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Effect of 9 mm Tibial Tuberosity Advancement on Cranial Tibial Translation in the Canine Cranial Cruciate Ligament-Deficient Stifle

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Objective—To assess the effect of 9 mm tibial tuberosity advancement (TTA) on cranial tibial translation (CTT) in a cranial cruciate ligament (CCL)-deficient canine stifle model.
Study Design—In vitro cadaveric study.
Animals—Canine pelvic limbs (n = 12).
Methods—Each stifle was placed in a jig at 135° with a simulated quadriceps force and tibial axial force. CTT distance was measured with the CCL intact (iCCL), transected (tCCL), and after performing TTA using a 9 mm cage.
Results—Mean CTT for iCCL was 0.42 mm, 1.58 mm after severing the CCL, and 1.06 mm post-TTA. The tCCL CTT measured without any quadriceps force was 2.59 mm. Differences between the intact and tCCL ($P < .0001$) and tCCL and TTA ($P = .0003$) were significant. The difference between the tCCL with and without the quadriceps force was not significant ($P = .0597$).
Conclusions—These data confirm that TTA does reduce CTT in tCCL stifles in this model. The CTT noted was less than that noted clinically. The addition of a simulated quadriceps force to a CCL-deficient stifle before a TTA, by itself, may not significantly lessen CTT.
Clinical Relevance—Whereas this in vitro model demonstrated that TTA reduced CTT in canine stifles with CCL transected, the model limitations preclude extrapolation to the effect of TTA in a live dog.

INTRODUCTION

RUPTURE OF the cranial cruciate ligament (CCL) is the most frequently diagnosed orthopedic disease in dogs.[1–3] CCL rupture results in femorotibial instability, specifically cranial tibial translation (CTT) and internal rotation during weight bearing, and predisposes to medial meniscal injury.[4,5] Lameness and progressive osteoarthritis are seen clinically in CCL-deficient dogs.[6] Many different surgical techniques have been proposed to stabilize the stifle after CCL rupture.[7]

The tibial tuberosity advancement technique (TTA) was designed to provide stability to the CCL-deficient stifle.[8,9] The TTA, theoretically, limits CTT by increasing the lever arm of the quadriceps muscle to resist CTT. In theory, the total joint force (combination of the ground reaction force and all muscles acting to counteract it) acts in a plane parallel to the patellar ligament. Forces acting in the plane of the neutral axis, which would preclude tibial motion in the cranial to caudal plane, are perpendicular to the tibial plateau (Fig 1). Part of the theory behind TTA is that all forces acting around the stifle can be simplified into the total joint force and the counteracting force of the quadriceps muscle. These forces can be reduced into a proximodistal vector and a craniocaudal vector. The proximodistal component force of the quadriceps muscle neutralizes the total joint force component; however, there is a residual cranial component that is not neutralized (Fig 1). If the patellar ligament is aligned parallel to the neutral axis, the total joint force will occur

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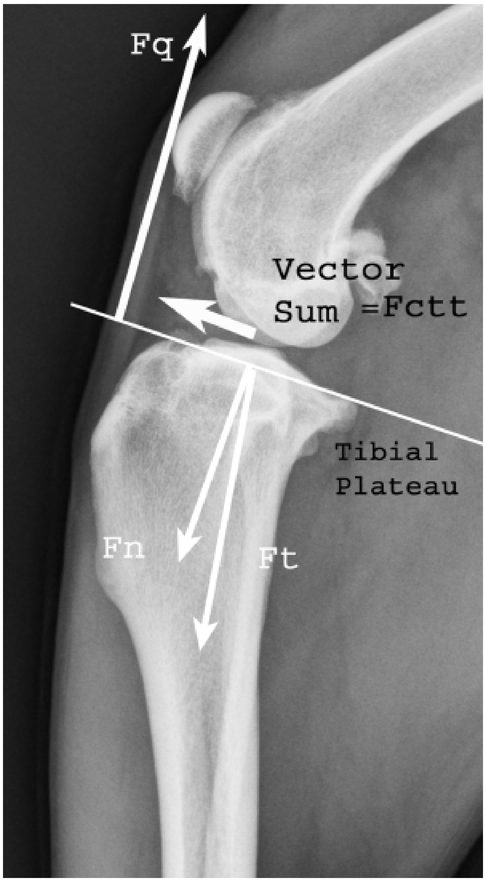

Fig 1. Radiograph of a diseased stifle demonstrating the forces acting on a cranial cruciate ligament (CCL)-deficient stifle. F_t, total force acting on the stifle; F_q, quadriceps force counteracting F_t; TPA, line of angle of tibial plateau; F_n, force in the neutral plane; vector sum, the sum of all the forces revealing a force creating cranial tibial translation (CTT) (F_{ctt}), because F_q does not balance F_t in the craniocaudal vector.

