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THE EFFECTS OF POST-SURGICAL LOADING ON THE BIOMECHANICAL AND HISTOLOGICAL PROPERTIES OF THE RABBIT PATELLAR TENDON AFTER REMOVAL OF THE CENTRAL 10% AND 40%

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By

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ABSTRACT

THE EFFECTS OF POST-SURGICAL LOADING ON THE BIOMECHANICAL AND HISTOLOGICAL PROPERTIES OF THE RABBIT PATELLAR TENDON AFTER REMOVAL OF THE CENTRAL 10% AND 40%

By

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Complications of using the central one-third of the patellar tendon as autogenous tissue for anterior cruciate ligament reconstruction may result from excessive hypertrophic scarring of the host patellar tendon. It is possible that this damage is a result of increased stresses generated in the host patellar tendon after removal of a portion for ligament reconstruction. In the present research, this hypothesis is tested by increasing the stresses in the rabbit patellar tendon by removing the central 10% and 40% of the tendon prior to subjecting the animal to post surgical exercise on a treadmill. In addition, one group of rabbits with the central 40% removed had a stress shielding device implanted to protect the host tendon from stresses during exercise. The biomechanical and histological properties of the tendons with the central 10% removed closely resembled those of the contralateral control tendons. The tendons with the central 40% removed showed significant hypertrophy and decreased material properties. Stress shielding of the tendon prevented scar formation but encouraged disuse atrophy of the tendon.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

I. INTRODUCTION

The knee is the most vulnerable of the large joints in the body. Located between the two longest lever arms in the body, it is often subjected to large mechanical forces and motions. The functions of the knee include transmitting loads, participating in motion, assisting in the conservation of momentum, and providing a force couple for activities that involve the leg. Kinematically, the knee rotates primarily in the sagittal and transverse planes. The sagittal plane, in which flexion and extension of the leg occur, is the most dominant plane of motion. Secondly, is the transverse plane of motion in which internal and external rotation of the knee occurs causing the helical screw axis phenomenon. Unlike the hip and shoulder, the knee is primarily a hinged joint with rotation and sliding that occurs during movement through its full range of motion.

Frequently, the knee must sustain loads of two to three times bodyweight. With the supporting structures of the knee being composed primarily of soft tissues like tendons, ligaments, and menisci, it is highly susceptible to injury. These soft tissues are also important in maintaining normal stability and alignment of the knee during motion.

Insufficiency of the ACL is the most common cause of knee instability[30]. The ACL attaches to the posterior femoral condyle and the anterior tibial plateau. The primary biomechanical function of the ACL is to resist, in various degrees of flexion, abnormal anterior displacement of the tibia in relation to the femur (Figure 1).

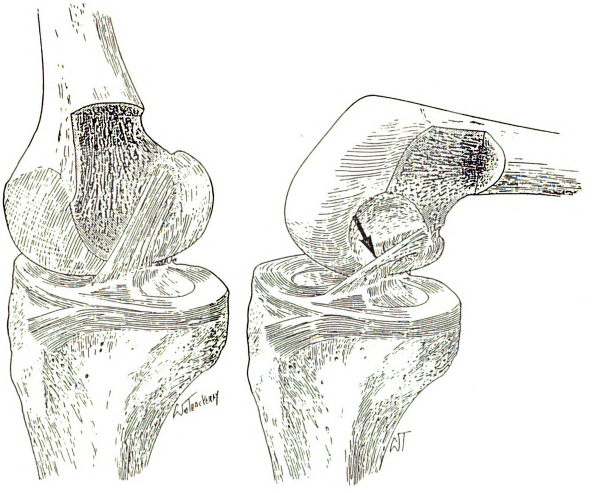


Figure 1: Location of the ACL in the knee and showing attachment sites. (taken from Arnoczky, 1983)

Midsubstance tears of the ACL occur due to excessive loads being quickly applied to the knee. These are usually caused by a deceleration, or "cutting" action of the knee joint[34]. Other mechanisms of injury causing failure of the ACL include external rotation and abduction of the leg at the knee joint, which occurs in skiing accidents (Figure 2), a direct posterior blow causing an anteriorly directed force on the tibia similar to the force generated during clipping in football, and lastly, complete dislocation of the knee joint caused by excessive hyperextension of the knee[50].

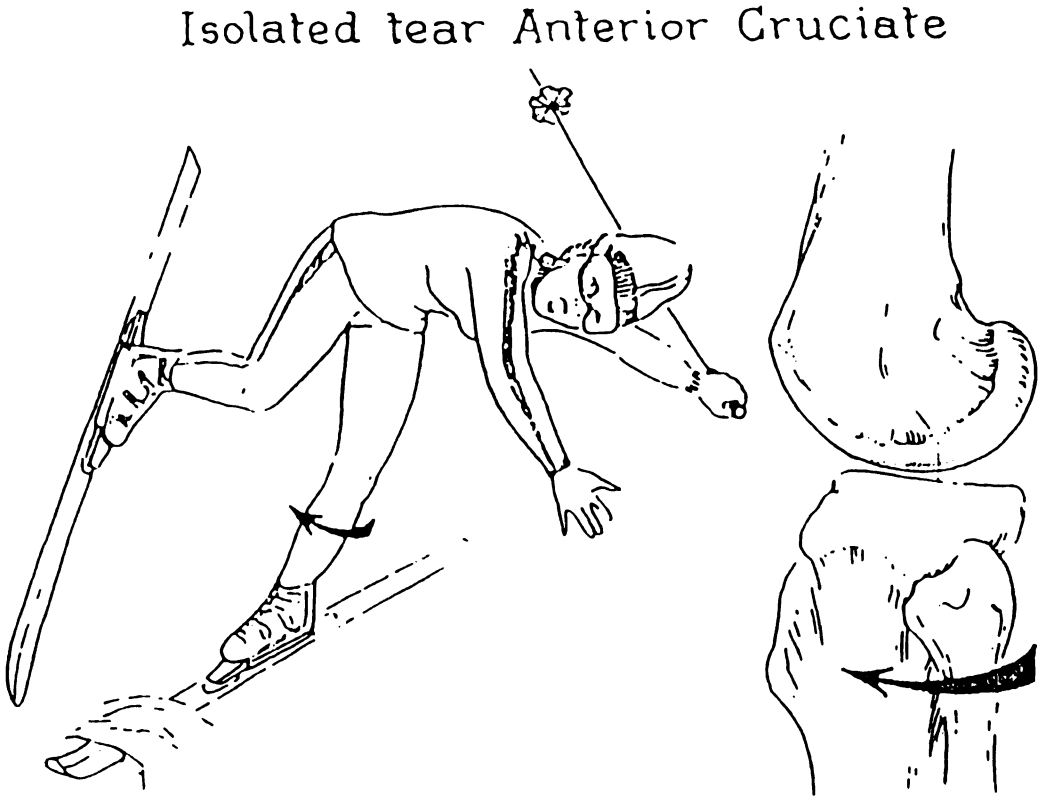


Figure 2: Possible mechanism for injury to the ACL (taken from Kennedy, 1974)

Failure to repair a torn ACL will result in knee instability, along with degenerative changes in the knee joint, and an increasing possibility of subsequent cartilage and menisci damage.

ACL reconstructions have been performed using a variety of techniques and tissues, each with their advantages and disadvantages. Whether the medial or central portion of the patellar tendon is used for ACL reconstruction varies among orthopaedic surgeons. However, the biomechanical properties of the central one-third of the human patellar tendon are superior to those of the medial one third. Its strength and accessibility has made this one-third portion of the patellar tendon the most common candidate for autogenous reconstruction of the ACL[3,25,28,33,47,69].

Todays trend of rehabilitation programs by most physicians, following ACL reconstruction using the central one-third of the patellar tendon, leans toward an aggressive physical therapy regimen. Many programs developed today consist of muscle strengthening and range of motion exercises to accelerate rehabilitation and increase knee stability[66,73,88]. However, these accelerated rehabilitation may cause increased stresses on the already surgically traumatized host patellar tendon. Increased scar formation, in addition to increases in cross-sectional area of the host patellar tendon, have been seen in recent studies involving animal models[18,43], as well as, human subjects[16,70]. Clinical studies involving magnetic

resonance imaging have shown increases in cross-sectional areas of the host patellar tendon, when compared to contralateral controls 12 to 24 months after ACL reconstruction using the central one-third of the patellar tendon[16,70].

This thesis will investigate the histological and biomechanical changes of the rabbit patellar tendon observed after removal of its central one-third. These changes include a significant amount of hypertrophic scarring often seen in the host tendon after exercise rehabilitation[16,18,43,54]. It is hypothesized that increased stresses generated in the host patellar tendon during exercise rehabilitation may structurally degrade the remaining patellar tendon. This in turn may cause an increase in collagen synthesis to accommodate the decreased material properties of the damaged host tendon.

The increase in stresses of the surgical patellar tendon were accomplished by removing the central 10% and 40% of the rabbit patellar tendon from different groups of animals. The rabbits were subjected to a six week rehabilitation program in which they were exercised on a treadmill for approximately 10 to 15 minutes daily. In addition, this thesis will present test data of a group of rabbits which had a stress shielding device implanted, in vivo, after removal of the central 40%. This augmentation device will completely unload the host patellar tendon of any stresses generated during exercise. Finally, the amount

of hypertrophic scarring of the host patellar tendon will be monitored with the use of magnetic resonance images taken one week after surgery, and again at sacrifice.

In addition, included in this thesis is two case reports presented on patients with quite different post-surgical rehabilitation programs. The surgical and contralateral legs of each patient were scanned by magnetic resonance imaging six months after ACL reconstruction using the central one-third of the patellar tendon. The objective was to determine if the patient with a more accelerated rehabilitation program incurred significantly more scar tissue formation in the host patellar tendon when compared to a patient that took a much less aggressive approach to rehabilitation of the surgical limb.

II. SURVEY OF LITERATURE

The ACL provides approximately 86% of the total resisting force to anterior displacement of the tibia on the femur[23]. Chen and Black[26] estimate the tensile forces in the human ACL during normal function range from 67 N for walking, to as high as 630 N for jogging. Grood and Noyes[40] calculated values of 200-400 N for young humans and 80-160 N for older humans, by assuming that the normal forces range from one-tenth to one-fifth of the breaking loads. Morrison[62] estimated forces in the ACL to be 67 N for ascending a ramp, and as high as 445 N for descending a ramp. To provide a factor of safety, it is important that the autogenous tissue strength is greater than the forces that it will be subjected to during normal and strenuous activities[64,67] (Figure 3).

It is also important to account for the weakening of the graft, due to tissue incorporation, after implantation of the replacement tissue[24,60]. For example, Noyes et al.,[65] had found that patellar tendon grafts in primates drop 15% of their pre-implantation strength by six weeks after surgery. This decrease in strength of the autograft is the primary reason that one-third of patellar tendon is often used for ACL reconstruction.

Most studies on the fate of intra-articular ligament reconstruction have shown the graft to have a prolonged weakness that requires a year or more to regain structural strength[4,11,29]. Experimental, and clinical, studies suggest that the patellar tendon grafts do survive within the joint and are functionally adequate replacements for the ACL. The structural strength of the tendon is directly related to its physical size and inherent mechanical properties. Long-term survival of these grafts are dependent on revascularization of the transplanted tissue.

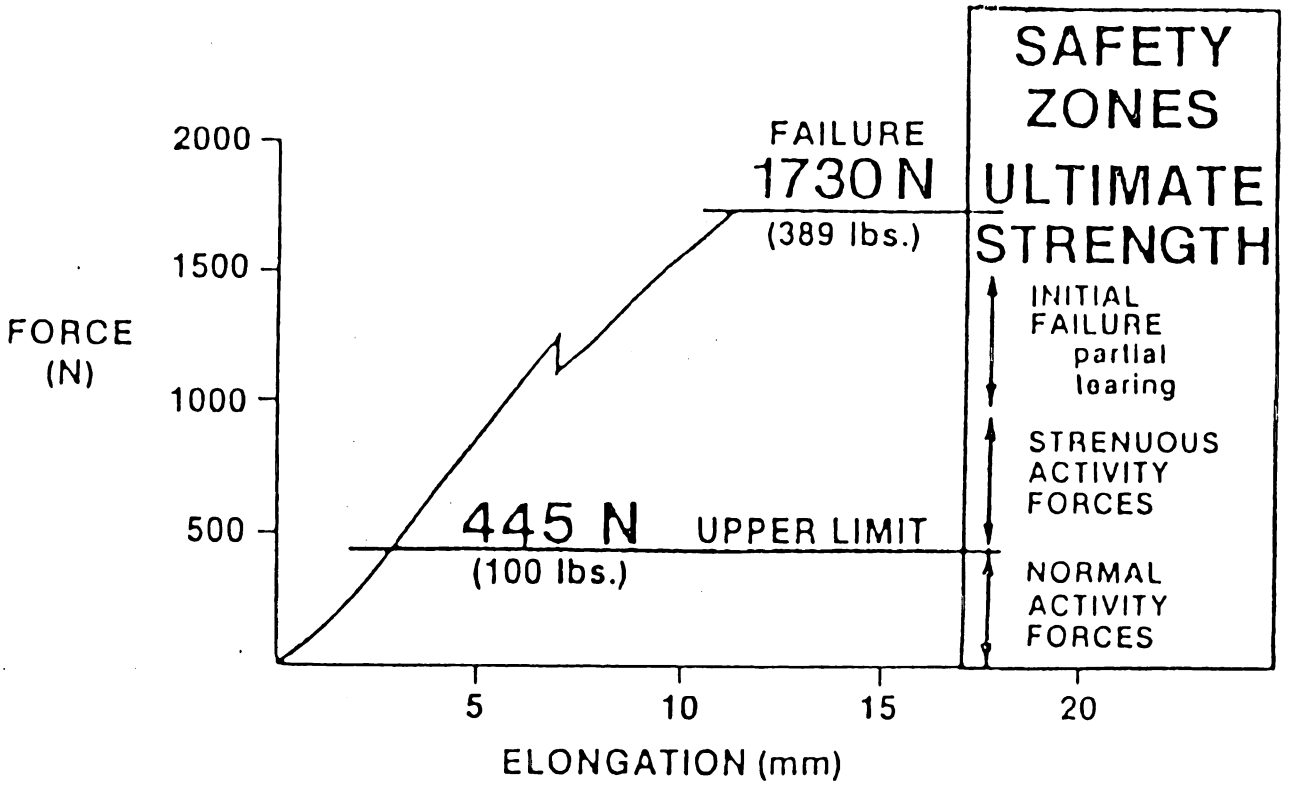


Figure 3: Hypothetical load-deformation curve for safety zones of the anterior cruciate ligament. (taken from Noyes, 1984)

Arnoczky, et al.,[12] in a study which used the medial one-third of the patellar tendon for ACL replacement in dogs, found that by 6 weeks after surgery the grafts were completely ensheathed in a vascular synovial envelope, and histologically showed avascular necrosis.

The vascular anatomy of the cruciate ligaments in dogs[11] and in rabbits[81] was studied using microangiography. The microvascular anatomy of the infrapatellar fat pad and the synovial membrane in both dogs and rabbits were found to be quite highly vascular. The blood supply to the cruciate ligaments in Arnoczky's study was found to originate predominantly from the infrapatellar fat pad and synovial membranes of the joint. Intrinsic revascularization of the patellar tendon autograft progressed from the proximal and distal portion of the graft towards the center. One year after surgery the vascular and histological appearance of the patellar tendon graft resembled that of the normal ACL. In a recent study by Horibe, et al.,[44] it was shown that the primary blood supply to the reconstructed ACL comes primarily from the bone marrow, and secondly from the infrapatellar fat pad and synovial tissues. The importance of getting nutrients and a blood supply to the graft tissue cannot be overlooked. It is therefore necessary to consider the effects of ACL reconstructive surgery on the infrapatellar fat pad.

Autogenous Tissue

There are many types of collagenous tissues available in the human body for intra-articular and extra-articular ligament reconstruction. The human patellar tendon properties are far superior to most structures in the area. The human patellar tendon grafts are extracted as a bone-patellar tendon-bone complex allowing bone to bone fixation in the knee. The patellar tendon autografts also show a strong ability to revascularize and survive within the knee joint after ACL reconstruction[11]. Few studies discuss injury to the patellar tendon after this surgical procedure[17,61]. Some disadvantages of ACL reconstruction using the central one third of the patellar tendon include a loss of quadriceps strength of 15-20% at two years post surgery, and a decrease in full range of motion[73,74].

A previous study looked at the mechanical properties of human patellar tendon grafts[64]. In this study they found that the central one-third of the human patellar tendon was far superior to all other graft tissues in both strength and structure. Their results indicate that the central and medial one-third sections of the human patellar tendon are 175% and 163%, respectively, stronger than the mean strength of the human anterior cruciate ligament (Figure 4). In comparison, the semitendinosus and gracilis tendons have only 75% and 49%, respectively, the strength of the human anterior cruciate ligament.

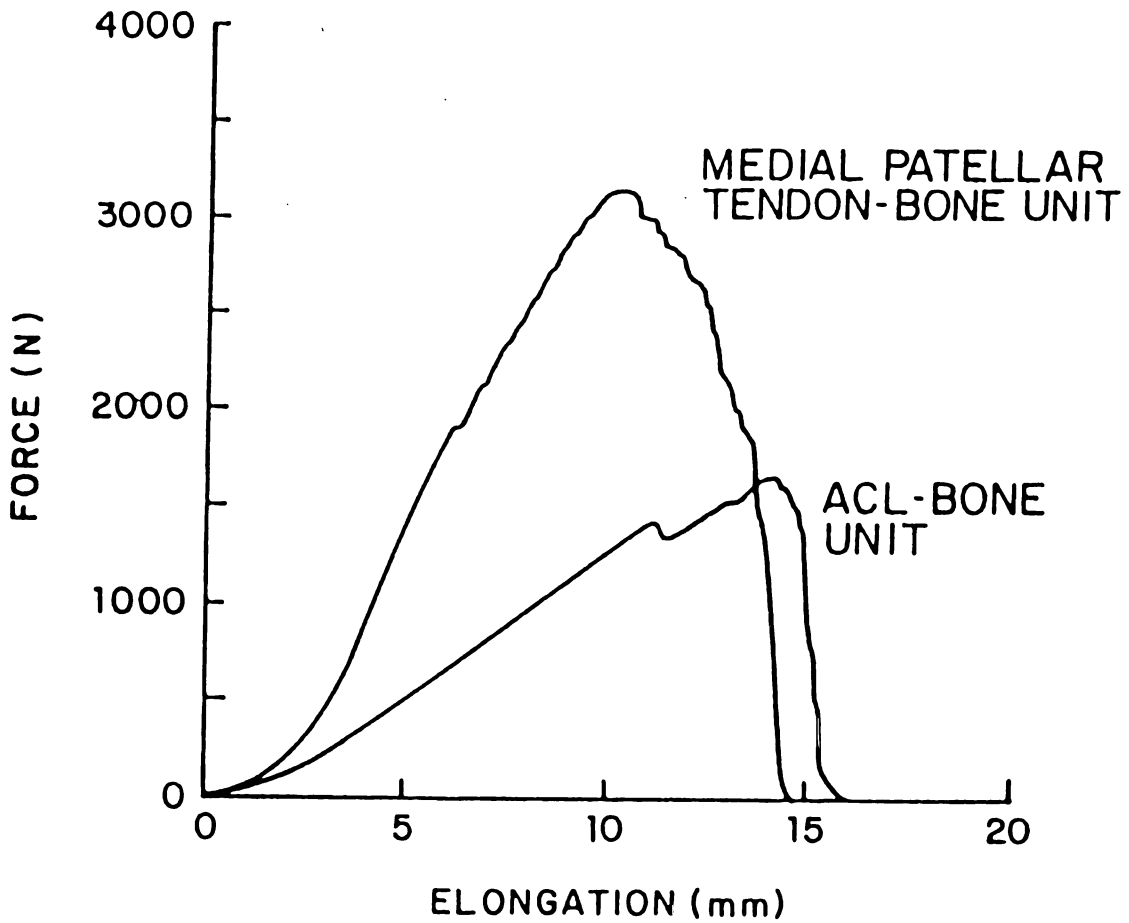


Figure 4: Graph of human patellar tendon vs ACL (Taken from Noyes, Ref. 64)

Butler, et al.,[22] also found that the maximum strain levels at failure were approximately 13-15% for both the anterior cruciate ligament and patellar tendon in humans (Figure 5). The bone-patellar tendon-bone units of the patellar tendon were also found to be three to four times stiffer than the anterior cruciate ligament itself. This allows a factor of

safety for the decrease in properties often seen during the revascularization period.

