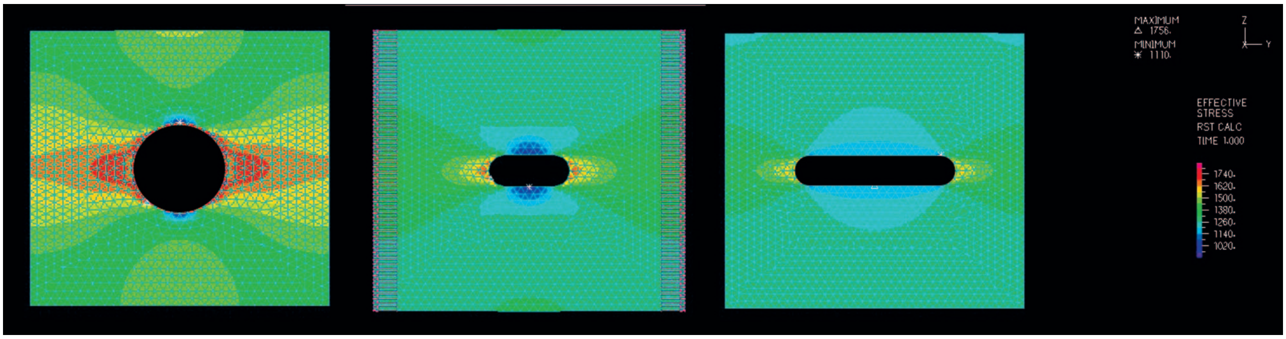

FIGURE 3. The model of changing the shape. The circular (left), elliptical (middle), and stick-shaped (right) keloid models are shown.

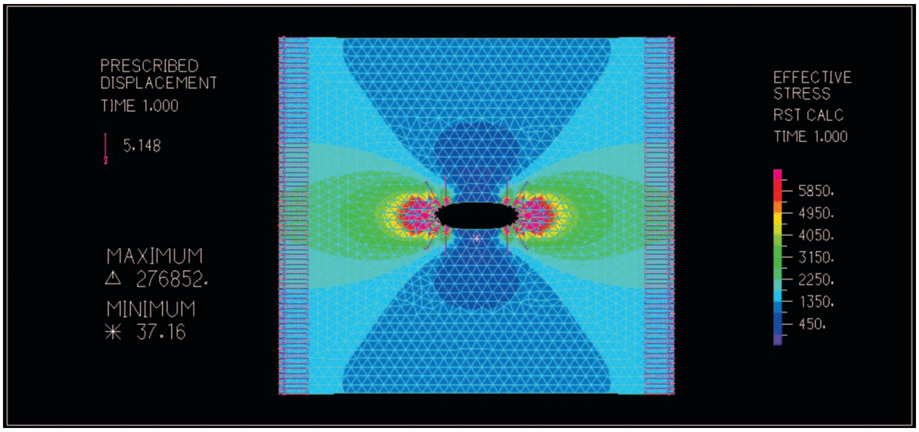

FIGURE 4. The model of shrinking and stretching. A stick-shaped keloid was pulled in all directions after it had shrunk. The crab's claw-shaped tension was clearly increased in the keloid.

Changing the Condition of Fat

Changing the hardness of fat from 15 kPa to 18 kPa (Fig. 7) and the thickness of fat by 2.5 (Fig. 8) did not increase the tension in any models. The highest tensions of each model were 4447 Pa (normal), 4561 Pa (hard fat model), 4130 Pa (thick fat model), and 4443 Pa (thin fat model). The hardness and thickness of the fat did not change the amount of tension.

