# Stress Shielding of Ligaments Using Nonabsorbable Suture Augmentation May Influence the Biology of Ligament Healing

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Nonabsorbable suture augmentation of ligament reconstruction has seen an increase in use over the past several years with the goal of protecting the newly reconstructed ligament while allowing early rehabilitation for a potential earlier return to activity and sport. By spanning the joint with a durable nonabsorbable suture, this construct shares the stress and load seen by the reconstructed ligament, thereby protecting it from forces that could result in an early failure during the early ligamentization phase of the tendon graft. However, stress shielding of the ligament via nonabsorbable suture augmentation is also a double-edged sword, as a reduction in the stress and load seen by the ligament during this healing phase may ultimately have an impact on the final strength and composition of the reconstructed ligament. Although the long-term effects of this stress shielding have yet to be studied or reported in human subjects, multiple biomechanical and animal studies have demonstrated overall changes in architecture, tensile strength, and mechanical properties of a stress-shielded autograft ligament reconstruction. *(J Hand Surg Am. 2022;47(3):275–278.* 

**Key words** Ligament augmentation, ligament rupture, stress shielding, suture augmentation.

## USE OF SUTURE AUGMENTATION IN LIGAMENT REPAIR AND RECONSTRUCTION

Ligament injuries are commonly encountered in competitive athletes but are also seen in the general population. Due to the importance of ligaments in maintaining joint stability, ligament repair or reconstruction is often the best option for these athletes to maximize their chances of returning to play. Restoring ligament biomechanics with respect to static stabilization of the joint while allowing the force transmission is of paramount importance. The recovery process takes time to allow for progressive ligament-to-bone healing, and, for this reason, athletic activities may be put on hold.

Recently, there has been an increase in the use of suture augmentation of these ligament repairs and reconstructions by placing a stout nonabsorbable suture spanning the joint paralleling the repaired or reconstructed ligament. This suture augmentation of ligament repairs and reconstructions has demonstrated statistically significant increased time-zero valgus stability and strength in cadaveric elbow models.[1] Dugas et al[2] demonstrated comparable findings of increased resistance to gapping in similar models evaluating elbow ulnar collateral ligament repair with suture augmentation compared with ulnar collateral ligament reconstruction. As such, many surgeons now advocate for the use of this nonabsorbable suture–augmented repair and reconstruction construct to propose an earlier rehabilitation and return to play. Additionally, this protection can also

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serve to prevent ligament failure during early healing stages and reduce the incidence of revision surgeries, which have a lesser likelihood for success.[3] Although these advantages are certainly valuable in mitigating the opportunity costs associated with longer rehabilitation and slower return to play, hand surgeons must also consider the natural biology of tendon and ligament healing to ensure adequate longevity of the repaired or reconstructed ligament.

## ROLE OF STRESS AND LOAD IN TENDON/LIGAMENT REMODELING

Tendon and ligament healing occurs in multiple phases analogous to traditional wound healing, which have been well described.[4] These include an early phase, a reparative phase, and, finally, a remodeling phase during which time the tensile properties of the tissue are developed.[5] While the reparative phase is associated with a mechanically weak environment, it tends to last only for several weeks and may represent a period during which time some form of augmentation can be beneficial. During the reparative phase, collagen levels increase rapidly and reach normal levels by 6 weeks, but with less type I collagen and inferior mechanical properties than a normal ligament.[6] Beyond this, however, the remodeling phase persists for months and responds to the direction of stress that is applied.[5] The mechanical stress on the healing tendon or ligament is important in facilitating the alignment of collagen fibers and tenocytes, thereby increasing the mechanical strength. It can, therefore, be inferred that shielding mechanical stress from a healing ligament during the remodeling phase may ultimately prove to be detrimental and contrary to innate physiologic healing mechanisms (Fig. 1). The proportion of type I collagen increases with time, and the strength, stiffness, and tissue quality continue to improve up to 1 year after injury.[7] Understanding the biologic basis for ligament healing can be beneficial when discussing ligament augmentation constructs.

