

## Patellar Tendon Augmentation after Removal of its Central Third Limits Joint Tissue Changes

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**Summary:** The central third of the patellar tendon is commonly used to reconstruct the injured anterior cruciate ligament. Some studies have noted changes in joint tissues following this procedure. It has been postulated that these changes may be associated with increased stress on the remaining tendon following harvest of the graft. In our study, the central third of the patellar tendon was excised in three groups of rabbits. The central tendon defects in two of the three groups were fitted with different augmentation devices to augment the host tendon during the healing process. All rabbits followed a daily treadmill exercise regimen for 12 weeks following the operation. Biomechanical testing of the tendon revealed that in nonaugmented tendons the cross-sectional area and the length of the patellar tendon significantly increased 112 and 16%, respectively. There was histological evidence of host-tendon remodeling throughout the cross section and extensive fibrosis in the infrapatellar fat pad. Augmentation of the tendon significantly reduced these changes, with the least change noted in the group with the greatest augmentation. The rabbits with augmentation devices retained tendon dimensions similar to those of the contralateral intact tendon, and tendon remodeling occurred only in the defect area. The rabbits with augmentation devices exhibited little to no fibrosis of the fat pad. Structural properties of augmented and nonaugmented tendons were similar despite the size differences, indicating higher tissue quality in the augmented tendons. This study suggested that complications of the knee joint (i.e., tendon proliferation and fat pad fibrosis) noted after anterior cruciate-ligament reconstruction with the autogenous patellar tendon may be limited by the implantation of an augmentation device.

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The central third of the patellar tendon has become the standard for reconstruction of a torn anterior cruciate ligament in patients with an active lifestyle (1). In humans, the strength and stiffness of the central third of the patellar tendon exceed those of the anterior cruciate ligament and bone plugs can be harvested to allow for bone-to-bone graft fixation (16). However, clinical and experimental studies have shown that this procedure may have detrimental and lasting effects on the host patellar tendon. Significant reductions in the strength and stiffness of the healing patellar tendon have been observed in experimental studies (4,9). In addition, both clinical (3) and experimental (9) studies have documented a doubled tendon cross section and

significant changes in the tendon's length. Other structures in the knee joint have also shown poor responses to this procedure. Clinical studies have documented tendon proliferation extending into tissues adjacent to the patellar tendon, such as the infrapatellar fat pad and joint capsule (3,15).

Remodeling and proliferation of the host patellar tendon are thought to result from increased stress in the tendon after resection of a third of the load-bearing material (4,9,12). Remodeling of the patellar tendon can be limited by joint immobilization (9); however, this protocol also results in complications (19,21,25) that may overshadow the benefits to the tendon. Related studies have investigated shunting the load across the tendon with a wire loop passed through lateral holes in the patella and tibial tubercle of both tendons with central defects (8) and entire tendons (27). Although this allows mobilization of the joint during the healing process, both studies show that complete stress-shielding of the patellar tendon results in atrophy with significant reductions in the material properties of the tendon. Other recent

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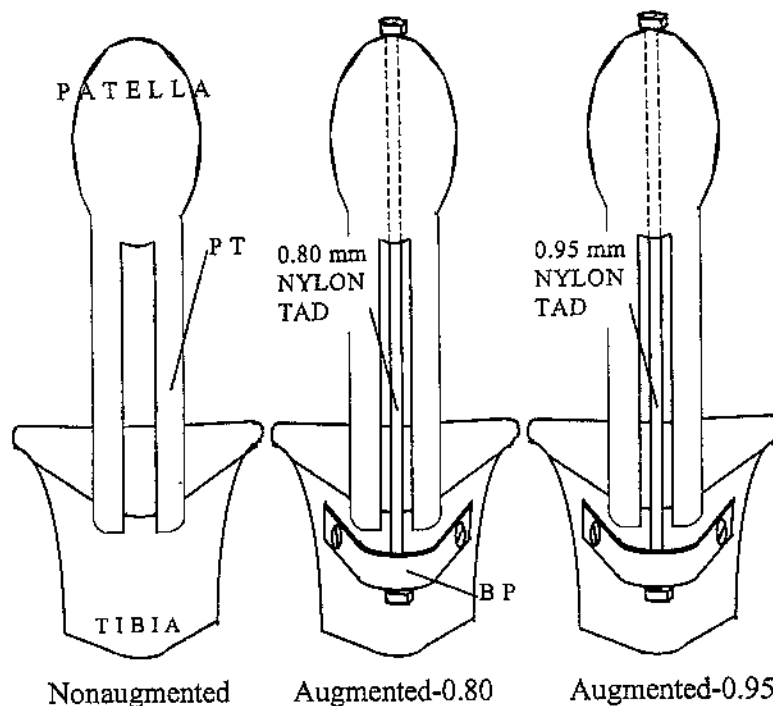


FIG. 1. The three groups operated on in the study. In the nonaugmented tendons, the central third of the patellar tendon was resected. In the augmented-0.80 and augmented-0.95 groups, the central third was resected and a tendon augmentation device (TAD) consisting of a stainless-steel bone plate (BP), a nylon monofilament (0.80 or 0.95 mm in diameter), and two stainless-steel clips was installed to rigidly fix the monofilament at the patella and bone plate.

studies on intact patellar tendons show that graded degrees of atrophy occur with graded partial shielding (14).