Adhering Keloid With Bone

When the keloid was adhered to bone, the highest tension value rose to 6155 Pa from 4447 Pa (Fig. 9). Adhesion to

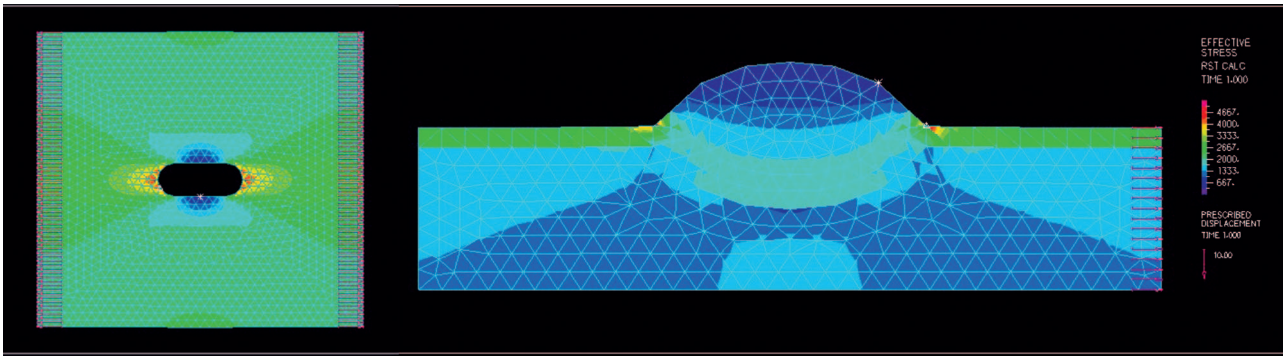

FIGURE 5. The model of changing the condition of normal skin (increasing hardness of the skin). The H-model (left) and the V-model (right) with change in the hardness of the normal skin from 20 kPa to 25 kPa. The highest tension value rose remarkably from 2977 Pa to 5081 Pa in the H-model, and from 4447 Pa to 5954 Pa in the V-model.

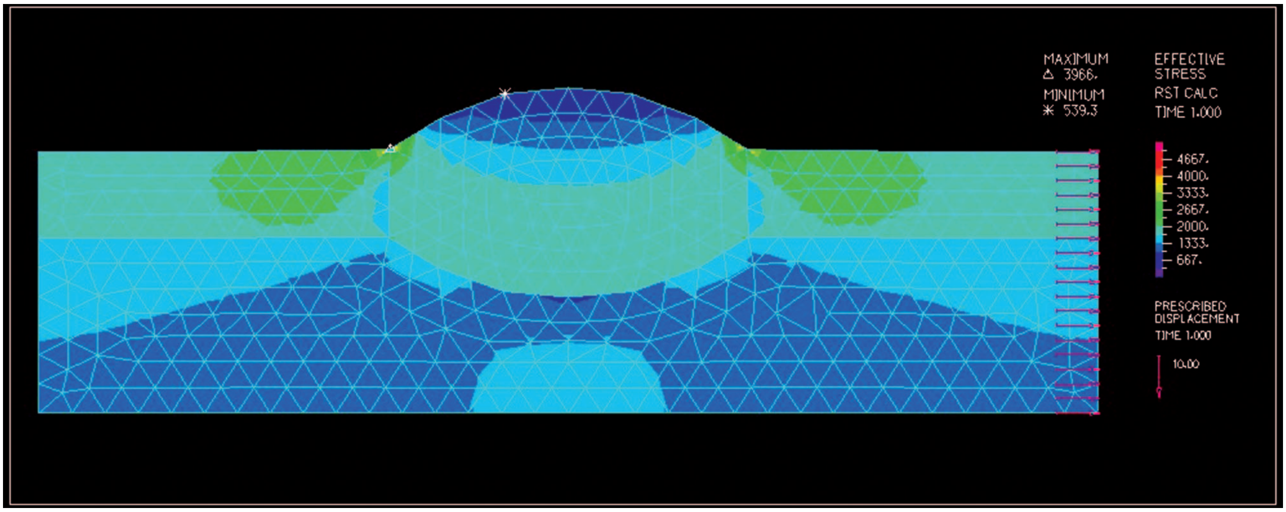

FIGURE 6. The model of changing the condition of normal skin (increasing thickness of the skin by 2-fold). The tension decreased from 4447 Pa to 3966 Pa when the thickness of the skin was doubled.

subcutaneous hard tissue greatly increased the tension applied to the keloid.

