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

By

Jonathan Mark Miller

Thesis submitted to the Faculty of the Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of

Master of Science in Biomedical and Veterinary Sciences

Approval Committee:

Chair: Peter K. Shires, BVSc, MS, Diplomate ACVS

Otto I. Lanz, DVM, Diplomate ACVS

Robert A. Martin, DVM, Diplomate ACVS

J. Wallace Grant, PhD

May 9, 2007
Blacksburg, Virginia

Keywords: Dog, Cranial cruciate ligament, Tibial tuberosity advancement, Biomechanics.

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

By

Jonathan Mark Miller

ABSTRACT

Objective-To assess the effect of 9 mm tibial tuberosity advancement (TTA) on cranial tibial translation (CTT) in cranial cruciate ligament (CCL) deficient canine stifles.

Study Design-In vitro cadaveric study.

Animals-Twelve canine pelvic limbs.

Methods-Each stifle was placed in a jig at 135° with a simulated quadriceps force and tibial axial force, and the distance of CTT was measured with the CCL intact (iCCL), transected (tCCL), and after performing a TTA using a 9 mm cage. In addition, a material testing machine was used to assess the force required to elicit CTT in each scenario.

Results-The 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 ($p<0.0001$) and tCCL and TTA ($p=0.0003$) were significant. The difference between the tCCL with and without the quadriceps force was not significant ($p=0.0597$). The force required to cause CTT was greater in the TTA than the tCCL up to 6mm ($p<0.0001$). As axial load increased, the force required to advance the tibia increased in both treatment groups (p value for overall weight effect =0.0002).

Conclusions- These data confirm that TTA does reduce CTT in tCCL stifles in this model. The addition of a simulated quadriceps force to a CCL deficient stifle prior to a TTA, by itself, may not significantly lessen CTT.

Clinical Relevance- While this in vitro model demonstrated that TTA reduced CTT in canine stifles with CCL transected, the modular limitations preclude extrapolation to the effect of TTA on the live dog.

ACKNOWLEGEMENTS

Grant Information: The author would like to recognize the American College of Veterinary Surgeons Surgeon-In-Training Grant for financial support.

Acknowledgements: The author would like to recognize Ms. Pam Arnold for her acquisition of specimens and laboratory expertise, Mr. Dave Simmons for his invaluable assistance in manufacturing and implementing the testing jig, and Mr. Bob Simonds for teaching the use of the materials testing machine. The author would additionally like to thank Dr. Stephen Werre for help with statistical analyses.

The author would be remiss to not thank the graduate committee members listed above for their support and encouragement throughout this project and acknowledge their immense and remarkable contribution to the author's professional career.

Dedication: The author would like to dedicate this manuscript to the 32 fallen students and faculty which occurred in horrific manner on April 16, 2007. Dave and Bob, above, were in the Norris Hall when the killings occurred, and Dr. Wally Grant was shot by the assailant. All of the biomechanical testing performed in the following thesis was accomplished with their help in Norris Hall, on the Virginia Tech campus. Let this dedication help us all to never forget.

TABLE OF CONTENTS

INTRODUCTION

Rupture of the cranial cruciate ligament is the most common cause of lameness of the hind limb in dogs.[1] The estimated cost of diagnosis and treatment of cranial cruciate ligament injuries in the USA in one year was over one billion dollars.[2] This pathologic process in dog's knees results in lameness, pain, instability, and degenerative joint changes. It is not surprising therefore that this disease has over the course of the last fifty years been the focus of much research and the development of over one hundred methods of treatment by the veterinary community to help affected dogs.

Dogs present to veterinarians with pain in the stifle with an associated lameness, ranging from mild to non-weight bearing. Depending on the chronicity of the injury, the stifle may demonstrate obvious effusion, soft tissue thickening, muscle atrophy, and instability. Instability is gauged by palpating the degree of cranial to caudal motion between the femur and tibia and reflects the presence of either complete or partial tearing of the cranial cruciate ligament. This ligament normally does not allow motion in a cranial to caudal direction, with the exception of less than one centimeter of movement in very young dogs. Two tests are available to assess the stifle for increased motion due to complete loss of the ligament: cranial drawer and cranial tibial thrust. To assess cranial drawer, the examiner stabilizes the distal femur with one hand and attempts to move the proximal tibia in a pure cranial direction with their other hand. Movement cranially is termed positive cranial drawer and is diagnostic of a cranial cruciate ligament rupture. This instability occurs when dogs walk with cranial cruciate deficient stifles, and is likely the cause of subsequent medial meniscal tears.[3,4] Cranial tibial thrust is assessed by holding the extended stifle with one hand and flexing the tibio-tarsal joint with the other hand. Obvious cranial motion of the proximal tibia will be noted when the cranial cruciate ligament is ruptured.

Radiographs of the affected stifle joint will often show joint effusion on the lateral view with displacement of the normal intraarticular fat pad cranially. On both the lateral and cranio-caudal radiograph, if the injury is chronic enough, evidence of osteoarthritis will be present in the form of osteophytes present on the poles of the patella, the femoral trochlear groove, and at the medial and lateral aspects of the tibial condyles, fabellae, and femoral intercondylar notch.

Conservative treatment of a cranial cruciate ligament rupture consists of pain relief medication, anti-inflammatory medication, weight loss, and exercise restriction for four to eight weeks. It was noted that 85% of dogs weighing less than 15 kilograms (kg) had improved at four months after conservative treatment, while only 19% of dogs over 15 kg showed any clinical improvement, even six months later.[5] In another conservative management study, a small group of dogs weighing more than 25 kg was evaluated three months post-treatment, and the owners felt that 73% were walking in the good to excellent range, while veterinarian evaluation rated 83% as good to excellent.[6] Thus a typical recommendation for small dogs and even cats is to attempt conservative treatment, but the recommendation for large breed dogs is less clear.

Surgical joint exploration is routinely recommended to the owners of large breed dogs suspected of having cranial cruciate ligament injury. Arthrotomy to inspect not only the cranial and caudal cruciate ligaments, but also the menisci is performed. Damaged ligamentous tissue is routinely excised. More recently, arthroscopic examination of the stifle joint was shown to be adequate in identifying damage and may offer some advantages in speed of recovery from surgery.[7] After surgical confirmation of the ruptured ligament, a procedure to assist the dog in controlling cranial drawer is performed. A large degree of success was reported with almost all procedures performed over the last fifty years, however osteoarthritic changes often progress with time to varying degrees.[8-10] It is suspected that early diagnosis and treatment followed by early return to motion may help improve outcome.[11] Obviously, the more durable the stabilization procedure is, the safer the implementation of early postoperative return to function.

The surgical technique that precipitated this study is aimed at assisting or empowering the active stabilization system of muscles around the stifle. Through this adjustment the effects of rehabilitation are expected to speed up and be enhanced, with a resultant earlier return to function for the patient.

CHAPTER I: Literature Review

A: History

1. Veterinary Literature

In the veterinary literature, cranial cruciate ligament rupture is the most common orthopedic condition seen in dogs.[1,12,13] More than 600 scientific articles have been published on the subject. Although cranial cruciate ligament (CCL) rupture was first reported in 1926, the first comprehensive description of the disease and treatment was published in 1952 by Paatsama.[14] The CCL, one of six major ligamentous structures associated with the canine stifle joint, was described traditionally as having three main functions: limiting cranial tibial translation, limiting stifle hyperextension, and in conjunction with the collateral ligaments, limiting internal rotation of the tibia. The CCL was originally thought to rupture in a traumatic fashion, secondary to sudden internal rotation of the tibia with the stifle flexed.[15] A pure hyperextension injury was also suspected to be involved in CCL rupture, as was hind limb conformation, and immune mediated arthropathies. Another theory proposed previously was a direct blow to the stifle in a cranio-caudal direction. Each of these theories may contribute to the pathogenesis, in some cases more than others; however, more recent work suggests that the most common mechanism is likely chronic and degenerative in origin, where CCL failure occurs after repetitive minor tearing of the ligament and subsequent biochemical and cellular changes within the stifle joint.[1,15]

Hundreds of surgical techniques have been proposed for cruciate ligament failure in dogs. They can be broadly classified into intracapsular stabilization, extracapsular stabilization, and bony alteration surgeries such as the tibial plateau leveling osteotomy

(TPLO) and tibial tuberosity advancement (TTA) procedures. Paatsama described an intracapsular technique to replace the CCL based on a human surgical technique. A strip of fascia lata from lateral and proximal to the stifle joint is harvested while maintaining its distal attachment.[14] This strip is then passed through tunnels drilled into the medial portion of the proximal tibia and the lateral femoral condyle along the same orientation of the original CCL. The fascial graft is then secured to the distal patellar ligament. This technique was intended to entirely replace the cranial cruciate ligament and all of its three major functions.