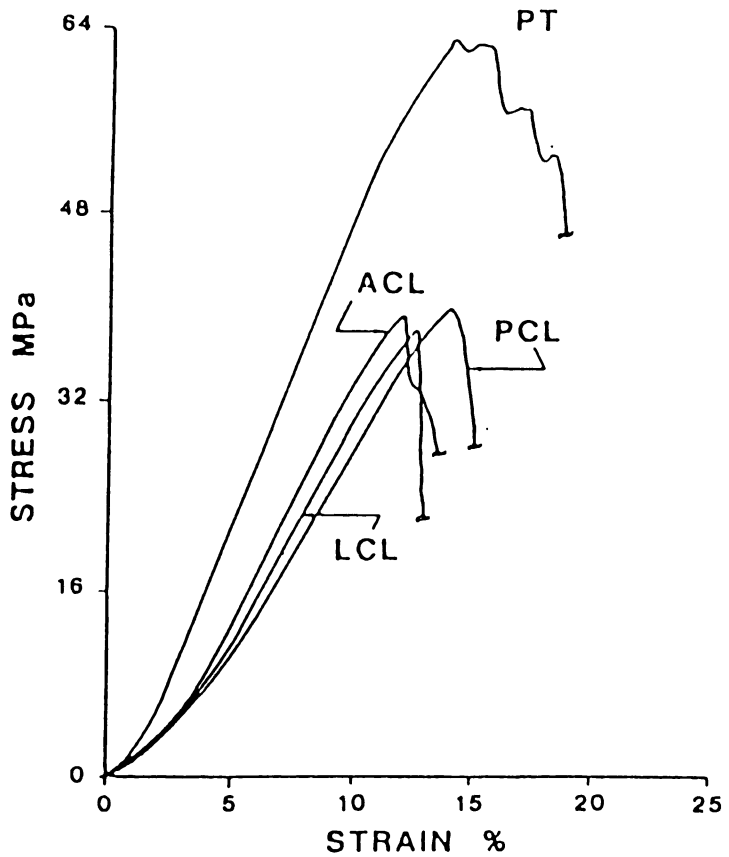


Figure 5: Stress-Strain curves for selected human ligaments versus the human patellar tendon. (Taken from Butler, Ref. 22)

Chun, et al.,[27] tested the material properties of the human patellar tendon in subunits. The human patellar tendon was divided into six equally spaced fascicles of bone-patellar tendon-bone units (Figure 6). The results indicate that the lateral and central thirds of the human patellar tendon have

a modulus that is twice that of the most medial one-sixth of the human patellar tendon.

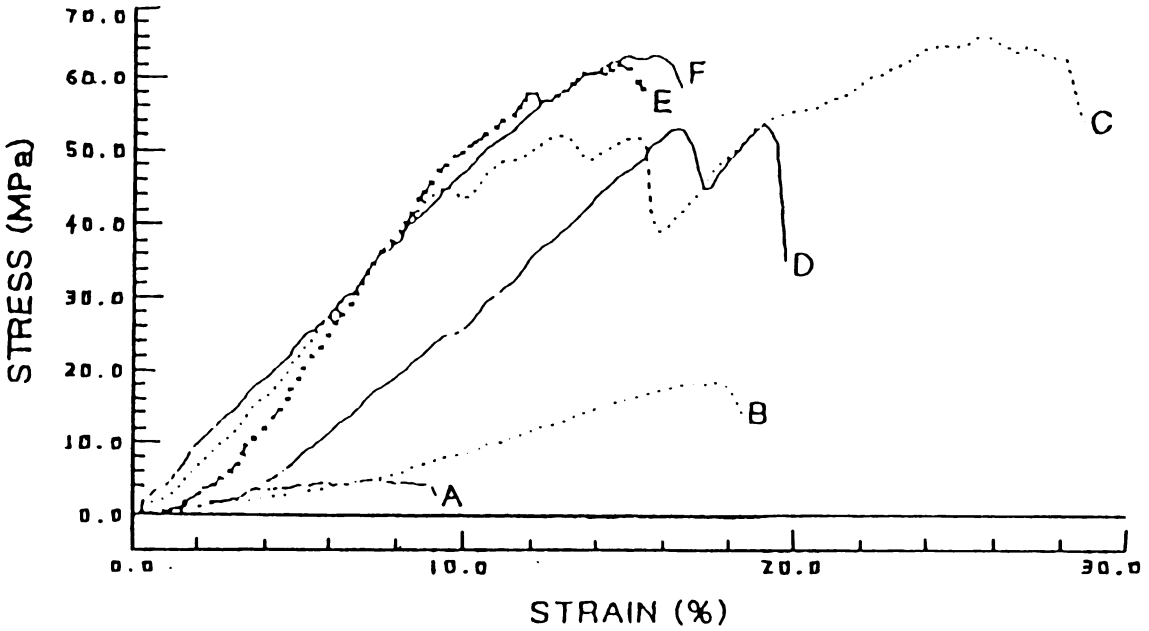


Figure 6: Stress vs. strain curves for the most medial (A) to most lateral (F) subunits of human PT. (Taken from Chun, 1989).

Surgical Procedure

In 1963, Jones outlined a surgical procedure for reconstruction of the anterior cruciate ligament. This procedure uses the central one-third of the patellar tendon as an autograft tissue, a procedure that is still currently used by many physicians[47,48]. Butler, et

al.,[30,45,51,59,70,74,82,88] and others are proponents of the autogenous patellar tendon graft, citing it as a superior substitute from a biomechanical standpoint. Its tensile strength and durability as a scaffold allow for adequate revascularization and subsequent proliferation of a new ligamentous tissue.

The usual width of the patellar tendon graft ranges from 10-13 millimeters, representing approximately one-third of the existing tendon. The two primary concerns in selecting the width of the host tissue is to not disturb patellofemoral tracking, and to prevent tissue rupture in the remaining patellar tendon. Bonamo, et al.,[17] has documented cases of rupture of the patellar tendon after removal of its central one-third. Sachs, et al.,[71] has documented patellofemoral problems, including flexion contracture, and patellofemoral pain after removal of the central one-third of the patellar tendon for ACL reconstruction. On the contrary, a recent study indicates patellofemoral contact pressures immediately after harvesting of the central one-third patellar tendon showed no significant alterations in patellofemoral contact pressures or pressure distribution[32].

Support for using the central one-third of the patellar tendon currently comes from its superior biomechanical properties and its lack of disturbing the patellofemoral contact pressures[21,32]. Conversely, critics of using the central portion of the patellar tendon cite problems ranging

from loss of quadriceps strength and failure to maintain normal thigh circumference[71,74]. While most experimental studies focus on the strength and composition of the intrarticular graft, few look at changes and effects of the surgery on the patellar tendon itself. Clinically documented loss of quadriceps strength and decreases in range of limb motion have been suggested to be in part related to inferior performance of the host patellar tendon after removal of its central one-third[18].

Clinical Relevance

Yasuda, et al.,[88] performed quantitative evaluations of knee instability and muscle strength after ACL reconstruction using the central one-third of the patellar tendon. Measurements were performed on 65 patients who were followed for 3 to 7 years. The authors checked for knee instability of each patient and found that 89% of the patients had differences of less than 2.5 mm between operated and non-operated knees. Quadriceps strength was measured with Cybex testing equipment and was found to be less than 50% the strength on the uninjured knee three months post-surgery. In men, quadriceps strength returned to only 85% at final follow-up. In women, quadriceps strength was only 70% the strength of the non-injured leg at final follow-up. The authors felt that although using the central one-third of the patellar tendon achieves good stability, quadriceps weakness occurs as a result of damage

to the knee extensor mechanism. Sachs, et al.,[71] also compared a group of patients with patellar tendon autografts, to a group with semitendinosus autografts. Again, the patellar tendon group had a significant reduction in quadriceps strength at 1 year post-surgery. Tibone and Antich[74] performed a two year evaluation of patients who had an intra-articular ACL reconstruction using the central one-third of the patellar tendon. Again, a decrease of approximately 15-20% of quadriceps strength was noted.

The current trend in rehabilitation is a more aggressive approach to regain range of motion, and muscle strengthening as soon as possible. Recently, Shelbourne and Nitz[73] looked at two groups of patients that were subjected to different rehabilitation regimens. In their study two groups were divided into an "aggressive" rehabilitation group and a "passive" rehabilitation group. All 450 patients in the study had an autogenous ACL reconstruction using the central one-third of the patellar tendon. Quadriceps strength and knee laxity were the parameters measured in the study. The results indicate that after a one year rehabilitation program there still remained a decrease in quadriceps strength of the surgical leg of approximately 10% in both groups[73].

Berg recently reported a case study that indicated uniform hypertrophic scar tissue formation in a patient eight months after removal of the central one-third of the patellar tendon for autogenous reconstruction of a torn

anterior cruciate ligament[16]. Despite an active life style and extensive physical therapy, the patient had a limited range of motion, walked with a limp, and had a 1.5 cm of measured thigh atrophy six months after surgery. Post-operatively the patient had continuous passive motion and was allowed partial weight-bearing after one month. Fearing infrapatellar contracture syndrome, the knee was re-opened to find that the central defect had filled and the infrapatellar fat pad was atrophic. Magnetic resonance images of the knee indicated that the cross-section of the patellar tendon had uniformly hypertrophied to approximately twice that of the contralateral control tendon. The fat pad was not distinguishable from the adjacent tissue.

Previous Animal Model Studies

A recent study by Burks, et al.,[18] indicates that a significant amount of hypertrophic scar tissue is observed in the dog patellar tendon six months after removal of its central one-third. While the mechanical properties of the healing tendon are significantly less than controls, the cross-section is found to be uniformly hypertrophic and approximately 4.5 times larger than the contralateral control tendons. The structural stiffness and tensile modulus of the operated tendon within the physiologic range were reduced to 70% and 33% of controls at 6 months post surgery, respectively. The authors also found a shortening of the patellar tendon of dogs three months after removal of

the central one-third of the patellar tendon. Upon gross inspection it was found that in all tendons the defect was filled completely with scar tissue. Histologically, at 3 and 6 months after surgery, a haphazard arrangement of collagen was noted throughout the entire tendon cross section, interwoven with more normal appearing patellar tendon.

A study performed by Cabaud, et al.,[24] in which the medial one-third of the patellar tendon was removed from dogs, had results that differed significantly than those of Burks. Failure testing of control and operated tendons showed slight decreases in strength and stiffness at 4 months. Cabaud found no abnormalities on gross inspection except for thickening of the undersurface where the fat pad had been dissected. Cross-sectional areas of the tendons did not differ significantly from control tendons.

A recent study by Linder[54] also investigated the removal of the medial one-third of the canine patellar tendon. Linder's results also differed significantly when compared to the results found by Cabaud. Linder found that at six months post surgery the operated tendons using the medial procedure had inferior structural and mechanical properties and a large increase in cross-sectional area when compared to controls. The suggested differences in test results were that Linder's dogs were allowed unrestricted activity immediately following surgery. In contrast, Cabaud's dogs were immobilized for six weeks immediately

following surgery, thus allowing time for the patellar tendon to develop sufficient scar tissue.

Recently Haut, et al.,[43] studied the effect of immobilization versus enforced exercise on the amount of hypertrophic scarring of the host patellar tendon. The animal models used in Haut's study were subjected to an aggressive exercise rehabilitation, or pin immobilized, after removal of the central one-third of the patellar tendon. Haut, much like Burks, also found an increase in cross-sectional area of 3.5 times in the operated tendons of the exercised group of rabbits when compared to contralateral control tendons three months after surgery. In contrast to the Burks study in which the length of the operated tendons decreased by 10%, Haut found a 12% lengthening of the operated patellar tendons in the exercised animals. The tendons from the animals with the pin immobilized legs showed very little scar formation in the operated tendons. The surgical leg was immobilized by inserting a Steinmann pin through the femur and tibial shaft after removal of the central one-third of the patellar tendon. The cross-sectional area of the immobilized tendons were not found to differ significantly from control tendons.

Burks suggested that the knee is put at a double disadvantage after ACL reconstruction because of patellar tendon shortening and the decreased material properties of the remaining tendon. Van Eijden, et al.,[77] used a mathematical model to look at the effects of changes in

length of the patellar tendon. They found that a short patellar tendon increased proximal tendofemoral compression pressures, increased anterior translation force on the tibia near full extension, and indicated a greater muscle force was needed to generate the same extensor moment toward terminal extension. Burk's suggested this effect as a possible reason for the clinical observations of decreased quadriceps strength.

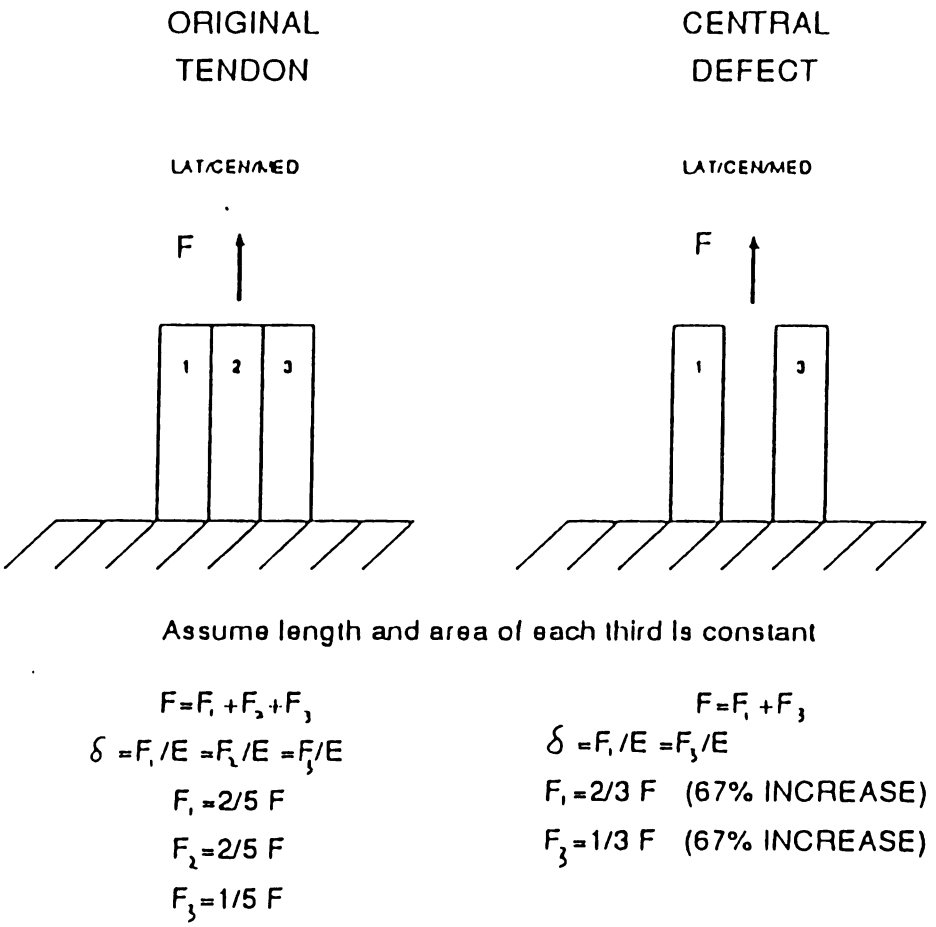


Figure 7: Mechanics of the tendon after removal of the central defect. (taken from Linder, 1992)

One explanation for the observed hypertrophic scar formation may be the mathematical concept developed by

Linder[54] (Figure 7). This mathematical model considered the variations in tensile stiffnesses across the thirds of the patellar tendons. In the human patellar tendon the tensile modulii of the central and lateral thirds are approximately twice that of the medial one-third[27]. When the central one-third of the patellar tendon is removed, as in Burk's study, the stresses in the remaining two-thirds may become excessive. Linder suggested that the increased stresses in the remaining tendon after removal of the central one-third may result in micro-damages of the remaining collagen fibers. This overstressing may act as a stimulant for fibroblasts to synthesize more new collagen fibers after removal of the central one-third to compensate for the detected increase in internal stresses.

This mathematical concept may explain the differences observed in the immobilized and exercised tendons in the previously mentioned study by Haut[43]. A previous study by Frank, et al.,[36] also found the collagen alignment in ligaments of pin immobilized legs to be superior than non-immobilized legs. Viidik, et al.,[79] suggests that tension in a tissue is likely to influence the alignment of its components. The tendons of the pin immobilized rabbits were subjected to low level isometric stresses throughout the three month rehabilitation period. Therefore, these uniaxial stresses may assist in the alignment of the newly synthesized collagen fibers. These well organized fibers

may be the reason for the decreased amounts of hypertrophic scar tissue observed in the pin immobilized tendons[36,43].

Structure of Tendons and Ligaments

The structure and chemical composition of ligaments and tendons are almost identical in humans and in many animal species such as rats, dogs, rabbits, and monkeys. Therefore, extrapolations regarding these structures in humans can be made from the results of studies on animal species[10].

