

# Does Graft Construct Lengthening at the Fixations Cause an Increase in Anterior Laxity Following Anterior Cruciate Ligament Reconstruction in vivo?

**Conrad K. Smith**

Biomedical Engineering Program,  
One Shields Avenue,  
University of California,  
Davis, CA 95616

**M. L. Hull<sup>1</sup>**

Department of Biomedical Engineering,  
One Shields Avenue,  
University of California,  
Davis, CA 95616;  
Department of Mechanical Engineering,  
One Shields Avenue,  
University of California,  
Davis, CA 95616  
e-mail: mlhull@ucdavis.edu

**S. M. Howell**

Department of Mechanical Engineering,  
One Shields Avenue,  
University of California,  
Davis, CA 95616

*A millimeter-for-millimeter relation between an increase in length of an anterior cruciate ligament graft construct and an increase in anterior laxity has been demonstrated in multiple in vitro studies. Based on this relation, a 3 mm increase in length of the graft construct following surgery could manifest as a 3 mm increase in anterior laxity in vivo, which is considered clinically unstable. Hence, the two primary objectives were to determine whether the millimeter-for-millimeter relation exists in vivo for slippage-resistant fixation of a soft-tissue graft and, if it does not exist, then to what extent the increase in stiffness caused by biologic healing of the graft to the bone tunnel offsets the potential increase in anterior laxity resulting from lengthening at the sites of fixation. Sixteen subjects were treated with a fresh-frozen, nonirradiated, nonchemically processed tibialis allograft. Tantalum markers were injected into the graft, fixation devices, and bones. On the day of surgery and at 1, 2, 3, and 4 months, Roentgen stereophotogrammetric analysis was used to compute anterior laxity at 150 N of anterior force and the total slippage from both sites of fixation. A simple linear regression was performed to determine whether the millimeter-for-millimeter relation existed and a springs-in-series model of the graft construct was used to determine the extent to which the increase in stiffness caused by biological healing of the graft to the bone tunnel offset the increase in anterior laxity resulting from lengthening at the sites of fixation. There was no correlation between lengthening at the sites of fixation and the increase in anterior laxity at 1 month ( $R^2 = 0.0$ , slope = 0.2). Also, the increase in stiffness of the graft construct caused by biological healing of the graft to the bone tunnel offset 0.7 mm of the 1.5 mm potential increase in anterior laxity resulting from lengthening at the sites of fixation. This relatively large offset of nearly 50% occurred because lengthening at the sites of fixation was small.*

[DOI: 10.1115/1.4001027]

## 1 Introduction

Following anterior cruciate ligament (ACL) reconstruction, clinically important increases in anterior knee laxity ( $>3$  mm [1]) occur in 9–22% of knees during the first 2 years [2,3]. One possible cause of an increase in anterior laxity is lengthening of the graft construct, particularly lengthening at the sites of fixation [4,5].

A millimeter-for-millimeter relation between an increase in length of the graft construct and an increase in anterior laxity has been demonstrated in vitro [6–8]. Based on this relation, if there is a 3 mm increase in length of the graft construct following surgery, then a 3 mm increase in anterior laxity or a clinically unstable knee might be expected. A previous in vivo study demonstrated that the millimeter-for-millimeter relation existed for a soft-tissue autograft fixed with an interference screw fixation [9], which has been demonstrated in vitro to be slippage-prone for soft-tissue grafts [10–13], but was not valid for a bone-patellar tendon-bone autograft fixed with an interference screw [9]. Therefore, the relation between lengthening of the graft construct and an increase in anterior laxity in vivo appears to be graft construct dependent.

Because the relation between lengthening of the graft construct and an increase in anterior laxity appears to be graft construct dependent in vivo, it would be beneficial to determine whether this relation exists for slippage-resistant fixation of tibialis allografts, which are recently becoming popular due to their availability, large cross-sectional areas, increased stiffness and strength compared with bone-patellar tendon-bone grafts, ability to mimic the anatomy of the original ACL (i.e., two functional bundles), reduced surgical time, lack of donor site morbidity, and similar clinical outcomes as autografts [2,3,14,15]. If the relation does not exist, then it would be of interest to determine to what extent the increase in stiffness caused by biologic healing of the graft to the bone tunnel [16] offsets the potential increase in anterior laxity resulting from lengthening at the sites of fixation. Therefore, the primary objectives were to determine whether the millimeter-for-millimeter relation exists in vivo for slippage-resistant fixation of a soft-tissue graft and, if it does not exist, then to what extent the increase in stiffness caused by biological healing of the graft to the bone tunnel offsets the potential increase in anterior laxity resulting from lengthening at the sites of fixation.

Roentgen stereophotogrammetric analysis (RSA) has been used to measure lengthening at the sites of fixation and the increase in anterior laxity in vivo [9,17]. Because systematic and random errors will affect the measurement of lengthening at the sites of fixation and the increase in anterior laxity, it is important to quantify these errors for the specific application of the measurement

<sup>1</sup>Corresponding author.

Contributed by the Bioengineering Division of ASME for publication in the JOURNAL OF BIOMECHANICAL ENGINEERING. Manuscript received July 13, 2009; final manuscript received January 5, 2010; accepted manuscript posted January 18, 2010; published online June 15, 2010. Assoc. Editor: Mohamed Samir Hefzy.

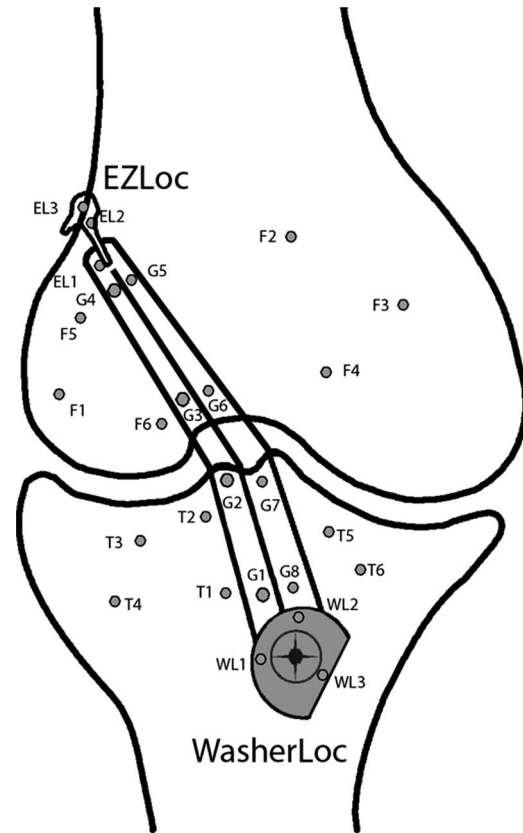
system. The bias (systematic) and precision (random) errors when using RSA to measure the distance between two markers have been determined to be 0.0 and 0.05 mm, respectively [18]. A factor that can inflate this error in measuring lengthening at the sites of fixation is migration of markers injected into soft-tissue grafts. A previous *in vitro* study determined that migration of markers injected into soft-tissue grafts does not occur [19]. However, *in vivo* it is possible that migration of injected markers could occur due to the biologic healing of the graft to the bone tunnel, remodeling of the graft substance, and nonaxial loading. Also, the precision of RSA to measure the increase in anterior laxity has been determined *in vitro* (0.33 mm) [20]; however, this precision may not be valid *in vivo* because of muscle activation. Therefore, the secondary objectives were to evaluate the precision of the measurement of anterior laxity *in vivo* and to determine whether migration of the implanted markers affects the measurement of lengthening at the sites of fixation.