In 1966, Dueland described an alternate intracapsular stabilization technique in which the middle one third of the patella ligament with a piece of patellar bone is placed through a hole in the lateral femoral condyle.[16] A modification of the Paatsama technique described in 1977, involved placing the fascial graft proximo-caudally through the joint and then through the femoral bone tunnel, thereby avoiding the tibial bone tunnel.[17] Arnoczky modified Dueland's technique further in the form of the "over-the-top", where the patellar ligament graft is pulled caudally through the joint and secured to the lateral femoral condyle with no need for bone tunnels.[18] Hulse and Shires reported a modification of Arnoczky's procedure, known as the "under-and-over", whereby a combined fascial and patellar ligament graft was pulled under the intermeniscal ligament cranially prior to passing it through the joint and securing it to the lateral femur with a screw and spiked washer.[19,20] More recently a hamstring graft was described experimentally.[21] The combined insertions of the semitendinosus and gracilis muscles were harvested, passed through a tibial bone tunnel, then caudally through the joint, and secured to the lateral femoral condyle. Use of allograft ligament, teflon, carbon fiber, polyester fiber, and skin were reported, but not used commonly due to success of autogenous tissue and concern of implant breakage and foreign body reaction.[22-26]

The above intracapsular techniques were designed to physically replace the CCL with autogenous fascia or patellar ligament. The disadvantages of performing intracapsular stabilization techniques have been enumerated in the literature. The harvesting of autogenous tissue and passing of the graft through the stifle joint are both technically demanding, requiring meticulous dissection and thorough knowledge of the articular anatomy.[8] The grafted tissue must be protected in the course of early surgical recovery lest it be subjected to stresses resulting in undue fatigue and ultimately failure. In a biomechanical evaluation of the under-and-over technique, the amount of cranial tibial translation occurring in dogs actually increased at four and twelve weeks following surgery demonstrating either stretching of the graft itself or weakening of the graft securement site on the lateral femur.[27] In vitro testing of the graft itself, however, demonstrated an increase in strength and stiffness of the tissue as postoperative time increased, suggesting that the autograft function may improve with time.[28] Histologic assessment of the graft showed that it becomes acellular and undergoes necrosis early after placement, but then revascularization occurs from the surrounding synovial fluid which also supplies new fibroblasts.[8] Because of this, severe exercise restriction for the first 2-3 months, with a short term bandage for support, was recommended postoperatively. The graft then remodels slowly over time, but likely becomes a substance similar in structure and function to the cranial cruciate ligament.[8]

Clinically, the intracapsular stabilization techniques provided good results in the medium term with 85 to 93% of dogs using the operated leg well.[19,29] However, in a long term, owner assessment study, owners reported a decline in function of the operated limb from 13 to 50 months postoperatively.[30] It was unclear given the study design, as to what caused this decline in perceived function. A prospective clinical study published in 2005, compared dogs that received an over-the-top procedure following CCL rupture to dogs with a TPLO or extracapsular repair.[31] The results based on force plate gait analysis showed that the dogs receiving the over-the-top procedure had significantly reduced function at two and six months after surgery. The results of these last two studies combined with the difficulty in surgical technique has led to alternate procedures such as the TPLO and extracapsular stabilization techniques becoming much more commonly performed in the treatment of CCL disease today.

Because of the disadvantages associated with intracapsular techniques, extracapsular techniques were described and used early on. After Paatsama's work, a variety of methods were employed to constrain cranial tibial translation in the CCL deficient dog. One of the first procedures described is a transposition of the long digital extensor tendon medially.[32] Since this tendon is attached to the lateral femoral condyle normally, a groove is made in the tibia and the tendon distal to the stifle joint is secured to the tibia. Good results were reported in 52 dogs.[32] The DeAngelis suture technique was introduced in 1970, and in some form is still widely used by veterinary surgeons today.[12,33] This stabilization technique, entitled lateral retinacular imbrication, involved application of suture material or stainless steel wire between the lateral femoro-fabellar ligament and the disto-lateral patellar ligament.[33] The primary goal of this procedure was to eliminate abnormal cranial tibial translation secondary to loss of the CCL, but it also limited internal tibial rotation. DeAngelis' imbrication technique was modified by Dr.

Flo in 1975 to include three sutures. Lateral and medial fabellar non-absorbable sutures are placed through a hole drilled in the tibial tuberosity, in addition to a supporting suture from the lateral fabella to the lateral parapatellar fascia.[34] She had 95% satisfactory outcome with 83 stifles and later, Gambardella reported 94% success with another modification.[34,35]

Modifications of the DeAngelis suture technique have been reported widely. The type of material used can affect the complication rate, with one study reporting a 21% rate of draining tracks with a braided, nonabsorbable suture.[36] Monofilament suture materials are preferred today.[37] Nylon fishing line and orthopedic wire appear to be the most widely used materials in contemporary veterinary surgery.[38-40] Utilization of autogenous tissue, in the form of a lateral fascial graft was also described.[41] In addition to different materials, the ideal location for placement of a lateral fabellar suture was also investigated. Work on finding the instant center of motion, the center point where motion occurs around during flexion and extension of the femoro-tibial joint, suggested that utilizing a lateral fabella to tibial tuberosity suture may allow for less disturbance in joint motion than a suture placed from the fabella to the patellar ligament.[42] More recently the use of a bone anchor in the distal femur was described as the proximal anchor point for the suture with good results.[43,44] The method of securing the suture has also received attention in the veterinary literature. Traditionally, the suture material is secured with multiple knots, but recent work suggests that using a specific metal crimp system provides a stiffer construct.[45,46]

Additional stabilization techniques include: in 1971, Pearson published a report in which tibial translation was stabilized by multiple imbricating Lembert sutures in the joint capsule and muscles lateral to the joint.[47] Hohn introduced a posterolateral capsulorrhaphy in 1975, in which the caudolateral joint capsule is imbricated in addition to advancing the musculature from the medial and lateral stifle up to the patellar ligament to prevent tibial translation and internal rotation.[48]

The fibular head transposition was introduced from the University of Pennsylvania in 1985 as an alternate extracapsular stabilization technique.[49] The fibular head and attached lateral collateral ligament are moved cranially, secured with a pin and wire, and a lateral retinacular imbrication is performed. The fibular head is moved cranial enough to eliminate intraoperative cranial drawer. Bandage for two weeks and exercise restriction for months was recommended. The original report of 71 surgically treated stifles resulted in 90% good to excellent function.[49] Complications related to the procedure, and operative difficulty when compared to a lateral fabellar suture procedure, have limited its widespread acceptance.[6,50]

All of the above extracapsular stabilization techniques rely on periarticular fibrosis for long term success as the implant is likely to fatigue and fail with time.[8] This knowledge and the postoperative exercise restriction required has led to the development of stabilization techniques that do not rely on soft tissue fibrosis, and therefore, allow the dog to return to function earlier after surgery. The tibial plateau leveling osteotomy was devised by Dr. Slocum as an alternative and reported in 1993.[51] This was an improvement upon his original cranial tibial wedge osteotomy designed to be an adjunct to one of the traditional stabilization procedures.[52] The rationale behind the TPLO was that cranial tibial thrust occurs during walking in the dog. This places tension on the CCL to restrain cranial tibial translation and subjects the CCL to chronic repetitive injury.