Tendons and ligaments are constructed primarily of type I collagen (75%) and elastin (15%), and a ground substance composed of proteoglycans with some degree of cellularity. Tendons and ligaments are subjected, under physiological conditions, to stresses of approximately one-third of their ultimate tensile strength. They are furthermore non-linear, viscoelastic materials. The collagen fibers in tendons are aligned in a nearly parallel orientation which makes them suitable to withstand high, unidirectional, tensile loads. Ligaments however, have collagen fibers that may be somewhat less parallel, but are closely interlaced with each other. The collagen molecule is synthesized by fibroblastic cells. Collagen molecules are formed by three alpha chains combined in a right hand triple helix which gives collagen its rodlike shape. The size of the collagen molecule is approximately 280 nm in length and 1.5 nm in diameter (Figure 8). The collagen molecules aggregate in the

extracellular matrix to form microfibrils and then fibrils. Cross links are formed between collagen molecules and it is these cross links that gives strength to tissue and allows them to function under mechanical stress.

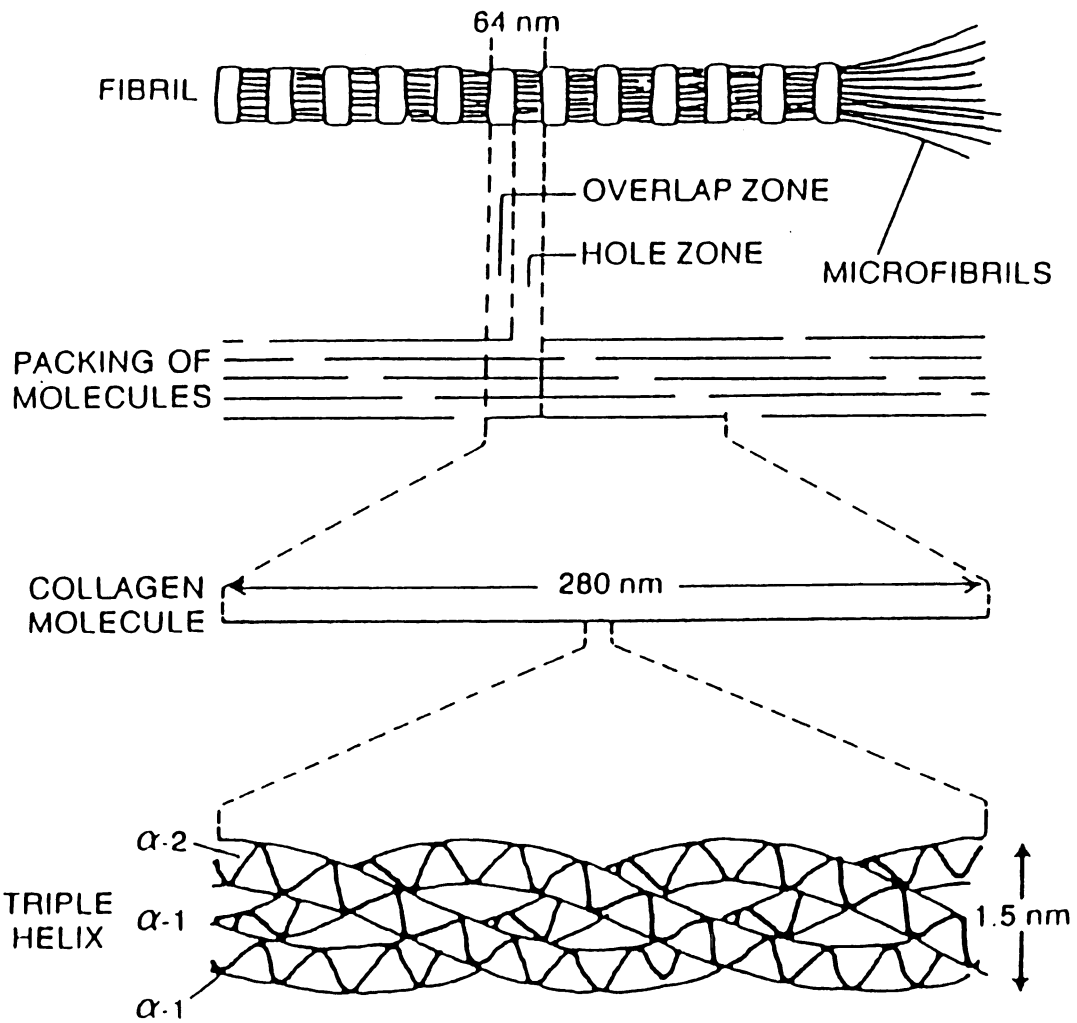


Figure 8: Schematic diagram of collagen molecule. (taken from Nordin/Frankel, 1989)

Since collagen alignment is more organized in tendons than ligaments, tendons have a shorter deformation prior to reaching the linear region when a tensile load is applied. Recall, in Figure 3, the shift in tensile response between the patellar tendon complex and the ACL[64]. This shift is likely caused by differences in the alignment of the constituent collagen fibers and content of these fibers between the patellar tendon and ACL.

Biomechanics of Tendons and Ligaments

Biomechanical properties of collagenous tissue can be described using a tensile response curve (Figure 9). The initial concave portion of the curve, region I of the graph, represents the "toe" region. The sequential uncrimping and progressive recruitment of relaxed collagen fibers occurs in this region. It is within the "toe" region that most physiological activities and clinical diagnosis takes place[63]. Haut and Little reported the "toe" region to have a strain value between 1.5 and 4.0%. This is where tissue preconditioning is often performed prior to tensile tests[42].

Region II is the "linear region" of the curve. The collagen fibers are further elongated in the linear region until first significant failure occurs at the linear load point[63].

In region III, a maximum loading occurs in which a series of small, sudden, force drops and fiber separations occur until maximum force is reached. At this point a

catastrophic failure of the tissue occurs. Finally region IV, the post-failure region, the tissue loses its load carrying capability even though some fibers may still appear to be intact.

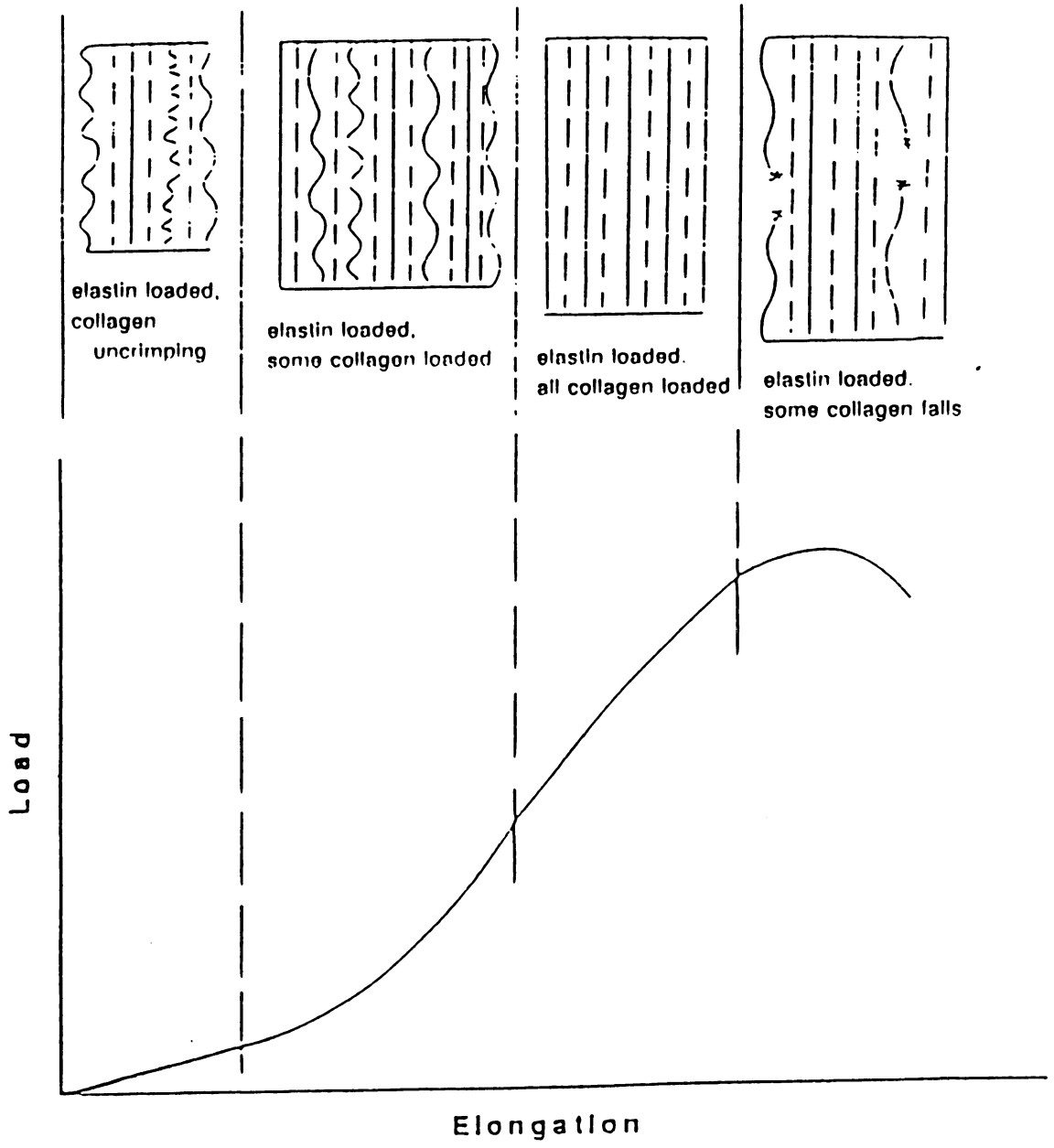


Figure 9: Tensile response of curve of a collagenous tissue. (taken from Noyes, 1977)

Repair Mechanism

When damage to a soft tissue occurs there are three primary phases of repair[13]. They include inflammation, fibroproliferation, and subsequent remodeling of the tissue. Inflammation involves a series of cellular mechanisms that produce an increase in vascular permeability and the accumulation of leukocytes, mostly neutrophils, and macrophages to begin the process of decontamination and debribement of the wound[13]. Manske and Gelberman[58] found that the surface layer of epitenon cells that migrate into a laceration site assume a phagocytic role, engulfing debris and old collagen fragments. Garner, et al.,[38] indicated that the epitenon cells also participate in collagen synthesis. Within three days after injury there is a migration of fibroblasts and endothelial cells into the defect that occurs in response to chemotactic factors. The fibroblasts began to proliferate and synthesize an abundant matrix, mainly in the form of type III collagen. During the final stages of healing there is a gradual shift from type III collagen to type I collagen. The type I collagen is then progressively cross linked to provide tensile strength.

Lindsay, et al.,[55] documented the events that occur after a laceration of the flexor tendon. Granulation tissue from the synovial sheath migrates into the wound gap along with fibroblasts derived from both extrinsic and intrinsic cells from the epitenon and endotenon during the first days after injury. Many of the cells proliferating to the injury

site have a phagocytic function. The fibroblasts in the wound lie perpendicular to the long axis of the tendon and by the fifth day actively secrete collagen. These fibrils are initially disorganized and are primarily aligned in a plane that is perpendicular to the axis of the tendon. The fibroblasts in the wound eventually align themselves with the long axis of the tendon, likely as a result of stress.

Fibroblasts in tendons and ligaments are arranged in long parallel rows in the spaces between the collagen bundles (Figure 10).

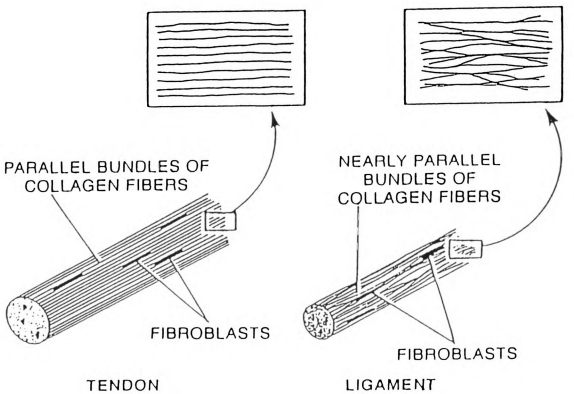


Figure 10: Fibroblast orientation in tendons and ligaments (taken from Nordin/Frankel)

Several tendon bundles form the tendon fascicle, which is surrounded by endotenon, and a number of fascicles are surrounded by epitenon to form a basic tendon unit. An elastic sleeve called paratenon surrounds the unit.

Functional alignment of collagen is usually complete after 2 months. Although scar tissue never achieves the strength of the original tendon, the remodeling phase can be influenced by load bearing and mechanical stimulation. The ultimate tensile strength of a connective tissue, and the mass average diameter of the constituent collagen fibrils have shown a positive correlation. Parry, et al.,[68] hypothesized that the ultimate size of the collagen fibrils in a tissue is dependent on the amount of stress under which the tissue functions. Consequently, large collagen fibril diameters are predicted to have a greater tensile strength than small diameter fibrils.

Immobilization versus Exercise

The universal goal of all orthopaedic treatments is for conditions involving ligamentous deficiency to reproduce or replace, as quickly as possible, the unique properties of the normal bone-ligament-bone complexes[35]. A superior system for ACL reconstruction would be to maximize graft strength and durability, without affecting the biomechanics of the joint. This theory also holds true for the host patellar tendon.

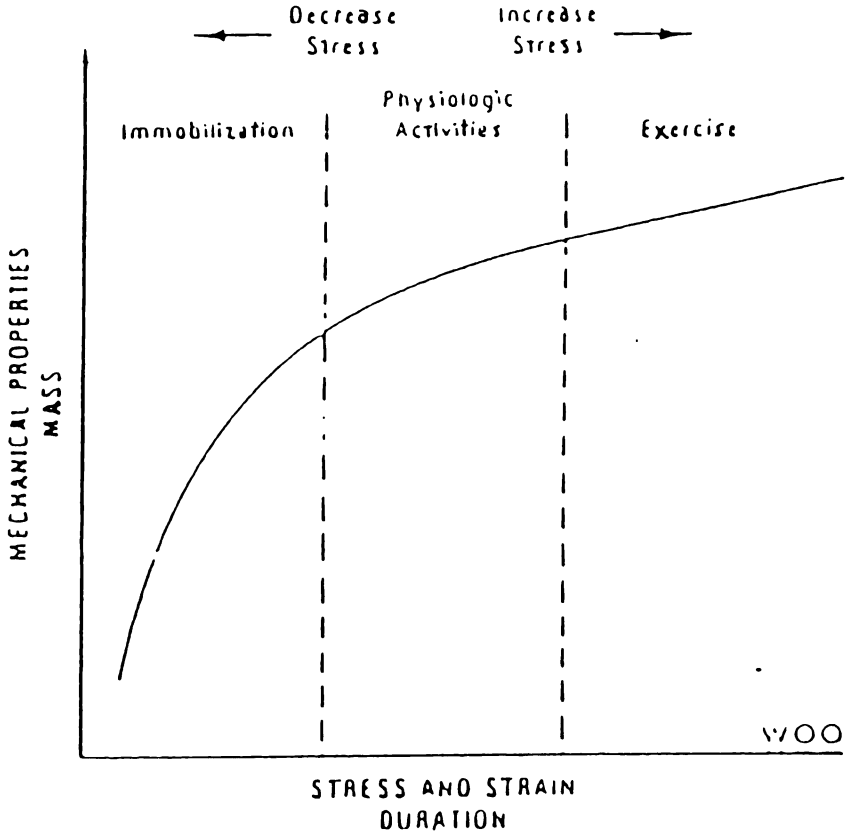


Figure 11: Homeostatic responses of biological soft tissues (taken from Woo, 1982)

In a stress induced and immobilized ligament model Woo documents that a relationship exits between strain levels and soft tissue homeostasis[46,85]. Whereas complete immobilization results in a rapid reduction of tissue strength, exercise tends to increase mechanical properties and mass of the tissue[85]. Woo developed the curve shown in Figure 11 that represents the stress and motion dependent homeostatic responses of biological soft tissues. Figure 12 depicts the effects of immobilization, exercise, and recovery on ligament properties and insertion sites. The

effects of immobility occur rapidly, and recovery of the ligament substance with remobilization occurs within a similar time frame. In contrast, the recovery curve for ligament insertion sites illustrates slow change over a prolonged period of time[84].

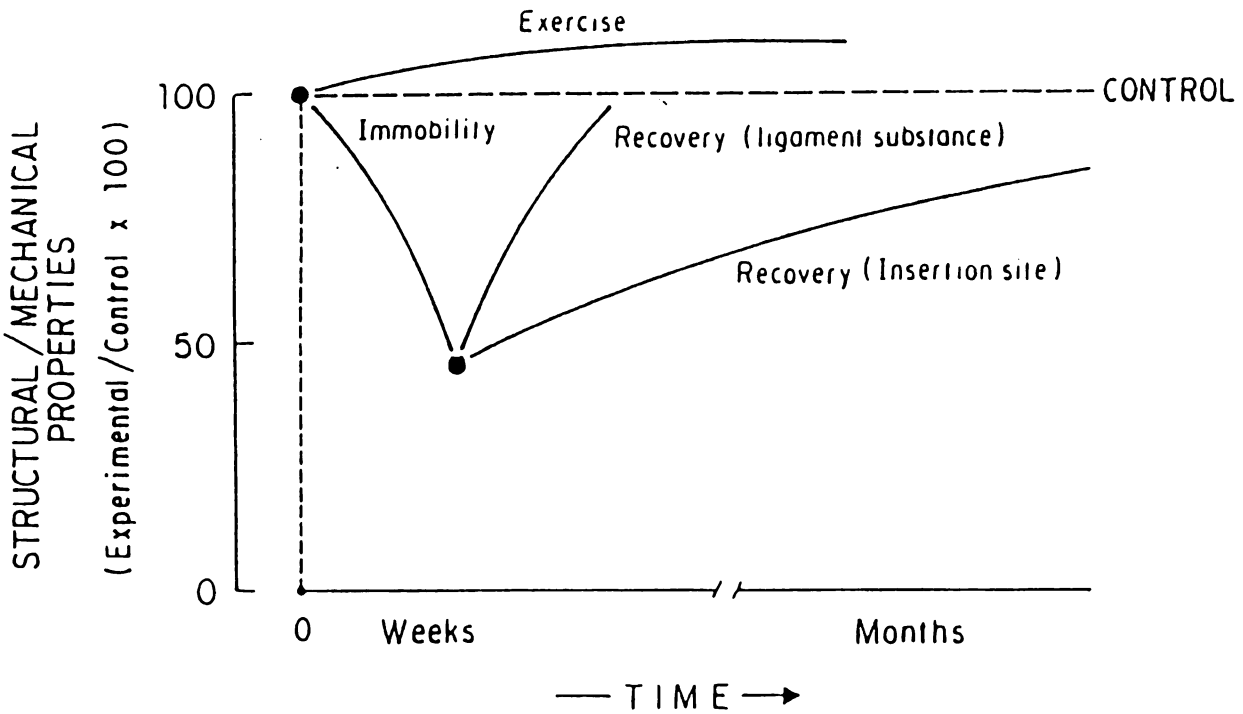


Figure 12: Summarization of homeostatic responses subjected to different activities. (taken from Woo, 1987)

During the course of treatment for injuries to ligaments and tendons, immobilization is often used as part of the rehabilitation process. The major drawback is that immobilization simultaneously induces reduction of the mechanical properties and disuse atrophy of the surrounding healthy tissues[2,8,84,85]. Studies have shown that short

term immobilization can have detrimental effects on cartilage, tendon, ligament, and bone. Noyes demonstrated that after 8 weeks of immobilization, twelve months of remobilization was required before the ACL-tibia complex structural properties returned to normal[63].