DISCUSSION

It has been proposed that the mechanical tension placed on a healing wound results in keloid formation.[6] Indeed, mechanical tension not only drives fibroblast proliferation and collagen synthesis but also dermal remodeling.[7] Anecdotal evidence suggests that incisions created parallel to skin tension lines rarely form abnormal scars.[8] And, abnormal scarring rarely develops in loosely skinned elderly patients.[9] This high-tension hypothesis has always implied that the earlobe is an exception as it is only subject to low tension. However, the earlobe is suspected to be subject to intermittent high tension caused by stretching on a pillow in the lateral or prone sleeping position.[3] It has been difficult to reveal an effect of irregular stimulation on the earlobe or a chronically infected site. So, in this study, we investigated the effect of tension simply by studying the effect of regular stimulation on a keloid or hypertrophic scar.

The FEM involves evaluating displacements for all points of the structure being studied by resolution of linear systems.[10] We investigated the effects of stress, according to the conditions of the components in this keloid model. Simple stretching revealed that high tension occurred around the edges of the keloid, and not in the entire keloid (Fig. 1). This finding is congruent with the blurred erythema observed clinically around keloids in white and Asians. Thus, the low tension in the center surface of keloids may be related to the better healing, which tends to occur in the center of large keloids. Vertical stretching dispersed the tension on the keloid edges, with no evidence of concentration of tension (Fig. 2). This dispersion supports the clinical observation that a new invasion from the middle of a keloid, which is vertical to the traction direction, rarely occurs. Adherence with the bottom floor would be required for such an invasion from the center of a keloid. In conclusion, keloid expansion occurs in the directions in which it is pulled.

The finite element analysis model indicated the same distribution of the tension (Fig. 3), but could not demonstrate the crab's claw deformity. Crab's claw-shaped tension in-

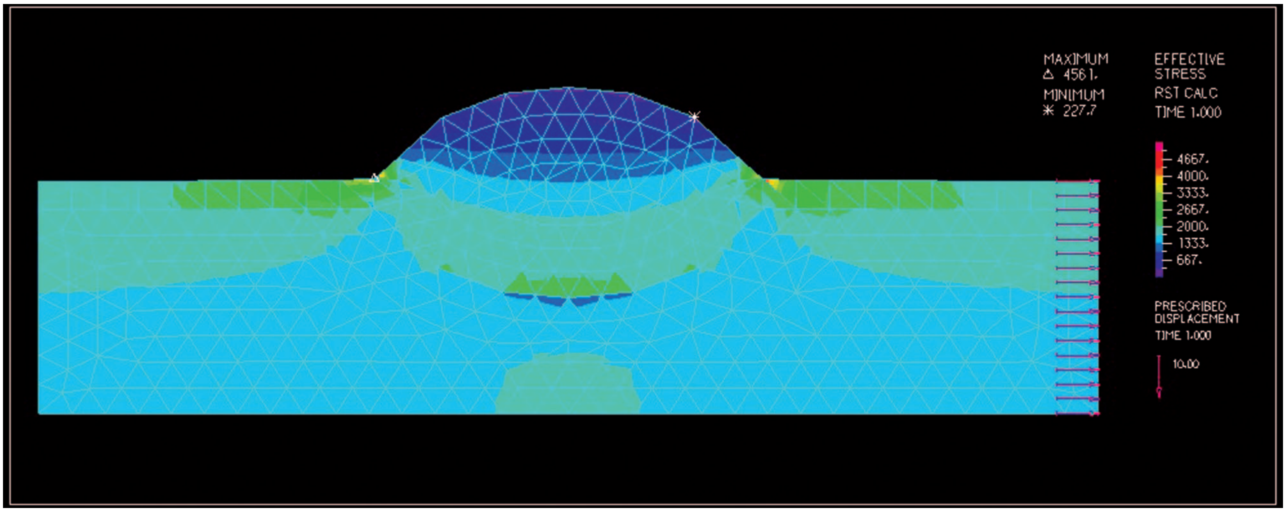

FIGURE 7. The model of changing the condition of fat (increasing the hardness). The V-model with change in the hardness of fat from 15 kPa to 18 kPa. The tension value was almost unchanged. The tension changed from 4447 Pa to 4561 Pa when the hardness of the fat was doubled.