## 2 Materials and Methods

All subjects with an ACL tear either chronic or acute, who were referred to the office of Barad and Howell, Sacramento, CA, between June 2007 and September 2008, and who were considering treatment with a tibialis tendon allograft, were considered for participation in this study. Chronic was defined as the date of the injury preceding the date of the surgery by more than 3 months. The inclusion criteria were: a torn ACL, intact posterior cruciate ligament (PCL), full motion at the time of reconstruction, no roentgenographic evidence of degenerative arthritis, no ligament injuries to either the medial or lateral collateral ligament, no previous ACL reconstruction procedures, and a willingness to participate in the study and be evaluated for up to 1 year post-operatively. Subjects were excluded from the study if they did not meet all of the inclusion criteria. Subjects who agreed to participate were given written information about the risks and benefits of allograft treatment. Twenty-three subjects were enrolled in the study and signed written consent forms approved by our institutional review boards (the ethical review board of Catholic Healthcare West and the IRB of UC Davis).

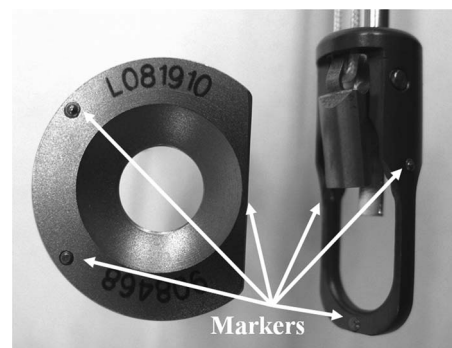
The arthroscopically assisted ACL reconstruction with a 9-mm in diameter, fresh-frozen nonirradiated, nonchemically treated tibialis allograft (Musculoskeletal Transplant Foundation, Edison, NJ) was performed under general anesthesia with use of a previously described technique [21]. Briefly, the tibial and femoral drill holes were made using a transtibial technique to avoid roof impingement, PCL impingement, and to match the tension pattern of the intact ACL during passive motion. Avoiding roof and PCL impingement and matching the tension pattern of the intact ACL is necessary to prevent lengthening of the graft from mechanical causes [22,23].

To provide references for computing the increase in anterior laxity and lengthening at the sites of fixation using RSA, a previously described technique was used to inject tantalum markers into the femur, tibia, and tibialis allograft during the surgical procedure and press-fit markers into the fixation devices prior to the surgical procedure [8]. Briefly, tantalum markers 0.8-mm in diameter (model 20401, Tilly Medical Products AB, Lund, Sweden) were implanted in the femur (N=6) and tibia (N=6) near the joint line and widely dispersed with use of a bead injector (model 20202, Tilly Medical) (Fig. 1). Tantalum markers 0.8-mm in diameter were injected into one strand (N=4) and tantalum markers 1.0-mm in diameter were injected into the other strand (N=4) of the tibialis allograft. The markers were positioned in the strands so that they were confined within the bone tunnels. Tantalum markers 1.0-mm in diameter were press-fit into holes milled into a slippage-resistant femoral fixation device (N=3) (9–10 mm standard EZLoc, Biomet Sports Medicine, Inc., Warsaw, IN) and a multispikey, slippage-resistant tibial fixation device (N=3) (size 18 WasherLoc, Biomet Sports Medicine, Inc.) (Fig. 2).

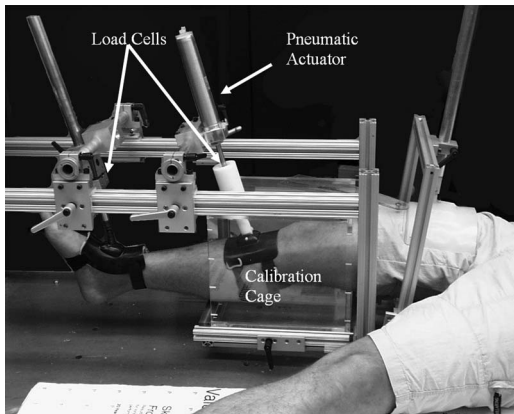


**Fig. 1** Schematic of the anterior-posterior view of the knee showing the locations of the 26 tantalum markers inserted in the femur (F1-F6), tibia (T1-T6), graft (G1-G8), femoral fixation device (EL1-EL3), and tibial fixation device (WL1-WL3). Markers with a diameter of 1.0 mm were injected into one strand (G1-G4) and markers with a diameter of 0.8 mm were injected into the other strand (G5-G8). All the markers in the allograft were inside the bone tunnels with markers G1, G4, G5, and G8 inserted 5 mm from the fixation device, and markers G2, G3, G6, and G7 inserted 5 mm inside the tunnel. The different diameters allowed the markers in each strand to be identified unambiguously.

After inserting the markers, the tibialis allograft was fixed to the femur, tension was manually applied to the distal end of the graft, and the knee was flexed and extended for 15 cycles to assure proper femoral fixation and seat the graft in the fixation



**Fig. 2** The locations of the 1.0-mm in diameter tantalum markers (arrows) are shown in the multispikey tibial fixation device (left) and the femoral fixation device (right). Three markers were press-fit into milled holes made with a 0.94-mm diameter ball end mill.

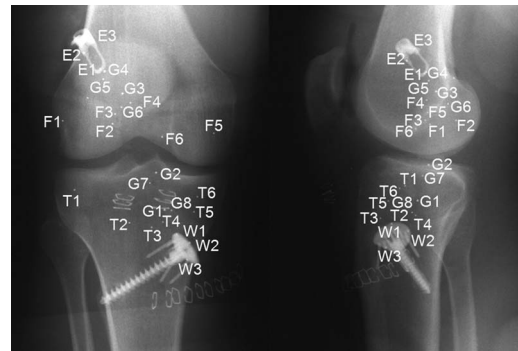


**Fig. 3** Photograph of the limb in the loading apparatus with the knee in 25 deg of flexion centered in the calibration cage, the ankle and thigh secured in supports, and a pneumatic actuator affixed to the proximal tibia. The pneumatic actuator applied posterior and anterior forces to the tibia, while the thigh and ankle were fixed. Load cells were used to measure the applied force and the reaction force at the ankle joint. The force transmitted at the knee was standardized (i.e., 150-N anterior force, 90-N posterior force) using a previously described technique.

device and the fixation device in bone. With the knee in maximum extension and the heel resting on a Mayo stand, a large, unmeasured, tensile force was manually applied to the distal end of the graft and the multiple-spike tibial fixation device was inserted and compressed with a cortical screw. Fixation was completed by inserting a 25-mm long, 8-mm in diameter autogenous bone dowel previously harvested from the tibial tunnel alongside the tibialis allograft, which increases stiffness and promotes circumferential tendon tunnel healing [16,24,25]. The wound was closed, 30 cc of bupivacaine with epinephrine mixed with 30 mg of ketorolac was injected in the knee and incisions, and 30 mg of ketorolac was administered intravenously. Compression and cold therapy were applied with a cuff (Knee Cryo Cuff, Aircast, Vista, CA).