The amount of thrust was dependent on the force applied across the joint, but perhaps equally as important, the slope of the top of the tibia: the tibial plateau.[51] Cranial tibial thrust was therefore reduced by reducing the caudally sloping angle of the tibial plateau in relation to the long axis of the tibia. During a TPLO the proximal tibia was osteotomized with a semicircular cut, the plateau leveled to about 5°, and the proximal tibia secured to the tibial shaft with a unique plating system. Dr. Slocum reported on 394 cases and had good to excellent clinical results in 94%, with many performance dogs returning to normal function.[51] Throughout the 1990s and early 21st century, the TPLO exploded in popularity to the point at which in 2005, it was reported to be the most common procedure used to stabilize a CCL deficient stifle in many specialty hospitals.[31]

Since the original 1993 article, TPLO was absent from the veterinary literature until 2001. Since then, over thirty articles have been published on various aspects of the technique. Conflicting reports on the significance of large tibial plateau angles were reported, with some studies correlating an increased tibial plateau angle to finding CCL ruptures, while others refuted that supposition.[53-55] Much attention was paid to methods and accuracy of the measurement of the tibial plateau angle on radiographs.[56-64] Additionally, work was done on how and where to make the osteotomy. The conclusions were that making the osteotomy perpendicular to the long axis of the tibia in cranio-caudal and proximo-distal planes was important to prevent angular and rotational deformities postoperatively.[65] The osteotomy should be reduced without regard to attempted alignment of the medial tibial cortices.[65] One report evaluated in bone models, the use of a variety of different osteotomies and fixation techniques, and found that all five variations on the TPLO were acceptable in vitro.[66]

Outcomes were assessed in dogs following TPLO in a number of reports. The most objective method to assess postoperative outcome is with force plate gait analysis.[67-69] One study in 2004, evaluated experimentally induced CCL rupture in dogs to compare gait in a normal dog before and at 8 and 18 weeks after CCL transection with TPLO stabilization.[70] Results showed reduction in peak vertical force (PVF), which is the amount of vertical or Z axis force applied by the dog when weight bearing on one limb, and reduction in vertical impulse (VI), which is the force placed in a craniocaudal or Y axis direction. The reduction correlated with lameness at the eight week assessment. At 18 weeks postoperatively, there was no significant difference compared to the normal preoperative values.[70] In a study from Iowa State University, a cohort of 32 dogs was assessed with force plate examination before and a mean of seven months after surgery.[71] It revealed that the PVF and VI improved significantly in dogs clinically affected by CCL rupture in which a TPLO was performed. They found that neither preoperative nor postoperative tibial plateau angle measurements had a significant effect on objective lameness when postoperative angles varied from 0 to 14°.[71]

In an excellent prospective force plate study, Dr. Conzemius evaluated 131 Labrador retrievers following CCL rupture stabilization with three different methods, one of which was a TPLO.[31] The TPLO group showed improved PVF and VI compared to preoperative levels at two and six months postoperatively, however, there was no significant difference between the TPLO group and a group of dogs that received a lateral fabellar suture.[31] Both groups failed to achieve normal force plate measurements long term. In a 2005 study, TPLO and lateral fabellar suture techniques were directly compared to evaluate the progression of radiographic osteophytosis.[72] It was found that osteophytosis progressed similarly in both groups up to a mean of 22 months after surgery, but those dogs that had severe progression were 6 times more likely to have had a lateral fabellar suture placed.[72] Another long term radiographic study on dogs receiving TPLO suggested that dogs in which only a limited arthrotomy was used to assess the medial meniscus had a slower progression of arthritic change over a mean of 20 months.[73] The authors recommended that the traditional arthrotomy to expose the entire stifle joint may be more harmful than is required to treat the intraarticular disease in dogs with CCL injury. In another study evaluating radiographic assessment of degenerative change in the canine stifle, a group of 40 dogs was followed for 6 months with radiographs.[74] Overall osteophytes worsened, however in 24 of the dogs, no progression was noted. No explanation was proven for a difference in the two groups.[74]

Complications associated with TPLO were noted to be different than those typically associated with other methods of CCL stabilization surgery. The reported complication rate ranges from 19 to 28%.[75-78] Incisional infection appeared to be the overall most common complication, which responded to antibiotic therapy. Osteomyelitis, when present in 14/193 and 7/397 cases, required plate removal.[76,77] Fracture of the fibula, presumably secondary to tension placed on it following tibial plateau rotation, occurred in 3.1%, 0.003%, 0.007% of cases and did not require treatment.[75-77] Postoperative fracture of the tibial tuberosity was noted in 6/193, 14/397, 28/696, and 19/219 stifles, usually prior to the 6-8 week radiographic recheck.[75-77,79] Conservative management of this complication seemed to be sufficient to resolve any setback in postoperative function, however the location of the osteotomy has since been recommended to be moved further caudal than the original description to avoid producing a thin tibial crest.[79] Additionally, it is recommended to place the temporary anti-rotational wire proximal to the insertion of the patellar ligament to avoid producing a stress riser distal to the attachment point.[75-77,79]

Another complication unique to the TPLO was patellar ligament desmopathy. This was reported to occur in 0.05% and 3% of dogs at a clinically apparent level.[75,77] However, when specifically assessed 80% of dogs had thickening of the patellar ligament on two month radiographic recheck, and 100% had radiographic thickening at one, two, and six months rechecks.[80,81] Thickening was most predominate distally. When the patellar ligament was assessed ultrasonographically, ligamentous thickening was observed in 89% and changes consistent with inflammation of the ligament were observed in 13/31 stifles at one month postoperatively and 3/13 stifles at six month recheck.[80] Proposed causes such as contact with the saw blade during the osteotomy and damage secondary to the anti-rotational pin placement have largely been discounted.[81] The most plausible theory was that the rotation of the weight bearing tibial surface caused an increased tensile force on the patellar ligament with each contraction of the quadriceps during weightbearing.[80,81]

One rarely reported complication secondary to TPLO was the development of osteosarcoma at the osteotomy site years after surgery in four dogs.[82] Use of the Slocum plate, which was determined to be a cast metal rather than an annealed or wrought metal, was shown to have crevice corrosion and an increased tissue reaction.[83,84] Tissue reaction secondary to corrosion materials released into the tissue surrounding the plate was proposed to be involved in the etiology of these tumors.[84]

The other major consideration when performing a TPLO procedure was what to do with the intact medial meniscus. Any damaged meniscal ligament is routinely removed, regardless of the stabilization procedure performed. The medial meniscus is damaged in over half of all stifles explored following CCL rupture.[4,85] Dr. Slocum, in his original report, recommended that all dogs receive a caudal pole medial meniscectomy to eliminate the possibility of postoperative medial meniscal tearing.[51] It was theorized that the increased pressure on the caudal medial meniscus would be amplified by rotation of the tibial plateau, resulted in wedging of the caudal meniscus between the femur and tibia subjecting it to crushing injury.[51,86] Slocum later suggested that a release of the intact medial meniscus, without excision, would accomplish the same goal of preventing future tearing by allowing it to slide caudally to avoid crushing.[87] Recently, the effects of medial meniscal release were investigated. When the medial meniscus was assessed by magnetic resonance imaging before and after a meniscal releasing procedure, the caudal horn did move caudally with simulated weight bearing.[88] The authors suggested that meniscal release eliminated the function of the medial meniscus, potentially subjected the femoral and tibial articular surfaces to increased stress, but did allow for reduced chance of postoperative medial meniscal damage.[88] In a radiographic study with simulated weight bearing forces, the effect of meniscal release was not different than caudal pole medial meniscectomy in relation to tibial translation in CCL deficient cadaver specimens.[89] Following TPLO, the caudal medial meniscus was unloaded, and thus the effect of meniscal release was reduced. The authors proposed that meniscal damage would actually be reduced in a TPLO stabilized stifle in comparison to an unstabilized CCL deficient stifle.[89] A recent retrospective study assessed the effect of meniscal release following TPLO in 254 stifles.[90] They found that 0.04% of dogs that had an arthrotomy performed without a meniscal release subsequently damaged the medial meniscus, whereas 0.02% of dogs with arthrotomy and a meniscal release returned for meniscal tearing.[90] When the meniscus was assessed arthroscopically without meniscal release, only 0.01% of dogs had a subsequent meniscal tear, so the authors recommended if the meniscus could not be assessed thoroughly, a releasing procedure should be performed.

