SKIN SCARS AND STIFFNESS

As a prelude to the storm, I am going to speak, first of all, of a world of harmony. This world is living matter. A world where everything is connected, where empty space does not exist, a world composed of intertwined fibrils, all different and arranged in an order that is not what we expect to encounter. Order that is apparently without regularity, without hierarchy, without stratification. In other words, real disorder. Chaos in the physical sense, and therefore not without meaning. This fibrillar chaos, since this is what it is, is to be found everywhere, in every corner of our organism. It seems to constitute our very makeup, and be responsible for our form. The cells nestle within this matrix.

These fibres and fibrils that ensure the architecture of our form are greatly variable in diameter, in total continuity with each other, and possess an inherent capacity for mobility. They are flexible, unstable architectures, which slide and stretch, with divisions enclosing liquids or gels in which one can sometimes see small bubbles ascending or descending. However, this chaotic world of non-linear mobility ensures total harmony. Flexibility, elasticity, and plasticity of all our structures are explained by these fibres, like the choreography of a ballet, with each element performing a mechanical function in complete harmony with the others in order to achieve the final gesture.

To watch these fibres (them) deal with any mechanical stress smoothly, and stretch without damage to their structure, is a testimony to optimum efficiency. These are not specific structures in a precise anatomical area. No, they are everywhere, and their outer limit is defined by our external membrane : the skin. Harmony exists between all the elements…. muscles, bones, tendons, vessels, nerves, and fat lobules. All are different, depending on which cells inhabit them. Adipocytes are yellow, either shiny and bright, or sometimes very pale, and juxtaposed against each other. Muscle myofibrils are a rich Bordeaux colour. On the other hand tendons, which are white, contain very few cells, with densely packed collagen fibres. They may be transparent and loose to allow for sliding , or highly densified to confer resistance to the dermis.

All these elements work together to sustain life, their lives, and therefore ours. This is what we could call homeostasis. Red blood cells deliver energy and transmit it to the cells. Blood flow and interstitial pressure stabilise in order to facilitate exchange via controlled pressure gradients. This is mediated by the fibrillar interior architecture of enclosed spaces filled with glycoaminoglycans.

Such is the flexibility, elasticity and mobility of the structures.
This chaotic world of non linear physics and deterministic disorder is constantly seeking equilibrium. It is not static. It moves constantly, dies, and is renewed constantly. But, one day everything changes. This closed world designed to live autonomously experiences sudden destruction and exposure to fresh air.

So what happens to this harmonious fibrillar architecture? How does it cope with the aggression?

Like the aftermath of an earthquake, this harmonious equilibrium disappears in an instant. Blood flows over these structures and erythrocytes infiltrate, penetrate, and stain the tissues. The trauma causes the collapse of all the microfibrils and enclosed spaces as well as the collapse of beams and frames within the tissue. It causes the rupture of vascular and nervous energy cables . The dermis, due to its fibrillar density, retracts more strongly than the other structures, which explains the invagination of the epidermis. Interstitial pressure collapses momentarily due to fluid leakage. The interior world gets to work on closing the gap as quickly as possible. Coagulation is initiated by thrombokinasis of blood platelets transforming prothrombin to thrombin, and fibrinogen to fibrin. A blood clot consisting of fibrin and blood cells acts as a temporary enclosure to protect the wound.

After a short phase during which prostaglandins cause a decrease of pressure in the interstitial space in order to facilitate capillary filtration, oedema appears, and all the chemical factors of the inflammatory reaction come into play. The debridement phase begins. The initial trauma, and oxidation through contact with the atmosphere, leads to the damage and deterioration of the fibrils and cells, and the appearance of proteolytic agents. These agents facilitate the release of inflammatory mediators such as histamine that increase capillary permeability.

Under the influence of alpha2B Integrins, the fibres stretch, and the multimicrovacuolar spaces expand, as the glycoaminoglycans are constantly in search of hydrating elements. This is the oedematous phase, with expansion and increased perfusion of fluids, and diapedesis, which is the migration of red blood cells and macrophages through the intact walls of the capillaries.

The epidermis and dermis are stretched and painful, with reduced mobility. At the bottom of the wound, red blood cells, already yellow, mix with an exudate rich in fibrin to form the fibrin-leukocyte coating. Following the exudation phase, cell production can be observed. A waltz of histiocytes, with the macrophages keeping time, opens the ball. Then, the fibroblasts, with their contractile properties, enter the dance, secreting the collagenic microfibrillar components of the extracellular matrix. All this under the baton of the newly formed capillaries, with their thin walls emerging from the capillary loops of the uninjured tissue. A tissue called young granulation tissue forms. It is oedematous, poor in collagen and blood vessels, but rich in fibroblasts and inflammatory cells.

Then, gradually, the concentration of inflammatory cells decreases in this tissue, which is enriched with type III collagen fibrils and blood vessels. Thanks to the integrins and mechanotransduction, the newly formed microfibrillar architecture modulates the migration of macrophages and fibroblasts. This is the morphodynamic influence on the composition of the extracellular matrix. Ultimately, the gap in the tissues will be filled. A transitional fibrous tissue called granulation tissue gradually evolves. Type 1 collagen fibres replace type III collagen. The vessels disappear. (are obliterated)
Myofibroblasts in the dermis bring the edges of the wound together. Then the regeneration of the epidermis starts from the periphery of the wound.

