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A comparison of joint stability between anterior cruciate intact and deficient knees: a new canine model of anterior cruciate ligament disruption

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Abstract

Transection of the canine anterior cruciate ligament (ACL) is a well-established osteoarthritis (OA) model. This study evaluated a new method of canine ACL disruption as well as canine knee joint laxity and joint capsule (JC) contribution to joint stability at two time points (16 and 26 weeks) after ACL disruption ($n = 5/\text{time interval}$). Ten crossbreed hounds were evaluated with force plate gait analysis and radiographs at intervals up to 34 weeks after monopolar radiofrequency energy (MRFE) treatment of one randomly selected ACL. Each contralateral ACL was sham treated. The MRFE treated ACLs ruptured approximately eight weeks (mean 52.5 days, SEM ± 1.0 , range 48–56 days) after treatment. Gait analysis and radiographic changes were consistent with established canine ACL transection models of OA. Anterior-posterior (AP) translation and medial-lateral (ML) rotation were measured in each knee at 30°, 60°, and 90° of flexion with and then without JC with loads of 40 N in AP translation and 4 N m in ML rotation. A statistically significant interaction in AP translation included JC by cruciate ($P = 0.02$), and there was a trend for a cruciate by time ($P = 0.07$) interaction. Significant interactions in ML rotational testing included the presence of joint capsule ($P = 0.0001$) and angle by cruciate ($P = 0.0012$). This study describes a model in which canine ACLs predictably rupture approximately eight weeks after arthroscopic surgery and details the contribution of JC to canine knee stability in both ACL intact and deficient knees. The model presented here avoids the introduction of potential surgical variables at the time of ACL rupture and may contribute to studies of OA pathogenesis and inhibition. This model may also be useful for insight into the pathologic changes that occur in the knee as the ACL undergoes degeneration prior to rupture.

Keywords

Anterior cruciate ligament; Knee; Canine; Joint stability; Radiofrequency energy; Osteoarthritis; Mechanical testing

Introduction

The canine model of osteoarthritis (OA) following anterior cruciate ligament (ACL) transection is one of the best characterized and mostly commonly used [10]. The gross changes that occur in ACL deficient canine knees are well documented [1,6,9,10]. It was recently reported that adaptive changes in the menisci and adjacent posterior joint capsule developed in ACL deficient goat knees and were associated with a reduction in the initial abnormal anterior tibial translation after ACL transection [4]. Though not objectively evaluated, it has long been

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assumed that capsular thickening in an ACL deficient canine knee functions to stabilize the joint over time [3,7]. A gait evaluation study in dogs showed a progressive return of weight bearing on the unstable limb to approximately 70% of the stable limb 2.5 years after ACL transection, suggesting some degree of joint stabilization in ACL deficient canine knees [12]. To our knowledge, an objective assessment of joint stability in ACL deficient canine knees has not been performed.

The two potential methods of naturally occurring canine ACL disruption include progressive elongation and weakening with eventual complete disruption or sudden, traumatic rupture [5, 11]. Experimental models involve the acute transection of the ACL through a stab incision, arthrotomy, or arthroscopically [1,5,10]. We have developed a model by which the canine ACL is over treated with monopolar radiofrequency energy (MRFE) using an arthroscopic approach. The ligament then undergoes progressive deterioration until complete disruption at a predictable time point after treatment. We undertook this project to evaluate the contribution of joint capsule (JC) to joint stability after progressive ACL deterioration leading to complete disruption in the dog. We hypothesized that JC significantly contributes to joint stability following ACL rupture. Further, we predicted that progressive OA changes would occur in ACL deficient knees despite any potential stabilizing influences from surrounding tissues

Materials and methods

Experimental design

This study was performed in accordance with Institutional and National Institutes of Health regulations governing the treatment of vertebrate animals. It was initiated after approval by the University of Wisconsin-Madison Animal Care Committee.