In 2003, the originators of the TTA from the University of Zurich published a report on an alternative to the TPLO. The surgical procedure, entitled the proximal tibial osteotomy, from the University of Zurich involved performing a double osteotomy starting caudal to the insertion of the patellar ligament to the caudal tibial cortex at the distal level of the tibial crest.[91] A wedge of bone was removed and the free portion of the caudal proximal metaphysis was moved forward and held in place with two screws. This procedure aimed to level the tibial plateau in a different method than the TPLO to avoid patent infringement.[91] Their preoperative planning, instead of measuring the tibial plateau angle, was to measure the angle of the wedge taken out to position the tibial plateau perpendicular to the patellar ligament. This change in planning was secondary to work performed by Dr. Tepic on the biomechanics of the dog stifle.[92] One hundred dogs with CCL ruptures were treated with the proximal tibial osteotomy and 86% were lame free. The technique was, however, not recommended due to a large number of intraoperative and postoperative complications.[91]

A later modification of the technique, reported from New Zealand, with the addition of a medial bone plate provided for improvement in complication rate.[93] In the report, 52 dogs received the proximal tibial intraarticular ostectomy, and author lameness scores and thigh circumference had improved in all but one dog at twelve month follow up compared to preoperative measurements.[93]

Tibial tuberosity advancement was first described by Drs. Montavon and Tepic in 2002 as an alternative to the patented TPLO.[94] In this procedure, the tibial tuberosity, and therefore the insertion point of the patellar ligament, was positioned more cranially such that the patellar ligament rests in its new position perpendicular to the tibial plateau. Titanium implants are placed to secure the advanced tibial crest to the remaining tibial shaft. They suggested that the TTA was less invasive, allowed for normal flexion of the stifle thereby eliminating the need for meniscal release, and advancement would reduce the femoropatellar pressure thus reducing patellar articular chondromalacia.[94] Montavon reported on 200 cases of TTA with good clinical results and 0.04% failure rate secondary to poor surgical procedure.[95]

A recent retrospective study was the first peer-reviewed article on the outcome of TTA in clinically affected dogs.[96] Clinically, 90% of the 57 dogs had improved postoperatively based on owner assessment. In this series, based on the inventors theory, no release of the medial meniscus was performed and 10% of the dogs with intact menisci subsequently required reoperation due to meniscal injury.[96] The authors recommended meniscal release in future cases. Only two other dogs required a second procedure: one dog had catastrophic failure of the implant requiring tibial crest repair with pin and tension band; the other dog required explantation due to suspicion of intraarticular screw placement. At a mean of eight weeks after surgery, mild osteophytosis progression was noted, and bone healing at the osteotomy had secured the tuberosity in its new position in most.[96]

2. Human Literature

Tibial tuberosity advancement was first described in human in 1976, as a method of reducing the pressure within the patellofemoral joint in knees.[97] People experiencing pain in the knee and having no other pathology besides patellar chondromalacia were diagnosed with patellofemoral pain syndrome.[98] Dr. Maquet theorized that by advancing the insertion of the patellar ligament, a longer lever arm existed for the quadriceps to be able to do the same work with less effort, i.e., increased efficiency, so that a reduced force was needed to hold the leg in extension during ambulation.[97] This subsequently reduced the force present between the patella and the femur. The procedure entailed making a transverse osteotomy of the tibial crest including the insertion of the patellar ligament that was not complete distally.[97] The osteotomy gap was held in position with a two to three centimeter piece of iliac crest at the proximal end, while distally the gap is filled with bone graft. Walking with crutches was encouraged the day after surgery and for two weeks. Full range of motion was to be regained by three months after surgery. He suggested that advancement of 20mm would reduce compressive forces within the patellofemoral joint by 50%.[99] Maquet originally reported on 41 knees receiving solely the procedure. At a mean of 4.7 years after surgery, 95% of patients reported absence of pain and improved function.[97] Postoperative results in people have been good overall in reducing patellofemoral pain since Maquet's original description, however not every case met with a successful outcome even with improvements in the technique over time.[100-107]

Patellofemoral joint pain, has since been described as the most common problem affecting the knee in humans.[108,109] This disease, also known as “runner’s knee”, was most prevalent in young women and constitutes 16 to 25% of all running injuries.[108,109] The diagnosis was often made with the historical findings of pain behind the patella exacerbated by sitting, stairs, or squatting coupled with abnormal patellar tracking in a knee without effusion.[109] The patella often would suddenly move laterally out of the line of the femoral trochlea as the knee is extended. Palpation of the patella in an extended knee may reveal abnormal tilting of the lateral patella out of the trochlea; this can be seen on radiographs of the knee taken to assess for osteoarthritis.[109] Surgical management of abnormalities of the patellofemoral joint were frequently reported, including the earlier described tibial tuberosity advancment.[110-112] However, more recently success was widely seen in non-operative management of patellofemoral pain.[113] Nonsurgical treatment recommendations include reducing running distance and cold application first, followed by in most cases physical therapy to increase quadriceps muscle strength or flexibility.[109] Success was noted in over 80% of people without surgery.[113] Non-steroidal anti-inflammatory drugs are used in most people, although their effectiveness has recently been questioned.[109] Taping of the knee and a variety of shoe inserts may help alleviate pain as well. Surgical treatment currently is reserved for those patients with persistent pain after completing a twelve month rehabilitation program.[109]

Another set of patients complain of patellofemoral pain following surgical management of anterior cruciate ligament injuries.[114,115] Pain may be secondary to postoperative osteoarthritic changes in the knee joint, shortening of the patellar ligament causing altered patellofemoral biomechanics, and loss of range of motion.[114,115]

Additionally, pain at the autograft harvest site and less than ideal placement of the reconstructed ligament were potential causes of postoperative patellofemoral pain.[116] These patients can be treated with either physical therapy or a second surgical procedure, which may include advancement of the tibial tuberosity.[116]

Anterior cruciate ligament (ACL) injury in humans is the subject of over 7,000 articles in the PubMed Database covering the last 50 years (accessed April 2007). It was estimated that over 200,000 new ACL injuries occur every year in the United States.[117] The ACL is the most frequently disrupted ligament in the human knee.[118] More than 100,000 ACL reconstructive surgeries are performed every year.[118] The ACL in the human knee accomplishes the same restraints as in the canine: control anterior tibial translation, limit internal tibial rotation, and help control hyperextension.[119] An additional function of proprioception has received attention more recently.[120] Injury is most often seen in young athletes, who become acutely lame secondary to ACL rupture commonly in conjunction with meniscal tearing, medial collateral sprain, and bone bruising.[118] Meniscal tearing is common, with 40% occurring concurrently; this increase to 80% in people with unreconstructed ACL tears at 10 years after the injury.[118] Risk factors for ACL injury include a high coefficient of friction between the athletes foot and the ground, the size of the ligament, sudden deceleration (cutting maneuvers or landing after a jump), and sudden unbalanced quadriceps contraction.[121]

A number of factors related to the knee contribute to injury of the ACL. A sudden, forceful contraction of the quadriceps muscle without concurrent contraction of the balancing hamstring muscle group was shown to strain the ACL when the knee was between full extension and 130° of extension.[118] This quadriceps force with the knee in extension results in anterior translation of the tibia and was suggested to be one mechanism for ACL rupture, whereas with increased flexion, ACL strain was reduced.[121] Valgus knee position and internal rotation of the tibia have also been proposed to increase risk of ACL injury.[118] Neuromuscular control of the knee was also shown to be a factor. Reaction time, muscle contraction balance, fatigue of muscle, and landing with hips and knees in extension will negatively impact the ability to spare the ACL ligament.[121] By training and improving the neuromuscular control of the knee, reduction in the incidence of ACL injury was noted in at-risk athletes.[122] Training included improving muscular reaction time with a balance board, strengthening the hamstring muscles, and risk awareness.

