Dynamic Chiropractic

SOFT TISSUE / TRIGGER POINTS

The Fibroblast: Friend and Foe

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Ever since I learned deep massage over a tendon causes an increase in proliferation of fibroblasts, I became a fan of this most important cell. It has been demonstrated clearly that friction massage and soft-tissue mobilization with instrumentation (such as with the Graston technique) can create a change at the cellular level.[1-3] Other soft-tissue fascial-release methods also may be responsible for fibroblastic proliferation. Additional explanations, such as the gel-sol model, the piezoelectical model, and recently, the mechanoreceptor theory,[4] have been used to explain the results of mechanical load on soft tissue.

All of the ground substance surrounding our cells is referred to as the extracellular matrix (ECM), where the cells and fibers are embedded. This ECM is composed of fibrous proteins embedded in a hydrated polysaccharide gel. Many fascial-release methods are directed toward freeing "densified gel," which is part of the cause of tissue restriction. The fibroblast is a mesenchymal cell responsible for the repair and maintenance of connective tissues,[5] and is the main producer of the ECM of connective tissue.[6]

Chondroblasts, osteoblasts, odontoblasts, tenocytes and cementoblasts are specialized forms of fibroblasts. Fibroblasts produce different collagen types, depending on the tissues in which they are present. For example, proteoglycans and structural arrangements are seen in the tendon, skin and cornea. Fibroblasts are not only limited to connective tissue, but produce supportive components to organs such as the liver, lungs, nerves, uterus and kidneys.[5]

In a macrotrauma (i.e., a wound), the initial damage to the connective tissue and blood vessels causes a release of plasma proteins and blood cells (growth factors) that cause the fibroblasts to migrate into the wound bed, proliferate, and synthesize a new collagen-rich matrix (granulation tissue). As soon as the wound closes, the remodeling of the granulation tissue by the fibroblasts occurs, which can take up to six weeks, depending on the size of the wound. At the end of the remodeling phase, the fibroblast reverts back to a fibrocyte. It is theorized that the use of instrument-assisted soft-tissue mobilization, resulting in the breakdown of the fragile blood vessels typically found in restrictive fibrous tissue, initiates or accelerates the inflammatory process to completion, which then encourages subsequent stages of healing to occur, i.e., the same remodeling phase that occurs within a wound.[2,3,7]

Fibroblasts, for all the good they do, can create excessive scar tissue in postsurgical situations[8] and are the main culprits in Dupuytren's contracture. Although the underlying cause of this contracture is unknown, fibroblasts intensively proliferate and transform to myofibroblasts, which abnormally remodel the extracellular matrix.[9]

Bunker and Anthony[10] included excessive fibroblastic proliferation as a potential cause of "frozen shoulder." Their histological and immunocytochemical findings showed that the pathological process was active fibroblastic proliferation, accompanied by some transformation to a smooth-

muscle phenotype (myofibroblasts). The fibroblasts laid down collagen, which appeared as a thick nodular band or fleshy mass. They appeared similar to those in Dupuytren's disease of the hand, with no inflammation or synovial involvement. They felt the contraction acted as a "checkrein" against external rotation, causing loss of both active and passive movement. Loss of active and passive external rotation is considered the first phase of loss of motion in the capsular pattern evident in adhesive capsulitis of the shoulder. It is interesting that the "itis" in adhesive capsulitis actually may be a misnomer, since the researchers did not find any inflammation. We already know this to be true for the so-called "tendonitis" in the elbow, Achilles tendon, patellar tendon and rotator cuff.

References

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