## BIOMECHANICAL AND ANIMAL STUDIES EVALUATING STRESS SHIELDING ON LIGAMENTS

The negative effects of stress deprivation on ligaments and tendons have been extensively studied in animal models. Through well-designed rabbit models simulating the effects of stress shielding on the patellar tendon while still allowing range of motion and return to full weight bearing, these studies were able to pinpoint various changes in the patellar tendon at the structural and cellular levels. Yamamoto et al[8] evaluated the mechanical properties of collagen fascicles in stress-shielded rabbit patellar tendons and compared them with collagen fascicles of non–stress-shielded patellar tendons. They found that stress-shielded patellar tendons at 2 and 3 weeks of stress shielding were significantly shorter than controls with larger cross-sectional areas.[8] The tangent modulus of the collagen fascicles in the stress-shielded model was significantly decreased at the 2- and 3-week timepoints.[8] The tensile strength of the collagen fascicles decreased to 74%, 44%, and 19% at 1, 2, and 3 weeks, respectively, when compared with time-matched controls.[8] The tensile strength of stress-shielded whole patellar tendons also decreased to 50%, 13%, and 9% at 1, 2, and 3 weeks, respectively.[8] There was also a statistically significant decrease in the strain at failure between the controls and the 2- and 3-week stress-shielded collagen fascicles.[8] Histologic analysis demonstrated that there was a marked time-dependent increase in the number of fibroblasts within the stress-shielded patellar tendons.[8]

In a follow-up study, Yamamoto et al[9] evaluated the transverse mechanical properties of stress-shielded patellar tendons using the same rabbit model. While their previous study evaluated the mechanical properties of collagen fascicles, transverse mechanical properties reflect the properties of interfibrillar ground substance matrix and interfiber and fiber–matrix interactions. Ground substances contribute to the viscoelastic properties of tendons and ligaments. This study was prompted because of the observation of an even more substantial decrease in the tensile strength of stress-shielded whole patellar tendon compared with the collagen fascicles only, indicating that stress shielding affected more than just the collagen fascicles. They found that the transverse tangent modulus and the transverse tensile strength were both significantly decreased, with the tensile strength in the transverse direction decreasing to 29% of the control value at 1 week.[9] There was no statistically significant difference in strain at failure in the transverse direction.[9] They were able to demonstrate that stress shielding had a negative impact on the tensile and viscoelastic properties of patellar tendons.

Stress shielding also affects the microstructure and ultrastructure of patellar tendons. Majima et al[10] performed light microscopy and transmission electron microscopy in the stress-shielded rabbit patellar tendon model to evaluate the effects of stress shielding on the microstructure and ultrastructure of patellar tendons. Using histology, they found a