Surgical reconstruction of ACL injuries in people has been the subject of great debate in the literature for years. Non-operative treatment has met with success in patients willing to restrict their athletic activity.[123,124] Increasing levels of osteoarthritis and risk of meniscal injury were reported, however.[118,123] Alternatively, some studies suggest individuals can return to high risk activity successfully without surgical stabilization.[125] Treatment consisted of rest and a controlled physical rehabilitation program for four to eight weeks.[124,125] Rehabilitation started with shifting of weight onto and off of the inured leg, then progressed to balance board and weight-bearing exercises, then to single leg standing and eventually straight line running.[122] Improvement in muscle strength and function of the knee was usually reported, though return to normal neuromuscular function was considered unlikely.[120]

A recent survey of sports medicine clinicians, the majority recommend surgical repair of ACL ruptures in people.[126] In most studies comparing non-operative treatment to surgical stabilization, those patients who have surgery routinely achieve superior results.[118] Surgical repair of ACL deficient knees blossomed in the 1970s, and has culminated in arthroscopically assisted autograft reconstruction as the procedure of choice.[118] Allograft tissue for ACL reconstruction has fallen out of favor due to cost and increased risk of infection. The two procedures with the most documented success, the four-strand hamstring graft and the bone-patellar tendon-bone graft, are similar to the canine intracapsular procedures described previously, in which the ACL is replaced by autogenous tissue to mimic the anatomical and functional aspects of the damaged ACL.[118] No significant differences in function between the two techniques were found in a multitude of controlled studies. People receiving surgical stabilization of the knee following ACL rupture were subjected to a variety of rehabilitation programs. Early joint motion, immediate weight-bearing, weight-bearing exercises, and use of neuromuscular stimulation were found to improve outcome, whereas cold therapy and the use of knee braces were not found to help.[118]

B: Canine Anatomy

The canine stifle joint is the articulation between the femur and the tibia, and the femur and the patella. Weight bearing occurs across the femoro-tibial joint, whereas the femoro-patellar articulation greatly increases the mechanical efficiency of the quadriceps mechanism to maintain stifle extension.[37] The quadriceps mechanism consists of the large rectus femoris muscle and three smaller muscles: the vastus lateralis, vastus medialis, and vastus intermedius. The patella, which is the body’s largest sesamoid bone, is imbedded on the femoral surface of the musculotendinous portion of the quadriceps and acts as a lever arm across the stifle joint. The patella articulates with the femoral trochlea before it divides onto the two caudally oriented femoral condyles. Distal to the patella, the quadriceps mechanism continues as the patellar ligament, or patellar tendon, until the insertion point on the tibial tuberosity. The tibial tuberosity is a cranial protuberance at the proximal end of the tibial crest. An infrapatellar fat pad is located caudal to the patellar ligament; on lateral radiographs of the stifle, this structure provides a convenient fat opacity to assist in defining the increased amount of fluid associated with pathologic stifle effusion. The quadriceps mechanism contracts during weight bearing to achieve stifle extension and prevent hind limb collapse. The so-called hamstring muscle group is caudal to the femur and through contraction it flexes the stifle. This muscle group, consisting of the biceps femoris, gracilis, semimembranosus, and semitendinosus, was suggested to provide some counter to cranial tibial translation in addition to the CCL.[51]

The canine stifle joint routinely allows for 140° of flexion-extension in most dogs, with a mean flexion angle of 42° and a mean extension angle of 162° in normal Labrador retrievers.[37,127] In a kinematic study, a normal dog traverses about 60° of flexion-extension while walking.[3] During flexion, internal rotation of the tibia with respect to the femur is noted, while the opposite: external rotation is seen with stifle extension. This provides for the stifle screw home mechanism.[37] About 10° of rotation occurred during walking in dogs, with the maximum internal rotation at maximum flexion.[3]

Within the stifle joint capsule are four major ligamentous structures. There are two cruciate ligaments: a cranial and a caudal (Figure 1). The major function of the caudal cruciate ligament, which originates from the lateral aspect of the medial femoral condyle and inserts on the caudal tibial plateau, is to prevent caudal translation of the tibia with respect to the femur.[37] The tough, thick, caudal cruciate provides for secondary restraint against stifle hyperextension and varus/valgus motion of the flexed joint. The other two major ligaments are the menisci: medial and lateral. These C-shaped fibrocartilaginous disks are located between the femoral condyles and the tibial plateau.[4] There are four functions of the menisci: prevention of joint capsule entrapment between the two bones, lubrication, stabilization by deepening the tibial articular surface, and transfer of the compressive load across the joint.[37] The amount of load transfer that the menisci provide was estimated to be 65% of force across the joint.[4] Following CCL transection, the force across the cranial portion of the medial meniscus doubled and increased across the caudal portion of the medial meniscus in one in vitro study.[86]

The lateral and medial collateral ligaments originate on the abaxial femur and insert on the abaxial tibia. These ligaments are actually outside of the joint capsule.[37] They provide for varus/valgus stability of the stifle joint. The cranial portion of the medial collateral ligament remains taut throughout stifle flexion and extension, while the caudal portion is only taut during extension.[37] The entire lateral collateral ligament is taut only in extension.[37] The long digital extensor muscle originates from the lateral femoral condyle and crosses the stifle joint, but no reports suggest any effect on stifle joint stability, though surgical reorientation of the tendon medially was once reported as a method of stifle stabilization following CCL rupture.[32]

The canine cranial cruciate ligament originates from the caudo-medial aspect of the lateral femoral condyle and inserts on the cranio-lateral intercondylar tibial plateau. It spirals 90 degrees externally as it courses distally.[1] The CCL has three major functions during load bearing: restrict cranial tibial translation, control tibial internal rotation, and restrict stifle hyperextension. The CCL is composed of a craniomedial band, which is taut in extension and flexion, and a caudolateral band, which is taut only in extension. The mean length of the CCL in dogs is 13.5 to 18.7 mm.[128] The CCL is supplied tenuously by vessels in the periligamentous tissue and by fluid from the synovial tissue of the stifle joint, which receives its blood supply from the genicular artery.[1,15] The blood supply is greatest at the proximal and distal ends of the ligament, while the inner portion is relatively hypovascular. The CCL is innervated by the branches of the saphenous, common peroneal, and tibial nerves with the greatest number of mechanoreceptors being present in the proximal portion of the ligament.[1] These mechanoreceptors contribute to many local, spinal, and central hindlimb muscle reflexes, which actively protect the CCL from tearing during motion and prevent joint damage.[128]

The micro-structure of the CCL, which is 70% water based on weight, consists of collagenous units which are subdivided into fascicles, subfascicular units, fibers, and fibrils.[1,128] This division of the CCL into multiple subunits allows for varied recruitment of fibers through various levels of tension at multiple orientations of elongation. The CCL is composed of 90% Type I collagen with the remainder being Type III collagen.[128] Fibroblasts are the main cell type present supporting the collagen and consist of 3 separate types: fusiform, ovoid, and spheroid.[1] The actions of these individual types of fibroblasts have not been completely elucidated.