This study tests the hypothesis that the detrimental geometrical and biomechanical changes in the patellar tendon following harvest of the central third are due to the increased load demands on the remaining host tissue. This was tested by surgically augmenting the tendon with nylon monofilaments of varying stiffnesses in the defect of the central third. We assumed that this material would bear load in the joint, allowing the host tissue to function under loading conditions more normal than those in rabbits with nonaugmented tendons. We postulated that this augmentation would minimize changes to the patellar tendon's mechanical properties while retaining a geometry more similar to that of the normal patellar tendon.

## MATERIALS AND METHODS

Twenty-four mature Flemish Giant rabbits (weight: 4.5-5.5 kg) were used in this study. The experimental protocols were approved by the Michigan State University All-University Committee on Animal Use and Care and adhered to the National Institutes of Health guidelines for the treatment and use of animals in research. The rabbits were randomly divided into four groups of six rabbits: time zero, augmented-0.80, augmented-0.95, and nonaugmented.

One individual (C.E.D.) performed all operations. The rabbits were given a preanesthetic dose of ketamine HCl (15 mg/kg) and xylazine (2 mg/kg) intramuscularly and atropine (0.04 mg/kg) subcutaneously. They were given oxygen and halothane (0.9-2.5%) during the operations. For all rabbits, the right hindlimb was

opened anteriorly at the stifle joint. The central third of the patellar tendon was measured and cut both longitudinally and from its patellar and tibial attachments (Fig. 1). No tibial or patellar bone blocks were harvested, and the anterior cruciate ligament was not reconstructed. In rabbits whose tendons were to be augmented, a stainless-steel plate was attached to the tibial tuberosity to install the tendon augmentation device: a nylon monofilament line with a 0.80-mm (augmented-0.80) or 0.95-mm (augmented-0.95) diameter. The components of the tendon augmentation device were sterilized with use of ethylene oxide. A 1.02-mm-diameter drill bit was used to create an inferior-superior hole through the patella to insert the device. The free ends of the device were secured with small stainless-steel clips, with the knee joint maintained at a flexion angle of approximately 20° during the installation. During its insertion, the device was tensioned just enough to initiate a slight crimping of the tendon. The wound was closed with absorbable subcutaneous (4-0 Maxon green monofilament polyglyconate; Davis and Geck, Ameri Cyanamid, Marati, PR) and nonresorbable (4-0 Ethilon nylon monofilament; Ethicon, Johnson and Johnson, Somerville, NJ, U.S.A.) sutures. Each rabbit was administered an analgesic, butorphanol, after the operation.

Preliminary studies were also conducted to determine the structural properties of the isolated and implanted tendon augmentation device. Testing was performed on specimens, 40 mm in length grip to grip (typical installed length), with the 0.80 and 0.95 mm diameter nylon monofilaments. These samples were equilibrated in a 37°C saline bath and extended to failure to evaluate the stiffness of the device. Next, the stiffness of the host tendon/tendon augmentation device complex was assessed in pilot experiments on six rabbits killed at time zero. The intact patellar tendon was elongated to failure on one limb in each of these specimens. In the contralateral limb, the central third of the patellar tendon was resected and a 0.80 mm diameter tendon augmentation device was installed. The patellar tendon was tested with the device installed. Comparisons of mechanical data from contralateral limbs were made to assess the effect of the device on the stiffness of the

complex. The stiffness of the augmented limb of the rabbits killed at time zero was normalized by the contralateral intact limb and compared with 67%, which is the normalized stiffness that has been shown to result following resection of the central third of the patellar tendon of the rabbit (9). This comparison provided an understanding of the effectiveness of an augmentation device.

All rabbits were trained to run on a treadmill prior to the operation. One week after the operation, the rabbits started treadmill exercise, running at approximately 0.3 mph for 10 min/day, 5 days/week. This degree of exercise has been seen in past studies to have a significant effect on the size and mechanical properties of the healing rabbit knee (8,9).