But what remains beneath the scar several weeks and months later? Has the fibrillar harmony of the past disappeared? Will we find it as it was? Let's explore this world that has been destroyed, then repaired Has the injured tissue been reconstructed identically, or is this just a simple repair job?

The critical factor is the nature of the initial trauma. The greater the destruction, the poorer the reconstruction. We can see straight away if the scar is wide or depressed, if the harmony of the cutaneous polyhedrons has been more or less restored, or if a vast, smooth desert has replaced it. This attempt at restoration is guided by an underlying principle : wound healing is not selective. This applies everywhere, and the healing tissue remains undifferentiated for several months. Time allows for the restoration of tissue specificity, and sometimes that of a specific function. However, nature does not reshape living human matter identically, but on the contrary, often in a very crude manner.

Let's first consider the case of a simple post-surgical scar. Re-opening the incision permits us to see an area of the epidermis that has been reconstituted with polyhedrons which allow movement similair to normal mobility. The tension lines cross the scar and shape it. The constraints have once again determined the orientation of the epidermal surface. Below, the dermis is thick, hard, and white in places, with no harmonious arrangement of vessels, while at the periphery we quickly come across normal fat lobules. Below the dermis, long adhesions appear sporadically. They are flexible, mobile, and slightly retractile, with little apparent impact on the overall flexibility. Forces can still accomplish the required action. That is not the case with this scar, even though it apparently seems to be well integrated, with almost normal reconstruction of polyhedral shapes at the surface.

However, below the surface, things are quite different. We see clearly in this sequence that traction of the area over the scar is not as easily accomplished as it is at the periphery. The dermis is very hard, as if fixed, and not very mobile, indicating less than perfect function. It has lost it's arborescent vascularisation. Instead, all we find are only newly formed capillaries in dispersed pattern .The hair follicles have lost their verticality, and resemble dead trees blown over by a storm.

Very quickly we discover adhesions between the dermis and the underlying sliding surfaces. Here, the fibres are thickened and packed tightly together, and almost devoid of microvacuoles. There are no more vessels. The adhesions spread out towards an area of an old section of tendon and we come across a sort of fibrillar apocalypse, with bulging fibres, intertwined like the masts of a ship after a shipwreck. We can see scattered ropes made of type I collagen. They are thick and irregular, without inherent mobility. It looks like a real forest decimated by a hurricane. The chaos has lost it's underlying order. Flexibility has disappeared, only to be replaced by adhesions resulting in functional stiffness as end result.

Sometimes, scars are unable to reach maturity. The cause is always some sort of irritation, either by a foreign body as a plate and screw after bone fracture, the section of a nerve , or some sort of functional tension. Such is the case with this scar, which is inflamed and violet, still scabby and painful after 3 months. Incision reveals the chaotic aspect of the vessels of the dermis, and areas that peel off in an abnormal way because fusion is incomplete, with the persistence of type III collagen fibres . Then at the bottom, we come across a reddish area, consisting of small clusters of vessels. This is a sign of inflammation that will soon be explained by the strangely humid adhesions with little bubbles in their framework. They terminate in the tendon, obstructing movement and maintaining the inflammatory reaction.

But sometimes the functional impairment is evident, as in this case, where the scar invaginates with each movement of flexion. During surgical exploration we discover swollen, oedematous tissue, which is thick, fatty, and covered with the small vessels of neovascularisation indicating the persistence of the inflammatory phase. The tissues are more fragile, so we must dissect carefully. Tissue differences are not as clearly defined. Little by little we manage to isolate the adhesion, which is all embracing, release it's grip on the tendon, and restore mobility. Functional recovery

will rapidly optimize the quality of the scar; We can make the same observation in the intraperitoneal folds as, for example, this adhesion between the ovary and the abdominal wall following a caesarian section. Its remarkable (exceptional) width, its stiffness (rigidity), its monolithic appearance, and its lack of mobility are noticeable.
Sometimes intraperitoneal adhesions are more supple (flexible), but even so they are still abnormal physical links which disturb the dynamic equilibrium of the viscera.
Inflammation is very different. It is always characterised by swelling found in the structures, but especially in the sliding systems around the muscles and tendons. We observe tissue that is opaque and less transparent, with the presence of small intratissular bubbles. These bubble formations are not artefacts. They may reflect the difficulty these tissues have in managing gas exchange.
One can sometimes come across localized oedema with distinct focal areas of hypervascularity. However, sometimes the vascular reaction is invasive, like ivy on an ageing tree, which explains the redness. In this type of oedema, the cellular response is real, but does not seem to be prolific. On the other hand, we notice the presence of fibres, or groups of fibres, which swell, creating stiffness and reducing mobility and sliding . However, the difference with these scars is that the structural elements have not been destroyed. They retain a capacity to recover once the cause has been treated, which is not the case in scars where the fibrillar architecture and mobility have been destroyed.

Therefore, following the destruction of tissue, and the ensuing disorder, both the surgeon, by re-establishing correct anatomical relationships, and the manual therapist working with the tissues to restore flexibility, can bring about significant improvement and restore meaning to the original fibrillar network.