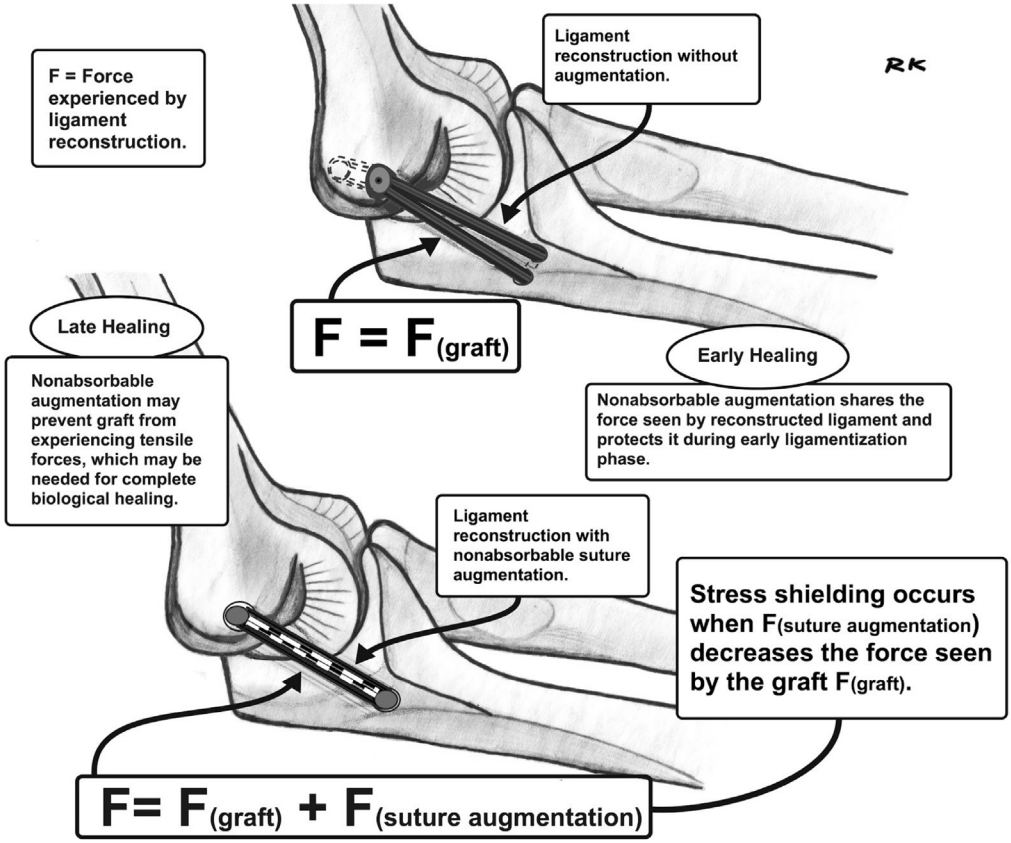


**FIGURE 1:** Schematic demonstrating the force experienced by the ligament reconstruction compared with the force that is shared by the graft and suture augmentation, which may result in stress shielding.

statistically significant increase in the mean cellularity of the stress-shielded patellar tendons with 2,383 cell nuclei/mm$^2$ and 2,053 cell nuclei/mm$^2$ compared with 410 cell nuclei/mm$^2$ and 459 cell nuclei/mm$^2$ in the sham group at 3 and 6 weeks, respectively.[10] There was an increase in the number of leukocytes, macrophages, and capillary endothelial cells in the stress-shielded patellar tendon group compared with the controls and sham groups.[10] Using transmission electron microscopy, it was found that stress-shielded tendons had a statistically significant decrease in the mean fibril occupation ratio with 49.0% and 55.1% compared with 67.7% and 66.5% in the sham group at 3 and 6 weeks, respectively.[10] Stress-shielded tendons at 6 weeks had a 58.8% decrease in the median fibril diameter compared with the control. Infiltration of leukocytes and macrophages can lead to the production of collagenase leading to catabolism and weakening of the tendon.[10] This increase in the inflammatory infiltration of stress-shielded patellar tendons was supported by Uchida et al[11] who showed that stress-shielded patellar tendons demonstrated overexpression of interleukin 1β, tumor necrosis factor α, and transforming growth factor β. In stress-shielded rabbit medial collateral ligaments, studies have demonstrated an increase in collagenase messenger RNA and that collagenase reduces collagen fibril size.[12,13]

## DISCUSSION

Nonabsorbable suture augmentation may protect the repaired or reconstructed ligament from early failure and is intended to allow the active patient or athlete a quicker return to rehabilitation and sport activities. The development of these techniques is based on our desire to help patients overcome substantial injuries in the shortest period of time possible. Although innovative methods that aim to accelerate recovery and allow an earlier return to play are attractive, we must be careful that our technological advancements do not disrupt the body's natural healing and potentially compromise long-term ligament integrity. One possible solution to this concern may be found in absorbable augmentation devices. This strategy would confer the advantage of transmitting forces away from the healing ligament in the period of early healing, yet allow the ligament to ultimately experience the full force that the joint transmits.

Despite an increase in the use of suture augmentation in ligament repair and reconstruction procedures, there remains a paucity of clinical studies evaluating the short- and long-term effects of potential tendon and ligament stress shielding. Without clinical studies, we must exercise caution in interpreting the conclusions demonstrated by animal models. Future clinical studies are needed to identify whether the concerning changes in architecture, tensile strength, and mechanical properties identified in animal studies translate into similar findings in human subjects.

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