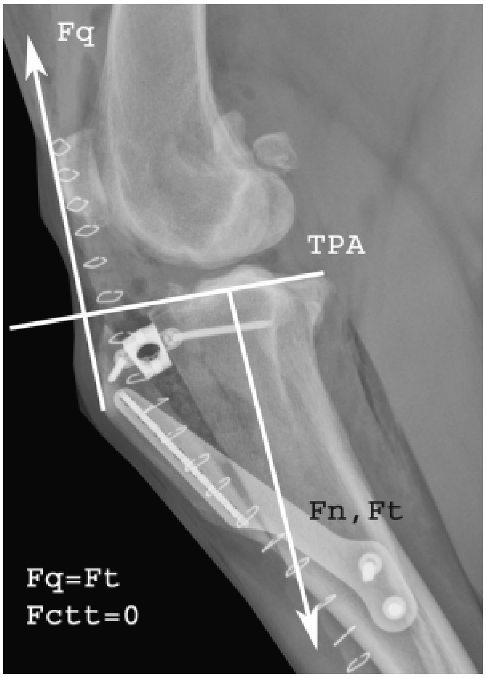

Fig 2. Radiograph of clinical tibial tuberosity advancement (TTA) demonstrating the forces acting on the stifle after surgery. Note now F_t and F_q act in the same plane thereby eliminating the cranial translation force vector. F_q, quadriceps force counteracting F_t; TPA, line of angle of tibial plateau; F_t, total force acting on the stifle; F_n, neutral plane of force.

in the same plane as the neutral force, thereby eliminating any unbalanced cranial vector, resulting in no CTT (Fig 2). The distance of advancement is therefore chosen to align the patellar ligament perpendicular to the tibial plateau. With the distal patellar ligament, i.e. the tibial tuberosity, in its new position the sum of forces acting on the stifle is directly countered by the quadriceps muscle, thereby eliminating CTT.[8,9] To our knowledge, no peer-reviewed biomechanical evidence has been reported to support this concept. An alternate theory for the method of action is shifting the cranial translation into a caudal translation, as in the tibial plateau leveling osteotomy (TPLO).[10,11] Another interpretation is that at 135° extension, the TTA will shift the positioning of the total forces acting on the stifle into a more neutral site by changing the geometry of the stifle, thereby minimizing CTT.[12]

Our purpose in this in vitro study was to assess the ability of TTA to reduce CTT in cadaveric canine stifles. Our null hypothesis was that the CTT in the CCL deficient stifle would be no different to CTT in the TTA stifle under simulated ground reaction and quadriceps muscle contraction forces. A second hypothesis was that in the CCL-deficient stifle before performing the TTA, a simulated quadriceps force would control CTT to a greater degree than without a quadriceps force.

MATERIALS AND METHODS

Specimen Collection and Preparation

Hind limbs of 6 large breed dogs (26–31 kg) euthanatized for reasons unrelated to this study were harvested at the coxofemoral joint, wrapped in moist saline (0.9% NaCl) solution-soaked towels, and stored in sealed plastic at −70°C until testing. Limbs were thawed overnight in a 5°C water bath before testing. Skin and soft tissues were removed sparing the patella, patella tendon, and collateral ligaments, the joint capsule and contents of the stifle joint. Throughout preparation and testing, the joint was kept moist with saline-soaked towels. The femur and tibia/fibula were cut transversely 13 cm from their respective stifle articular surfaces. The femur and tibia were individually potted to a depth of 6 cm using a polyester resin (Bondo, Bondo Corporation, Atlanta, GA). A plastic straw was used to form an air-filled tube through the cranial aspect of the femoral potting material to allow for the placement of a simulated quadriceps mechanism. Digital radiographs of each specimen were made to document skeletal maturity and absence of degenerative stifle disease. From these, tibial plateau angle (TPA) measurements were made using a previously described method.[13]