Within 2 hours following surgery, the leg was inserted into a loading apparatus, which consisted of a calibration cage, ankle and thigh supports that restrained the movement of the leg under load, a pneumatic actuator that applied posterior and anterior forces to the shank, two load cells: one at the ankle support and one at the pneumatic actuator, and two portable X-ray machines (model HF80H+, MinXray Inc., Northbrook, IL) (Fig. 3) [8]. The knee was centered in a calibration cage (model 10, Tilly Medical Products) and a goniometer was used to position the knee in 25 deg of flexion. The ankle and thigh supports were secured. The pneumatic actuator was secured about the proximal tibia 12.5 cm distal to the joint line of the knee. The portable X-ray machines were positioned to expose anterior/posterior and lateral views of the knee (i.e., biplanar) at a distance of 87 cm from their respective film plane. Measurements were obtained so that the setup of the limb, load apparatus, and X-ray machines could be reestablished when testing the knee at each follow-up visit.

Surface electromyography was used to monitor muscle activation and the biplanar radiographs were taken when there was no muscle activation present. Surface electrodes (Bagnoli-8, DelSys, Boston, MA) were positioned over the vastus lateralis, long head of the biceps femoris, and medial head of the gastrocnemius muscles because contraction of each of these muscles affects anterior laxity [26–29]. To detect muscle activation, we relied on a previous study, which determined the optimal electromyogram (EMG) processing parameters necessary to detect activation [30]. The processing parameters used resulted in zero error in detecting



**Fig. 4** Sample biplanar radiographs of a right knee showing the anterior-posterior view (left image) and the medial-lateral view (right image). The 26 tantalum markers fixed to the femur (F1-F6), tibia (T1-T6), graft (G1-G8), femoral fixation device (EL1-EL3), and tibial fixation device (WL1-WL3) are indicated. The tantalum markers fixed to the fixation devices can be distinguished because the devices are made from less radiopaque metals.

the onset of activation. The onset of muscle activation turned on a light, which provided visual feedback to both the examiner and subject.

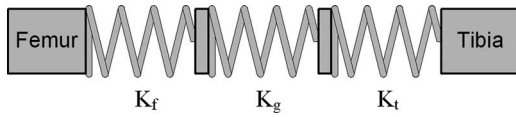
Biplanar radiographs of the knee were obtained to define an anatomical coordinate system at the center of rotation of the knee [8,20]. A line connecting three tantalum markers that were fixed in the housing of the pneumatic actuator defined the anterior direction (+*x*-axis) of the anatomical coordinate system. The cross product of the *x*-axis and the line passing through the centers of the femoral condyles defined the distal direction (+*z*-axis) and the cross product of the *z*-axis and *x*-axis defined the lateral direction (+*y*-axis) of the anatomical coordinate system [8].

Anterior and posterior forces were applied to the tibia and biplanar radiographs were obtained to determine the initial anterior position of the tibia with respect to the femur and the reference position of each marker for the subsequent computations of lengthening at the sites of fixation. The knee was not preconditioned because preconditioning might have caused lengthening at the sites of fixation causing an underestimation of the subsequent computation of lengthening at the sites of fixation. Using a previously described technique, standardized forces of 90-N posterior and 150-N anterior were transmitted at the knee [20]. After transmitting the 90-N posterior force at the knee, the 150-N anterior force was transmitted at the knee and biplanar radiographs were obtained when the muscle activation light was unlit.

Following testing on the day of surgery, the subject was instructed to weight-bear as tolerated without a brace, begin flexion and extension exercises, and was discharged from the hospital. Rehabilitation was self-administered with the goal of walking without crutches by 1 to 2 weeks, jogging by 8 weeks, and return to sport by 4 months [31,32].

Follow-up tests were performed at 1 month (29 ± 3 days), 2 months (60 ± 4 days), 3 months (94 ± 4 days), and 4 months (124 ± 8 days) (mean ± standard deviation). The position of the limb was reestablished in the loading apparatus. The knee was preconditioned by applying ten cycles at 1/4 Hertz of a 90-N posterior force followed by a 150-N anterior force transmitted at the knee. Biplanar radiographs were exposed with a 150-N anterior force transmitted at the knee.

RSA was used to determine the three-dimensional positions of the markers. Analysis of the radiographs was performed using a customized RSA data analysis system described previously [18]. Briefly, each radiograph was scanned (Epson 1600, Epson America Inc., Long Beach, CA) to provide a 300 dpi digital image (Fig. 4). The two-dimensional coordinates defining the centroid of each marker were measured with use of image analysis software



**Fig. 5 Springs-in-series model of the knee.  $K_f$  represents the stiffness of the femoral fixation,  $K_g$  represents the stiffness of the graft, and  $K_t$  represents the stiffness of the tibial fixation. The stiffness of the graft construct ( $K_{gc}$ ) is determined from  $K_{gc} = (K_f K_g K_t) / (K_f K_g + K_f K_t + K_g K_t)$ .**

(Scion Image 1.0, Scion Corporation, Frederick, MD). A customized software program was written with use of commercial software (MATLAB version 6.0, The Mathworks Inc., Natick, MA) to determine the position of the tibia with respect to the femur along the  $x$ -axis and lengthening at each site of fixation. The bias and random error of this RSA system are 0.0 and 0.05 mm, respectively [18].

Lengthening at the sites of fixation was determined as the change in position of the fixation devices and of the markers in the graft along the direction of the tunnels. A previously described methodology was used that defines the directions of the tunnels from the markers in the graft, computes both the 3D position of the markers in each fixation device with respect to the bone, and the position of the markers in the graft within the tunnels with respect to the fixation devices [8,33]. The change in the position over time defines lengthening between the fixation devices and their respective bone and the lengthening between the graft and each fixation device. The lengthening at each site of fixation (i.e., tibia and femur) was the sum of the lengthening between the fixation device and bone and between the graft and the fixation device. Total lengthening at the sites of fixation was the sum of the lengthening at both sites of fixation.

The increase in anterior laxity was determined by computing the difference in the position of the tibia with respect to the femur between each follow-up test and the position found on the day of surgery along the  $x$ -axis of the anatomical coordinate system under application of the 150-N anterior force transmitted at the knee.

To determine whether the millimeter-for-millimeter relation exists, a simple linear regression on the pooled subject data was used. The dependent variable was the increase in anterior laxity and the independent variable was total lengthening at the sites of fixation. This was performed for the month when the average total lengthening at the sites of fixation had the greatest incremental increase as this increase was expected to translate into an increase in anterior laxity based on the millimeter-for-millimeter relation.