The rabbits were killed 12 weeks after the operation with use of 2 ml of an intravenous experimental euthanasia drug under patent to Michigan State University, after which the joints were opened and photographed. The infrapatellar fat pad was dissected free and placed in 10% buffered formalin for histological analysis. The patella/patellar tendon/tibia complexes were isolated in the limbs not operated on and in those operated on, while the joint was continuously moistened with room-temperature saline solution. The tendon augmentation device was carefully cut and removed without disrupting the remaining tendon. An area micrometer was used to measure the cross-sectional area of the patellar tendon (6) by taking the average over three sites (proximal, central, and distal) with an applied pressure of 0.12 MPa. The length of the patellar tendon was measured with calipers by a single observer by applying a minimal force to straighten the tendon. The length was defined as the distance from the inferior pole of the patella to its distal insertion into the tibia (4,12).

Biomechanical experiments were conducted on the patellar tendons of limbs that had been operated on and contralateral limbs with use of previously established methods (8,9). The tibia was potted in a steel cylinder with room-temperature curing epoxy. The patella was mounted in an epoxy-filled steel box. Care was taken to not allow contact of soft tissue with the epoxy. The preparation was mounted in a servohydraulic testing machine (model 1331; Instron, Canton, MA, U.S.A.) with the following sagittal-plane orientation: the patellar tendon was oriented vertically by the patella being directly positioned over the tendon's tibial insertion, and the distal tibia was rotated posteriorly 70° from the vertical axis. A load cell (Lebow, Troy, MI, U.S.A.) and linear variable differential transformer connected to the crosshead of the machine were sampled at 500 Hz during mechanical testing. The joint preparation was equilibrated in 37°C physiological saline solution for 5 minutes prior to testing. Each preparation was preconditioned by a cyclic stretch to 3% strain at 1 Hz for 20 cycles; this was immediately followed by a failure test at 50% strain per second. The mode of tensile failure was recorded, and the patellar tendon and patella were retained in 10% buffered formalin.

Load-displacement data obtained during tensile tests on tendons exhibit a recognized initial nonlinear (toe) response, which is followed by a linear response until failure of the tendon. Because the toe response is a critical portion of the physiological loading range of tendons *in vivo* (23), an iterative curve-fitting program (2) was used to analyze both the toe and linear regions of the load-elongation data obtained in this experiment. The program quantitatively and graphically describes the stiffening response of collagen fibers under uniaxial tension as a gradual collagen-fiber recruitment, such that the stiffening is attributed to the number of fibers loaded. The fibers are unloaded at zero deformation and are loaded sequentially according to a recruitment function in which  $x$  denotes tensile elongation of the tendon (mm):

$$R(x) = \{1/\sigma(2\pi)^{1/2}\} \exp[-(x - \mu)^2/2\sigma^2].$$

This recruitment function is a normal distribution centered on  $\mu$  ( $\mu$ ), which estimates the tensile displacement necessary to recruit 50% of the fibers with a standard deviation  $\sigma$  ( $\sigma$ ). The

computer program developed for this tendon model fit the linear range response first to determine the linear-range stiffness. It was then repeated to calculate the best fit of the nonlinear region with use of the two recruitment parameters  $\mu$  and  $\sigma$ . The average values of the three fitting parameters ( $\mu$ ,  $\sigma$ , and stiffness) were used along with a modified version of the computer program already described to generate a load-displacement curve representing the average tensile response of each group. Finally, the stiffness was normalized, with use of the cross-sectional area and length of the tendon, as an estimate of the tensile modulus for the tendon substance (9,22).

After fixation in formalin, the tendon and fat pad were processed in paraffin by standard histological methods, sectioned at 8-10  $\mu$ m, and stained with hematoxylin and eosin. The sections were examined under light microscopy at 12-400 power by one author (C.D.M.) in blinded fashion. A semiquantitative five-point histological rating scale was developed specifically for this study: 1 signified a normal tissue, and 5 signified the greatest change from the normal tissue. The defect and host regions of the patellar tendon were scored independently for hypercellularity and cell maturity. Cell maturity was defined simply as follows: rounded cells indicate immaturity, and spindle-shaped cells indicate maturity (12). The fat pad was scored for the degree of collagen infiltration (fibrosis), where 1 indicated 0-20% collagen infiltration, 2 indicated 20-40% collagen infiltration, and so on. The histological scores were presented as the median and range.

Seven parameters were analyzed statistically: the curve-fitting parameters  $\mu$  and  $\sigma$ , cross-sectional area, length, structural stiffness, tensile modulus, and ultimate load. The data have been reported as the mean  $\pm$  SD. The histological data were not statistically analyzed. Statistical significance was set at a level of  $p < 0.05$ . A one-way analysis of variance was used to test for significant differences between test groups. *Post hoc* multiple pairwise comparisons were performed with the Student-Newman-Keuls test to isolate specific differences. Paired Student *t* tests were used to detect differences between contralateral intact sides and sides that were operated on.