Testing Device

Each limb was placed in a custom built jig to secure the stifle at 135° (Fig 3). The potting material was secured by set screws within the cylindrical receptacles for the femur and tibia. The position of the potting material was marked, so that the limb could returned to the same position between testing scenarios. A 2.4 mm Kirschner wire was placed transversely through a predrilled hole in the center of the patella. Braided wire, with loops formed by crimp tubes, was placed on the medial and lateral sides of this pin. The braided wire was threaded through the static femoral pot, and over a pulley to a platform on which weights were placed to simulate a quadriceps contraction force of 10% body weight (bw). The tibial pot was attached to a constrained mobile portion of the jig, which allowed for proximodistal, craniocaudal, and internal and external rotational motion of the distal limb. Weights attached to a lever arm were placed to load the tibia in an axial direction to simulate ground reaction forces. The length of the lever arm in this model effectively doubled the force transferred to the distal tibia, i.e. if 20% bw was required, then 10% bw was added to the end of the lever arm.

Specimen Testing

A dial indicator (Mitutoyo No. 2416, Japan), with a sensitivity of 0.0025 mm, was used to measure the distance the tibial tuberosity translated cranially during load application. The dial indicator was positioned at the tibial tuberosity and routinely readjusted between testing scenarios to maintain the position. A mass of 20% bw was directed axially up the distal tibia, while a mass of 10% bw was placed on the patella, through the pulley system, pulling it proximally. During pilot studies using 20% bw on the tibia and 10% bw on the patella allowed CTT to occur in unstabilized CCL-deficient specimens.

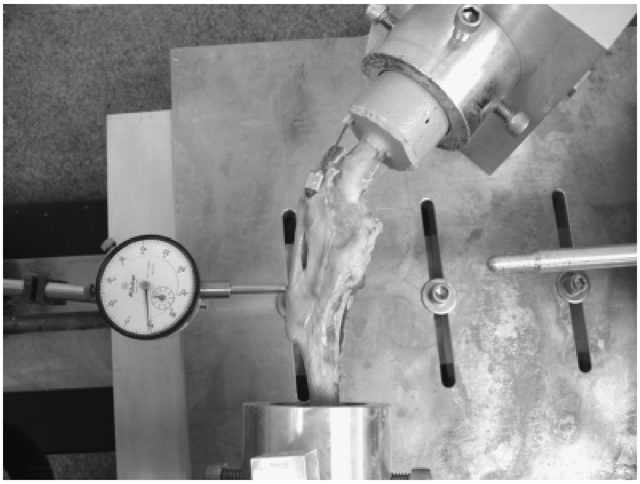

Fig 3. Stifle secured to testing jig. Note the wire starting at the patella and extending proximally through a hole drilled in the potting material at the top and the dial indicator on the left.

CTT distance was measured 5 separate times with axial and quadriceps loads applied to a stifle with an intact CCL (iCCL), after a 2 cm medial joint capsule incision. The CCL was then sharply transected (tCCL) and CTT distances were again measured with the same patella and tibial loads applied. Additionally, to document the effect of the quadriceps muscle, CTT was measured without the quadriceps load in the tCCL stifles. A TTA procedure was then performed by making a medial to lateral osteotomy of the tibial crest just cranial to the cranial prominence of the extensor groove proximally to the distal extent of the tibial crest. A TTA fork and plate were attached to the tuberosity and a 9 mm cage was secured on either side of the proximal extent of the osteotomy with two 2.4 mm screws. The plate was attached to the tibial shaft with a 2.7 mm screw (Fig 4). CTT was measured as above.

Each stifle was explored fully once the experiment was completed to ensure the presence of a completely transected

Fig 4. Post tibial tuberosity advancement (TTA) experimental specimen with a 9 mm cage.

CCL, an intact caudal cruciate ligament, and intact menisci. The angle between the bone (medial aspect of the tibia and cranial aspect of the femur) and the base of the potting material was measured in each specimen with a goniometer.

