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Joint Surgery Creates Increased Adhesion Formation

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We are taught that when there is macrotraumatic injury, such as in tendons, paratenon and surrounding peritendinous tissues, an inflammatory cascade occurs, enabling the body to heal. Why is it that with surgery, an inflammatory reaction occurs that often results in overdevelopment of adhesions and eventual restricted joint movement?

Adhesion formation with subsequent stiffness and deformity is one of the foremost obstacles in achieving predictably beneficial results in tendon surgery and all surgery around joints.¹ Doctors using soft tissue techniques often see postsurgical soft tissue adhesions occurring in the painful knee, shoulder, spine or foot; the operation was a success, but the pain lingers. The inflammatory response to the injury creates the adhesions. We are taught that inflammation is necessary for healing to occur, although in the fetus, healing occurs without a marked inflammatory response, and there is no scar tissue formation at all.²

It has been accepted that adhesions are part of the natural healing process, supplying the fibroblasts and vascularity necessary for repair.¹ Steroids are often prescribed to reduce the postsurgical inflammation and associated adhesions, but studies have shown decreased strengths of healed tendons versus controls with steroid use.³ Mulhall, et al.,¹ performed an interesting experiment to determine if thermal preconditioning prior to surgery would decrease the inflammatory response. It appears from their findings that if a cell is first subjected to significant stress, such as from heat, there is a reduced susceptibility to a subsequent traumatic stress.

An experiment was performed on New Zealand rabbits in which their Achilles tendons were incised. Eighteen hours before the operation, half of the rabbit group had their core temperature raised from 38 C to 41.5 C; the other half served as control subjects. In the rabbits preconditioned by heat, there was significant reduction, and in some cases, complete elimination of postoperative inflammation and peritendinous adhesions, without detrimental side-effects on healing and tendon strength. As in the fetus, adequate healing

occurred histologically and biomechanically, without a marked inflammatory response and scar formation.¹ In other words, normal healing occurred after macrotrauma in the absence of the usual inflammatory cascade.

It is known that tissue stress, such as from trauma, ischemia and hyperthermia, cause cellular production of "heat-shock proteins," which are intracellular cytoprotective proteins. These interact with other proteins and protect the cells and organism from additional traumatic, ischemic and inflammatory damage.

How this information relates to soft tissue methods, such as friction massage and instrument-assisted soft tissue mobilization, remains to be seen. However, we do know that soft tissue methods are extremely effective in the treatment of postsurgical paratendinous and muscular scarring.

References

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