C: Pathophysiology

CCL rupture is proposed to occur in two main ways: acute traumatic disruption and chronic degeneration. In traumatic disruption, the CCL is torn because of a large external force, sometimes resulting in multiple ligamentous rupture, as is seen in deranged stifles.[1] The limb is often in a hyperextended or internally rotated position, which has the CCL in a taut position, and then an additional force applied externally which strains the ligament past the ultimate failure point (the point at which rupture occurs). In cadaver experiments, midsubstance rupture occurred in most CCL ruptures at 51 megapascals (MPa)/kg body weight and 148 MPa in two separate studies.[28,129] Some CCLs did avulse from the origin or insertion points.[28] The strain (amount of elongation) at time of failure was noted in ACL to be about 15%.[119] Both the stress and strain at time of failure are affected by the rapidity at which the ligament is stretched. This is due to the viscoelastic nature of ligamentous tissue which allows for creep (elongation of the ligament under a constant force) and stress relaxation (subsequent elongation that occurs over time with the ligament secured at a set length). The force required to rupture the CCL and ACL was found to be equal in dogs and humans: 1700 newtons.[28,119] Rupture in the midsubstance occurred in most failure tests of cruciate ligaments due to stress dissipation at the enthesis, or insertion site, of the ligament.[130] An alternative method of traumatic CCL rupture is juvenile avulsion of the CCL origin or insertion. Avulsion of the relatively weaker bone of the distal insertion point of the CCL occurs in immature dogs, and a portion of the tibial bone can be seen radiographically within the stifle joint.[131]

More commonly, CCL rupture is seen as part of a continuum of CCL degeneration. These dogs are commonly older and have midsubstance tears of the CCL.[15] The CCL in these dogs are subjected to repeated strain due to cranial tibial translation which is not controlled by the dog’s secondary stifle stabilizers.[51]

Degeneration of the CCL was attributed to neutering, obesity, sedentary lifestyle, conformation with an excessively large tibial plateau angle, and increased body size.[1,15,128] The evidence of the effect of tibial plateau angle on CCL rupture has recently been questioned by studies showing no difference between dogs with CCL rupture and without.[54,55] Smaller dogs were noted in one study to experience CCL injury significantly later in life.[5] Neutering seems to increase the risk of CCL rupture, but whether this was due to a skewed study population was not clear.[132]

Osteoarthritis inevitably follows in the early stages of CCL rupture, and likely contributes to further fiber rupture as degradative enzymes are upregulated. It is unclear whether stifle instability begins the process or whether an immune mediated component of arthritis initiates it. Inflammatory changes have been documented in the synvioum, periligamentous tissue, and synovial fluid.[128] These changes are mediated by upregulated matrix metalloproteinases, cathepsin K, and tartrate-resistant acid phosphatase resulting in recruitment of inflammatory cells and collagen degeneration.[1,133,134] Histologically, degeneration of the ligament fibroblasts in naturally occurring CCL rupture was noted along with degeneration of the extracellular matrix.[135] The changes noted in collagen, loss of normal crimp and separation of fibers supported the idea that the CCL was subject to mechanical overload. Cells surrounding the CCL appeared to be attempting to remodel the CCL, but no actual reparation was noted.[135] Likely, CCL degeneration is multifactorial, including joint inflammation, mechanical loading, collagen fiber injury, and ischemia.[128] Collagen remodeling, CCL laxity, and osteoarthritis eventually lead to CCL rupture induce femorotibial instability and its associated clinical lameness in dogs. Even after stifle stabilization, osteoarthritis routinely progresses.[6,9,72-74,96]

D: Biomechanics

1. Canine Cranial Cruciate Stabilization Techniques

The biomechanical variables of the intact CCL in dogs were assessed in a number of studies. Stress is defined as a force per unit area, while strain is a change in length. Stiffness is the ratio between those two, which gives an idea as to how much force is required to deform a substance. The stiffness of cadaveric femur-intact CCL-tibial constructs was reported to be 93, 120, and 348 newtons (N) per millimeter (mm).[28,41,136] The maximum stress the CCL could endure before failure was 148 megapascals and 70 megapascals in two studies in which it was measured.[28,129] The maximum force applied before construct failure was 1656 N, with failure occurring midsubstance ligament in 27% of CCLs in one study.[28] In another study, the angle of the stifle affected not only the maximum force before CCL failure, but also whether the failure occurred within the ligament or from the bony attachment points.[137] In full extension, over 1,000 N were required for failure and most of these failed at the tibial insertion point.[137] With the stifle in 45° and 90° of flexion, failure loads were lower, about 400 N, and at 90° almost all failed by midsubstance ligament fiber tearing.[137] While these in vitro data provide information on the magnitude of loading the CCL is able to withstand, the in vivo situation in the dog is much more complex. The stifle of the dog must withstand weight bearing forces which were estimated to be anywhere from 45% of the dog's body weight to up to 90% body weight at a trot.[138,139] This 90% translates to 300 N in a 75 pound dog, which was less than the failure force noted in cadavers. In vivo, stifle stabilizers other than the CCL play a role. The caudal cruciate twists around the CCL and contributes to stability throughout flexion and extension, however dogs were reported to do well without reconstruction of a caudal cruciate rupture.[140] The menisci provide for joint stability by limiting varus/valgus motion and by increasing the articular surface area for contact with the femoral condyles.[88] The collateral ligaments provide additional control, though in an in vitro study, the joint capsule was found to not provide any cranial to caudal stability by itself.[141] However, in another study the joint capsule significantly reduced cranial to caudal translation.[142]

Muscle surrounding the stifle joint is of paramount importance to stifle stability with the quadriceps controlling extension and the hamstrings flexion, though the specific effect of muscle function is very difficult to assess in dogs. Information was gathered on the motion of joints through kinematic gait analysis.[143] Using these data and morphometric measurements of individual muscles around the knee, Shahar devised a mathematical model of the muscular forces affecting the canine knee.[144] Using this theoretical biomechanical model, the CCL was found to be subjected to 12% to 25% body weight force.[144,145] Since no in vivo measurements of muscle forces around the stifle exist, this model can be used to predict forces, but the validity in walking dogs is still unproven. This computer model was used to evaluate the effect of TPLO on forces that the CCL was subject to. Following a TPLO with the recommended 5° of tibial plateau angle, the force was reduced, but still present; however at 0°, the force on the CCL was eliminated and shifted onto the caudal cruciate.[145]

Intracapsular stabilization of the CCL deficient stifle was tested in vitro to assess stiffness in two early reports. In one, the patellar tendon was used as a graft, and the stiffness increased from 22 N/mm to 109 N/mm over the course of 6months in the dog.[28] The graft was however still only 30% as stiff as the original CCL. The graft could endure only 10% of the stress compared to the CCL before failure.[28] The stiffness of both a patellar tendon graft and a fascial lata graft was noted to be less than 10% of the CCL in a cadaver study.[136] One study evaluated patellar tendon graft stabilization in dogs.[146] The dogs were euthanized at 12 weeks postoperatively and the bone graft bone construct tested. They found that the graft had a stiffness of 54 N/mm (control CCL = 130 N/mm) and a maximum force to failure of 176 N (CCL = 626 N), and all failed with midsubstance graft tearing. Another study found similar results with a bone patellar tendon bone graft failing, after 12 weeks of healing, at a force of 258 N and stiffness of 63 N/mm.[147] Similar results found in a 2006 study (stiffness 70 N/mm and maximum force before graft failure 200 N) confirm that grafts are routinely weaker than the intact CCL.[148]

The majority of biomechanical studies have focused on measuring the distance of tibial translation following various manipulations. In a study on the difference in cranial tibial translation (CTT), the mean CTT with intact CCL was 1.8 mm.[27] Following a patellar tendon intracapsular graft placement, CTT was 2.9 mm after surgery, and then increased to 5.2 and 4.1 mm at four and twelve weeks after surgery, respectively. By six months, however, the CTT had reduced to 2.5 mm.[27] Normal tibial translation with an intact CCL was noted to be 0.72 mm and after cutting the CCL, 9.44 mm of translation was seen.[136] In this study, the stifle was loaded in a cranial direction with 80 N and caudal direction with 65 N; so, pure CTT was not measured. Measuring CTT using the same methods after stabilization with implants the following was reported: 3.3 mm with patellar tendon graft, 2.0 mm with a fascia lata graft, 2.3 mm with a lateral fabellar suture, and 0.8 mm with a fibular head transposition.[136] The authors suggested a significantly more stable repair could be made by performing a fibular head transposition.