Statistical Analysis

CTT means in the iCCL, tCCL, and TTA specimens were compared using 2-way ANOVA and post hoc Tukey's test using statistical software (SAS version 9.1.3, Cary, NC). Associations between dial indicator CTT measurements and potting material angles were assessed using bivariate scatter plots followed by nonparametric Spearman's correlation coefficients. Significance was set at $P<.05$

RESULTS

Mean CTT for 12 iCCL stifle joints, with 20% bw loaded axially to the tibia and 10% bw patella load representing the quadriceps mechanism, was 0.42 ± 0.21 mm in our model (Fig 5). Mean CTT for tCCL stifles with the patella load was 1.58 mm (range, 0.55–7.35 mm). With no patella load CTT was 2.59 ± 2.07 mm (Fig 6). After the 9 mm TTA was performed, mean CTT was 1.06 ± 0.55 mm. The difference between mean iCCL and tCCL was significant ($P<.0001$) as was the difference between the tCCL and the TTA ($P=.0003$). No significant difference was found between tCCL with and without a quadriceps load ($P=.0597$). Although, TTA reduced CTT, there was a significant difference between the TTA and the iCCL, showing that the TTA did not eliminate CTT entirely ($P=.0428$).

Each stifle was explored after testing, and all caudal cruciate ligaments, menisci, and collateral ligaments were intact. The mean angle of the femur to the potting material was 89° ± 2°, whereas the angle of the tibia in the pot was 91° ± 3°. Preoperative mean TPA was 19° ± 3°. No association between these variables and CTT was noted in the analysis (all P-values were $>.05$).

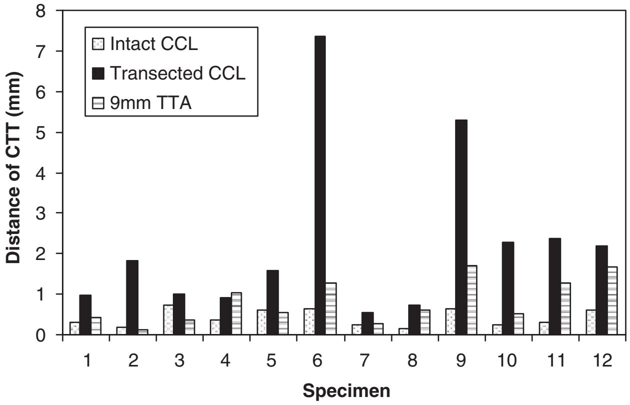

Fig 5. Direct measurement of the distance in mm of cranial tibial translation (CTT) (y-axis) was recorded for the intact CCL, transected CCL with 10% body weight applied to the patella, and the TTA with 9 mm advancement.

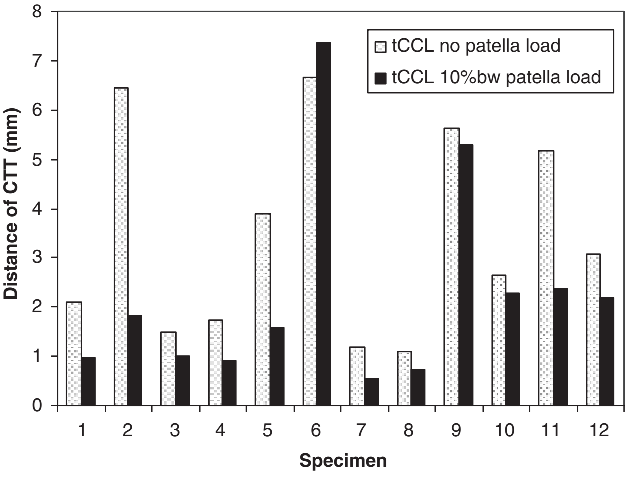

Fig 6. Direct measurement using the dial indicator of the distance in mm (y-axis) was recorded for the transected CCL with and without 10% body weight (bw) applied to the patella.