## RESULTS

The *in situ* length of the tendon augmentation device was approximately 40 mm. Tensile experiments on the nylon monofilament with use of the 40-mm gauge length indicated that the stiffnesses of the augmented-0.80 and augmented-0.95 materials were approximately 28 and 35 N/mm, respectively, and the tensile response was linear. The stiffness of the 0.80 mm tendon augmentation device/host tendon complex implanted at time zero was  $118 \pm 26$  N/mm ( $n = 6$ ). This was 79% of the  $148 \pm 31$  N/mm ( $n = 6$ ) for the intact contralateral tendon. The normalized stiffness of the augmented-0.80 complexes was significantly greater than 67%, the normalized stiffness without augmentation (9).

The cross-sectional area and the length of the normal tendons did not vary between the groups operated on, averaging  $15.0 \pm 2.3$  mm<sup>2</sup> and  $20.7 \pm 2.4$  mm, respectively. The cross-sectional area of the nonaugmented tendons that were operated on was significantly greater than that of the contralateral patellar tendon (Table 1), and augmentation reduced this effect. No difference in tendon length was seen between the three test groups. However, the patellar tendon

**TABLE 1.** Curve-fitting parameters, dimensions, and biomechanical data of the patellar tendon (mean  $\pm$  SD)

Parameter	Test groups			
	Not operated on	Nonaugmented	Augmented-0.80	Augmented-0.95
$\mu$ (mm) <sup>a</sup>	0.19 $\pm$ 0.11	0.51 $\pm$ 0.29 <sup>b</sup>	0.39 $\pm$ 0.15 <sup>b</sup>	0.19 $\pm$ 0.07 <sup>c</sup>
$\sigma$ (mm) <sup>a</sup>	0.09 $\pm$ 0.07	0.30 $\pm$ 0.22 <sup>b</sup>	0.19 $\pm$ 0.09	0.09 $\pm$ 0.04
CSA (mm <sup>2</sup> )	15.0 $\pm$ 2.3	30.1 $\pm$ 13.1 <sup>b</sup>	20.1 $\pm$ 2.4 <sup>b</sup>	18.0 $\pm$ 2.4 <sup>b,c</sup>
Length (mm)	20.7 $\pm$ 2.4	23.2 $\pm$ 4.6 <sup>b</sup>	22.1 $\pm$ 2.3 <sup>b</sup>	21.5 $\pm$ 1.2
Stiffness (N/mm)	192.9 $\pm$ 30.6	145.3 $\pm$ 10.8 <sup>b</sup>	123.7 $\pm$ 33.8 <sup>b</sup>	147.7 $\pm$ 24.3 <sup>b</sup>
Modulus (MPa)	266.1 $\pm$ 40.4	122.6 $\pm$ 31.1 <sup>b</sup>	138.2 $\pm$ 47.1 <sup>b</sup>	179.6 $\pm$ 42.2 <sup>b</sup>
Ultimate load (N)	827.1 $\pm$ 243.2	640.1 $\pm$ 167.3	683.1 $\pm$ 346.3	715.4 $\pm$ 168.1

In the group not operated on, the central third of the patellar tendon of the rabbits was not excised; in the nonaugmented group, the central third was excised but not augmented; in the augmented-0.80 group, the tendon was augmented with a 0.80 mm diameter nylon line; and in the augmented-0.95 group, the tendon was augmented with a 0.95 mm diameter nylon line. The augmentation device was removed prior to dimensional and biomechanical analysis. CSA = cross-sectional area.

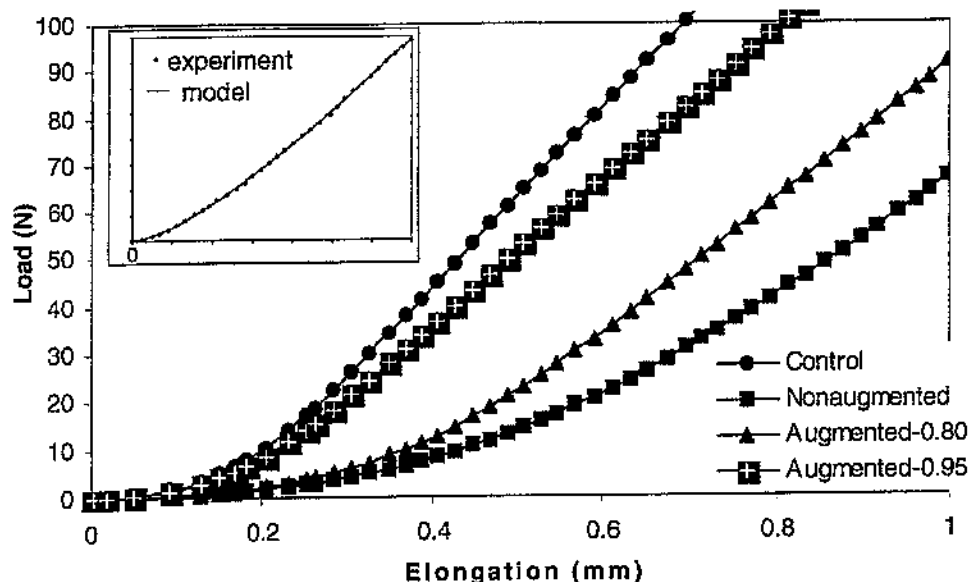
<sup>a</sup> Recruitment parameters  $\mu$  ( $\mu$ ) and  $\sigma$  ( $\sigma$ ). The recruitment function is a normal distribution centered on  $\mu$ , which estimates the tensile displacement necessary to recruit 50% of the fibers with a standard deviation  $\sigma$ .