During immobilization there is an increase in collagen crosslinking that may play a role in the contracture process that often occurs with immobilized tendons[18,87]. Previous studies using pin immobilization have observed better collagen alignment in healing ligaments than in the ligaments of a mobilized group[36,43]. In these studies the rabbits surgical legs were pin immobilized to prevent joint motion, but uniaxial stresses were present in the tendon during the healing phase. As mentioned previously, it has been suggested that collagen will align in the direction of stresses[79]. The collagen alignment of the healing tendons in both studies were superior than that of the non-immobilized tendons. In a recent study Amiel, et al.,[5,8,41] suggests that since ligament size and collagen mass change minimally during the first stages of immobilization, the observed structural weakening must be due to changes of the ligament substance itself, and not solely to atrophy. It is because of these forementioned reasons that immediate remobilization of the limb following surgery is the protocol called out by many physicians today.

Recently Yamamoto, et al.,[87] installed an augmentation device to completely stress shield the rabbit patellar

tendon. They found that after 6 weeks of stress shielding there was a large increase in the number of fibroblasts present in the patellar tendon after six weeks. Also, the stress shielding decreased the tensile modulus to 9% of the control tendons; whereas, Woo found a decrease of only 54% of the control tendon modulus when the knee was immobilized. Majima, et al.,[56] also found that complete stress shielding of the patellar tendon significantly decreased the properties of the tissue while simultaneously increasing the number of fibroblasts present.

Increased stress and motion, on the other hand, increases the mass and strength of the tissue. The effects of exercise on ligament strength are well documented[1,19,53,75,76]. Several studies show that increased stresses on collagen causes increased collagen synthesis[9,36,78,80]. Mobilization during ligament healing has shown to result in greater strength and stiffness upon biomechanical testing[31]. Schaberg, et al.,[72] suggests that the larger cross-sectional area of the mobilized healing ligaments leads to a higher structural stiffness when compared to the stiffness of the stress immobilized ligament. In contrast, Haut et al.,[43] found a decrease in stiffness of the non-immobilized versus pin immobilized healing patellar tendons, although the cross-sectional areas were 3.5 times that of the contralateral control tendons.

Significance of Present Research

The reason for this study is based on the following hypothesis. It is postulated that the increased stresses developed in the remaining two-thirds of patellar tendon of the exercise group in previous studies by Haut, et al.,[43] and Burks, et al.,[18] may have been caused a structural breakdown of the remaining host patellar tendon. Possibly, in addition to the degradation of the mature collagen their was a simultaneous increase in immature collagen synthesis. The newly synthesized immature collagen fibers are more pliable, have reduced stiffness, and are randomly dispersed which results in the tendon being inferior to resisting tensile forces than a undisturbed tendon. Therefore, to compensate for the lower structural properties of the newly laid collagen, an increased amount collagen is produced which results in an increase in the amount of hypertrophic scar tissue.

If the data generated from this study supports this hypothesis, it would be imperative that a augmentation device be developed. This device would be required to allow immediate remobilization of the knee, while simultaneously protecting the host patellar tendon from excessive stresses.

III. MATERIALS AND METHODS

Tendon Preparation

Forty two Flemish giant rabbits, weighing 5.2 ± 0.6 kg, were divided into four groups. The first group consisted of 12 rabbits used for immediate sacrifice (time zero data) and testing of the patellar tendon after removal of the central 10% and 40% of the patellar tendon. The contralateral limb was used for control data. The second (n=8) and third groups (n=5) consisted of animals with the central 10% and 40%, respectively, surgically removed from the patellar tendon. The fourth group had 7 animals in which the central 40% of the patellar tendon was also removed, but in addition a stress shielding device was implanted to protect the remaining 60% of the patellar tendon from in vivo stresses developed during treadmill exercise.

A pre-operation injection of Robinul V was induced prior to surgery, at a doseage of 0.01 mg per kilogram of bodyweight, to decrease secretions and maintain heartrate during surgery. Intramuscular injections of 15 mg/kg of bodyweight of Ketamine, and a 2 mg/kg of bodyweight of Rompun were administered. The rabbits were maintained on 2% Isoflurane for the duration of the surgical procedure. The rabbits were placed supine, with the right hind limb exposed for surgery. The selected amount of the central patellar

tendon was then extracted in the following fashion. The patellar tendon was exposed with a medial-longitudinal cut through the skin and surrounding fascia layer. The width of the patellar tendon was measured using a stainless steel metric rule and then the central 10% or 40% of the patellar tendon was sharply excised leaving the infrapatellar fat pad intact. The stress shielding device consisted of a No. 40 monofilament line inserted medial to lateral through a 1.0 mm diameter hole located in the central region of the patella and tibial tuberosity. The original length of the tendon was measured. The monofilament line was tied on the lateral side of the tibial tuberosity after the length of the tendon was reduced by an average of 4.5 mm, as shown in the diagram in Figure 13.

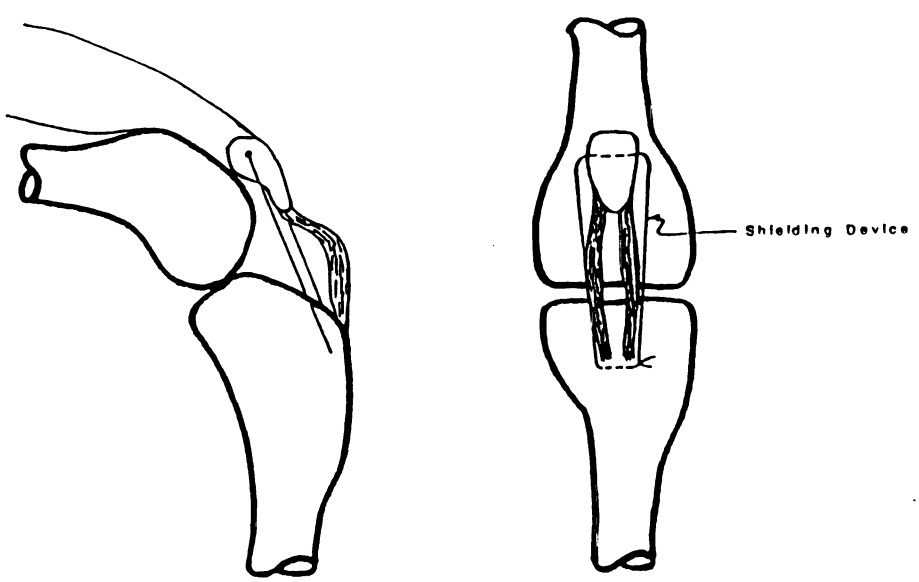


Figure 13: Diagram showing implanted stress shielding device

The contralateral limb of all groups served as control data. The tendon defect in all groups was left open. The subcutaneous fascia was then closed using resorbable suture and the skin closed with 4-0 nylon suture.

All three surgical groups were allowed one week of cage activity following surgery and then were subjected to magnetic resonance imaging (MRI). Upon completion of the six week exercise program the rabbits were sacrificed with a 2 milliliter intravenous injection of Ecklemide and again were subjected to MRI.

Mechanical Test Preparation

Immediately following sacrifice the patella-patellar tendon-tibia complexes from both the surgical and contralateral limbs were extracted. The patella-patellar tendon-tibia complexes were cleaned of all soft tissue except for the patellar tendon. The fat pads were carefully resected and placed in 10% buffered formalin for histology. The tibia was cut off approximately four inches distally from the tibial plateau using a reciprocating bone saw (Stryker Inc.). A 1.0 inch inside diameter aluminum tube, 4 inches long with a .125 inch wall thickness was placed vertically under a ventilator hood. A room curing fiberglass resin (NAPA board Fiberglass reinforced resin 6371) was mixed with a compatible hardener and poured into the tube until it was approximately three-quarters full. The tibia was cleaned of fatty residue with alcohol. It was

then inserted into the epoxy filled tube until the tibial insertion point of the patellar tendon was just above the epoxy. Care was taken to ensure that the tendon did not come into contact with the epoxy resin. The potted configuration was allowed to harden at room temperature for approximately 40 minutes. The tendon remained wrapped in saline soaked gauze, throughout this procedure (Figure 14).

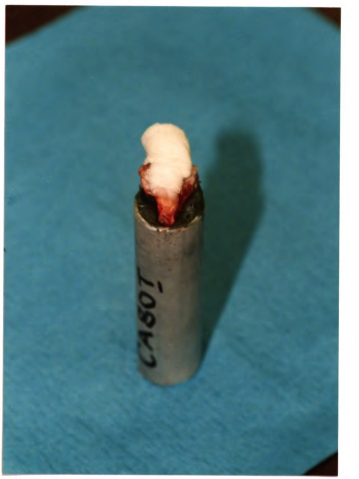


Figure 14: Potted patella-patellar tendon-tibia complex

The total time between potting and testing was approximately 1-3 hours.

Prior to testing the patellar tendon measurements were made by a single examiner using vernier calipers (Figure 15). Width and thickness measurements were taken at three locations along its length. The cross sections were assumed rectangular and their averages were used to calculate the average cross-sectional area.

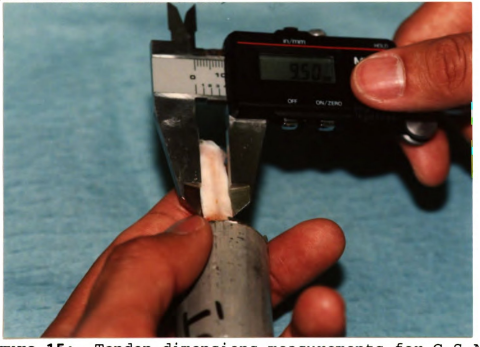


Figure 15: Tendon dimensions measurements for C.S.A.

The cross-sectional areas of the time zero tendons with the central 10% removed were measured as described above and then the average cross-sectional area was decreased by 10%. The time zero tendons with the central 40% removed were measured in three places along its length and width as previously mentioned. These dimensions were taken on both the lateral and medial sections of remaining tendon and the

average cross-sectional area of both sections were added. Tendon length was measured from the inferior pole of the patella to the superior-posterior attachment of the tendon on the tibia. The tendon was immediately wrapped in physiological saline (0.9% NaCl) soaked gauze.

Prior to the mechanical tests the patella was potted in a special stainless steel box-like grip (0.5 x 0.5 x 0.75 inches)[18,54]. The inside was sprayed with a silicone based lubricant to allow easy removal of the potted patella after tensile tests. The same epoxy resin hardener mix, described previously, was used to secure the patella. The box was nearly filled with the epoxy resin. The patella was then pushed into the resin up to the anterior surface and the front plate of the box was secured. The apex (inferior pole) of the patella was allowed to rest against the bottom of the box. A slot cut in the bottom of the box allowed the tendon to pass through without restricting its motion. The resin was allowed ten minutes to harden.

The tendon complex was then mounted into a specially designed fixture which allowed alignment of the tendon along the load axis (Figure 16). The fixture was composed of three horizontal 1/4 inch stainless steel plates, a vertically mounted rotating dish, and a 1/8 inch thick plexiglass saline bath. The bottom plate was secured to the base of the Instron machine. Screw drives were mounted between each sets of plates to allow 2 dimensional motion of the fixture. The x-y table configuration enabled the

operator to precisely align the tendon with the axis of the Instron actuator.

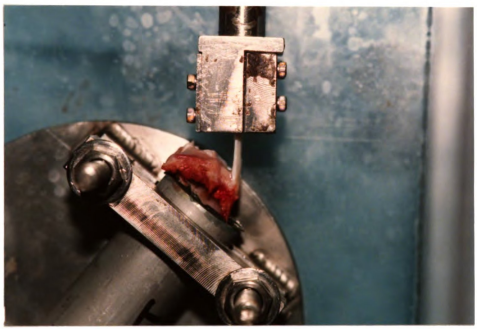


Figure 16: Patella-tendon-tibia complex mounted for testing

The rotating disc, located inside the bath, was secured by three 1/4-28 stainless steel bolts. Its primary function was to provide a secure mounted mechanism for the potted tibia. A secondary function of the rotating disc was to allow for angle changes between the tibia and load axis. An inclusive angle of 110 degrees was used to simulate a physiological loading configuration, and orient the patellar tendon in tension without a tearing or cutting action at the tibial tuberosity.

A standard servo-hydraulic tensile testing machine (Instron model 1331) was used to mechanically extend the preparation. The upper part of the loading mechanism consisted of a 500 lb load cell mounted in line with the

actuator. Below the load cell a universal joint was used to correct minor offsets in alignment of the tendon preparation with the machine actuator. The box containing the potted patella was connected to the load cell.

Physiological saline was heated to 37 degrees Celsius by an external heat exchanger and pumped into the bath after the specimen was properly aligned and secured. The patellar tendon complex was allowed to equilibriate in the heated saline for approximately five minutes prior to mechanical testing.

Pre-Conditioning

Viscoelastic properties of tendons are often used for the understanding of its physiological function[83]. Specimen preconditioning was performed on each specimen in this study prior to failure testing. A 1.5 N preload was applied to the patella-patellar tendon-tibia (PPT) complex prior to cyclic testing. The specimen was then cycled at 3% strain (3% of the tendons original length) with a one hertz, haversine function, for 20 cycles. A standard linearly variable displacement transducer (accurate to 0.016 mm) with the Instron actuator measured the grip to grip deformation. A Nicolet oscilloscope and compatible disk drive continuously stored the load and deformation data at a rate of 100 samples per second. The data generated was not analyzed in this study.

Tensile Tests

The specimen was allowed to stabilize for two minute following the pre-conditioning cycles. The tendon complex was then failure tested at a nominal rate of 100% strain per second. Continuous load and deformation data were collected and stored in the same manner as the cyclic tests at 1000 samples per second. The mechanism of failure in the PPT complex was visually determined and documented.

Force versus deformation plots were generated from the failure experiments. The structural parameters documented in each experiment were the maximum load and time to peak load. The stiffness in the linear range was then calculated from the load-deformation plots (Figure 17). The tensile modulus was calculated as the product of the stiffness times the ratio of initial length to cross sectional area.

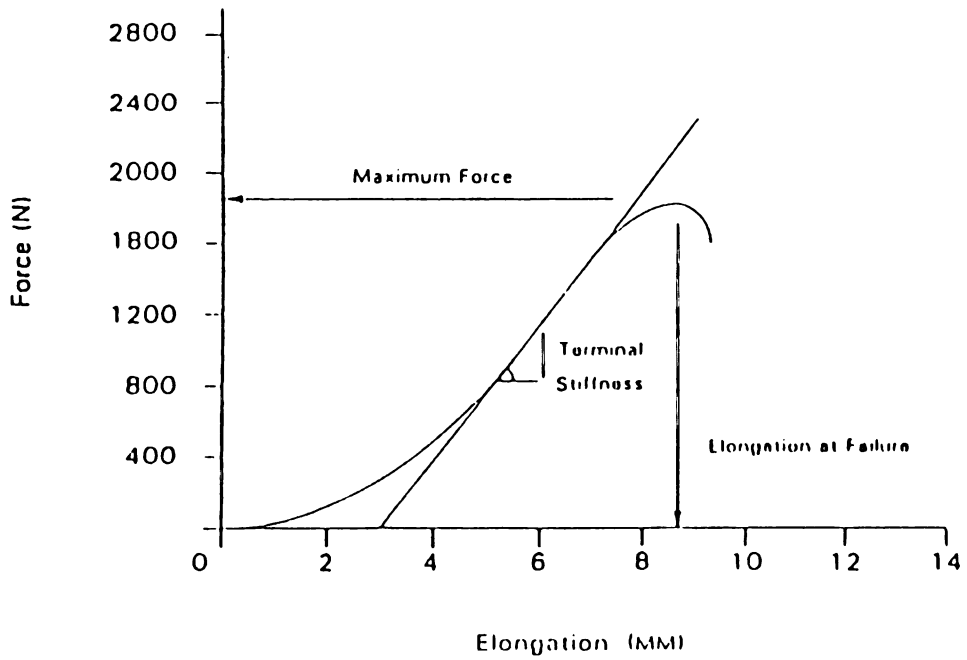


Figure 17: Stiffness recorded from load-deformation curve

Microstructural Model

Most studies describe the structural and mechanical properties of tendons and ligaments in the linear range of the tensile response curve. However, it is documented that most physiological activity occurs at much lower strain levels. Strain imposed on the ACL during clinical diagnostic tests is rarely thought to extend beyond the non-linear "toe" response[20]. Noyes suggests that the maximum loading on the ACL for normal activities rarely exceeds 445 N[64]. A widely accepted value for the amount of strain placed on tendons and ligaments during normal physiological activities is 2-4% strain[37,49]. As shown on most stress-strain response curves, these low strain levels are usually included in the "toe" region.

A mathematical model, previously developed by Belkoff[15], based on Lanir[52], was implemented to help quantitatively describe the "toe" region of load-deformation curves generated from the failure tests of the rabbit patellar tendons. Belkoff's model was originally used to model the tensile response of the human patellar tendon.

The mathematical model assumes a natural waviness (crimp) of collagen fibers in a stress-free tendon. As the tendon is deformed, the collagen fibers are sequentially uncrimped, until fibers are straight. Once straightened, the fibers are able to resist deformation and transmit load. It is assumed that collagen fibers are straightened gradually with elongation of the tendon such that the

distribution of slack lengths is described by a normal Gaussian distribution function (Figure 18).

Using the Marquardt method, Belkoff's mathematical model was used to curve fit the load-deformation data. With this curve fitting procedure it was possible to calculate two model parameters that help describe the toe region of the curve, namely μ (mu) and σ (sigma). In the model the value of μ describes the mean amount of deformation required to straighten 50% of the collagen fibers, whereas, σ describes the standard deviation, or degree of dispersion, in the collagen crimping.