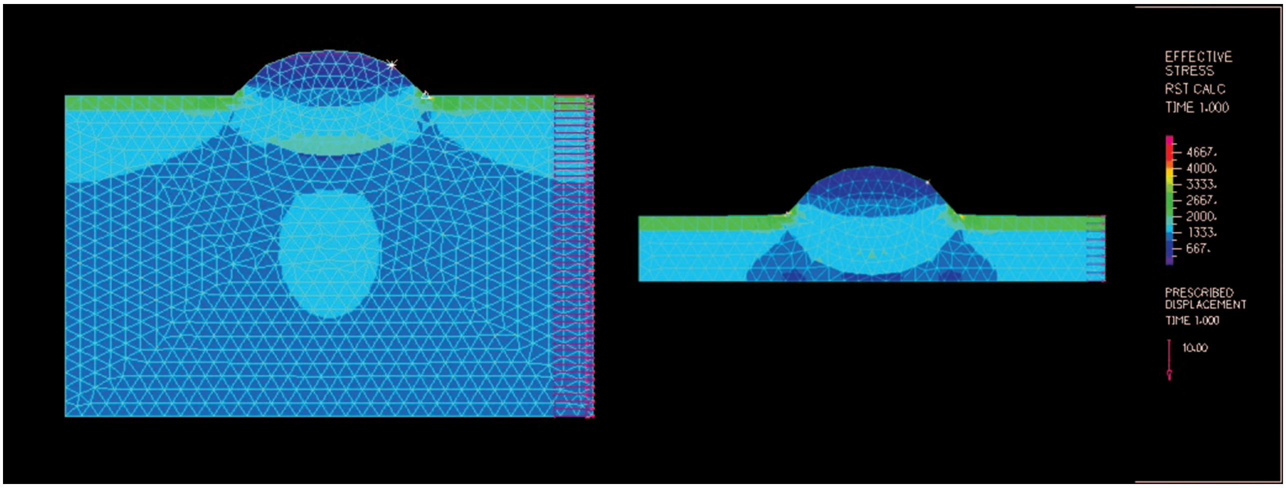

FIGURE 8. The model of changing the thickness of fat [increasing (left) and decreasing (right) the thickness]. V-model with change in the thickness of fat by 2-fold (left) and by half (right). The tension changed slightly from 4447 Pa to 4130 Pa (left), to 4443 Pa (right).

creased in a keloid pulled in all directions after it had shrunk (Fig. 4). Crab's claw-shaped invasion (which appears to be similar to malignant tumor growth) is apparently caused solely by increased stretching tension.

When the skin in the circumference of the keloid was stiff, the tension applied to the keloid increased greatly. Some clinicians have noted that the skin of keloid patients is dark, dry, and without luster,[11] but the skin condition has not been officially characterized. The hardness of the normal skin around keloids has been postulated to be related to the occurrence of keloids. A bow-shaped crease appears in the superior abdominal region of obese individuals (Fig. 10). This crease is the boundary of tension surrounding the bilateral anterior superior iliac spines. The high incidence of keloids in the superior abdominal region was previously suggested to be caused by hair,[12] but our results suggest that keloid growth in the superior abdominal region may be explained by tension.