To determine the extent to which an increase in stiffness from biological healing of the graft to the bone tunnel offset the potential increase in laxity resulting from lengthening at the sites of fixation, the graft construct was modeled using three springs in series (Fig. 5) [13]. The three springs represented the stiffness of the site of femoral fixation ( $K_f$ ), the stiffness of the graft ( $K_g$ ), and the stiffness of the site of tibial fixation ( $K_t$ ). On the day of surgery, the initial graft construct stiffness was represented by

$$K_{gc0} = \frac{K_f K_g K_t}{K_f K_g + K_f K_t + K_g K_t} \quad (1)$$

where  $K_{gc0}$  was the overall initial graft construct stiffness. Known values for the stiffness of the graft (400 N/mm) [34] and tibial fixation (565 N/mm) [24] were used, while the stiffness of the femoral fixation was estimated to be 200 N/mm. These values yielded a stiffness of the graft construct ( $K_{gc0}$ ) of 108 N/mm. On the day of surgery, the predicted contribution of the springs-in-series model to anterior laxity ( $\delta_0$ ) was computed from  $\delta_0 = F / K_{gc0}$  where  $F$  was the force transmitted at the joint line or 150 N (assuming the majority of the force transmitted at the joint line is transmitted to the ACL) [35,36], making  $\delta_0 = 1.4$  mm.

The increase in stiffness from biologic healing of the graft to the bone tunnel and from shortening of the graft also was determined using values of the individual springs in the model. At 1 month after surgery when biologic healing of the graft to the bone tunnel has occurred to a large extent [16,37,38], stiffness of all components of the graft construct increased. Therefore, the tibial and femoral fixation stiffness values were increased to 1194 and 304 N/mm, respectively, at 1 month based on previous studies, which have demonstrated a 136% increase [16] and 52% increase [39] in stiffness at the sites of tibial and similar cross-pin femoral fixation during the first month, respectively. Healing of the graft to the tunnel wall also increased the stiffness of the graft to 1200 N/mm by effectively shortening the graft from 95 mm to 30 mm assuming the graft was healed solidly at the aperture of the bone tunnel entrance into the intra-articular space. Thus, the computed stiffness of the graft construct ( $K_{gc1}$ ) at 1 month nearly doubled to 205 N/mm and the contribution to anterior laxity ( $\delta_1$ ) was 0.7 mm. The increase in anterior laxity ( $\Delta AL$ ) predicted by the springs-in-series model due to the graft construct stiffness in conjunction with lengthening at the sites of fixation ( $L_{SF}$ ) can be determined from

$$\Delta AL = \delta_1 + L_{SF} - \delta_0 \quad (2)$$

To determine the precision of the anterior laxity measurement, three sets of biplanar radiographs were obtained in 12 randomly selected subjects 1 month post-operatively. Each set of biplanar radiographs was obtained after removing the knee from the loading apparatus, reestablishing its position, preconditioning for ten cycles, and applying a 150-N anterior force transmitted at the knee. The variance in the measurement of the position of the tibia with respect to the femur from the three sets of radiographs was determined for each subject. The precision of the anterior laxity measurement was the square root of the pooled variance of these 12 subjects.

To determine whether migration of the implanted markers used to measure lengthening at the sites of fixation affects the measurement of lengthening at the sites of fixation, a correlation analysis was performed. The change in position of each of the eight markers injected into the graft with respect to the bone (i.e., distance of the marker from the bone-fixed coordinate system origin) was computed. The change in length of the vector between each marker pair (i.e., near the fixation and near the joint line) in each strand in each tunnel along the axis of the respective tunnel was also determined. Pearson's correlation coefficients between the change in length of the vector between marker pairs and the change in position of the markers with respect to bone were computed to assess whether migration occurred systematically for the markers either near the joint line or near the fixations.

### 3 Results

Seven of twenty-three subjects that consented to participate in the study were subsequently excluded for several technical reasons. Two subjects were excluded intraoperatively because of a mechanical problem with the bead injector that prevented installation of the markers. Two subjects were excluded during testing on the day of surgery, one because he/she could not tolerate the application of force due to knee pain, and one because of an equipment malfunction that prevented an accurate measurement of anterior laxity. In three subjects, the markers were not injected into the graft properly and lengthening at the sites of fixation could not be computed. Therefore, data was collected from 16 subjects, 13 males and three females with a mean age of  $36 \pm 10$  years (range, 18–47). Of these 16 subjects, five had acute tears (less than 3 months), ten had chronic tears, and one subject could not be classified.

Lengthening at the sites of fixation did not correlate with increases in anterior laxity 1 month after surgery ( $R^2 = 0.0$ , slope = 0.2). The average lengthening at the sites of fixation had its

**Table 1 Total lengthening at the sites of fixation and increase in anterior laxity for 16 subjects at the 1 month follow-up. A millimeter-for-millimeter relation did not exist.**

| Subject             | Total lengthening at sites of fixation (mm) | Increase in anterior laxity (mm) |
|---------------------|---|----------------------------------|
| 1                   | 2.9   | 2.4                              |
| 2                   | 1.5   | 0.2                              |
| 3                   | 1.8   | 0.3                              |
| 4                   | 2.3   | -0.2                             |
| 5                   | 1.5   | 2.7                              |
| 6                   | 1.6   | -1.4                             |
| 7                   | 1.1   | 1.1                              |
| 8                   | 1.3   | -1.6                             |
| 9                   | 1.1   | 1.9                              |
| 10                  | 0.5   | 3.2                              |
| 11                  | 1.9   | -0.4                             |
| 12                  | -0.1  | -0.4                             |
| 13                  | 1.2   | 2.0                              |
| 14                  | 0.9   | -3.3                             |
| 15                  | 2.8   | 1.0                              |
| 16                  | 1.1   | 1.6                              |
| Average ( $\pm$ SD) | 1.5( $\pm$ 0.8)                             | 0.6( $\pm$ 1.8)                  |

greatest increase of  $1.5 \pm 0.8$  mm (average  $\pm$  standard deviation) at 1 month and the average increase in anterior laxity was  $0.6 \pm 1.8$  mm (Table 1, Fig. 6).

The change in the anterior laxity predicted from Eq. (2) after 1 month was  $\Delta AL=0.8$  mm. Hence the increase in stiffness of the graft construct after biologic healing of the graft to the bone tunnel offset the potential increase in anterior laxity that would have occurred with no biologic healing of the graft to the bone tunnel (i.e.,  $L_{SF}$ , which was 1.5 mm) by 0.7 mm.

The precision of the anterior laxity measurement was 0.5 mm. Migration of the implanted markers did not affect the measurement of lengthening at the sites of fixation as demonstrated by the