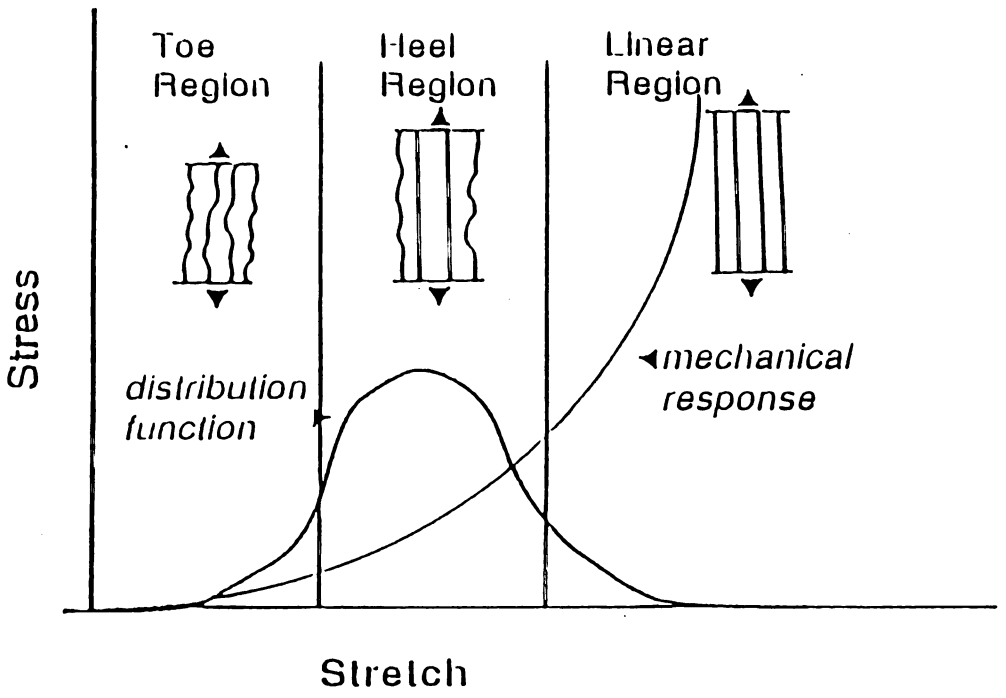


Figure 18: Tendon crimp organization

Intuitively the model equation can be used to quantify the realignment of collagen during tensile elongation. For example, skin has a extremely unorganized collagen matrix compared to ligaments, or tendons. This model would predict a large μ and σ for skin[14], compared to that of tendon[15], because it would require a larger deformation to straighten out the collagen fibers. With a more randomized organization, the "toe" region would be less accentuated and more gradually stiffening.

Magnetic Resonance Imaging

Rabbit Study

One week prior to surgery the rabbits were placed on the treadmill for approximately 5-10 minutes daily. All three surgical groups were allowed one week of cage activity following surgery and then were subjected to magnetic resonance imaging (MRI). The animals were anesthetized prior to being placed in the MRI chamber with intramuscular injections, per kilogram of bodyweight, of 35 mg/kg Ketamine, 5 mg/kg Rompin, and 0.01 mg/kg Butorphenol. The rabbit was placed in a supine position and positioned so a small elliptical surface coil could be placed slightly above the knees. A pulse repetition time (TR) of 1.0 second, an echo time (TE) of 16.0 milliseconds, and 128 phase encoding steps were used to produce relatively high signal-to-noise (SNR) images. Other imaging parameters included a 100 mm field-of-view (FOV), 3 mm slice thickness, and 3 mm

innerslice spacing. The slice spacing is measured from the center of adjacent slices, so all slices were spatially continuous. Total imaging time was approximately 8 minutes and 30 seconds for each animal. All data was acquired on a 4.7 Telsa OMEGA CSI system equipped with S-250 Accustar shielded gradients (Bruker Instruments, Fremont, CA). After completion of the MRI the animal was given a intravenous injection of 0.25 ml Yohimbine. The following day each rabbit began a six week aggressive exercise program in which they were required to run on a treadmill for approximately 10-15 minutes daily at a speed of 0.013 meters/sec. After six weeks each rabbit was sacrificed and again subjected to MRI.

Human Study

It was fortuitous that during the course of our animal studies two surgical patients (N.L. and G.M.) were available for a comparative analysis using MRI. Six months previously both patients had undergone ACL reconstruction using the central one-third of the patellar tendon. Each patient had undergone quite different rehabilitation programs, varying in degrees of aggressiveness, since their surgery.

The subjects were scanned in a 1.5 Tesla Signal clinical imaging system (General Electric Medical Systems, Milwaukee, WI). The patient's legs were placed in the transmit/receive extremity coil and rotated laterally 15 to 20 degrees to facilitate visualization of the anterior

cruciate ligament. The surgical and contralateral limbs were imaged using four pulse sequences from the standard knee protocol used at Michigan State University. The first images taken were axial T-1 weighted, 5 mm thick slices. The second and third image sets, sagittal and coronal views respectively, were also T-1 weighted. The fourth and final acquisition consisted of multi-echo (balanced and T-2 weighted) 5 mm sagittal images.

The T-1 weighted MR images showing the cross-section of the patellar tendon at the level of the meniscus were scanned (300 pixels/inch) using a Microtek scanner and digitized using a public domain software program (Image 1.44) on a Macintosh II computer. From the digitized images we approximated the ratio of the cross sectional areas of the surgical and contralateral control patellar tendons.

Histology

Fat Pads

The infrapatellar fat pads were resected from the patellar tendon immediately following extraction of the patella-patellar tendon-tibia complex from the rabbit. Each infrapatellar fat pad was placed, superior side down, on filter paper with the orientation noted. The paper and fat pad were then immersed in 10% phosphate buffered formalin. After fixation the fat pad was trimmed into three sections, two longitudinal and one cross section, and processed according to standard paraffin procedures. Samples, 6

micrometers in thickness, were cut on a Microtome and stained with hematoxylin and eosin.

Tendons

The patellar tendons from each rabbit were used for histological analysis after failure testing. The tibial heads were removed from the tibias with a reciprocating saw (Stryker Inc.) immediately following failure tests. Each was wrapped in 0.9% phosphate buffered saline soaked gauze, inserted into plastic bags, wrapped tightly and stored overnight at a temperature of 4 degrees Celsius. Adjacent cross sectional samples from the mid-tendon area were sectioned the following day for transmission electron microscopy (TEM) and histology. The TEM specimens were immediately fixed in Karnovsky fix (4% Paraormaldehyde, 5% Glutaraldehyde) and sent for analysis. These samples are currently being processed and these data are not included in this thesis. The histology specimens were fixed in 10% buffered formalin. After fixation, the patellar tendon specimens were processed for routine histology, embedded in paraffin, and sectioned on a rotary Microtome at a thickness of 6 micrometers. The specimens were stained with hematoxylin and eosin (H & E) and observed under a light microscope. The specimens were also observed under polarized light to identify the amount of mature collagen present.

Statistics

The statistical analysis used for this study was performed on CRUNCH (Crunch Software Corp.). The results are presented as mean ± one standard deviation. Initially, an ANOVA (Analysis of variance) was performed on each variable to determine whether the sample means came from the same populations. If a significant difference was noted ($p < 0.05$) with the ANOVA, then post hoc tests were performed. These tests make pairwise comparisons of the means. The Student-Newman-Keuls test (S-N-K) was chosen for the post hoc analysis. The S-N-K test compares the maximum and minimum means for each variable between groups. If the range is not significant then no further testing is required.

IV. RESULTS

The rabbits with the stress shielding device did not appear to have any gait abnormalities related to the device. Two rabbits with the stress shielding device were excluded from the study due to complications that occurred from a broken device and a broken patella. After extraction of one of the rabbit's patella-patellar tendon-tibia complexes, it was noticed that the stress shielding device had become untied sometime during the exercise program. Severe hypertrophic scar tissue was observed throughout the patellar tendon. The rabbit with the broken patella was involved in the exercise program for nearly six weeks. Three days prior to sacrifice the rabbit became lame and was removed from the program. Inspection of the knee during tissue recovery showed that the patella had fractured at the location of the 1.0 mm hole. Visual inspection of the patella showed that the hole had been drilled relatively close to the inferior end of the patella. The increased stresses generated during running probably caused the patella to fracture. Biomechanical data could not be generated, but the specimen was used for histological analysis.

Control Data

The post-hoc results for the time zero control tendon data and six week control tendon data for this study showed no significant difference in any comparisons made between all the variables. These groups were then combined to increase the statistical power of subsequent tests. All test groups were compared to the two control groups (combined) using the method of contrasts.

Tissue Dimensions

The surgical tendons, upon gross inspection and dimensional analysis of the cross-sectional area, of seven rabbits in the 40% removed without the stress shielding device group showed no signs of severe hypertrophic scarring after completion of the six week exercise program. After discussions with the animal technician, it was noted that two of these rabbits developed "sore hocks" during the exercise program and were unable to run a majority of the time. The other two rabbits were classified as "poor exercisers" and did not run well on the treadmill. The reason the patellar tendons of the remaining three rabbits in this group did not become hypertrophic cannot be explained. The data from this group of animals was considered outlier data and removed from the 40% without stress shielding group. Analysis of these data proved to be interesting, and relevant to this study, so this data was

categorized in a separate group. The new group is referred to as the "40% removed without hypertrophic scar tissue."

The average length of the control tendons was 20.7 ± 1.4 mm (n = 33), as shown in Table 1. The average length of the tendons with the central 10% removed and the 40% without hypertrophic scar formation did not differ significantly from the control tendons after the six week exercise program. However, the lengths of tendons with 40% removed, with and without a stress shielding device, differed significantly from control values. The tendons without stress shielding increased in length, on the average, by 16% while the stress shielded tendons decreased in length by 9%, when compared to controls.

The average cross sectional area of the control tendons was 14.6 ± 1.8 mm^2. The average thickness of the control tendons was 2.00 ± 0.15 mm and the average width recorded was 7.25 ± 0.60 mm. The average cross sectional area of the tendons with the central 10% removed was not significantly different than controls. The average cross sectional areas of the tendons with the central 40% removed, with and without stress shielding devices, as well as, the tendons with the central 40% removed without hypertrophic scar tissue were significantly larger than controls. These increases were due to a significant increase in both the width and thickness of the operated tendons. The cross sectional area of the operated tendons with the central 40% removed, and no stress shielding device, increased 3.0 times

in cross section when compared to contralateral controls. In comparison the cross sectional area of the tendons with the central 40% removed and with the stress shielding device increased 1.5 times compared to controls. Similarily, the cross-sectional area from the group of tendons with the central 40% removed that did not develop hypertrophic scar tissue increased in cross section by 1.3 times compared to controls (Figure 19).

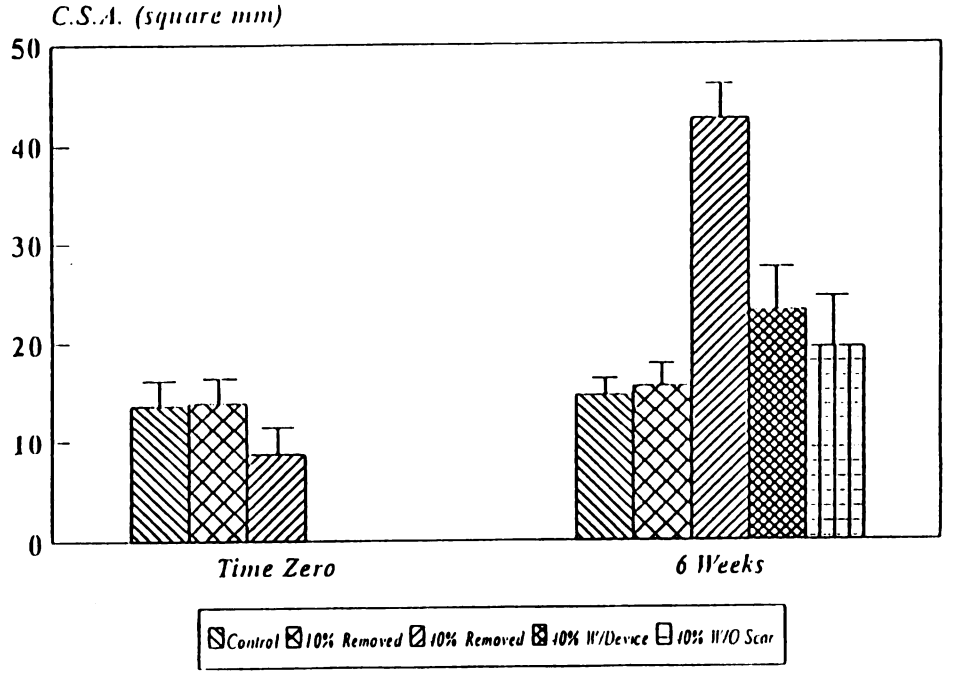


Figure 19: Histogram of cross-sectional areas

As expected, the time zero tendons with the central 40% removed differed significantly from controls. The thickness and cross-sectional area of these tendons were 70% and 60% of controls, respectively. This was due to the removal of the central portion of the patellar tendon at sacrifice.

Table 1: Tissue dimensions of control versus surgical tendons (Data is presented as Mean ± 1 S.D.)

Specimen	**Length (mm)**	**Thickness (mm)**	**C.S.A (mm^2)**
Control	20.7 ±1.4	2.0 ±0.2	14.6 ±1.8
10% Removed (T=0 Wks)	21.1 ±2.0	1.9 ±0.2	14.0 ±2.6
10% Removed (T=6 Wks)	19.7 ±1.9	2.1 ±0.2	15.6 ±1.9
40% Removed (T=0 Wks)	19.8 ±1.7	**1.4*** ±0.3	**8.7*** ±2.5
40% Removed (T=6 Wks)	**24.2*** ±2.6	**4.1*** ±0.5	**42.6*** ±3.6
40% W/Device	**18.8*** ±1.5	**2.6*** ±0.2	**23.1*** ±4.1
40% W/O Scar	20.7 ±1.9	**2.3*** ±0.2	**19.6*** ±4.2

* Denotes value that is significantly different than control ($p < 0.05$)

Structural Properties

All tendon specimens were elongated in tensile tests sufficiently to cause a mechanical failure of the patella-patellar tendon-tibia complex. The mean and standard deviation values of the structural stiffness are shown in Table 2. The structural stiffness was determined as the slope of the linear region of the load-deformation curve.

Table 2: The mechanical properties of the control versus surgical data

Specimen	**Stiffness (N/mm)**	**Modulus (MPa)**
Control	129.3 ±26.7	198.3 ±68.4
10% Removed (T=0 Wks)	130.9 ±15.2	206.7 ±61.9
10% Removed (T=6 Wks)	144.3 ±20.6	183.3 ±29.1
40% Removed (T=0 Wks)	**97.0*** ±23.4	213.7 ±70.7
40% Removed (T=6 Wks)	**85.8*** ±14.8	**49.1*** ±10.2
40% W/Device	**93.9*** ±32.9	**68.2*** ±29.9
40% W/O Scar	138.1 ±30.9	159.0 ±60.6

* Denotes value that is significantly different than control ($p < 0.05$)

The control tendons had a average structural stiffness of 129.3 ± 26.7 N/mm. There was no significant difference in structural stiffness between the tendons with the central 10% removed, at time zero and 6 weeks, and the tendons with the central 40% removed without hypertrophic scar formation when compared to controls. The two groups of tendons with the central 40% removed, with and without stress shielding, showed significant decreases in stiffness at time zero, and 6 weeks, when compared to controls (Figure 20).

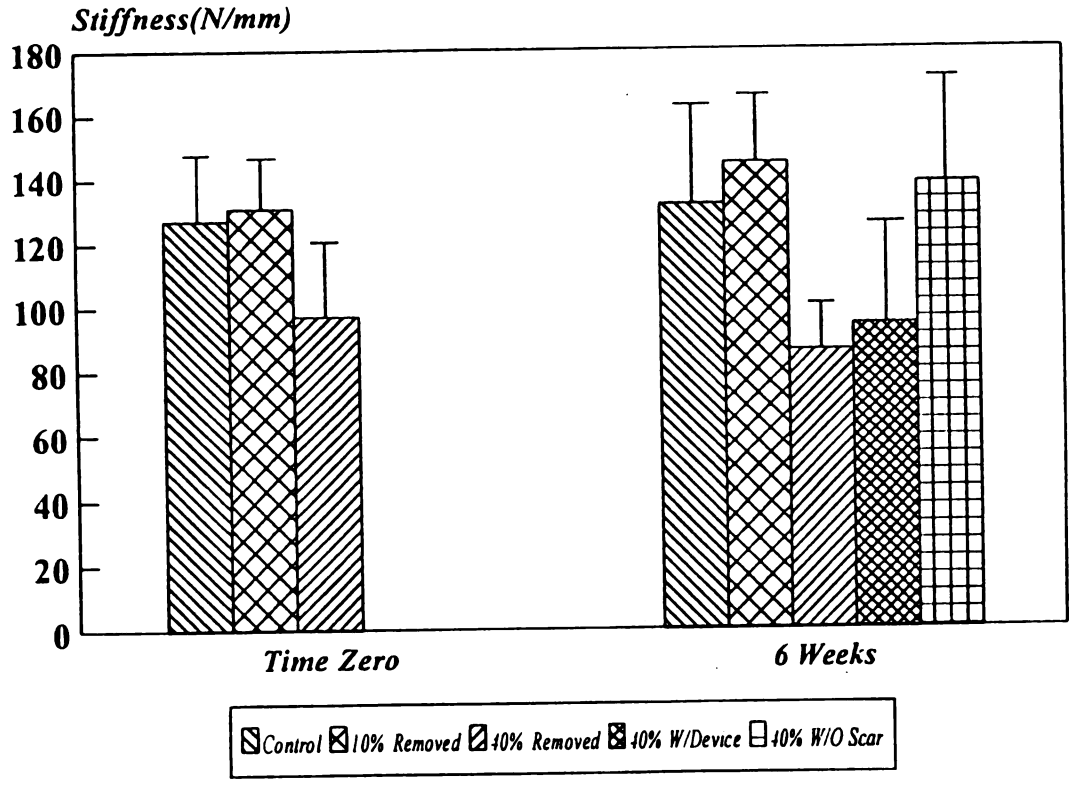


Figure 20: Structural stiffnesses of tendons after 6 weeks. Bar indicates One Standard Deviation.

The structural stiffnesses of the 40% removed tendons, with and without stress shielding, were 73% and 66% of controls, respectively. The time zero tendons with the central 40% removed recorded a stiffness that was 25% less than the control values. Additionally, these time zero tendons also recorded stiffness that was very similar to the stress shielded tendons at 6 weeks.

The failure loads for the control tendons were, on the average, 661.7 ± 175.9 N as shown in Table 3. There was no statistically significant difference in failure load of the tendons with the central 40% removed, and the tendons that

had the central 40% removed but did not develop hypertrophic scar tissue, when compared to controls. In contrast, the tendons with the central 10% removed and the tendons with the stress shielding device were significantly different than control values. The average failure load of the tendons with the central 10% removed was, on the average, 73% of control values. The low average failure loads of the 10% removed tendons are due to one specimen failing in the mid-substance area, on the lateral side only, at a relatively low load. Additionally, two specimens failed by patella avulsion at extremely low loads. Had these values been treated as outlier data, then the failure loads of this group would not be significantly different than controls. The tendons with the stress shielding device failed, on the average, at loads 57% of the failure loads of control tendons. The mechanism of failure in approximately one-half of the specimens in both groups was by mid-substance failure.

Expectedly, the average energy at failure of these same groups were significantly different than controls. The energy at failure for the tendons with the central 10% removed and the stress shielded tendons were, on the average, 63% and 44% of controls, respectively.