<sup>b</sup> Different from the contralateral intact knee (paired Student *t* test, two-tailed,  $p < 0.05$ ).

<sup>c</sup> Different from the nonaugmented group (one-way analysis of variance,  $p < 0.05$ ).

was significantly longer in the nonaugmented and augmented-0.80 tendons than in the contralateral limbs within the same animal (Table 1). The length of the host patellar tendon for the tendons augmented 0.95 mm was not statistically different from that of the contralateral limbs. The cross-sectional area of the augmented-0.95 tendons was significantly smaller than that of the nonaugmented tendons. The mechanism of tensile failure was the same for all groups: failure of the tendon at its patellar insertion. The ultimate load for the intact preparations was  $827 \pm 243$

N, and the ultimate load carried by all preparations operated on was approximately 80% that of the intact limbs (Table 1). The structural stiffness of the intact tendons was  $193 \pm 31$  N/mm and did not vary significantly between groups. The intact limbs at 12 weeks were significantly stiffer than they were at time zero. The structural stiffness of tendon defects from the nonaugmented tendons was reduced to approximately 75% that of the intact limbs. Although augmentation of the host tendon reduced the amount of tissue proliferation, the structural stiffness of the augmented



**FIG. 2.** Composite load-elongation curves, for the group with the intact tendon and for the three groups operated on, constructed with use of the average values of the three parameters ( $\mu$ ,  $\sigma$ , and stiffness) for each group (Table 1). These curves represent the typical responses of the four groups of tendons. The parameters were determined by a nonlinear curve-fitting program. The correlation coefficients ( $r^2$ ) of the model fit to the load-elongation data from each patellar tendon were high ( $r^2 > 0.98$ , see inset). In addition, the correlation coefficients of each group's composite elongation curve to all tendon data they represent were calculated ( $r^2$  values for each group: normal = 0.89, augmented-0.95 = 0.93, augmented-0.80 = 0.81, and nonaugmented = 0.76).

**TABLE 2.** Histology scores, presented as the median, of the infrapatellar fat pad and of the host and defect cells of the patellar tendon

Parameter	Test groups			
	Not operated on	Nonaugmented	Augmented-0.80	Augmented-0.95
Fat pad fibrosis	1 (1-2)	4 (2-5)	3 (2-4)	2 (2-3)
Host cell maturity	1 (1-1)	4 (3-5)	2 (2-3)	1 (1-2)
Defect cell maturity		5 (4-5)	3 (3-4)	2.5 (2-3)
Host hypercellularity	1 (1-1)	4 (3-5)	2 (2-3)	1 (1-2)
Defect hypercellularity		5 (4-5)	3 (3-4)	3 (2-3)

In the group not operated on, the central third of the patellar tendon of the rabbits was not excised (therefore, there was no defect in this group); in the nonaugmented group, the central third was excised but not augmented; in the augmented-0.80 group, the tendon was augmented with a 0.80 mm diameter nylon monofilament line; and in the augmented-0.95 group, the tendon was augmented with a 0.95 mm diameter nylon monofilament line.