Our results indicate that the hardness and thickness of fat do not influence tension, though the adhesion with subcutaneous hard tissue greatly increases the tension applied to a keloid (Fig. 9). The anterior chest wall is a common site for keloids,[13] but even in this location, keloids may occasionally have some normal scarring (Fig. 11). In women, partial healing of keloids in the anterior chest has been suggested to be caused by the pressure exercised by a brassiere, but the patient in this figure is a man. The increased tension because of adhesion to the sternum could explain keloid formation on the sternum, whereas regions with low tension without adhesion to the sternum could allow normal scarring to occur. In addition, the inferior part of this keloid in the epigastric region (Fig. 11) may be caused by the tension exercised by

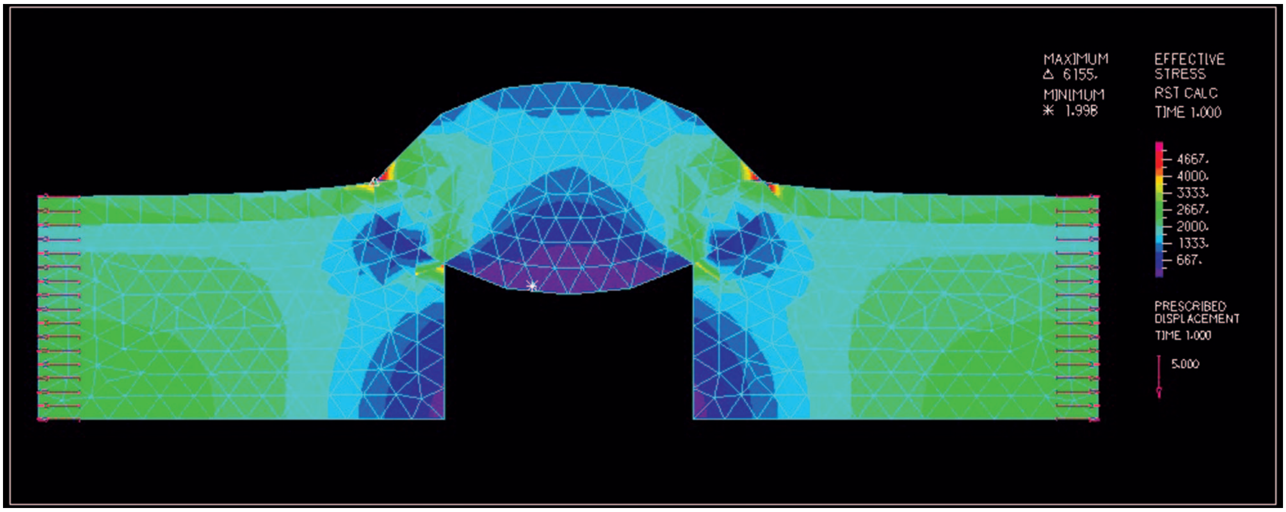

FIGURE 9. The model of adhering keloid with bone. When the keloid was adhered to the bone, the highest tension value rose to 6155 Pa from 4447 Pa.