The time zero tendons with the central 40% removed had results that were similar to the tendons with stress shielding. The failure load of the time zero, 40% removed tendons, was only 8% larger than the stress shielded tendons

at six weeks. The energy at failure results show an increase of 29% for the time zero, 40% removed tendons compared with stress shielded tendons.

Table 3: The structural properties of the control versus surgical patellar tendons

Specimen	Failure Load (N)	Energy at Failure(J)	Failure Mode
Control	661.7 ±175.9	2.0 ±1.1	29/33 failed by avulsion
10% Removed (T=0 Wks)	707.0 ±192.3	2.2 ±1.5	4/6 failed by avulsion
10% Removed (T=6 Wks)	**481.0*** ±156.7	**1.1*** ±0.4	4/8 failed by avulsion
40% Removed (T=0 Wks)	**412.9*** ±215.0	**1.2*** ±0.8	0/8 failed by avulsion
40% Removed (T=6 Wks)	509.9 ±80.7	1.9 ±0.6	6/6 failed by avulsion
40% W/Device	**378.4*** ±91.8	**0.9*** ±0.5	3/7 failed by avulsion
40% W/O Scar	568.4 ±169.2	1.7 ±1.2	3/6 failed by avulsion

* Denotes value that is significantly different than control ($p < 0.05$)

Mechanical Properties

The tensile modulus was derived from the linear region of the tensile response curve after normalization of the load-deformation data, as shown in Table 2. The average modulus of the control tendons was 198.3 ± 68.4 MPa. Again,

the results of the tendons with the central 10% removed, at time zero and 6 weeks, the tendons without hypertrophic scar formation, and the time zero tendons with the central 40% removed did not differ significantly from controls. In contrast, the tensile modulus of tendons with the central 40% removed, with and without stress shielding, decreased significantly from the control values (Figure 21). The tensile modulus of the 40% removed, with and without stress shielding, decreased by 34% and 25% of controls, respectively.

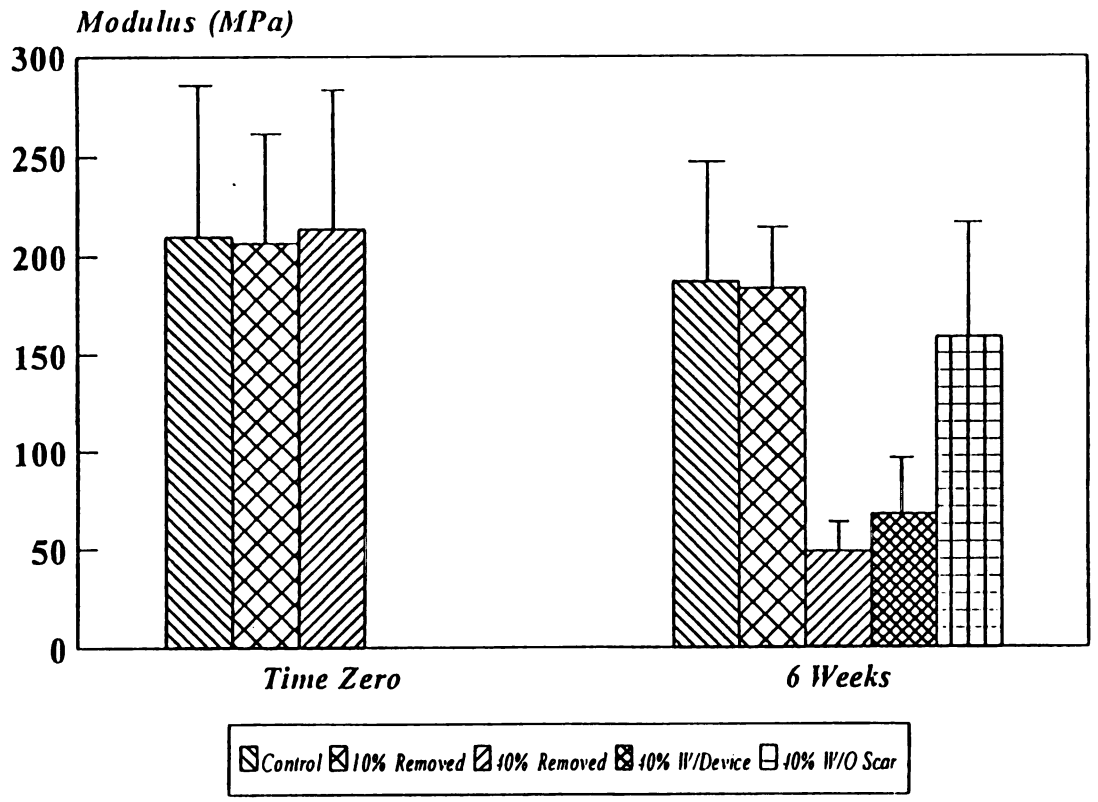


Figure 21: Tensile modulus of tendons after 6 weeks

Microstructural Model

The mathematical microstructural model fit the experimental data very well, especially in the toe region of the load-deformation curve (Figure 22). The parameter μ was, on the average, .27 ± .06 mm for control tendons (Table 4). The value of μ for tendons with the central 40% removed was the only group significantly different than controls. Statistical analysis of these tendons showed an increase in μ of 55% versus controls. Interestingly, two groups of tendons had σ values that were significantly different than control values.

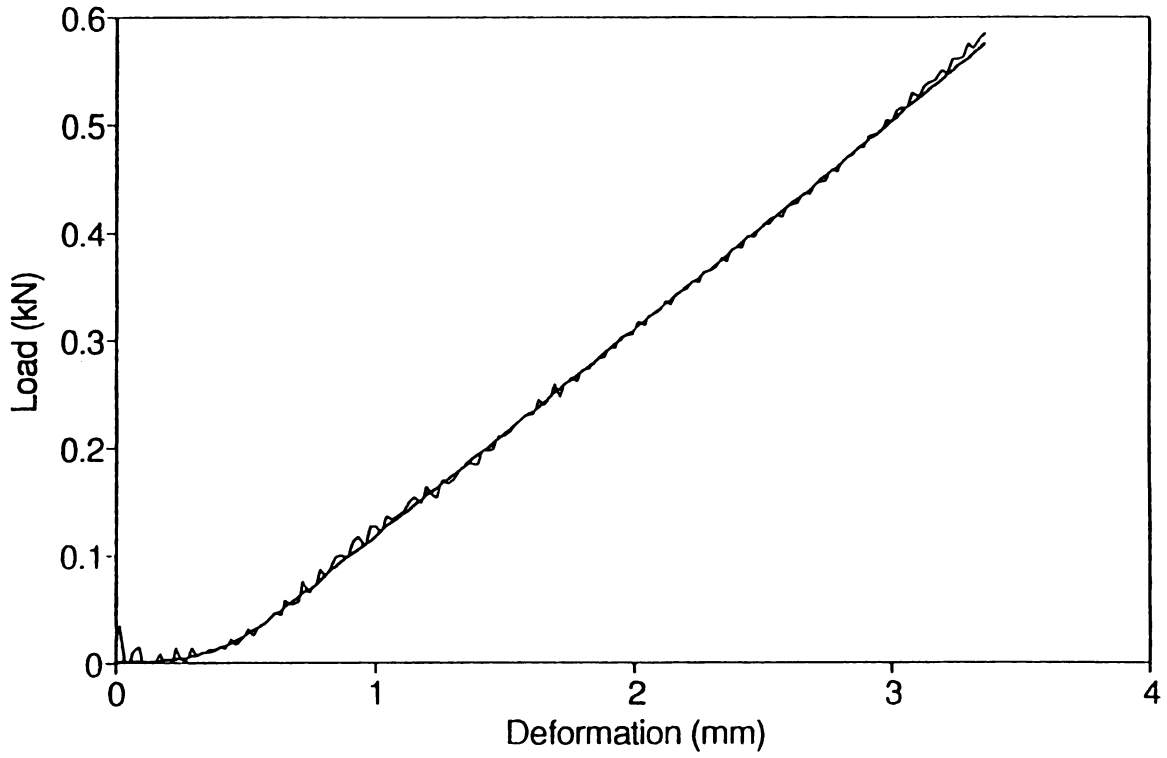


Figure 22: Experimental data and model fit

The tendons with the central 40% removed, as before, and the tendons from the group with the central 40% removed that did not show significant hypertrophic scarring had increased σ values. The control tendons had an average σ of .15 ± .08 mm. This was approximately 35% less than the tendons with the central 40% removed that did not develop hypertrophic scar tissue, and 54% less than the tendons with the central 40% removed that showed significant increase in cross-sectional area.

The μ and σ values were averaged for each group of tendons after the 6 week exercise program. These values, in addition to the average structural stiffness of each group, were input back into the model equations and the estimated "toe" regions and linear stiffnesses were plotted (Figure 23).

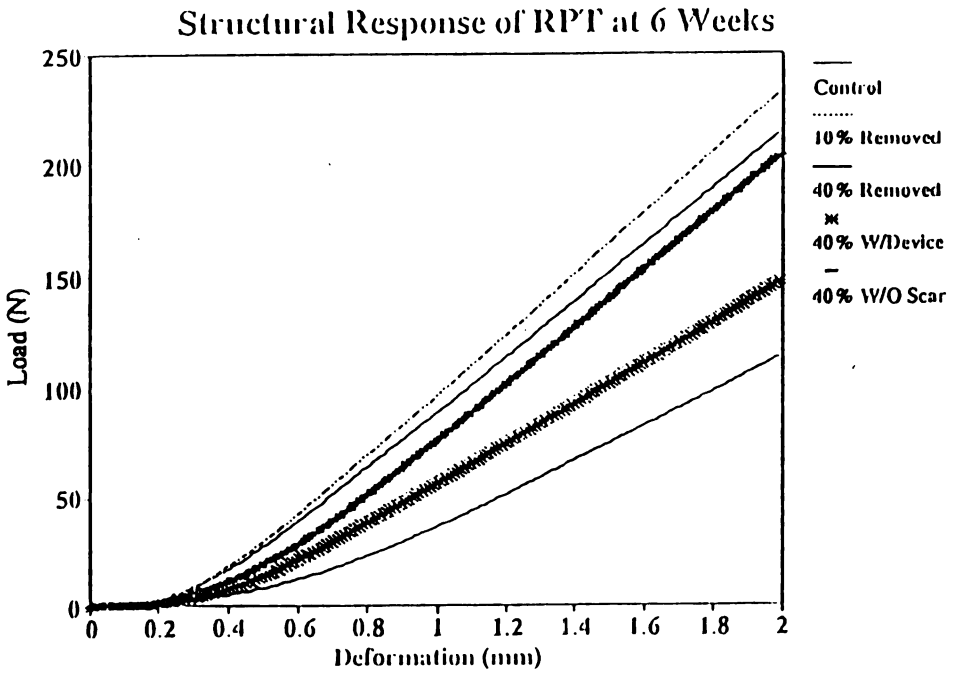


Figure 23: Estimated "toe" region and structural stiffness

Accordingly, the same procedure was used with the average tensile modulus for each group (Figure 24). These plots are valuable in showing comparisons of the toe regions for each group of tendons.

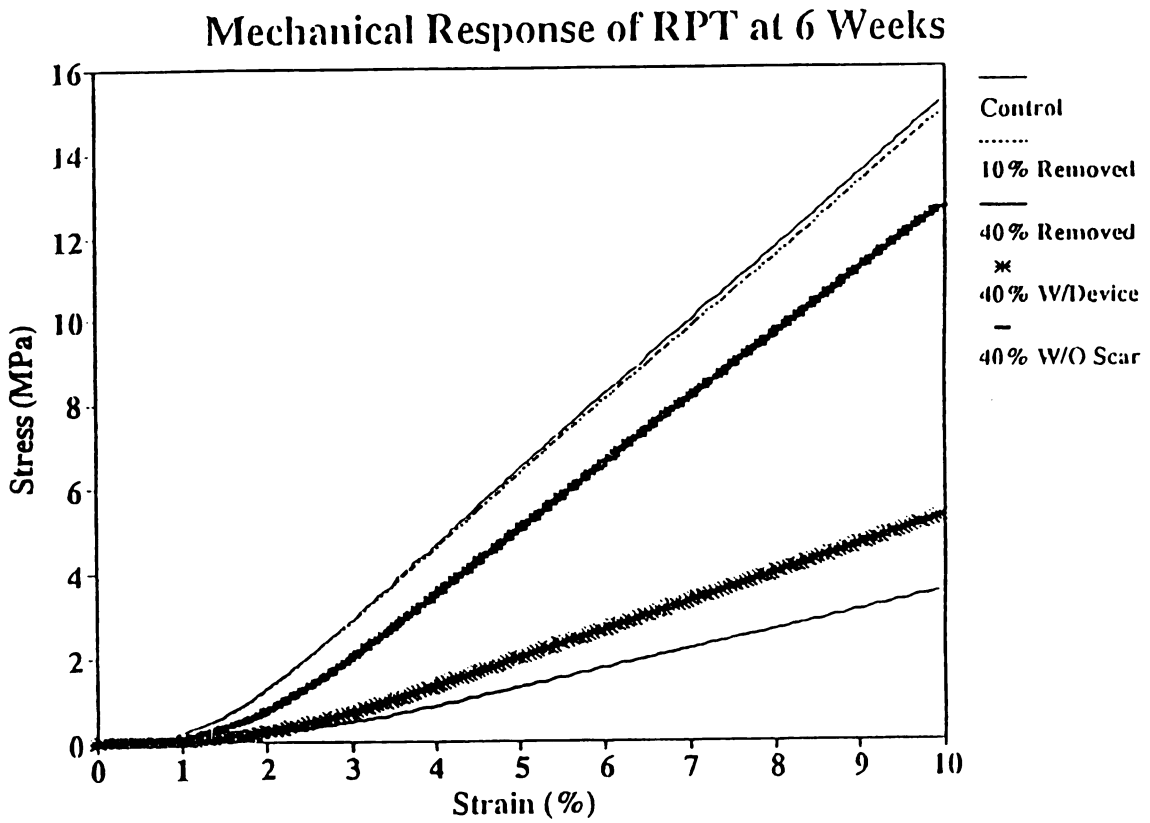


Figure 24: Estimated "toe" region and tensile modulus

The data shown in Table 4 suggests that the tendons with the central 40% removed had a much larger "toe" region than control tendons.

Table 4: Curve fitting parameters for tendons at 6 weeks.

Specimen	μ	σ
Control	0.27 ±0.08	0.16 ±0.07
10% Removed	0.27 ±0.12	0.17 ±0.07
40% Removed	**0.49*** **±0.17***	**0.35*** **±0.15***
40% W/Device	0.37 ±0.12	0.19 ±0.11
40% W/O Scar	0.36 ±0.16	**0.25*** **±0.09***

* Denotes value that is significantly different than control ($p < 0.05$)

Histology (Fat Pads)

A comparative analysis was made between the fat pads extracted from the surgical knees, after six weeks of exercise, and from the contralateral control knees. The analysis of the fat pads was subjective and performed on two longitudinal and one cross sectional samples taken from each specimen. The specimens were evaluated on the fat cell quality, amount of collagen present in the fat pad, and amount of vascularity.

The control infrapatellar fat pads were primarily composed of fat cells. Also present was small amounts of

collagen, randomly dispersed throughout the specimen, along with a small number of visible blood vessels (Figure 25).

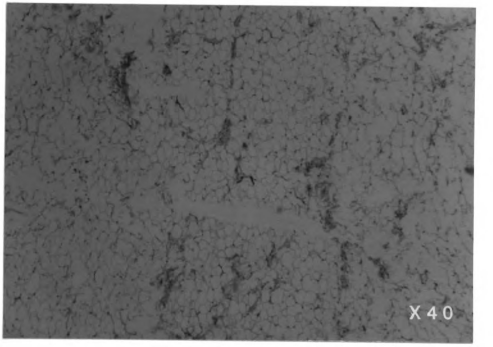


Figure 25: Histology of the control infrapatellar fat pads

The fat pads extracted from the surgical knees of rabbits with the central 10% removed did not appear to be significantly different than the contralateral control fat pads in either collagen content or vascularity (Figure 26). The rabbits with the central 40% removed that did not become hypertrophic also did not appear to be significantly different than their contralateral control tendons in either collagen content, or vascularity (Figure 27).

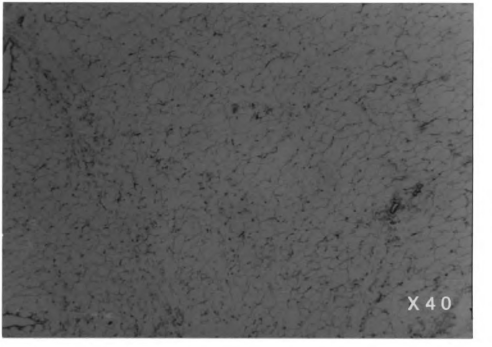


Figure 26: Histology of a infrapatellar fat pad with the central 10% removed

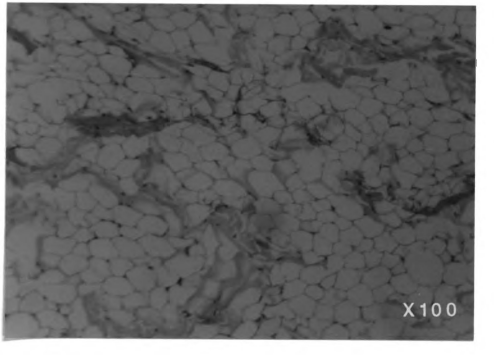


Figure 27: Histology of a infrapatellar fat pad with the central 40% removed that did not become hypertrophic

In contrast, the infrapatellar fat pads from the group of rabbits with the central 40% removed showed a small increase in the amount of vascularity and collagen (Figure 28).

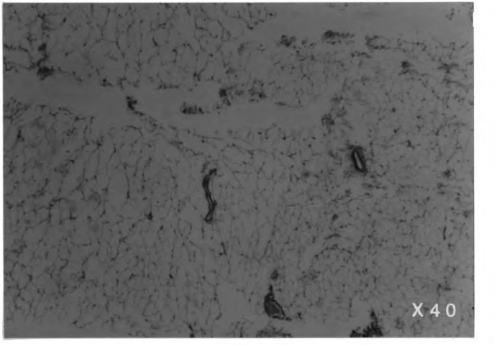


Figure 28: Histology of a infrapatellar fat pad with the central 40% removed

The infrapatellar fat pads from the surgical knees with the stress shielding device differed significantly from all other groups. These specimens showed large increases in collagen content, amount of vascularity, and cellularity (Figure 29).

Histology (Tendons)

A comparative analysis was made between the time zero control tendons and the six week operated tendons. The analysis was subjective and performed on the cross section samples taken from both limbs.