Ten skeletally mature female crossbreed dogs, weighing 24–37 kg each (27 ± 1.3 kg) (mean $\pm$ SEM), were used in this study. MRFE was applied to the ACL of one randomly selected knee joint of each dog arthroscopically. An identical operation was performed on the contralateral joint with the exception that no energy was applied to the ligament. Treated ACLs ruptured approximately 53 days after surgery. Animals were sacrificed 16 weeks (n=5) or 26 weeks (n=5) (Beuthanasia-D Special, Schering-Plough Animal Health, Union, NJ, USA) after rupture of the treated ACL, and the hind limbs were stored at -70 °C until biomechanical testing. Comparisons were made between knees with ruptured ACLs with and without JC and control joints with and without JC. Assessment parameters included manual knee examination, radiographs, gait analysis, and instrumented anterior-posterior (AP) translation and medial-lateral (ML) rotation testing. Synovial fluid levels of the OA markers 3B3(-) (proteoglycan) and COL2-3/4C long (collagen type 11) were determined after each radiograph, and are reported elsewhere [2]. Open dissection confirmed that all treated ACLs were ruptured.

Surgical procedure

All animals were pre-medicated with a subcutaneous injection of 0.10 mg/kg acepromazine (Acepromazine maleate, Vedco, St. Joseph, MO USA) and 0.2 mg/kg butorphanol (Torbugesic, Fort Dodge Animal Health, Fort Dodge, IA, USA). After 20 min, 5 mg/kg thiopental (Pentothal, Abbott Laboratories, North Chicago, IL, USA) was administered intravenously for induction of anesthesia. The dogs were intubated and maintained on halothane in oxygen administered with a semi-closed circle system. Each animal received an intravenous preoperative injection of cephazolin (20 mg/kg) (Cephazolin, Faulding Pharmaceutical Co., Elizabeth, NJ, USA) for prophylaxis. Each knee was physically examined for drawer, range of motion, swelling, temperature, crepitus, patellar tracking, and varus-valgus. A standard surgical scrub with chlorhexidine followed by a wash with sterile water was performed.

The knee to receive treatment was distended with 7 ml of sterile saline. A 0.5 cm incision was made through skin and J C on the lateral aspect of the knee 0.5 cm proximal to the tibia and just cranial to the lateral digital extensor tendon. A 2.7 mm blunt trocar in a 3 mm cannula was used to enter the joint through the incision. The trocar was replaced by a 2.7 mm, 30° arthroscope (Storz, Goleta, CA). Joint distension was maintained with sterile saline using gravity flow. A 4.0 mm synovial shaver (APEX, Linvatec, Largo, FL) was placed in the joint through a 0.5 cm incision in the medial aspect of the joint, just proximal to the tibial plateau and just medial to the patellar tendon. Synovium and infrapatellar fat pad were removed until the origin and insertion of the ACL could be clearly seen. A 2 mm diameter MRFE probe (Tac-C probe, Smith & Nephew, Inc., Andover, MA) was used to treat the entire anterior surface of the ACL in transverse passes across the ligament at a rate of 1–2 m d s at temperature and power settings of 70 °C and 25 W, respectively, using a MRFE generator (Electrothermal System ORA-50™, Smith & Nephew, Inc., Andover, MA). The joint was lavaged for 1 min with sterile saline, and incisions were closed routinely. An identical operation was performed on the contralateral joint except that no energy was applied to the ACL.

Gait analysis

Force plate gait analysis was performed pre-operatively and at 4, 8, 12, 16, and 24 weeks after surgery for all dogs, and at 34 weeks after surgery for five dogs using a force plate (OR6-6-1000 Biomechanics Platform with SGA6-4 Signal Conditioner/Amplifier, Advanced Medical Technology, Inc., Newton, MA) connected to a commercially available satellite data acquisition system (VETDATA v2.03, Acquire v5.0, Mininet v4.0 and Update v1.1 from Sharon Software Inc., Dewitt, MI). A single handler trotted dogs for all gait trials. A trial was considered successful if a forepaw contacted the force plate followed by contact of the ipsilateral hind paw at a velocity of 1.80–2.80 m/s and acceleration of 0.9 to –0.9 m/s². The velocity range was designed to include a comfortable trot rate of each dog included in the study. Three successful passes for each limb were recorded at each time point. Peak vertical impulse (PVI) values, defined as total force applied over time, were normalized to body weight for comparison across time.

Radiographic evaluation

Radiographs (posteroanterior and lateromedial) were taken pre-operatively and at 4, 8, 12, 16, and 24 weeks after surgery for all dogs, and at 34 weeks after surgery for five dogs. All radiographs were performed with the knees in full extension (approximately 30° of flexion). Radiographs were evaluated for signs of OA including the presence of osteophytes, enthesiophytes, subchondral bone sclerosis, and, following ACL rupture, anterior displacement of the tibia. All radiographs were performed with the animals sedated (acepromazine, 0.10 mg/kg; butorphanol, 0.2 mg/kg; both administered subcutaneously).