In a study, using 40 N of cranial and caudal loading on the cadaver stifle, intact CCL stifles had 2.4 mm of translation, following transection of the CCL, 4.7 mm was noted, and after removal of the joint capsule for 6.6 mm of translation was present.[142] After dogs lived without a CCL for 34 weeks, CTT remained increased at 6.0 mm.[142] Another study in which 50 N of cranial and caudal loading was used, revealed that a bone-patellar tendon-bone graft allowed double the CTT of the intact CCL.[146] In one study, a 65 N caudal force and a 80 N cranial force was used to assess translation.[41] In Harper's study using 65 N, 1.8 mm of CTT was found with the intact CCL, 12.0 mm with the CCL transected, and 3.2 mm following a standard lateral fabellar suture.[41] This report also assessed a lateral fascia graft for the fabellar suture material; there was no significant difference in translation when compared to traditional suture material. It is noteworthy that no standard force for measuring tibial translational distance exists in the literature.

One study evaluated translational distance with radiographs.[149] The authors used 44.5 N of cranial and caudal force, and then measured distance off of the radiographic film. They found a total translation of 1.7 mm with intact CCL and 6.3 mm in stifles with CCL rupture. Interestingly they reported that the cranial portion of translation consisted of only 1.1 mm in the intact CCL group and 3.2 mm in the CCL ruptured group.[149] This suggests that pure CTT comprised only a fraction of the translational distance in many of the previous reports.

Other investigators have focused on measuring CTT in the live dog. In a landmark kinematic study, veterinarians at the University of Illinois described three dimensional motion of the dog stifle before and after transection of the CCL.[3] They described during manual palpation an increase from less than 1 mm to 11 mm following CCL transection. The dogs seemed to keep the stifle in more of a flexed position to avoid the cranial translation that occurred during full extension. When using kinematics for assessment, the cranial tibial translation was noted to increase only from less than 1 mm to 2mm secondary to an unopposed cranially directed force.[3] The authors suggested that in the live dog CTT was reduced by joint compression and muscle forces, but could not elaborate specifically. In another kinematic report, CTT increased from 0.4 mm to 10.1 mm after the CCL was severed.[150] This CTT remained uncontrolled throughout the 2 year study. Additional changes noted after CCL severance were increased stifle flexion (which improved with time), increased stifle adduction, and a small amount of increased medial translation.[150]

Effect of TPLO on tibial translation has received attention recently in the literature. In the first study, cadaver hindlimbs were subjected to tibial plateau leveling and translation noted based on measurements from radiographs of the intact CCL, following plateau leveling to 0°, and leveling to the point when CTT was eliminated.[151] Specimens were set to simulate in vivo forces by securing the stifle at 135°, applying turnbuckles to simulate the quadriceps and gastrocnemius muscles to a point where the stifle and tibiotarsal joints were kept steady when an axial force of 30% body weight was applied to simulate weight bearing. The amount of CTT in the intact CCL was not reported, but following transection CTT was 18.9 mm. After plateau leveling, a caudal translation was noted of 6.3 mm. By approximating the minimum plateau angle to control CTT, an ideal angle of 6.5° was described.[151] During the same study, the strain on the caudal cruciate ligament was noted to increase after plateau leveling and the authors suggested that over rotation of the plateau could subject the caudal cruciate to harmful amounts of strain.

In another study, the effect of TPLO on tibial translation was assessed using an axial force of 22 N.[152] Cadaver stifles without the patella were placed at 130° and CTT was measured after CCL transection and after the plateau was leveled to 5°. No measurement of intact CCL was reported. The mean CTT with no CCL was 14 mm, while after the TPLO a caudal translation of 2 mm was noted.[152] The authors also assessed the force resisting CTT by pulling cranially on the tibia until a sudden CTT was noted. They described the resistance to CTT as caudal tibial thrust and noted an increase in that force from 4 to 8 N when increasing axial force (13 to 45 N) was applied.[152]

In a study evaluating a variety of tibial plateau leveling procedures in a bone model, Hildreth reported reduction in construct stiffness in all methods when compared to the original model.[66] The authors could recommend any of the five different procedures in the clinical setting. In a cadaver study evaluating TPLO, investigators used a stifle angle of 145° with turnbuckles applied to keep the stifle and tibiotarsal joints at walking angles.[153] CTT was recorded based on radiographs while the limb was subjected to 30% body weight axial force. The study was meant to assess the difference in position of the TPLO osteotomy site. They found a CTT of 0 mm in the intact CCL group, 15 to 16 mm following CCL transection, and 1.5 mm after the better of the two osteotomy locations.[153] The distally oriented osteotomy still had a CTT of 9.0 mm, and thus was not recommended.

In a study of the effect of medial meniscal release on the canine stifle, the authors used radiographic measurement of CTT with the stifle positioned at 105° under a 20% body weight axial force.[89] They noted a 28 mm increase in CTT after CCL transection. The caudal pole of the medial meniscus moved 0.8 mm with an intact CCL and 1.9 mm with a transected CCL stifle in both meniscal release and caudal medial hemimeniscectomy groups of stifles prior to TPLO.[89] After TPLO, CTT was reduced to 1.7 to 2.4 mm. Following TPLO, the medial meniscus motion actually decreased to 0.5 mm, thus the authors suggested the reduction in CTT secondary to TPLO may actually spare future meniscal damage by decreasing the wedging of the medial meniscus between the femur and tibia. The authors proposed that routine use of medial meniscal releasing procedures may not be necessary after TPLO.[89]

In the above literature review it can be seen that much variation exists between testing methods. The stifle was held at 105 °, 130°, 135°, and 145° of extension. This undoubtedly has some effect on biomechanical studies. The consensus from kinematic studies is that the normal stance angle of the stifle in a walking dog is 135°.[3,143] This normal angulation of the femorotibial joint was confirmed in radiographic studies.[154,155] The amount of axial force applied to cadaver limbs was also noted to vary greatly in previous studies: 20% of body weight, 30% of body weight, and 22 N. In other studies specific loading of the CCL in cranial and caudal directions was utilized at forces from 40 to 80 N. This lack of standardized values in individual experiments makes it difficult to compare results between studies.

2. Human Tibial Tuberosity Advancement

Contraction of the human quadriceps was shown to increase tension on ACL reconstructed grafts at flexion angles of 5 to 80°, but reduce tension with further flexion.[119] Maquet theorized that as the quadriceps contracts, the patellar ligament is pulled taut, which increases the force between the patella and the trochlear groove of the femur.[97] By reducing this force, patellofemoral pain may also be reduced. When the tibial tuberosity, or distal end of the patellar ligament, is moved cranially, the efficiency of the quadriceps muscle is increased, so it can do the same work with less effort.[97] Based on theoretical modeling, Maquet proposed that a 20 mm advancement would reduce the patellofemoral force by 50% at the beginning of weight bearing. An early assessment study showed that knee extension strength improved at a mean of 20 months postoperatively.[156]

In a study using human cadaver knees, the authors confirmed that as the tibial tuberosity was moved cranially, the patella was consequently shifted distally as a function of TTA distance and length of tibial osteotomy.[157] They also showed a significant reduction in the force between the patella and femur, which generally decreased as TTA length increased and as angle of knee flexion increased. The authors concluded that 10 mm TTA was sufficient to reduce patellofemoral forces.[157] Another study using mathematical calculations based on radiographic anatomy concluded that TTA increased the moment arm of the patellar ligament and reduced patellofemoral forces by increasing the efficiency of the quadriceps muscle.[158]