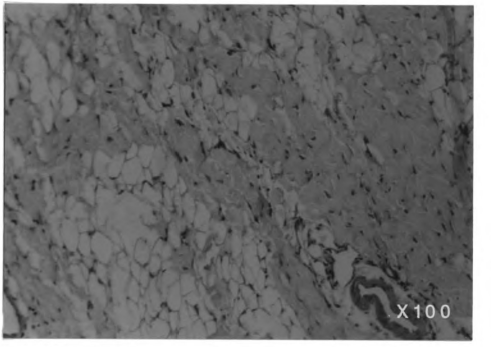


Figure 29: Histology of infrapatellar fat pad from 40% with stress shielding

These specimens were analyzed to determine if significant differences in tissue quality and characteristics were present. The control tendons of time zero, and 6 week, were composed of primarily mature collagen, when observed under polarized light. The cross sections of these patellar tendons consisted of low cellularity tissue with long, spindle shaped fibroblasts spaciously located throughout the cross section (Figure 30).

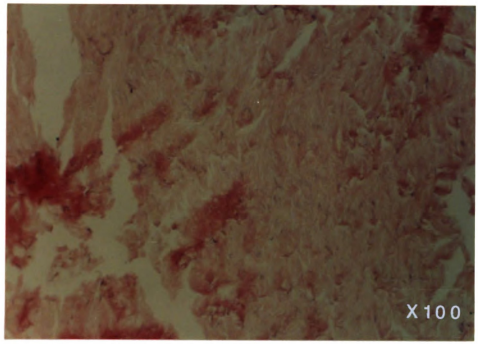


Figure 30: Histology cross section of control tendon

The four specimens of the tendons with the central 10% removed also was composed of "normal looking" tissue. Under polarized light the tendon was composed primarily of mature collagen, with relatively new collagen located in the central defect. The central defects of these specimens were completely filled with hypercellular tissue. The cells in the defect were rounded in shape, rather than spindle shaped that is often seen in normal tendon, and appeared to have penetrated slightly into the mature collagen nearest the defect (Figure 31).

The histology specimens of the three groups of tendons with the central 40% removed varied significantly in tissue quality. The patellar tendons that did not develop

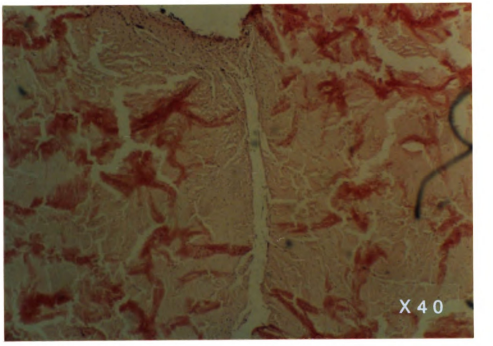


Figure 31: Histology cross section of 10% removed tendon

hypertrophic scar tissue were superior in quality compared to the other groups. These histology specimens showed mature collagen in the locations of the original tendon. The regions of mature collagen, similar to control tendons, had relatively few spindle shaped fibroblasts randomly located throughout two regions. The defect was partially filled with hypercellular tissue that contained rounded fibroblasts. The areas of mature collagen nearest the defect showed signs of increased cellularity (Figure 32).

The specimens from the tendons that were stress shielded showed two areas of normal appearing, mature

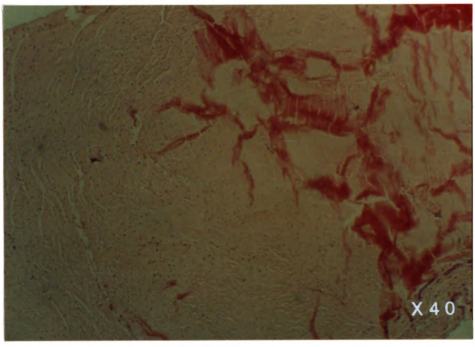


Figure 32: Histology cross section of tendons that did not become hypertrophic

collagen, as seen under polarized light, along with the defect being filled with hypercellular tissue. The soft tissue in the defect appeared to have fat pad herniation along with rounded fibroblasts. The tissue from the defect appeared to partially encompass the two areas of mature collagen. The two areas of mature collagen showed an approximate twofold increase in the number of the fibroblasts present within the boundaries. Although increased in number, these fibroblasts maintained their size and spindle shaped appearance (Figure 33).

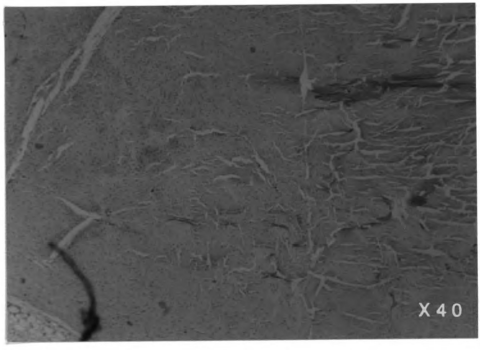


Figure 33: Histology of tendon with 40% removed and stress shielding device

The 40% removed tendons that were not stress shielded and scarred were composed of primarily immature and highly disorganized collagen, when viewed under polarized light. The entire cross section was composed of highly cellular tissue with only rounded fibroblasts present throughout the cross section. The defect area was not clearly visible due to the increase in cellularity of the entire tendon. Fat pad herniation was also present in the defect location in some of these specimens (Figure 34).

Magnetic Resonance Imaging

Rabbit studies

The MR images taken of each rabbit was used to follow the degree of hypertrophic scarring present in the surgical

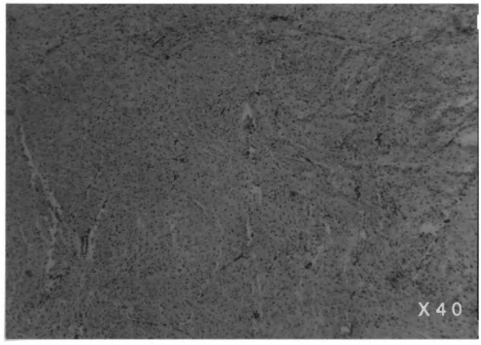


Figure 34: Histology of tendon with 40% removed and no device

patellar tendon. This data has not been analyzed and will not be discussed in this study.

Human Case Studies

CASE REPORT 1:

A 15 year old female suffered a torn anterior cruciate ligament while making a quick stop and pivot move during a soccer game on July 27, 1992. The patient's knee had a significant drawer motion when a Lachman test was performed by the examining physician. The patient had no previous knee injuries and was an active intramural athlete in basketball, soccer, and volleyball. The patient's knee was reconstructed on Aug. 12, 1992 with a central one-third patellar tendon graft. The central defect was left open.

After 1 week of immobilization the patient was given a functional leg brace and encouraged to maintain range of motion. The leg brace was worn all day for 8 weeks. Weight bearing was not encouraged until four weeks after surgery. The patient performed quadriceps contraction exercises three times a day, for 5 minutes each time, during rehabilitation. Overall, an aggressive rehabilitation was not followed, although weights were occasionally used by the patient. The rehabilitation program was not monitored by a therapist. The patient experiences pain under the kneecap after playing sports, especially after running. She also experiences pain when she gets up from sitting for long periods of time, and occasionally has difficulty in kneeling and extending the leg. Also, the knee tends to swell after any type of physical activity.

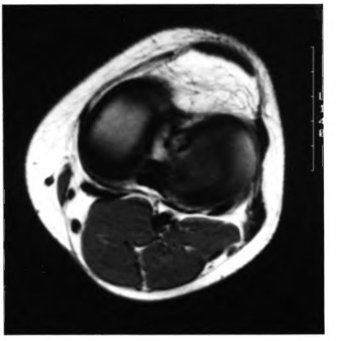
CASE REPORT 2

A 24 year old male patient suffered a torn anterior cruciate playing basketball on May 15, 1992. The knee had been subjected to a lateral blow while the knee was locked and the leg was fully extended. The injured knee had previously undergone two arthroscopic medial menisectomies. The patient conservatively treated the injury for approximately 3 months before MRI revealed a torn anterior cruciate ligament. Surgical intervention and reconstruction using the central one-third of the patellar tendon was performed. The surgical limb was not subjected to

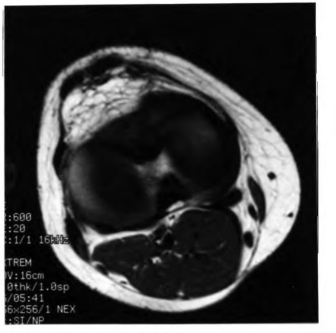
continuous passive motion following surgery. The patient was encouraged to aggressively rehabilitate the surgical limb. A soft cast was prescribed with limited motion and weight-bearing being encouraged the day after surgery. Quadriceps exercises were performed three times a day for approximately 20 minutes per session beginning 1 week after surgery. Weights were used during the rehabilitation program. The patient was an active intramural athlete prior to the injury. Limited running and hockey was resumed at approximately 6 months. The patient was squat lifting 275 pounds 6 months after surgery. The patient often experiences pain in the patellar tendon and medial portion of the knee. His knee occasionally hurts when he gets up from sitting for long periods of time or walks up stairs. He also has difficulty kneeling.

Human Study

At six months post-surgery both patients were given MRI scans. The axial MRI images of the female subject showed that the defect was still observed in the central portion of the host patellar tendon (Figure 36 a,b). The digitized MRI images of the surgical and contralateral knees indicated a slight (9.3%) increase in cross-sectional area when compared to the contralateral control. T-2 weighted sagittal images showed that the hypertrophy observed in the defect was not caused by edema, hemorrhage, or fat. A uniform MRI response was noted throughout the entire host tendon.



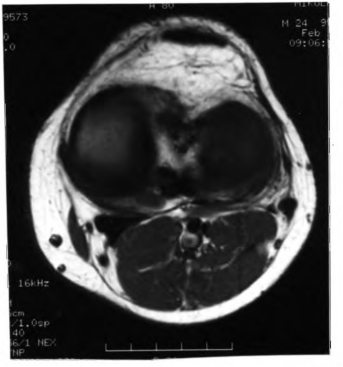
(a)



(b)

Figure 36: T-1 weighted image of a 16 year old female who was reconstructed with a central one-third PT. After 6 months of minimal exercise the (a) control tendon and (b) the surgical tendon showed the defect was filled an little hypertrophic scar was evident elsewhere.

The digitized MRI images of the male subject indicated that the cross sectional area of the host patellar tendon had increased by 136% in comparison to the contralateral control (Figure 37 a,b). T-2 weighted sagittal images showed hypertrophy in the surgical tendon was not caused by edema, hemorrhage, or fat. The tendon was uniformly enlarged, and uniformly hypertrophic throughout its cross-section. The infrapatellar fat pad in the surgical knee appears to have become highly vascular and possibly fibrotic.



(a)

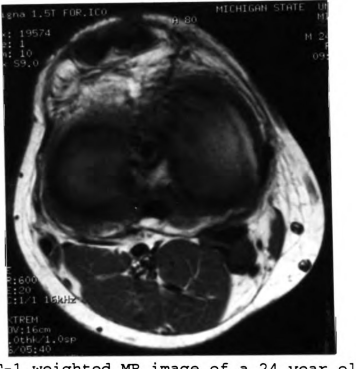


Figure 37: T-1 weighted MR image of a 24 year old patient reconstructed with a central one-third PT. After 6 months of aggressive rehabilitation the (a) control tendon and (b) the surgical tendon which shows that the surgical defect was still evident and significant scarring had developed throughout the PT. Also, the infrapatellar fat pad was altered.

V. DISCUSSION

The primary objective of the study was to determine if the excessive hypertrophic scarring of the patellar tendon often seen in animals, as well as in humans, after removal of the central one-third of the patellar tendon was the result of mechanical overstressing of the remaining host tendon. This hypothesis was tested by surgical removal of the central 10% and 40% of the rabbit patellar tendon. In addition, one group with the central 40% removed had a stress shielding device implanted to eliminate post-surgical stresses in the host tendon.

Six weeks after surgery the tendons with the central 10% removed showed a small increase in cross-sectional area and histologically appeared to be very similar to the control tendons. Similarities in the structural and mechanical properties of tendons with the central 10% removed, when compared to the control tendons, suggests that increased hypertrophic scar formation is not a physiological response to the surgical "cutting" of the host tendon. Furthermore, the slight increase in stress applied to the remaining tendon actually results in a slight increase in stiffness and a slight decrease in tensile modulus. This is paralleled with a slight increase in cross-sectional area.

The morphology of the host tendon, however, seemed to be preserved.

Linder suggested that with removal of the central one-third of the patellar tendon, there is a 67% increase in stress applied to each portion of the remaining tendon[54]. The tendons of rabbits with the central 40% removed showed a significant amount of hypertrophic scar, likely due to the increased stresses in the host tendon. The increase in cross-sectional area of the host tendon corresponded with the human MR image for the aggressive rehabilitator, as well as, a previous case study presented by Berg[16]. However, there were a group of animals with the central 40% removed that showed little or no increase in hypertrophic scar formation. These rabbits were, for the most part, classified as "poor" exercisers. I theorize that these animals applied sufficient stress to prevent disuse atrophy, yet did not apply excessive stresses on the healing tendon. The appearance of these tendons during gross inspection appear to be similar to the MR images of the human subject that was a non-aggressive rehabilitator. These data suggest that an optimum rehabilitation program likely exists.

Increases in cross-sectional area of the surgical tendons, accompanied a decrease in structural and mechanical properties of the host patellar tendon, after removal of the central 40% of the patellar tendon, and paralleled the results seen in previous studies. Haut, et al.,[43] found a 28% decrease in the structural stiffness of the operated

tendons versus controls. In addition, the surgical tendons increased in cross section by approximately 4.0 times the control values. Similarly, Burks, et al.,[18] found a decrease of structural stiffness that was 20% of control tendons, and a increase in cross section of 4.5 times the control values. In this study, the structural stiffness of tendons with 40% removed, with and without stress shielding, decreased to 73% and 66% of control tendons, respectively. The 40% removed tendons without the stress shielding also had a cross-sectional area 3.0 times controls. Burks, et al.,[18] also found a 10% shortening in the surgical tendons of dogs. The length of the surgical tendons in this study, with the central 40% removed increased 16%, similar to Haut's study. This lengthening may due to overstressing the host tendon and subsequent damage. Conversely, the length of the stress shielded tendons decreased 9% in length, and increased 1.5 times in cross section compared to controls. The decrease in length was most likely due to a contracture process that often occurs in an immobilized, healing tendon[5-7].

Amiel, et al.,[5] suggests that with stress deprivation there is a increase in collagen turnover in ligaments. With a less mature collagen fiber being more pliable, there may also be a reduced stiffness. The healing tissue is composed primarily of newly synthesized, highly unorganized collagen, with low material properties. Stresses applied it may result in permanent deformation and lengthening of the

tendon. Eventually the newly laid collagen is aligned in the direction of stress loading. These observations correlate well with the results of this study for both the 40% removed, and the stress shielded tendons. I hypothesized that the original mature collagen of the tendons with the central 40% removed was overstressed during exercise. This caused an increased synthesis of immature collagen to help repair the ensuing microdamages. The decreased mechanical properties of this newly synthesized collagen then causes the host patellar tendon to increase in cross-sectional area, so the total stiffness of the tendon increases towards normal.

The tendons with the central 40% removed showed a significantly increased "toe" region. The increased "toe" region would suggest an increase in the amount of unorganized collagen present within the composition of the tendon. The decrease in tensile modulus also suggests that these tendons are primarily composed of immature collagen. This is also supported by the analysis of the histological cross sections from these specimens. The "toe" region of the stress shielded tendons was more similar to controls than the group with the central 40% removed. Most likely this was due to the larger amount of organized collagen present in the stress shielded tendons, as seen histologically under polarized light. The stress shielding device may have protected much of the original tendon from overstressing and subsequent microtrauma. However, the

decrease in modulus observed in these tendons was likely due to the effects of disuse atrophy or stress deprivation.

The decrease in mechanical properties of the stress shielded tendons with the central 40% removed was consistent with a previous study by Yamamoto, et al.[87] The tensile modulus of rabbit tendons in Yamamoto's study decreased 9.0%, whereas, the tensile modulus of the stress shielded tendons in this study decreased 34%, after six weeks of stress shielding. The differences in results are probably due to the central 40% being removed in the stress shielded tendons for this study, while no surgery was performed in the Yamamoto study. In addition to decreases in mechanical properties, the cross-sectional area used in this study for the normalization of load-deformation data included a significant amount of unorganized, immature collagen that probably was not load bearing during tensile failure. The results of Yamamoto's study also showed an average increase in cross section of 1.3 times larger than control tendons, and a 15% decrease in length for the stress shielded tendons. However, the cross sectional area of the stress shielded tendons in this study was 1.5 times larger than controls, and the initial length decreased by only 9% of control values. The increase in cross-sectional area of the stress shielded tendon in this study was due to the newly laid scar tissue encompassing areas of original tendon.

Previous studies have shown that lack of joint movement by pin immobilization results in better alignment of

collagen in a healing tendon[36]. Similarly, Schaberg postulated in a study involving stress immobilized legs, and non-stressed immobilized legs, that pin immobilization prevents flexion and extension of the joint but allows uniaxial longitudinal stresses which promote collagen alignment[72]. Haut also found similar results in the immobilized group of rabbits in his study. Haut used a Steinmann pin immobilization technique, which probably allowed newly laid collagen to align itself in the direction of isometric stresses. In a recent study by Gomez, et al.,[39] rabbit MCL's were medially transected, permitted to heal for 4 weeks, and then placed under increased isometric tension for 8 additional weeks. Gomez found that there was no significant difference in mechanical properties between surgical and control MCL's after 12 weeks, and the newly laid collagen in the defect area appeared to be well aligned. Majima, et al.,[56] found that the degree of stress shielding affects the mechanical properties of the patellar tendon. Majima concluded that complete stress shielding significantly decreases the mechanical properties of patellar tendon after six weeks[56,57].

The scar formation in the central defect of the stress shielded tendons in this study was relatively unorganized according to the results generated from the mathematical model. Most likely the lack of stress in the completely stress shielded tendon made it difficult for the immature collagen to align itself in any predominant direction.

Although the stress shielding device did not allow for any stresses to be placed on the patellar tendon during collagen synthesis, it did allow for normal range of motion of the limb during the exercise program.

In many studies it is believe that exercise helps to stimulate tendon remodeling and increase ligament strength[86]. It is likely that this effect is shown in the results of the tendons with the central 10% removed. Most studies, however, focus on the clinical complications that often occur after ACL reconstruction using the central one-third of the patellar tendon, and do not consider the possibility of overstressing the operated tendon during physical rehabilitation. Although exercise has been shown to increase strength of a normal ligament, or tendon, the excessive stresses generated during exercise rehabilitation may be detrimental to the already traumatized host patellar tendon after removal of a central 40%.