Mechanical testing

A P translation and ML rotation were performed with the knee at 30°, 60°, and 90° of flexion. Each A P translation test was followed by a ML rotation test before the joint angle was changed. All testing was first done with an intact JC and then with J C dissected away. JC was always completely detached from all femoral and tibial insertions, though small pieces were left attached to periarticular structures as necessary to ensure that the structures were not disrupted. The quadriceps muscle was transected 6 cm proximal to the knee for all testing, and the quadriceps, patella, and associated tendons were reflected in the anterior direction when JC was removed.

The tibia and femur were cut to a length of 10 cm from the joint line and were potted with polyester resin in thick-walled aluminum cylinders for attachment to mechanical testing system (MTS 858, Minneapolis, MN). Musculature was removed 6 cm above and below the joint line.

The specimen was then mounted in the testing system with the tibial cylinder rigidly fixed to a base attached to the force-moment sensor and the femoral cylinder rigidly fixed to the actuator in custom made positioning fixtures (Fig. 1). The positioning fixtures permitted placement of the knee in three different flexion angles, 30°, 60°, and 90°. It accommodated six degrees of freedom for positional adjustment. The degrees of freedom were referenced to the anatomic planes of motion: AP, ML, and superior-inferior displacement, flexion-extension, varus-valgus, and internal-external rotation. During adjustment of knee flexion angle, internal-external rotation of the tibia was unlocked and free to rotate. At the desired flexion angle, the tibia was rotated internally and externally until the midpoint of rotation was established, and the tibia was locked into position. Internal-external rotation of the tibia relative to the femur was locked while A P loads were applied across the tibiofemoral joint to yield reproducible load-displacement data.

A P translation was evaluated by applying four complete AP load cycles to each specimen. The first two load cycles were used to pre-condition the specimen and to demonstrate reproducible load-displacement of the tibia relative to the femur that occurred between the limits of the 20 N anterior and 20 N posterior loads. The joints were then tested non-destructively through two cycles under load control (ramped over 12 s per cycle). AP knee translation was defined as the AP displacement of the tibia relative to the femur that occurred between the limits of the 40 N anterior and 40 N posterior loads.

Following each AP translation test, a ML rotation test was performed before the angle of the joint was changed for the next test. For this study, ML rotation refers to internal (medial) rotation of the femur relative to the tibia followed by lateral (external) rotation of the femur relative to the tibia. The joints were tested non-destructively through four cycles as described previously. For pre-conditioning, two cycles of 2 N m were applied to the femur first in the medial direction followed by the lateral direction. For testing, a torque of 4 N m was applied to the femur medial to lateral in two cycles (ramped over 12 s per cycle). ML rotation was defined as the ML rotation of the tibia relative to the femur that occurred between the limits of the 4 N m medial and lateral loads.

Statistical analysis

Comparisons in AP translation and ML rotation were made among knees with ACL rupture with and without JC and control joints with and without JC. The SAS least squares mean and SEM was determined for each property of interest. A factorial model with all interactions was used to compare time, capsule status, ligament status, and angle. Because all treatments except time were within-animal treatments, a split plot model for incomplete data was used. Standard errors for the means were based on the root means squared error from the ANOVA. The mean and SEM for PVI, velocity, and acceleration were determined for each treatment group at all time points. ANOVA was used to determine differences in velocity or acceleration among trials (treated and sham treated ACLs at each time interval) and among time intervals for PVI of each treatment group. Tukey's post-hoc analysis was used to evaluate significant differences among time intervals. Student's t-tests were used to determine differences in PVI among treatment groups at each time interval. Significance for all analyses was set at $P < 0.05$. Statistical analysis was performed with commercially available software programs (SAS Release 6.12, SAS Institute, Cary, NC, USA or GraphPad Prism v3.0, GraphPad Software, Inc., San Diego, CA, USA).

Results

ACL rupture

Rupture of treated ACLs was detected by sudden onset of non-weight bearing lameness, joint effusion, and a positive drawer sign. The exact dates of rupture were not observed in two dogs. The values from these animals were not used to calculate the mean rupture date. Treated ACLs ruptured approximately 53 days after treatment (mean 52.5 days, SEM ± 1.0 , range 48–56 days).