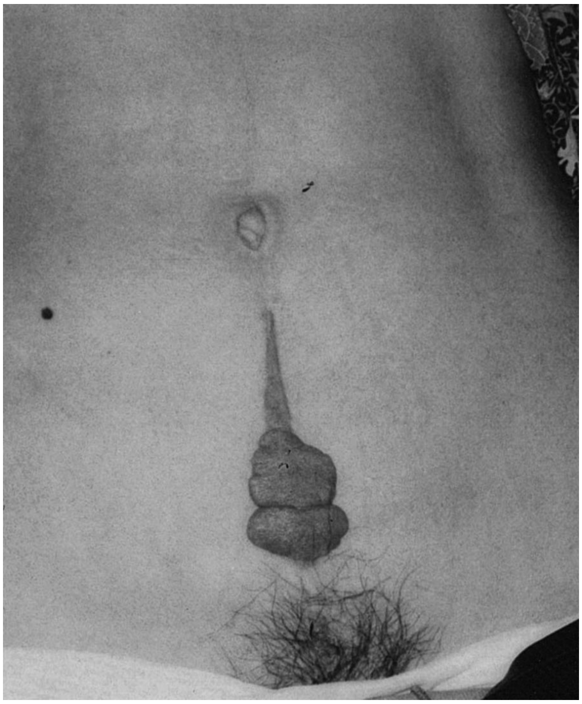

FIGURE 10. A case with both keloid and hypertrophic scar. In this case, the keloid and hypertrophic scar exist simultaneously. According to our results, the difference between keloid and hypertrophic scar generation relates to the boundary of the tension of the surrounding skin, not to blocked sebaceous glands or ingrown hairs.

abdominal breathing. Finally, it is important to show how the information presented in this study will be of use to basic science researchers and clinicians. Skin inflammation causes keloids or hypertrophic scars because of abnormal regulation of the collagen equilibrium, and leads to a large collagenous mass, which distinguishes it from a normal scar. Skin inflam-

FIGURE 11. A case with both keloid and normal mature scar. The anterior chest wall is a common site for keloids, yet keloids in this region sometimes contain normal mature scar tissue. According to our results, this case could be explained by adhesion of the keloid to subcutaneous hard tissue, which would greatly increase the tension applied to the keloid.

mation is caused by various stimuli, such as chronic infection, blocked sebaceous glands, ingrown hairs, foreign bodies, and stretching tension. We suppose that stretching tension also causes skin inflammation and accelerates the inflammation induced by other factors, such as infection; however, nobody knows if stretching tension leads to skin inflammation or not. Hence, we suggest a new hypothesis termed the "Neurogenic inflammation theory" of keloid and hypertrophic scar. Neurogenic inflammation is defined as cutaneous antidromic vasodilatation and plasma extravasation, which is mediated by the release of neuropeptides from sensory endings.[14] The activity of neuropeptides in skin inflammation can be observed in the form of erythema, edema, hyperthermia, and pruritus.[15] This phenomenon coincides with the appearance of keloids and hypertrophic scars.

Mechanosensitive nociceptors with unmyelinated axons (C-fibers) are stimulated when the skin is stretched.[16] Additionally, local erythema results from axon reflex and antidromic sensory nerve stimulation-induced release of vasodilative factors (substance P, calcitonin gene-related peptide).[15] Substance P upregulates the transforming growth factor-β1 gene, which is expressed at higher levels in keloid fibroblasts.[17,18] Conclusive evidence supporting this new theory comes from leprosy, a granulomatous disease caused by *Mycobacterium* that leads to neuropathy, but never to hypertrophic scaring.[1] Neurogenic inflammation is caused by neuropeptides; the loss of neuropeptides-immunoreactive fibers in leprosy is thought not to cause tension-induced inflammation.

The Greek word "keloid" means "crab's claw." Keloids, which have always been extremely difficult to treat, have a hereditary tendency and can be expansive. Various characteristics associated with the occurrence of keloids have been proposed.[19] But we propose that the most important cause of keloids is tension induced on the keloid, and the fat and bone surrounding the multiple forms of keloids, as we observed in our previous studies. Computer analysis of keloids, as in this study, may form the foundation of research that will advance our understanding of keloids.

CONCLUSION

We propose the following based on our results employing the FEM. (1) High tension occurred around the edges of keloids, and not in the entire keloid, when the keloid was stretched. (2) The center of the keloid had low tension, which explains the central healing tendency of keloids. (3) Keloid expansion occurred in the direction in which it was pulled. (4) The crab's claw-shaped invasion was caused solely by increased stretching tension. (5) When the skin in the circumference of the keloid was stiff, the tension applied to the keloid increased greatly. (6) The hardness and thickness of fat did not change the amount of tension. (7) The adhesion with subcutaneous hard tissue greatly increased the tension applied to the keloid.

The simulations performed in this study led to the development of a digital keloid model. These stretching results advanced understanding of the formation of keloids in various conditions.

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