A previous study by Majima, et al.,[56] indicates that partial stress shielding (at 30% of physiological loads) results in no significant difference in the tensile strength after six weeks. These data, along with the data presented in the current study showing protection of the host tissue from a proliferative response, would suggest that some degree of augmentation may help protect the host patellar tendon from excessive stresses during early exercise rehabilitation. This augmentation device should allow full range of motion to prevent disuse atrophy in the joint

tissues, while simultaneously sharing the forces applied to the remaining portions of the host patellar tendon. The extent of the load sharing should initially be high, and decrease with time and the degree of healing. The device used in this study was probably too stiff resulting in complete shunting of loads from the tendon. An optimal device would share loads with the host tendon. This would protect the host tendon from excessive stresses, yet allow development of newly formed collagen.

Future studies should consider using devices with different stiffnesses to better control the amount of loading on the tendon. The best scenario would probably be a device that will ultimately decrease in stiffness as the host tendon increases in stiffness by development of quality repair tissue.

VI. CONCLUSIONS

1. The results for tendons with the central 10% removed suggest that increases in cross-sectional area and decreases in the structural and mechanical properties of surgical tendons seen in earlier studies, are not a result of a surgical "cutting" of the host patellar tendon.

2. Mechanical and histological results from tendons with the central 40% removed are similar to the results from previous studies by Burks et al[18] and Haut et al[43]. Six weeks after surgery the operated tendons show significant increases in cross-sectional area and significant decreases in the structural and material properties of the host tendon. Excessive stress on the host tendon after surgery results in complete remodeling of the host tissue.

3. The stress shielded tendons also showed a decrease in stiffness, modulus, and original length six weeks after surgery. These changes were probably due to disuse atrophy resulting from a stiff augmentation device. However it seemed clear that the host tendon morphology was preserved.

4. A number of animals, after having 40% portion of tendon removed, showed little hypertrophic scar formation or change in tendon morphology.

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APPENDIX A

Table 1: Tissue dimensions of the rabbit patellar tendon prior to the six week rehabilitation program.

Control

Specimen	Length (mm)	Thickness (mm)	C.S.A. (mm^2)
A2LF	19.220	1.350	8.720
A2RF	20.400	1.470	10.650
116CF1	21.250	1.930	16.550
23RBCF	20.510	2.040	14.320
88J1CF	19.920	2.090	13.690
GO1CF	21.500	2.110	15.570
FL1CF	24.600	2.070	15.640
DEMCF	19.300	2.050	14.180
Mean	20.840	1.889	13.670
Std.	1.730	0.302	2.680

10% Removed

Specimen	Length (mm)	Thickness (mm)	C.S.A. (mm^2)
JW2LF	21.500	1.560	9.300
CK2RF	19.020	1.880	13.650
23RBTF	19.610	2.010	16.800
88J1TF	20.52	2.020	14.460
GO1TF	21.330	2.000	14.100
FL1TF	24.600	2.070	15.640
Mean	21.100	1.923	13.990
Std.	1.970	0.189	2.570

40% Removed

Specimen	Length (mm)	Thickness (mm)	C.S.A. (mm^2)
BB9LF	19.700	1.260	6.170
BB9RF	18.470	1.180	7.580
CK2LF	19.900	1.050	5.240
NTLF	20.850	1.390	8.960
NTRF	20.400	1.310	7.810
116F1	22.850	1.600	9.290
PETLTF	17.240	1.860	12.480
DEMTF	19.290	1.780	11.750
Mean	19.840	1.428	8.660
Std.	1.660	0.290	2.520

Table 2: Tissue dimensions of the rabbit patellar tendon after completion of the six week rehabilitation program.

Control

Specimen	Length (mm)	Thickness (mm)	C.S.A. (mm^2)
RS043CF	19.680	1.960	13.450
GB4CF	21.730	2.170	16.170
CKJOFCF	19.750	2.120	14.460
3GJ3CF	21.320	1.969	13.430
GH1CF	19.810	1.967	13.790
9207CF	19.610	1.926	13.350
JOEYCF	20.780	1.927	13.180
KN5CF	20.790	1.846	12.670
H3CF	20.590	1.680	13.020
JCK1CF	20.510	1.960	11.740
I40CF	19.250	1.960	13.200
I9CF	19.380	2.010	16.040
I59CF	22.410	2.083	15.000
I52CF	19.620	2.163	15.750
I51CF	20.270	1.967	14.560
I4CF	21.740	2.076	16.740
I36CF	18.370	1.948	13.880
I35CF	19.010	2.070	15.630
EN10CF	19.130	2.133	14.890
CKB5CF	18.760	1.970	12.490
CKOKCF	20.770	2.126	16.310
I50CF	20.600	2.177	16.720
I61CF	22.280	2.080	16.310
Mean	20.268	2.103	14.469
Std.	1.115	0.117	1.507

10% Removed

Specimen	Length (mm)	Thickness (mm)	C.S.A. (mm^2)
RS043TF	19.280	1.880	13.220
GB4TF	20.510	1.880	13.400
CKJOFTF	18.710	2.080	13.540
I4TF	23.100	2.100	16.870
I36TF	20.090	2.230	16.990
EN10TF	17.360	2.180	15.830
CKOKTF	20.860	2.350	17.810
I54TF	17.320	2.110	17.010
Mean	19.650	2.101	15.584
Std.	1.930	0.162	1.898

Table 2: (Continued)

40% Removed

Specimen	Length (mm)	Thickness (mm)	C.S.A. (mm^2)
GH1TF	23.320	3.730	37.670
9207TF	24.710	3.970	42.150
I40TF	21.780	4.990	45.300
I35TF	22.570	3.980	41.020
I60TF	28.450	3.770	46.670
Mean	24.170	4.090	42.560
Std.	2.630	0.520	3.570

40% W/Stress Shielding Device

Specimen	Length (mm)	Thickness (mm)	C.S.A. (mm^2)
JOEYTF	18.200	2.560	21.900
KN5TF	20.300	2.500	19.430
172TF	18.290	2.229	17.100
I59TF	17.780	2.360	22.350
I51TF	16.610	2.719	25.100
I61TF	20.700	2.800	26.740
Mean	18.650	2.528	22.103
Std.	1.560	0.214	3.543

40% W/O Hypertrophic Scar Formation

Specimen	Length (mm)	Thickness (mm)	C.S.A. (mm^2)
3GJ3TF	22.170	2.410	18.870
H3TF	21.910	2.240	16.690
I9TF	19.360	1.990	15.470
CKB5TF	17.400	2.653	26.370
Mean	20.210	2.320	19.350
Std.	2.260	0.280	4.890

APPENDIX B

Table 3: Structural properties of the rabbit patellar tendon prior to the six week rehabilitation program.

Control

Specimen	Failure Load (N)	Stiffness (N/mm)	Energy at Failure (J)
A2RF	1032.00	166.00	4.20
116CF1	675.00	144.00	2.07
23RBCF	729.80	105.37	2.24
88J1CF	480.60	123.48	1.08
GO1CF	680.90	126.14	2.02
FL1CF	667.50	107.31	0.69
DEMCF	538.45	116.07	1.59
Mean	678.41	126.91	1.99
Std	164.57	21.59	1.13

10% Removed

Specimen	Failure Load (N)	Stiffness (N/mm)	Energy at Failure (J)
JW2LF	735.00	134.00	1.95
CK2RF	672.00	144.00	1.95
23RBTF	783.20	103.59	2.92
88J1TF	396.10	123.77	0.83
GO1TF	987.90	136.98	4.60
FL1TF	667.50	142.88	0.67
Mean	706.95	130.87	2.15
Std.	192.32	15.22	1.45

40% Removed

Specimen	Failure Load (N)	Stiffness (N/mm)	Energy at Failure (J)
BB9LF	178.40	67.00	
BB9RF	518.00	109.00	1.27
CK2LF	104.00	62.00	0.11
NTLF	248.00	96.00	0.39
NTRF	448.00	101.00	1.02
116F1	632.00	130.00	1.62
PETLTF	698.65	117.78	2.60
DEMTF	476.15	93.04	1.69
Mean	412.90	96.98	1.24
Std.	214.99	23.37	0.84

Table 4: Structural properties of the rabbit patellar tendon after completion of the six week rehabilitation program.

Control

Specimen	Failure Load (N)	Stiffness (N/mm)	Energy at Failure (J)
RS043CF	825.00	126.00	3.65
GB4CF	446.20	115.00	0.84
CKJOFCF	461.20	141.00	1.50
3GJ3CF	668.40	200.00	1.38
GH1CF	488.30	154.00	0.81
9207CF	428.50	123.00	0.98
JOEYCF	447.80	172.00	0.69
KN5CF	357.40	92.00	1.10
H3CF	502.30	148.21	0.96
JCK1CF	718.50	198.11	1.69
140CF	738.70	104.80	2.68
19CF	856.60	171.23	2.94
159CF	574.05	112.13	1.89
152CF	874.43	81.28	4.04
151CF	338.20	121.09	0.89
14CF	743.20	174.95	1.10
136CF	654.15	103.33	2.07
135CF	734.25	94.30	1.86
EN10CF	365.00	114.43	0.44
CKB5CF	910.00	116.07	3.29
CKOKCF	754.30	120.22	2.35
150CF	754.30	123.63	2.52
161CF	694.20	122.71	2.88
CA80CF	623.13	143.53	1.19
F04CF	814.10	99.47	3.78
R379CF	998.00	150.00	3.58
Mean	645.01	131.72	1.97
Std	187.27	31.78	1.10

10% Removed

Specimen	Failure Load (N)	Stiffness (N/mm)	Energy at Failure (J)
RS043TF	558.90	154.00	1.50
GB4TF	248.00	103.00	0.78
CKJOFTF	346.70	158.00	0.82
14TCF	678.63	145.99	1.58
136TF	654.15	162.37	1.15
EN10TF	335.10	126.21	0.58
CKOKTF	536.23	142.75	1.12
154TF	490.00	162.28	1.47
Mean	480.96	144.32	1.13
Std.	156.69	20.60	0.37

Table 4: (Continued)

40% Removed

Specimen	Failure Load (N)	Stiffness (N/mm)	Energy at Failure (J)
GH1TF	439.40	84.00	1.23
9207TF	485.70	107.14	1.45
140TF	631.90	88.70	2.58
135TF	547.35	65.59	1.93
160TF	445.00	83.56	2.22
Mean	509.87	85.80	1.88
Std.	80.71	14.84	0.55

40% W/Stress Shielding Device

Specimen	Failure Load (N)	Stiffness (N/mm)	Energy at Failure (J)
JOEYTF	215.60	133.60	0.28
KN5TF	317.50	51.00	0.93
172TF	421.10	108.00	0.92
159TF	362.68	85.33	1.05
151TF	416.08	121.54	0.16
161TF	411.63	50.64	1.53
F04TF	503.90	106.90	1.27
Mean	378.36	93.86	0.88
Std.	91.79	32.90	0.50

40% W/O Hypertrophic Scar Formation

Specimen	Failure Load (N)	Stiffness (N/mm)	Energy at Failure (J)
3GJ3TF	508.70	148.00	0.81
H3TF	348.00	157.55	0.62
19TF	489.50	182.60	1.19
CKB5TF	596.30	112.60	1.92
CA80TF	612.90	98.29	1.96
R379BTF	854.80	129.40	3.83
Mean	568.37	138.07	1.72
Std.	169.20	30.88	1.17

APPENDIX C

Table 5: Mechanical properties of the rabbit patellar tendon prior to completion of the six week rehabilitation program.

Control

Specimen	Tensile Strength	Modulus (MPa)	% Strain at Failure
A2RF	96.90	316.00	36.56
116CF1	40.79	322.00	27.00
23RBCF	50.96	150.92	34.90
88J1CF	35.11	179.67	23.45
GO1CF	43.73	174.18	27.70
FL1CF	42.68	168.79	31.05
DEMCF	37.97	157.98	32.40
Mean	52.45	209.94	30.44
Std	21.24	75.14	4.65

10% Removed

Specimen	Tensile Strength	Modulus (MPa)	% Strain at Failure
JW2LF	79.03	309.00	27.19
CK2RF	49.23	202.00	30.30
23RBTF	46.62	120.92	40.60
88J1TF	27.39	175.65	42.00
GO1TF	70.06	207.22	46.20
FL1TF	42.68	225.48	33.10
Mean	52.50	206.71	35.57
Std.	18.89	61.91	7.45

40% Removed

Specimen	Tensile Strength	Modulus (MPa)	% Strain at Failure
BB9LF	28.91	215.00	15.70
BB9RF	68.34	270.00	29.80
CK2LF	19.85	108.00	12.00
NTLF	27.68	214.00	15.10
NTRF	57.36	265.00	22.40
116F1	68.03	322.00	24.50
PETLTF	55.98	162.70	39.60
DEMTF	40.52	152.74	36.90
Mean	45.83	213.68	24.50
Std.	19.10	70.73	10.26

Table 6: Mechanical properties of the rabbit patellar tendon after completion of the six week rehabilitation program.

Control

Specimen	Tensile Strength	Modulus (MPa)	% Strain at Failure
RS043CF	61.34	192.00	36.20
GB4CF	27.59	153.00	19.30
CKJOFCF	31.89	176.00	26.10
3GJ3CF	49.77	312.00	20.00
GH1CF	35.41	211.00	17.80
9207CF	32.10	162.00	25.90
JOEYCF	33.98	336.00	14.85
KN5CF	28.21	167.00	21.30
H3CF	38.58	234.47	19.50
JCK1CF	61.20	345.93	22.75
I40CF	5.96	152.84	35.10
I9CF	53.40	206.88	33.10
I59CF	38.27	167.52	26.00
I52CF	55.52	101.25	49.00
I51CF	23.23	168.58	22.90
I4CF	44.40	227.21	23.40
I36CF	47.13	136.76	35.20
I35CF	46.98	114.69	40.80
EN10CF	24.51	147.02	20.70
CKB5CF	72.86	174.34	41.10
CKOKCF	46.25	153.10	21.80
I50CF	45.11	152.32	32.20
I61CF	42.56	153.97	37.90
CA80CF	33.55	168.67	22.60
F04CF	58.86	140.28	42.30
R379CF	62.85	200.00	31.20
Mean	44.49	186.72	28.42
Std	13.25	61.68	9.03

10% Removed

Specimen	Tensile Strength	Modulus (MPa)	% Strain at Failure
RS043TF	42.28	211.00	27.20
GB4TF	18.51	166.00	24.10
CKJOFTF	25.61	227.00	21.00
14TCF	40.23	199.90	22.50
136TF	38.50	192.00	26.80
EN10TF	21.17	138.41	19.90
CKOKTF	30.11	167.20	33.20
154TF	28.81	165.24	29.60
Mean	30.65	183.34	25.54
Std.	8.91	29.10	4.52

Table 6: (Continued)

40% Removed

Specimen	Tensile Strength	Modulus (MPa)	% Strain at Failure
GH1TF	11.66	53.00	26.90
9207TF	11.52	62.81	25.80
140TF	13.95	42.64	39.80
135TF	13.34	36.09	38.90
160TF	9.54	50.94	31.30
Mean	12.00	49.10	32.54
Std.	1.73	10.22	6.56

40% W/Stress Shielding Device

Specimen	Tensile Strength	Modulus (MPa)	% Strain at Failure
JOEYTF	9.84	37.50	21.90
KN5TF	16.34	52.00	32.40
172TF	24.63	124.00	26.80
159TF	16.23	68.01	31.10
151TF	16.58	80.43	26.00
161TF	15.39	39.20	20.00
F04TF	17.55	75.94	26.70
Mean	16.65	68.15	26.41
Std.	4.33	29.91	4.47

40% W/O Hypertrophic Scar Formation

Specimen	Tensile Strength	Modulus (MPa)	% Strain at Failure
3GJ3TF	26.96	182.00	18.10
H3TF	20.85	206.82	15.70
19TF	31.64	228.51	24.90
CKB5TF	22.61	74.30	38.20
CA80TF	26.78	99.47	28.85
R379TF	25.52	163.00	36.20
Mean	29.63	159.02	26.99
Std.	10.19	60.63	9.22

APPENDIX D

Table 7: Curve fit parameters recorded from time zero tendons

Control

Specimen	μ	σ
A2RF	0.330	0.081
116CF1	0.263	0.314
23RBCF	0.237	0.166
88J1CF	0.245	0.110
GO1CF	0.222	0.064
FL1CF	0.252	0.098
DEMCF	0.290	0.187
Mean	0.263	0.146
Std.	0.037	0.087

10% Removed

Specimen	μ	σ
JW2LF	0.464	0.442
CK2RF	0.378	0.351
23RBTF	0.165	0.131
88J1TF	0.377	0.251
GO1TF	0.171	0.076
FL1TF	0.246	0.360
Mean	0.300	0.269
Std.	0.124	0.142

40% Removed

Specimen	μ	σ
BB9LF	0.189	0.320
BB9RF	0.537	0.433
CK2LF	0.255	0.291
NTLF	0.135	0.259
NTRF	0.228	0.173
116F1	0.563	0.169
PETLTF	0.182	0.074
DEMTF	0.265	0.102
Mean	0.294	0.228
Std.	0.163	0.120

Table 8: Curve fit parameters recorded on 6 week tendons.

Control

Specimen	μ	σ
GB4CF	0.197	0.219
CKJOFCF	0.369	0.147
3GJ3CF	0.393	0.168
GH1CF	0.222	0.125
9207CF	0.345	0.226
JOEYCF	0.198	0.117
KN5CF	0.286	0.105
H3CF	0.333	0.176
JCK1CF	0.419	0.137
I40CF	0.369	0.209
I9CF	0.199	0.085
I59CF	0.318	0.191
I52CF	0.216	0.043
I51CF	0.150	0.104
I4CF	0.227	0.099
I36CF	0.246	0.118
I35CF	0.286	0.286
EN10CF	0.232	0.140
CKB5CF	0.177	0.121
CKOKCF	0.156	0.180
I50CF	0.204	0.091
I61CF	0.186	0.290
Mean	0.260	0.153
Std	0.081	0.064

10% Removed

Specimen	μ	σ
RS043TF	0.219	0.148
GB4TF	0.271	0.087
CKJOFTF	0.327	0.246
I4TF	0.387	0.249
I36TF	0.233	0.195
EN10TF	0.128	0.072
CKOKTF	0.456	0.216
I54TF	0.114	0.134
Mean	0.267	0.168
Std.	0.119	0.069

Table 8: (Continued)

40% Removed

Specimen	μ	σ
GH1TF	0.534	0.392
9207TF	0.555	0.258
I40TF	0.222	0.143
I35TF	0.476	0.546
I60TF	0.671	0.407
Mean	0.492	0.349
Std.	0.167	0.154

40% W/Stress shielding Device

Specimen	μ	σ
JOEYTF	0.612	0.371
KN5TF	0.350	0.190
172TF	0.311	0.086
I59TF	0.347	0.205
I51TF	0.393	0.278
I61TF	0.226	0.072
Mean	0.373	0.200
Std.	0.130	0.114

40% W/O Hypertrophic Scar Formation

Specimen	μ	σ
3GJ3TF	0.499	0.354
H3TF	0.359	0.358
I9TF	0.158	0.199
CKB5TF	0.230	0.140
Mean	0.311	0.263
Std.	0.150	0.110