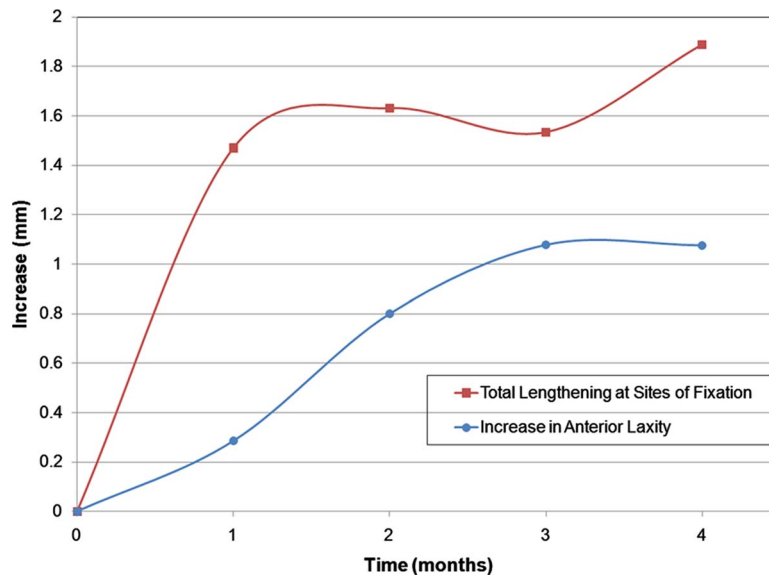
**Table 2 Summary of Pearson's correlation coefficients between the change in length of the vector between marker pairs injected into the tendon and the change in position of the markers with respect to the bone. The correlation analysis demonstrates that correlation coefficients for the markers at one location (near the joint line) were not consistently higher than those at the other location (near the fixation).**

| Marker pairs      | Marker near joint line | Marker near fixation |
|-------------------|------------------------|----------------------|
| Femur (G3 and G4) | 0.50                   | 0.31                 |
| Femur (G5 and G6) | 0.06                   | 0.67                 |
| Tibia (G1 and G2) | 0.24                   | 0.59                 |
| Tibia (G7 and G8) | 0.78                   | 0.40                 |

correlation coefficients between the change in length of the vector between marker pairs and the change in position of the markers with respect to bone which were not consistently higher for markers near the joint line compared with markers near the fixation (Table 2).

#### 4 Discussion

For soft-tissue grafts fixed with slippage-prone fixations, anterior laxity has been shown to increase greater than 3 mm, which is the threshold for a clinically unstable knee, and this increase was due to lengthening at the sites of fixations [9]. Accordingly, for soft-tissue grafts fixed with slippage-resistant fixations, we wanted to determine whether the millimeter-for-millimeter relation exists, and to what extent an increase in stiffness from biologic healing of the graft to the bone tunnel offsets the potential increase in laxity resulting from lengthening at the sites of fixation. There was no correlation between lengthening at the sites of fixation and the increase in anterior laxity ( $R^2=0.0$ , slope=0.2). The stiffness increase in the graft construct from biologic healing offset half of the potential increase in anterior laxity resulting from lengthening at the sites of fixation. The precision of the anterior laxity mea-



**Fig. 6 Graph showing the average total lengthening at the sites of femoral and tibial fixations together with the average increase in anterior laxity over the 4-month time period of the study. At 1 month, there was no one-to-one correspondence between the total lengthening at the sites of fixation and the increase in anterior laxity. After 1 month, the total lengthening did not increase appreciably, which is a consequence of biologic healing of the graft in the bone tunnels, whereas the increase in anterior laxity continued to increase until 3 months presumably as a result of a decrease in the stiffness of the graft due to remodeling.**

surement was high (0.5 mm) and migration of the markers implanted into the graft did not systematically affect the measurement of lengthening at the sites of fixation. Prior to discussing these results, two methods issues and a few limitations need to be addressed.

One methods issue that might affect the results of the study is the timing of the initial measurement of anterior laxity. The initial position of the tibia, with respect to the femur under application of the 150-N anterior force, was taken within hours following surgery when there was a bandage applied to the knee, 30 cc of bupivacaine with epinephrine mixed with 30 mg of ketorolac was injected into the capsule, and the knee was effused. Because the increase in anterior laxity is computed from the initial position of the tibia with respect to the femur under application of the 150-N anterior force, it is possible that the millimeter-for-millimeter relation could be affected. The combined effects of the bandage, the injected ketorolac, and the effusion of the knee following surgery might be expected to cause an increase in stiffness of the joint and an underestimation of the initial position of the tibia with respect to the femur. This underestimation of the initial position would cause an overestimation of the increase in anterior laxity. Because the millimeter-for-millimeter relation was not demonstrated due to larger values of lengthening at the sites of fixation ( $1.5 \pm 0.8$  mm average  $\pm$  standard deviation) than the smaller increases in anterior laxity ( $0.6 \pm 1.8$  mm), and it is possible that the increase in anterior laxity was overestimated, this methods issue does not affect the lack of correlation between lengthening at the sites of fixation and the increase in anterior laxity.

A second methods issue that might affect results is the time of the load application. Time dependent response in the form of creep should not have affected the results because the time of loading was relatively short (approximately 10 s) so that significant creep did not have time to develop [34].

The method used to apply the initial tension also might have affected our results. Initial tension to the graft was applied manually, was unmeasured, and was not reacted by the tibia. This method was chosen because it is commonly used clinically and results in a clinically stable knee as demonstrated by our results (Fig. 6) and those of others [40] when used in conjunction with the other independent variables of our surgical technique which define the graft construct (i.e., graft type, tunnel placement, and fixation methods).

One limitation of the present study is that the springs-in-series model was not useful for estimating the total (as opposed to a change in) anterior-posterior laxity for two reasons. One reason is that this model cannot account for the posterior reference position, which was defined by the posterior force transmitted by the knee. A posterior reference position defined under an applied posterior force reduced variability of anterior-posterior laxity measurements when determined with a zero-load posterior reference position [41–44]. A second reason is that the knee has both high and low-stiffness regions [45] so that the anterior laxity measured under the 150-N anterior force applied in our study results from the sum of two contributions, one in each region. Because the springs-in-series model applies to the high-stiffness region and because the majority of the laxity is due to the contribution from the low-stiffness region, the model is inherently limited in estimating the total laxity. However, our interest was in the increase in anterior laxity from the day of surgery to 1 month post-surgery. Because the knee was in the high-stiffness region due to the magnitude of load applied, the model was useful for estimating the decrease in anterior laxity that occurred solely due to a change in stiffness.

A second possible limitation of the springs-in-series model is that it can only account for a change in stiffness due to biologic healing of the graft to the bone tunnel, whereas it is also possible that a change in stiffness could result from remodeling of the graft. A previous study demonstrated a 162% decrease in stiffness of allografts during the first 6 weeks following ACL reconstruction in a goat model [46]. This decrease in stiffness increases  $\Delta$ AL

by 0.2 mm, which corresponds to a decrease in the offset of lengthening at the sites of fixation on anterior laxity from 0.7 mm to 0.5 mm, which is small.

A final limitation of the springs-in-series model is that the stiffness of the femoral fixation had to be estimated at time zero because it has not been previously measured. To apply the springs-in-series model for our purposes, the stiffness of each of the three components was necessary. The stiffness at the site of tibial fixation and the stiffness of the graft were measured previously so these values were taken directly from the literature. The stiffness at the site of femoral fixation has not been measured previously and the value used was taken from similar types of fixation devices [47,48]. The largest standard deviation (worst case) of the measured stiffness of the femoral fixation presented in these previous studies is 68 N/mm. The value (200 N/mm)  $\pm$  1 standard deviation (132 and 268 N/mm) causes  $\Delta$ AL to vary from 0.7 to 0.9 mm. This changes the offset of lengthening at the sites of fixation on anterior laxity by  $\pm 0.1$  mm, which is minimal.