Gait analysis

The velocity and acceleration of all gait trials were 2.2 ± 0.03 m/s and -0.6 ± 0.07 m/s (mean $\pm$ SEM), respectively. There were no significant differences in velocity or acceleration among trials over time or among trials for limbs with treated or sham treated ACLs.

PVI values were significantly lower on limbs with treated ACLs than limbs with sham treated ACLs beginning 12 weeks after treatment (Fig. 2). The differences in values between limbs with treated and sham treated ACLs decreased over the period of the study, though they were still significant 34 weeks after treatment. Mean PVI on the treated ACL limb improved from a minimum of 36% of that on the leg with the sham treated ACL 12 weeks after treatment to a maximum of 67% of that on the limb with the sham treated ACL 34 weeks after treatment.

PVI values on limbs with sham treated ACLs at 12 weeks post-operatively were significantly higher than those pre-operatively, four and eight weeks post-operatively. Values from 16 weeks post-operatively were significantly higher than those pre-operatively and four weeks post-operatively. Limbs with treated ACLs had significantly lower PVI values from pre-operative values at 12, 16, 24, and 34 weeks post-operatively; from four week post-operative values at 12, 16, and 24 weeks post-operatively; and from eight week post-operative values at 12 and 16 weeks post-operatively.

Radiographic evaluation

All pre-operative and four week post-operative radiographs were within normal limits. Radiographs of sham treated ACL limbs remained normal for the length of the study. Five of the radiographs performed eight weeks post-operatively on knees with treated ACLs demonstrated significant joint effusion and anterior displacement of the tibia on the lateral view. There was anterior displacement of the tibia in all lateral 12 week post-operative radiographs. Significant joint effusion, which was present for the remainder of the study, was apparent in all joints, and seven joints with treated ACLs had very small osteophytes on the proximal trochlear ridges. Radiographs performed on limbs with treated ACLs 16 weeks post-operatively showed varying degrees of osteoarthritic changes on the tibial intercondylar eminences and femoral trochlear ridges. By 24 weeks after treatment, significant radiographic changes consisted of osteophytosis of the tibial articular surface and the trochlear ridges of the femur and slight subchondral bone sclerosis in the treated ACL limbs. Thirty-four weeks after treatment, the osteophytes and subchondral bone sclerosis were more pronounced. The severity of radiographic changes in the 16, 24 and 34 post-operative radiographs varied between animals, but all joints with treated ACLs had the described changes.

Anterior-posterior translation testing

ANOVA showed a significant interaction to include JC by cruciate ($P = 0.02$) and a trend for a cruciate by time interaction ($P = 0.07$) (Figs. 3 and 4). Joints with no ACL and the joint capsule removed had significantly greater AP motion (6.6 ± 0.42 mm) (mean $\pm$ SEM) than those with no ACL and intact JC (4.7 ± 0.42 mm) and joints with intact ACLs with (2.4 ± 0.42 mm) or without (2.7 ± 0.42 mm) JC (Fig. 3). Joints with no ACL but with intact JC had significantly greater motion than those with intact ACLs with or without JC. There was no significant

difference in AP translation 24 or 34 weeks post-treatment (16 or 26 weeks post-rupture) among joints with no ACLs (24 weeks, 5.3 ± 0.49 mm; 34 weeks, 6.0 ± 0.49 mm) or among those with intact ACLs (24 weeks, 2.8 ± 0.49 mm; 34 weeks, 2.3 ± 0.49 mm). Joints without ACLs had greater motion at both time points than those with intact ACLs. The difference in the amount of motion between those joints with and without ACLs was greater at 34 weeks than at 24 weeks post-operatively.

Medial-lateral rotation testing

Significant interactions included the presence of JC ($P=0.0001$) and angle by cruciate ($P=0.0012$). There were significant differences in ML rotation between joints with intact JC ($17^\circ \pm 1.6^\circ$) and those without JC ($22^\circ \pm 1.6^\circ$) (Fig. 5). Also significant to the amount of ML rotation was the angle of flexion and presence or absence of the ACL (Fig. 6). The amount of rotation at 30° with ACL ($14^\circ \pm 1.9^\circ$), 30° without ACL ($15^\circ \pm 2.0^\circ$) and 90° with ACL ($15^\circ \pm 1.9^\circ$) were not significantly different from one another. All three were significantly less than 60° with ACL ($20^\circ \pm 2.0^\circ$), 60° without ACL ($30^\circ \pm 2.0^\circ$), and 90° without ACL ($25^\circ \pm 1.9^\circ$), which were all significantly different from one another. The 60° without ACL testing protocol produced the greatest rotation followed by 90° without ACL, and then 60° with ACL.