DISCUSSION

CTT distance, as measured by the dial indicator, was significantly reduced by advancement of the tibial tuberosity 9 mm in this experimental model when TTA was compared with tCCL; however, TTA did not entirely eliminate CTT. Thus, there was sufficient statistical support to reject the null hypothesis, and this is evidence that the TTA procedure reduces CTT in CCL-deficient canine stifles in this in vitro model. In each specimen it was noted, with additional tibial axial load beyond 20% bw, the tibia was translated further demonstrating the CTT measured here was not beyond the physiologic limits of the system. Interspecimen variation was noted in the model. The individual stifle TPA and the angle of the bone to the potting material did not seem to relate to the variation seen, though because of the small number of specimens tested, a type II error could account for this. The jig allowed for tibia motion in 3 dimensions: proximodistal, craniocaudal, and rotational. Craniocaudal movement was measured directly with the dial indicator. Proximodistal motion was limited by the stifle joint capsule, bony contact, and the stifle ligaments. Rotational differences between specimens was noted during testing, but was not measured. The variation in CTT results may have been affected by the rotation of the tibia and the resulting effect of this on the remaining stifle ligaments to constrain CTT.

The quadriceps muscle force is integral to hindlimb weight bearing, providing not only extension of the stifle, but also aiding in stifle joint stability.[14] The TTA theory proposes that active contraction of the quadriceps muscle is necessary to achieve CTT control.[15,16] The actual force exerted by the quadriceps muscle is unknown in dogs, but logically varies with the bw, phase of weightbearing, and speed of locomotion. Quadriceps force can be estimated experimentally[17–19] by measuring muscle torque, physiologic cross-sectional area, or morphometric analysis, and a wide variety of estimations were proposed. The use of a quadriceps force of 10% bw was chosen in this model after pilot studies using this jig revealed that forces greater than this would not allow CTT without large axial loads in the tCCL specimens. For example, using 1/3 of the bw for the quadriceps force, the model required 123 N to elicit any CTT in a tCCL specimen. There was no significant difference in CTT noted between the tCCL stifles with and without the 10% bw quadriceps force. This would suggest that the small amount of force we used does not, by itself, contribute to controlling CTT.

The stifles we used were tested after being thawed from -70°C. Ideally, specimens would be tested immediately after euthanasia, but this is often not feasible. Storage temperatures ranging from 4°C to -80°C have been shown to have minimal negative biomechanical effects on ligaments, but no ideal storage temperature has been identifed.[20–22] It is possible that the freezing of the hind limbs used in this experiment produced some minimal changes that could have affected the biomechanical function of the ligaments and therefore the results.

Study limitations included an inability of this model to mimic the 10 mm amount of CTT noted in live dog experimental studies.[4,23] The magnitude of CTT elicited in the first part of this study was lower than some other in vitro studies testing CTT (4.7–19 mm).[11,24–26] This may be because of differences in the testing jig, the angle of the stifle, and the magnitude of tibial axial loads and quadriceps loads used.

Variability between individual legs in CTT was noted. No association between TPA or perpendicularity of the bone in the potted material could be found. Rotation of the tibia may have played a role in the measurements obtained for CTT, but because recording of the degree of this motion was not performed, its significance in unknown.

Removal of all structures superficial to the joint capsule and collateral ligaments was performed to reduce sources of variation in the data obtained. An alternate method of testing could use the entire hind limb with the foot and calcaneal mechanism intact, which may reflect in vivo anatomy better, but also, may introduce more uncontrolled variables. TTA theory suggests that forces beyond the quadriceps can be fully accounted for by using the total force acting around the stifle, which is how we tested in this experimental model. None of the cadavers we used had evidence of stifle pathology on radiographs or on gross examination. Therefore, this model may not correlate to a dog with cruciate disease, because of the associated stifle osteoarthritis and the ensuing ligamentous, cartilaginous and joint capsular changes. This in vitro model used a cadaver stifle at only 135° with only the quadriceps muscle force simulated. More complex models could be developed to include the hamstring or gastrocnemius muscles and investigate CTT at multiple joint angles. This may provide further evidence of the mechanism of action of CCL rupture and TTA.

In conclusion, the ability of TTA to limit CTT during simulated weightbearing in this in vitro model was documented, although with CTT less than that observed clinically. Future studies of the TTA investigating the effect of larger loads, the effect of differing joint angles, the effect of varying cage sizes, and the effect of other muscles acting around the stifle are warranted.

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