Our result that lengthening at the sites of fixation did not produce a corresponding increase in anterior laxity in vivo ( $R^2 = 0.0$ ) is different than the millimeter-for-millimeter relation determined in vitro [6–8]. Because the millimeter-for-millimeter relation did not exist for slippage-resistant fixation of a tibialis allograft and because biologic healing of the graft to the bone tunnel might offset the lengthening at the sites of fixation, a springs-in-series model was used to assess this offset. Our result of a 50% decrease in the increase in anterior laxity from the effects of biologic healing of the graft to the bone tunnel demonstrates that biologic healing of the graft to the bone tunnel appears to offset to a large degree the increase in anterior laxity, resulting in a more stable knee.

Although biologic healing of the graft to the bone tunnel can offset the increase in anterior laxity caused by lengthening at the sites of fixation when the stiffness of the fixations increases, this is not the case when the stiffness decreases as demonstrated in a previous study [9]. In this previous study, lengthening at the sites of fixation was 3 mm and the increase in anterior laxity was 3.3 mm at 6 weeks following ACL reconstruction thus confirming the millimeter-for-millimeter relation determined in in vitro studies [6,8,49]. One reason that might explain the millimeter-for-millimeter relation found in the previous study is the disparity in stiffness caused by the apparent lack of biologic healing of the graft to the bone tunnel. An ovine study, which used soft-tissue grafts and interference screw fixation demonstrated that the stiffness at the site of tibial fixation decreased 40% during the first 4 weeks following ACL reconstruction [16]. Therefore, fixation devices that promote biologic healing of the graft to the bone tunnel when used with soft-tissue grafts may be necessary to offset the increase in anterior laxity caused by lengthening at the sites of fixation. Furthermore, the use of slippage-resistant fixations is advantageous to limit the amount of lengthening at the sites of fixation because the amount of lengthening that can be offset is limited.

The precision of the anterior laxity measurement (0.5 mm) is improved over previous in vivo studies which used RSA. One previous in vivo study demonstrated a precision of 0.8 mm [50], another 0.9 mm [9], while another study demonstrated a 1.1 mm precision [17]. The main reason for the precision in our study being improved over those of the previous studies is probably attributed to our choice of a center-of-rotation coordinate system [20] and the use of electromyography to detect muscle activation. Because our goal was to accurately detect an increase in anterior laxity of 3 mm and because the precision of our system was 0.5 mm, we are confident in our ability to detect clinically important increases in anterior laxity.

Even though a previous in vivo study demonstrated no migration of markers in portions of the graft inside the bone tunnels [9], and a previous in vitro study demonstrated no migration of markers injected into single-looped grafts [19], the possibility of

marker migration was assessed as any migration effects could bias the measurement of lengthening at the sites of fixation. The assessment was a worthwhile objective because different grafts and fixations were used (i.e., distal fixation versus aperture fixation) and because different dynamics such as the windshield washer effect [51] might have caused the markers near the joint line to migrate independently of the markers near the fixations. Indeed, as a result of the latter effect, it was expected that if systematic migration occurred, then it would be restricted to the markers near the joint line. However, the results of the correlation analysis (Table 2) demonstrated that correlation coefficients for the markers at one location were not consistently higher than those at the other location. Accordingly, migration of markers did not systematically affect the results of our measurement of lengthening at the sites of fixation.

The *in vivo* findings of the relatively small amount of lengthening at the sites of fixation for slippage-resistant fixations in the present study, and the relatively large amount of lengthening at the sites of fixation for the interference screw with a soft-tissue graft in the previous study [9] raises a question as to whether the results of *in vitro* studies, which measure the performance of fixation devices, carry over to the *in vivo* environment. For the slippage-resistant fixations used in our study, *in vivo* lengthening at the fixations was less than that in previous *in vitro* studies [4,11,13,52]. One study in particular reported that the femoral fixation device used in our study failed for all specimens using porcine tibia [52]. Given the results of our study, it is clear that their results do not carry over to the *in vivo* environment because no femoral fixations failed. For slippage-prone fixation devices, lengthening of interference screw fixation of a soft-tissue graft *in vivo* was greater than that in *in vitro* studies [9,11]. Therefore, *in vitro* testing of slippage-prone fixation devices should be interpreted cautiously particularly because the material properties of animal models such as porcine and older human bone do not duplicate those of young human bone [53].

In conclusion, there was no correlation between increases in lengthening at the sites of fixation and increase in anterior laxity at 1 month for a soft-tissue graft fixed with slippage-resistant fixation in our *in vivo* study. Further an increase in graft construct stiffness from biologic healing of the graft to the bone tunnel offset to a large degree the potential increase in laxity resulting from lengthening at the sites of fixation. This occurred because lengthening at the sites of fixation was small.

## Acknowledgment

The authors are grateful to the Musculoskeletal Transplant Foundation for providing financial support.