Discussion

Transection of the canine ACL resulting in significant, progressive OA is one of the most popular OA models [1,6,10]. Over treatment of the canine ACL with MRFE using an arthroscope is a highly predictable and reproducible model of progressive joint instability and OA. An arthroscopic approach for transection of the canine ACL has been previously described and promoted as avoiding disadvantages of an open arthrotomy including soft tissue dissection and injury to periarticular structures [10]. The model described here has the added advantage of about eight weeks between surgery and ACL disruption. The time period permits incisions to heal and synovial fluid to return to a steady state. This model mimics the clinical condition in which a normal joint undergoes progressive osteoarthritic changes following ACL degeneration and subsequent rupture, and avoids any variables introduced by surgery at the time the knee becomes ACL deficient.

Radiographic changes in the ACL deficient knees in this study were consistent with previous studies evaluating OA changes following canine ACL disruption. Radiographic evidence of OA became apparent approximately four weeks after rupture (12 weeks after treatment). The timing of radiographically evident OA changes is consistent with previous models and further supports the consistency and totality of this model [13].

There were no significant effects of surgery on PVI values in this study. There was a non-significant difference apparent in mean PVI values between limbs with treated and sham treated ACLs eight weeks after treatment, likely since not all treated ACLs had ruptured by the eight week gait trial. Mean PVI values on limbs with treated and sham treated ACLs were significantly different beginning about 12 weeks after treatment. A significant decrease occurred on limbs with treated ACLs with a compensatory significant increase on limbs with sham treated ACLs. Mean PVI on limbs with treated ACLs returned to 76% of limbs with sham treated ACLs six months after rupture. The onset and partial resolution of lameness associated with ACL rupture is consistent with previous gait evaluation studies of dogs with transected ACLs [12].

Following disruption of the ACL, the canine knee undergoes changes which confer stability to the joint [1,3,7,9]. Tissue changes that might contribute to joint stability occur in JC, articular cartilage, extra-articular connective tissues, medial collateral ligament, and the menisci [3,7,9]. These changes have been noted in the dog and have been established in the cat and goat,

but the specific contribution of JC to knee joint stability in ACL deficient canine knees has not been determined, to our knowledge [4,8]. In this study, the presence of JC in ACL deficient knees significantly affected the amount of AP translation in the joint, though this was not the case in knees with intact ACLs. Regardless of the presence of JC, however, the ACL deficient knees had significantly greater motion than those with intact ACLs. In this investigation, JC prevented approximately 1.9 mm or 29% of the motion in ACL deficient canine knees. This percentage should be viewed as an estimate since the patellar apparatus (quadriceps, patella, and patellar tendon) may have conferred some stability to the joint and was reflected when JC was removed. It can be assumed, however, that any stability conferred by the patellar apparatus was negligible during testing since the origins of the quadriceps were disrupted prior to testing.

There was no apparent increase in canine ACL deficient knee AP stability in the 10 week period between the two time points evaluated in this study (16 and 26 weeks after rupture). Statistically, there was a significant increase in the differences between mean values from ACL intact and deficient knees at the two different time points. An increase in mean PVI on ACL deficient knees and a decrease on ACL intact knees, though not statistically significant between time points, seems to indicate greater joint stability at the later time point. There are several potential reasons for this apparent contradiction between mechanical testing and gait evaluation data. The time period between mechanical evaluations was, perhaps, not long enough to detect a change in joint stability using this model. The testing loads may not have been large enough to reveal slight changes in AP motion, though they were within the range reported for evaluation of canine knee AP motion and were selected to avoid potential tissue damage at more extreme loads [2,14].