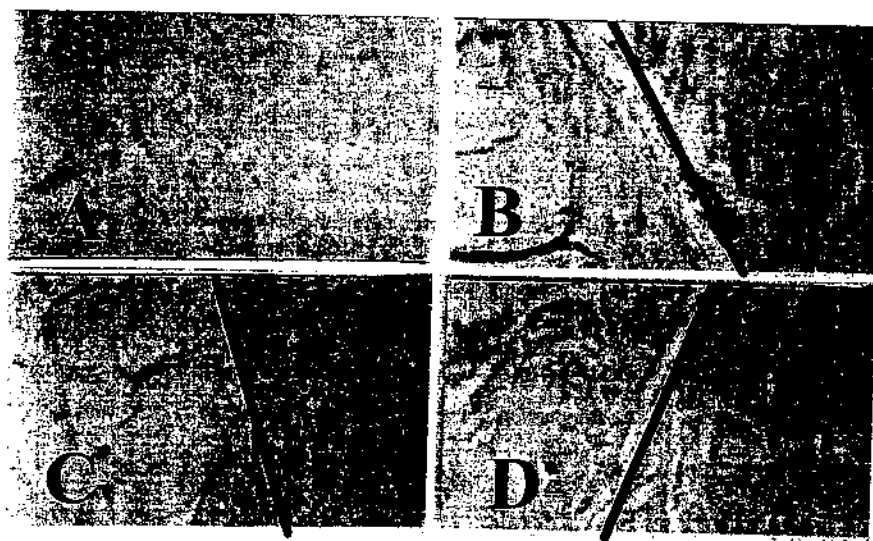
tendons did not differ from that of the nonaugmented tendons (Table 1). The tensile modulus of the intact tendons was estimated to be  $266 \pm 40$  MPa. The modulus of all tendons operated on was significantly less than that of the intact limbs: the modulus of the nonaugmented limbs was 46% that of the intact limbs, whereas the moduli of the augmented-0.80 and augmented-0.95 limbs were 52 and 56%, respectively, that of the intact limbs (Table 1).

For the nonaugmented tendons, changes in  $\mu$  and  $\sigma$  coupled with changes in the stiffness resulted in a shift in the nonlinear toe response to the right along the elongation axis (Fig. 2). Surgical augmentation of the tendon defect shifted the response curves to the left, more closely approximating the response of the intact tendons. Compared with the intact tendons, the values of  $\mu$  and  $\sigma$  were approximately tripled in the group with nonaugmented tendons, doubled in the augmented-0.80 group, and not different in the

augmented-0.95 group (Table 1). This indicated that twice as much elongation was required for the nonaugmented tendons to develop a load of 40 N compared with the intact tendons or the augmented-0.95 tendons.

The appearance of all normal joint tissues was grossly similar: the patellar tendon was white and glistening, and the fat pad was slightly yellow with firm attachments to the patella and tibial plateau. The nonaugmented tendons had a darkened, clearly visible defect. The fat pads appeared grossly darker than the fat pads from the normal joints, were extensively integrated into the tendon defect, and were detached from the patella and compressed into the distal-anterior joint space. Gross changes in the tendon and fat pad were reduced with augmentation, and no fat pads detached from the patella.

Histologically, the intact tendons exhibited a normal arrangement of collagen and tissue cellularity (Fig. 3), whereas those operated on exhibited in-



**FIG. 3.** Typical cross sections at the host-defect junction of the normal patellar tendon and the patellar tendons operated on (hematoxylin and eosin; magnification:  $\times 40$ ). The black line demarcates the host (left) from the defect (right). A: Cross section of the normal tendon. Note a minimal distribution of mature fibroblasts. B: Nonaugmented tendon. Both host and defect tissue exhibit immature, hypercellular tissue indicative of remodeling. C: Augmented-0.80, and D: augmented-0.95. Augmentation limits changes in the host and remodeling is limited to the defect.



**FIG. 4.** Transverse sections of fat pads showing gradation of fibrosis (hematoxylin and eosin; magnification:  $\times 40$ ). The numbers indicate the percentage of fibrosis in the sections in 20% increments: 1 indicates 0-20% fibrosis (typical of a fat pad from an intact joint), 2 indicates 20-40% fibrosis, and so on. Without augmentation, the median score was 4; augmentation reduced these scores to 3 and 2 for the rabbits with tendons augmented with 0.80 and 0.95 mm diameter devices, respectively (Table 2). The most common change was a focal increase in tissue fibrosis with associated neovascularization (arrow) and, to a lesser extent, a chronic inflammatory response.

creased cellularity in the defect and in the adjacent host tissue (Table 2). The nonaugmented tendons exhibited significant hypercellularity throughout the cross section, often obscuring the borders between the host and defect areas. The line of demarcation was clearly evident in the augmented tendons. All tendons operated on showed evidence of neovascularization in the defect and in the host tissue of the rabbits with the nonaugmented tendons. The nonaugmented tendons exhibited a hypercellular response composed predominantly of immature-appearing fibroblasts, whereas the augmented tendons exhibited less hypercellularity and the fibroblasts appeared more elongated and mature. The tendons augmented with the tendon augmentation device showed no signs of inflammation. The fat pads from the intact joints generally showed small amounts of collagen throughout the adipose tissue (Fig. 4). The fat pads from the joints operated on exhibited significantly higher levels of collagen infiltration (Table 2), and the rabbits with nonaugmented tendons had the most fibrosis. In cases of the greatest fibrosis, there was also proliferation of the endothelial lining of the synovium.

## DISCUSSION

This study was designed to investigate the effects of increased loads across the patellar tendon on the mechanical and histological properties of the tendon after removal of its central third. We hypothesized that extensive proliferation of the host tendon, significant reductions in the biomechanical properties of the tendon, and remodeling of the fat pad are associated with increased load in the host tendon after removal of its central third. Furthermore, we suggested that mechanical augmentation of the tendon defect would attenuate alterations to knee-joint tissues by retaining a more normal load state in the tendon.