## References

- [1] Daniel, D. M., Stone, M. L., Sachs, R., and Malcom, L., 1985, "Instrumented Measurement of Anterior Knee Laxity in Patients With Acute Anterior Cruciate Ligament Disruption," *Am. J. Sports Med.*, **13**(6), pp. 401–407.
- [2] Chang, S. K., Egami, D. K., Shaieb, M. D., Kan, D. M., and Richardson, A. B., 2003, "Anterior Cruciate Ligament Reconstruction: Allograft Versus Autograft," *Arthroscopy: J. Relat. Surg.*, **19**(5), pp. 453–462.
- [3] Nyland, J., Caborn, D. N., Rothbauer, J., Kocabay, Y., and Couch, J., 2003, "Two-Year Outcomes Following ACL Reconstruction With Allograft Tibialis Anterior Tendons: A Retrospective Study," *Knee Surg. Sports Traumatol. Arthrosc.*, **11**(4), pp. 212–218.
- [4] Roos, P. J., Hull, M. L., and Howell, S. M., 2004, "Lengthening of Double-Looped Tendon Graft Constructs in Three Regions After Cyclic Loading: A Study Using Roentgen Stereophotogrammetric Analysis," *J. Orthop. Res.*, **22**(4), pp. 839–846.
- [5] Smith, C. K., Hull, M. L., and Howell, S. M., 2006, "Lengthening of a Single-Loop Tibialis Tendon Graft Construct After Cyclic Loading: A Study Using Roentgen Stereophotogrammetric Analysis," *ASME J. Biomech. Eng.*, **128**(3), pp. 437–442.
- [6] Cuppone, M., and Seedhom, B. B., 2001, "Effect of Implant Lengthening and Mode of Fixation on Knee Laxity After ACL Reconstruction With an Artificial Ligament: A Cadaveric Study," *J. Orthop. Sci.*, **6**(3), pp. 253–261.
- [7] Grover, D. M., Thompson, D. M., Hull, M. L., and Howell, S. M., 2006, "Empirical Relationship Between Lengthening an Anterior Cruciate Ligament Graft and Increases in Knee Anterior Laxity: A Human Cadaveric Study," *ASME J. Biomech. Eng.*, **128**(6), pp. 969–972.
- [8] Smith, C., Hull, M. L., and Howell, S. M., 2008, "Roentgen Stereophotogrammetric Analysis Methods for Determining Ten Causes of Lengthening of a Soft-Tissue Anterior Cruciate Ligament Graft Construct," *ASME J. Biomech. Eng.*, **130**(4), p. 041002.
- [9] Khan, R., Konyves, A., Rama, K. R., Thomas, R., and Amis, A. A., 2006, "RSA Can Measure ACL Graft Stretching and Migration: Development of a New Method," *Clin. Orthop. Relat. Res.*, **448**, pp. 139–145.
- [10] Ahmad, C. S., Gardner, T. R., Groh, M., Arnouk, J., and Levine, W. N., 2004, "Mechanical Properties of Soft Tissue Femoral Fixation Devices for Anterior Cruciate Ligament Reconstruction," *Am. J. Sports Med.*, **32**(3), pp. 635–640.
- [11] Coleridge, S. D., and Amis, A. A., 2004, "A Comparison of Five Tibial-Fixation Systems in Hamstring-Graft Anterior Cruciate Ligament Reconstruction," *Knee Surg. Sports Traumatol. Arthrosc.*, **12**(5), pp. 391–397.
- [12] Giurea, M., Zorilla, P., Amis, A. A., and Aichroth, P., 1999, "Comparative Pull-Out and Cyclic-Loading Strength Tests of Anchorage of Hamstring Tendon Grafts in Anterior Cruciate Ligament Reconstruction," *Am. J. Sports Med.*, **27**(5), pp. 621–625.
- [13] Magen, H. E., Howell, S. M., and Hull, M. L., 1999, "Structural Properties of Six Tibial Fixation Methods for Anterior Cruciate Ligament Soft Tissue Grafts," *Am. J. Sports Med.*, **27**(1), pp. 35–43.
- [14] Caborn, D. N., and Selby, J. B., 2002, "Allograft Anterior Tibialis Tendon With Bioabsorbable Interference Screw Fixation in Anterior Cruciate Ligament Reconstruction," *Arthroscopy: J. Relat. Surg.*, **18**(1), pp. 102–105.
- [15] Pearsall, A. W., Hollis, J. M., Russell, G. V. Jr., and Scheer, Z., 2003, "A Biomechanical Comparison of Three Lower Extremity Tendons for Ligamentous Reconstruction About the Knee," *Arthroscopy: J. Relat. Surg.*, **19**(10), pp. 1091–1096.
- [16] Singhatat, W., Lawhorn, K. W., Howell, S. M., and Hull, M. L., 2002, "How Four Weeks of Implantation Affect the Strength and Stiffness of a Tendon Graft in a Bone Tunnel: A Study of Two Fixation Devices in an Extraarticular Model in Ovine," *Am. J. Sports Med.*, **30**(4), pp. 506–513.
- [17] Friden, T., Ryd, L., and Lindstrand, A., 1992, "Laxity and Graft Fixation After Reconstruction of the Anterior Cruciate Ligament. A Roentgen Stereophotogrammetric Analysis of 11 Patients," *Acta Orthop. Scand.*, **63**(1), pp. 80–84.
- [18] Roos, P. J., Hull, M. L., and Howell, S. M., 2004, "How Cyclic Loading Affects the Migration of Radio-Opaque Markers Attached to Tendon Grafts Using a New Method: A Study Using Roentgen Stereophotogrammetric Analysis (RSA)," *ASME J. Biomech. Eng.*, **126**(1), pp. 62–69.
- [19] Smith, C. K., Hull, M. L., and Howell, S. M., 2005, "Migration of Radio-Opaque Markers Injected Into Tendon Grafts: A Study Using Roentgen Stereophotogrammetric Analysis (RSA)," *ASME J. Biomech. Eng.*, **127**(5), pp. 887–890.
- [20] Roos, P. J., Neu, C. P., Hull, M. L., and Howell, S. M., 2005, "A New Tibial Coordinate System Improves the Precision of Anterior-Posterior Knee Laxity Measurements: A Cadaveric Study Using Roentgen Stereophotogrammetric Analysis," *J. Orthop. Res.*, **23**(2), pp. 327–333.
- [21] Matsumoto, A., and Howell, S. M., 2005, "EZLoc: A Simple, Rigid, Femoral Fixation Device for a Soft Tissue Anterior Cruciate Ligament Graft," *Oper. Tech. Orthop.*, **20**(3), pp. 238–244.
- [22] Simmons, R., Howell, S. M., and Hull, M. L., 2003, "Effect of the Angle of the Femoral and Tibial Tunnels in the Coronal Plane and Incremental Excision of the Posterior Cruciate Ligament on Tension of an Anterior Cruciate Ligament Graft: An *In Vitro* Study," *J. Bone Jt. Surg., Am. Vol.*, **85-A**(6), pp. 1018–1029.
- [23] Howell, S. M., Wallace, M. P., Hull, M. L., and Deutsch, M. L., 1999, "Evaluation of the Single-Incision Arthroscopic Technique for Anterior Cruciate Ligament Replacement. A Study of Tibial Tunnel Placement, Intraoperative Graft Tension, and Stability," *Am. J. Sports Med.*, **27**(3), pp. 284–293.
- [24] Howell, S. M., Roos, P., and Hull, M. L., 2005, "Compaction of a Bone Dowel in the Tibial Tunnel Improves the Fixation Stiffness of a Soft Tissue Anterior Cruciate Ligament Graft—An *In Vitro* Study in Calf Tibia," *Am. J. Sports Med.*, **33**(5), pp. 719–725.
- [25] Matsumoto, A., Howell, S. M., and Liu-Barba, D., 2006, "Time-Related Changes in the Cross-Sectional Area of the Tibial Tunnel After Compaction of an Autograft Bone Dowel Alongside a Hamstring Graft," *Arthroscopy: J. Relat. Surg.*, **22**(8), pp. 855–860.
- [26] Solomonow, M., Baratta, R., Zhou, B. H., Shoji, H., Bose, W., Beck, C., and Dambrosia, R., 1987, "The Synergistic Action of the Anterior Cruciate Ligament and Thigh Muscles in Maintaining Joint Stability," *Am. J. Sports Med.*, **15**(3), pp. 207–213.
- [27] Feller, J., Hoser, C., and Webster, K., 2000, "EMG Biofeedback Assisted KT-1000 Evaluation of Anterior Tibial Displacement," *Knee Surg. Sports Traumatol. Arthrosc.*, **8**(3), pp. 132–136.
- [28] Fleming, B. C., Renstrom, P. A., Ohlen, G., Johnson, R. J., Peura, G. D., Beynon, B. D., and Badger, G. J., 2001, "The Gastrocnemius Muscle is an Antagonist of the Anterior Cruciate Ligament," *J. Orthop. Res.*, **19**(6), pp. 1178–1184.
- [29] Markolf, K. L., Graff-Radford, A., and Amstutz, H. C., 1978, "In Vivo Knee Stability. A Quantitative Assessment Using an Instrumented Clinical Testing Apparatus," *J. Bone Jt. Surg., Am. Vol.*, **60**(5), pp. 664–674.
- [30] Hodges, P. W., and Bui, B. H., 1996, "A Comparison of Computer-Based Methods for the Determination of Onset of Muscle Contraction Using Electromyography," *Electroencephalogr. Clin. Neurophysiol.*, **101**(6), pp. 511–519.
- [31] Howell, S. M., and Hull, M. L., 1998, "Aggressive Rehabilitation Using Hamstring Tendons: Graft Construct, Tibial Tunnel Placement, Fixation Properties,