It is apparent from the results of this study that the JC contributes significantly to rotational joint stability in both ACL intact and deficient canine knees, with all flexion angles considered together. There were no differences in ML motion between time points evaluated in this study, likely due to the factors discussed previously. The fact that the presence or absence of the ACL did not significantly affect rotation with all angles considered together, while the presence of JC apparently did can be explained by the mechanical contributions of both structures to joint stability. The ACL is known to contribute to prevention of medial rotation of the tibia relative to the femur in a normal standing knee angle in the dog (approximately 60° of flexion) [11]. This function is a result of the relationship of the posterior and anterior cruciates, that is, they twist around each other as the joint is flexed from full extension, preventing internal joint rotation. As the joint is extended, this function is lost due to the mechanical rearrangement, “untwisting”, of the two structures [11]. It can similarly be assumed that when the joint is placed in extreme flexion, 90°, that restraint against internal joint rotation is compromised. The JC is taut and lax in different areas depending on the relative relationship of the femur and tibia, but the restraint function is not as dependent upon the relationship of two structures. Hence, when considering all angles together, there was no apparent contribution of the ACL for inhibition of ML rotation, while the JC was significant at all angles and in the presence or absence of the ACL.

In this study, the greatest ML rotation occurred at 60° of flexion, the normal standing knee joint angle of the dog, when rotation was compared among flexion angles [11]. This is consistent with the fact that soft tissue impingement and mechanical restraints should be minimal at a standing joint angle, while the function of the cruciates should be ideal as discussed previously. At full extension (30° flexion) there appeared to be considerable inhibition of rotational joint motion, making it impossible to distinguish significant differences in motion between ACL intact and deficient knees. At a flexion angle of 90°, full flexion, ML rotation was significantly decreased from 60° of flexion, though differences between ACL intact and deficient knees were statistically distinguishable. These findings support the function of the JC and other secondary constraints in canine knee joint stability. Based on our findings, it

appears that 60° of flexion is the best angle at which to evaluate rotational stability of the canine knee.

This study presents a new approach to an established OA model. The degeneration and rupture of MRFE treated canine ACLs was highly predictable and reproducible. This study confirmed the stabilizing influence of JC to ACL deficient knees in A P motion. Studies evaluating methods to enhance joint stability following ACL disruption should be especially stringent in documenting the stability conferred by the stabilization technique versus that conferred by the JC. The importance of JC for ML rotational stability of the canine knee in both ACL intact and deficient knees was documented in this study, and 60° of flexion appears to be the best angle to evaluate rotational stability. This model may be useful to evaluate treatments to inhibit OA progression without any surgical variables associated with ACL disruption. In general, an ACL disruption model without potential surgical variables and knowledge of JC contribution to joint stability in ACL deficient canine knees will contribute to the study of OA pathogenesis and inhibition. This model may also be useful for insight into the pathologic changes that occur in the knee as the ACL undergoes degeneration prior to rupture.

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Fig 1.

The knee-testing fixture allowed six degrees of freedom to position the joint. The rotational axes of the fixture were aligned with the rotational axes of the canine knee. Load (AP or ML) was applied to the femur through the Materials Test System while the tibia was held in the horizontal plane. All rotations were locked while loads were applied and then unlocked for adjustment of knee flexion angle. Joints were tested in all three flexion angles (AP displacement followed by ML rotation) with and then without JC.

Fig 2.

PVI (%I body weight x sec) (mean $\pm$ SEM) on limbs with treated and sham treated ACLs up to 34 weeks after treatment. PVI values were significantly lower on limbs with treated ACLs than on limbs with sham treated ACLs beginning at 12 weeks after treatment and continued for the remainder of the study. The mean rupture date is indicated by a dotted line. * = significant difference between limbs with treated ACLs and those with sham treated ACLs ($P < 0.05$).

Fig 3.
Anterior-posterior translation (mean $\pm$ SEM) of canine knees with and without ACL or JC. Columns with different letters are significantly different from one another ($P < 0.05$).

Fig 4.

Anterior-posterior translation of canine knees (mean $\pm$ SEM) without and without ACLs 24 and 34 weeks post-treatment (16 and 26 weeks post-rupture). Columns with different letters are significantly different from one another ($P < 0.05$).

Fig 5. Medial to lateral rotation (mean $\pm$ SEM) of canine knees with and without JC. Columns with different letters are significantly different from one another ($P < 0.05$).

Fig 6. Medial to lateral rotation of canine knees (mean $\pm$ SEM) at different flexion angles with and without ACLs. Columns with different letters are Significantly different from one another ($P < 0.05$).