Our results support these hypotheses, demonstrating limited changes in the tendon when augmentation was utilized. We documented a significant proliferation of the nonaugmented patellar-tendon defect at 12 weeks. Although the cross-sectional area of the ten-

don was nearly doubled, its structural stiffness was approximately 25% less than that in the contralateral intact limbs. Newly synthesized and disorganized tissue was observed throughout the tendon that was operated on, and its poor tensile qualities were confirmed by a significantly reduced tensile modulus (4,12). In contrast, mechanical augmentation of the tendon defect led to a significant reduction in tissue proliferation. The tensile response of the augmented tendons was also more comparable with that of the intact limbs, as evidenced by the qualitative response within the toe region (Fig. 2). In particular, the  $\mu$  and  $\sigma$  values quantitatively describing the toe region (Table 1) were more similar to those of the intact tendon. These parameters may better describe the effect of tendon remodeling on rabbits with augmented and nonaugmented tendons by reflecting the degree of disorganization of the collagen and the overall laxity of the tendon. For example, twice as much elongation would be required for the nonaugmented tendons to develop a load of 40 N, compared with the augmented-0.95 group or those left intact (Fig. 2). Trends in the data for  $\mu$  and  $\sigma$  were reflected in the histological studies, which indicated that much of the host tendon remained more nearly normal with augmentation.

The biomechanical data from the limbs that were operated on are similar to past studies (4,8,9,12). A previous study using the same rabbit model and time point (9) also documented approximately 30 and 13% reductions in tendon stiffness and ultimate load, respectively, with concomitantly large increases in cross-sectional area and length of the tendon. There was histological evidence of remodeling in both the host and defect tissue, as observed in the current study. Furthermore, similar increases of 15 and 188% in tendon length and cross-sectional area, respectively, and increases of 69 and 127% in the recruitment parameters  $\mu$  and  $\sigma$ , respectively, have been documented in a similar study with the rabbit model at 6 weeks (8). Similar studies on the canine patellar tendon (4,12) also show large changes in tendon size and significant remodeling throughout the host tissue. The mean es-

estimated tensile modulus of the intact limbs in the current study was 266 MPa. Kamps et al. used the same animal model and reported a similar estimated modulus of 270 MPa (9). However, these moduli data are based on grip-to-grip tests and cannot be directly compared with studies in which local surface strains are measured. Butler et al. reported that grip-to-grip strain data are typically 2.5 times greater than average local strain data (5). This suggests that a modulus calculated with local strain data would be four times greater than one calculated with grip-to-grip data. This might explain why the modulus data from the current study are approximately 25% of that reported for Japanese White rabbits (1,300 MPa) with use of local strains (14,27).

The increased stiffness of the normal patellar tendon at 12 weeks compared with stiffness at time zero suggested a stiffening of the tendon had occurred. One explanation may be associated with the daily exercise regimen. Although the exposure to exercise was relatively minimal (10 min/day on a treadmill), past studies suggest that exercise may alter the mechanical properties of tendons and ligaments (24,26). The rabbits used in our study were raised in cages until the start of the study. Thus, the daily exercise they received was essentially their primary exposure to exercise and may explain, in part, the observed changes. Another potential explanation may be that a contralateral effect occurred. Such an effect was documented by Frank et al. (7) in a model of a rabbit medial collateral-ligament defect. However, in contrast with our study, the biomechanical properties of the normal medial collateral ligament in that study were decreased. This might be due to different alterations in the load demands on the normal medial collateral ligament compared with those of the patellar tendon.

We observed two postoperative complications with the infrapatellar fat pad of the nonaugmented tendons. First, we documented detachment and retraction of the fat pad into the distal joint space. Second, we documented the replacement of adipose tissue in the fat pad with fibrous tissue. We hypothesize that these complications may have resulted from a sequence of related events. First, the patellar tendon was observed to significantly elongate, which may have caused detachment of the fat pad from its proximal attachment to the distal pole of the patella. Second, a concomitant doubling of the cross-sectional area of the tendon may have then compressed the fat pad into the distal joint space. The fat pad may have then been subjected to repeated, compressive microtrauma during daily exercise. This may have altered the state of stress in the fat pad and caused the soft adipose tissue to be replaced by collagen. Clinical studies (13,17) have implicated repeated microtrauma to explain hardening

of fat pads, as evidenced by significant infiltration of collagen. Alternately, in the current study, augmentation significantly limited increases in tendon length and cross-sectional area, with associated reduction in changes to the fat pad.