- and Clinical Outcome," *Am. J. Knee Surg.*, **11**(2), pp. 120–127.
- [32] Howell, S. M., and Taylor, M. A., 1996, "Brace-Free Rehabilitation, With Early Return to Activity, for Knees Reconstructed With a Double-Looped Semitendinosus and Gracilis Graft," *J. Bone Jt. Surg., Am. Vol.*, **78**(6), pp. 814–825.
- [33] Smith, C. K., Hull, M. L., and Howell, S. M., 2008, "Can Markers Injected Into a Single-Loop Anterior Cruciate Ligament Graft Define the Axes of the Tibial and Femoral Tunnels? A Cadaveric Study Using Roentgen Stereophotogrammetric Analysis," *ASME J. Biomech. Eng.*, **130**(4), p. 044503.
- [34] Haut Donahue, T. L., Howell, S. M., Hull, M. L., and Gregersen, C., 2002, "A Biomechanical Evaluation of Anterior and Posterior Tibialis Tendons as Suitable Single-Loop Anterior Cruciate Ligament Grafts," *Arthroscopy: J. Relat. Surg.*, **18**(6), pp. 589–597.
- [35] Butler, D. L., Noyes, F. R., and Grood, E. S., 1980, "Ligamentous Restraints to Anterior-Posterior Drawer in the Human Knee. A Biomechanical Study," *J. Bone Jt. Surg., Am. Vol.*, **62**(2), pp. 259–270.
- [36] Markolf, K. L., Burchfield, D. M., Shapiro, M. M., Davis, B. R., Finerman, G. A., and Slauterbeck, J. L., 1996, "Biomechanical Consequences of Replacement of the Anterior Cruciate Ligament With a Patellar Ligament Allograft. Part I: Insertion of the Graft and Anterior-Posterior Testing," *J. Bone Jt. Surg., Am. Vol.*, **78**(11), pp. 1720–1727.
- [37] Zacharias, I., Howell, S. M., Hull, M. L., and Lawhorn, K. W., 2001, "In Vivo Calibration of a Femoral Fixation Device Transducer for Measuring Anterior Cruciate Ligament Graft Tension: A Study in an Ovine Model," *ASME J. Biomech. Eng.*, **123**(4), pp. 355–361.
- [38] Rodeo, S. A., Arnoczky, S. P., Torzilli, P. A., Hidaka, C., and Warren, R. F., 1993, "Tendon-Healing in a Bone Tunnel. A Biomechanical and Histological Study in the Dog," *J. Bone Jt. Surg., Am. Vol.*, **75**(12), pp. 1795–1803.
- [39] Zantop, T., Weimann, A., Wolle, K., Musahl, V., Langer, M., and Petersen, W., 2007, "Initial and 6 Weeks Postoperative Structural Properties of Soft Tissue Anterior Cruciate Ligament Reconstructions With Cross-Pin or Interference Screw Fixation: An In Vivo Study in Sheep," *Arthroscopy: J. Relat. Surg.*, **23**(1), pp. 14–20.
- [40] Howell, S. M., Gittins, M. E., Gottlieb, J. E., Traina, S. M., and Zoellner, T. M., 2001, "The Relationship Between the Angle of the Tibial Tunnel in the Coronal Plane and Loss of Flexion and Anterior Laxity After Anterior Cruciate Ligament Reconstruction," *Am. J. Sports Med.*, **29**(5), pp. 567–574.
- [41] Anderson, A. F., and Lipscomb, A. B., 1989, "Preoperative Instrumented Testing of Anterior and Posterior Knee Laxity," *Am. J. Sports Med.*, **17**(3), pp. 387–392.
- [42] Daniel, D. M., 1991, "Assessing the Limits of Knee Motion," *Am. J. Sports Med.*, **19**(2), pp. 139–147.
- [43] Edixhoven, P., Huiskes, R., de Graaf, R., van Rens, T. J., and Slooff, T. J., 1987, "Accuracy and Reproducibility of Instrumented Knee-Drawer Tests," *J. Orthop. Res.*, **5**(3), pp. 378–387.
- [44] Fleming, B. C., Beynon, B. D., and Johnson, R. J., 1993, "The Use of Knee Laxity Testers for the Determination of Anterior-Posterior Stability of the Knee: Pitfalls in Practice," *The Anterior Cruciate Ligament: Current and Future Concepts*, D. Jackson, ed., Raven, New York, pp. 239–250.
- [45] Eagar, P., Hull, M. L., and Howell, S. M., 2001, "A Method for Quantifying the Anterior Load-Displacement Behavior of the Human Knee in Both the Low and High Stiffness Regions," *J. Biomech.*, **34**(12), pp. 1655–1660.
- [46] Jackson, D. W., Grood, E. S., Goldstein, J. D., Rosen, M. A., Kurzweil, P. R., Cummings, J. F., and Simon, T. M., 1993, "A Comparison of Patellar Tendon Autograft and Allograft Used for Anterior Cruciate Ligament Reconstruction in the Goat Model," *Am. J. Sports Med.*, **21**(2), pp. 176–185.
- [47] Milano, G., Mulas, P. D., Ziranu, F., Piras, S., Manunta, A., and Fabbriani, C., 2006, "Comparison Between Different Femoral Fixation Devices for ACL Reconstruction With Doubled Hamstring Tendon Graft: A Biomechanical Analysis," *Arthroscopy: J. Relat. Surg.*, **22**(6), pp. 660–668.
- [48] To, J. T., Howell, S. M., and Hull, M. L., 1999, "Contributions of Femoral Fixation Methods to the Stiffness of Anterior Cruciate Ligament Replacements at Implantation," *Arthroscopy: J. Relat. Surg.*, **15**(4), pp. 379–387.
- [49] Grover, D. M., Howell, S. M., and Hull, M. L., 2005, "Early Tension Loss in an Anterior Cruciate Ligament Graft. A Cadaver Study of Four Tibial Fixation Devices," *J. Bone Jt. Surg., Am. Vol.*, **87**(2), pp. 381–390.
- [50] Karrholm, J., Selvik, G., Elmqvist, L. G., Hansson, L. I., and Jonsson, H., 1988, "Three-Dimensional Instability of the Anterior Cruciate Deficient Knee," *J. Bone Jt. Surg., Br. Vol.*, **70**(5), pp. 777–783.
- [51] Brucker, P. U., Lorenz, S., and Imhoff, A. B., 2006, "Aperture Fixation in Arthroscopic Anterior Cruciate Ligament Double-Bundle Reconstruction," *Arthroscopy: J. Relat. Surg.*, **22**(11), pp. 1250.e1–1250.e6.
- [52] Conner, C. S., Perez, B. A., Morris, R. P., Buckner, J. W., Buford, W. L., and Ivey, F. M., 2009, "Biomechanical Comparison of Three Femoral Fixation Devices for ACL Reconstruction," *Annual Meeting of the ORS, Las Vegas, NV*.
- [53] Nurmi, J. T., Sievanen, H., Kannus, P., Jarvinen, M., and Jarvinen, T. L. N., 2004, "Porcine Tibia is a Poor Substitute for Human Cadaver Tibia for Evaluating Interference Screw Fixation," *Am. J. Sports Med.*, **32**(3), pp. 765–771.