It has been suggested that fibrosis of knee-joint tissues, such as the fat pad and extraarticular capsule, could be a generalized systemic effect due to their close proximity to, and shared vasculature with, the healing patellar tendon (10,18). In particular, Hoffa's syndrome is a clinically recognized condition in which a fibrotic and hardened fat pad is associated with anterior knee pain (3,11,13,17,19,20), often requiring surgical resection of the fat pad to alleviate the pain (17). Several studies (13,19) presented histological data from patients with Hoffa's syndrome in which fat pad sections appear grossly similar to fat pad grades 4 and 5 of our study (Fig. 4). We were not able to assess a painful knee, but the rabbits generally appeared not to be in significant pain. Future studies will need to be designed to measure some aspects of a painful joint, such as altered loading during stance or gait. The studies may also be able to evaluate changes in pain levels after surgical resection of the fibrotic fat pad.

One method to prevent abnormal loading of the tendon tissue defect, and thus potentially prevent adverse joint alterations, is to immobilize the joint. In a study using the same rabbit model (9), the stifle joints were immobilized with Steinmann pins in an attempt to reduce postoperative stresses in a patellar tendon defect. In these cases, little or no tissue proliferation was documented at 12 weeks in the host. However, the resulting structural stiffness of the tendon was approximately 60% that of contralateral limbs. Recent biomechanical literature has documented some of the major drawbacks of immobilization (25). In our study, by augmenting the host tendon with the larger augmentation device, there was limited proliferation of the tendon (approximately 20% at 12 weeks) and the structural stiffness was nearly 78% that of contralateral tendons. The limbs that were operated on were allowed normal joint mobility and regular exercise. The cross-sectional area of the augmented tendons was similar to that in the immobilization studies (9); however, we documented an improved stiffness of the tendon with augmentation and mobilization of the limb that was operated on.

The actual degree of stress-shielding provided by the tendon augmentation device during the healing process was not quantified in this study. The 0.80 mm device/tendon complex was tested at time zero, and the stiffness of the complex was on average 79% that of the contralateral intact limb. A previous study shows that removal of the central third without augmentation in the rabbit results in a stiffness of approximately 67% that of the contralateral limb (9). Thus, in our study,

implantation of the 0.80 mm device yielded a 12% greater stiffness than is to be expected without augmentation. This suggests that the device was load-bearing and, therefore, participating in some degree of load-sharing with the tendon at time zero. Although the 0.95 mm device/tendon complex was not tested at time zero, grip-to-grip tests of the 0.80 and 0.95 mm diameter nylon material were conducted and showed stiffnesses of 28 and 35 N/mm, respectively. This represents a 25% increase in stiffness between the two devices. Data from the 0.80 mm device/tendon complex at time zero, as well as the grip-to-grip data on the device material, suggested that the augmented-0.95 tendons would have participated in load-sharing at a greater level than those augmented with the 0.80 mm device. These data are representative of the installed condition at time zero. The device fixations may have loosened and the nylon material may have crept during *in vivo* usage. However, notwithstanding these considerations, the rabbits with augmented tendons consistently showed significantly less tissue proliferation in the fat pad and tendon. If we assume that some degree of augmentation existed, this supports our hypothesis that reducing the load demands on the host tendon limits the proliferative response. Additional considerations may also influence these results; it may simply be that implantation of any device, drilling of the holes, or the geometrical characteristics of the device may induce healing effects. For example, the surface area, diameter, and cross-sectional area of the 0.95 mm diameter tendon augmentation device were 16, 19, and 42% greater, respectively, than those of the 0.80 mm diameter device. Thus, the 0.95 mm diameter device occupied more of the defect volume, and this may have affected the alignment and production of newly synthesized collagen. As the new collagen entered the defect from the medial and lateral host tendon, the tendon augmentation device may have prevented the interaction of these fibers at the center of the defect. This may have resulted in a more optimal alignment along the longitudinal axis of the tendon.

In our experiment, no bone blocks were excised during resection of the central third of the patellar tendon. In addition, the central third was not actually used to reconstruct the anterior cruciate ligament. These digressions from the clinical scenario were adopted in an effort to isolate effects due solely to surgical alterations of the patellar tendon.

In conclusion, postoperative proliferation of the host patellar tendon was minimized by mechanical augmentation. We believe that graded, partial augmentation of the tendon defect is partly responsible for the limited proliferative response. Furthermore, we documented a reduction in the degree of fibrosis in the fat pad when the defect of the patellar tendon was mechanically augmented. Although this experi-

mental study was limited to 12 weeks after the operation, we suggest that alterations in the fat pad and tendon themselves may contribute to long-term clinical complications in the joint. We believe these data, with use of a small-animal model, could provide a basis for investigations toward methods, operative or rehabilitation-centered, that will help control remodeling of the patellar tendon defect after its use in the reconstruction of a damaged anterior cruciate ligament.

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