Following earlier supportive biomechanical studies of Maquet's TTA, descriptions refuting benefits were published. A study using a contact pressure film measurement device, revealed that in normal cadaver knees, no significant reduction in patellofemoral force was noted after TTA.[159] In a human postoperative study, the quadriceps lever arm was increased by 20%, however, the patellofemoral force was actually increased in most patients despite improvement in mobility and relief of pain.[160] One study suggested a reduction in patellofemoral force up to 20%, but only with less than 20° of flexion, while another reported a reduction in patellofemoral force by 30% in the operated knee compared to the contralateral knee.[161,162]

A recent report used a three dimensional mathematical model of the knee to evaluate the effect of TTA on patellofemoral force and force on the ACL.[163] Advancement of 25 mm resulted in a reduction of patellofemoral force by 78% at full extension. This reduction decreased as the knee was flexed to about 11% at 90° flexion. This was the first study evaluating the effect of TTA on ACL biomechanics in humans. Results revealed a reduction from 143 N to 32 N of force on the ACL at full extension.[163] At large flexion angles, the posterior cruciate was increasingly subjected to force following a 25 mm TTA.

3. Canine Tibial Tuberosity Advancement

Tepic applied the theoretical model of the Maquet procedure to the dog stifle and introduced the tibial tuberosity advancement.[92,94] The TTA, theoretically, limits cranial tibial translation by increasing the lever arm of the quadriceps muscle to resist CTT. There is no expectation that TTA would control excessive internal rotation or hyperextension. 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. The sum of all forces acting in the plane of the neutral axis would be balanced, which would preclude tibial motion in the cranial to caudal plane (Figure 2). The neutral axis is perpendicular to the tibial plateau.

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 broken down into the proximodistal vector and the craniocaudal vector. As seen in Figure 2, 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. If the patellar ligament is changed to be parallel to the neutral axis, the total joint force will occur in the same plane as the neutral force, thereby eliminating any unbalanced cranial vector, resulting in no cranial tibial translation (Figure 3). The force resulting in CTT is greatest with the stifle in full extension, and as the stifle is flexed this cranially directed force decreases until it is near zero at the point of flexion where the patellar ligament is positioned perpendicular to the tibial plateau.[164] In a radiographic study of canine stifles, at a stifle angle of 90° of flexion a crossover point is present. A cranially directed force on the tibia was present in extension up to 90° of flexion and beyond this point the force on the tibia from the quadriceps muscle should be directed caudally.[164] At this crossover point, the patellar ligament was noted to be perpendicular to the tibial plateau. If the patellar ligament can be moved to perpendicular to the plateau while the stifle is at a standing angle of 135° of extension, then any cranially directed force would be neutralized, however based on these data, movement of the patellar ligament past perpendicular may subject the stifle to a caudally directed force as is noted in TPLO with to less than 6.5° of leveling.[151] A small, but significant, difference in the angle between the patellar ligament and the tibial plateau was noted in one study comparing dogs with partial CCL ruptures to normal stifles, which may support this theory.[165]

The distance of advancement is therefore chosen to place 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.[94,95] An alternate theory for the method of action is shifting the cranial translation into a caudal translation, as in the tibial plateau leveling osteotomy.[54,151] Another interpretation is that at 135° of extension advancement of the tuberosity 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.[92] What happens at stifle angles other than 135° was not considered by the TTA theory, but the angle and moment of the quadriceps muscle group are likely to be changed as flexion of the joint occurs.

After completion of the described experiment below, a report evaluating the effect of TTA on CTT was published.[166] In this cadaver study, turnbuckles were used to simulate the quadriceps and gastrocnemius muscles to secure the stifle at 135°. An axial force of 30% body weight was applied while CTT was measured off of radiographs. CTT in the intact specimens was not reported, and CTT after CCL transection was 11 mm while the angle between the patellar ligament and tibial plateau (PTA) changed from 106° to 80°.[166] A variable distance of TTA was performed in order to elucidate the PTA at which CTT was zero with axial loading. At a PTA of 90°, CTT was 2.3 mm despite stabilization of the joint; this occurred at a mean TTA of 10 mm.[166] With further TTA beyond this point, the tibia was translated caudally. The results of this study seemed to support that TTA can eliminate CTT in CCL deficient stifles in their model.

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

A: Objectives

The purpose of this in vitro study was to assess the ability of the TTA to reduce CTT in cadaveric canine stifles. The null hypothesis was that the CTT in the CCL deficient stifle would be no different to the 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. The amount of force require to cause CTT was also investigated.

B: Materials and Methods

1. Specimen Collection and Preparation

Hindlimbs of six large breed dogs (26-31kg) euthanized for reasons unrelated to this study were harvested at the coxofemoral joint, wrapped in moist saline, and stored in sealed plastic at -70º C until the day of testing. The limbs were subjected to an overnight thaw in a 5º C water bath prior to testing. Skin and soft tissues were removed sparing the patella, patella tendon, and the 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 measurements were made using a previously described method.[58]

2. Testing Device

The limb was placed in a custom built jig to secure the stifle at 135º (Figure 4). 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 as to return the limb to the same position between testing scenarios. A 2.4 mm Kirschner wire was placed transversely through a pre-drilled hole in the center of the patella. Braided wire, with loops secured 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 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.

3. Specimen Testing

In the first part of the experiment 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 force equal to 20% bw was directed axially up the distal tibia, while a force equal to 10% body weight was placed on the patella, through the pulley system, pulling it proximally. During pilot studies using 20% bw on the tibia, and a 10% bw on the patella, these allowed cranial tibial translation in unstabilized CCL deficient specimens.

The CTT distance was measured five separate times with axial and quadriceps loads applied to a stifle with an intact CCL (iCCL), following 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 (Figure 5) 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 (Figure 6). CTT was measured as above.

For the second part of the experiment, after each set of CTT measurements using the dial indicator, the force required to elicit CTT in the tCCL with quadriceps load and 9mm TTA specimens was measured using a material testing machine and load cell (MTS Systems Corp., Model 661.11B-02. Minneapolis, MN) (Figure 7). The machine applied a load through a metal rod to the caudo-proximal tibial bone in a cranial direction at 1 mm/sec up to 100 newtons. In the tCCL and TTA groups the force and distance were simultaneously recorded as the machine applied the load cranially. In both cruciate ligament scenarios, 6 sets of axial loads were placed on the tibia, 1, 2, 3, 4, 4.54, and 5.54 kg (10 to 54 N), and data collected for each of the subsequent 12 configurations. Recalibration to zero mm and zero newtons was performed between each trial following patella and axial load application. The test was terminated in each situation when the testing machine recorded 100 newtons.

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

4. Statistical Analysis

CTT means in the iCCL, tCCL, and TTA specimens were compared using two-way analysis of variance (ANOVA) and post hoc Tukey's test. Mean force required for CTT in the tCCL and TTA groups, and mean force noted with increasing axial loads, were compared using the Mixed procedure of SAS statistical software (SAS version 9.1.3, Cary NC). Subsequently, a mixed effects analysis of variance model was fitted to the data with treatment, weight, and distance as class variables (fixed effects), and specimen as a random effect. Associations between dial indicator CTT measurements and potting material angles were assessed using bivariate scatter plots followed by non-parametric Spearman correlation coefficients. Significance was set at $p<0.05$.

C: RESULTS

The mean CTT for the twelve 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 (Table 1). The mean CTT for the tCCL stifles with the patella load was 1.58 mm (range 0.55 to 7.35 mm). With no patella load CTT was 2.59 +/- 2.07 mm (Table 2). After the 9 mm TTA was performed, the mean CTT was 1.06 +/- 0.55 mm. The difference between the mean iCCL and tCCL was significant ($p<0.0001$) as was the difference between the tCCL and the TTA ($p=0.0003$). No significant difference was found between the tCCL with and without a quadriceps load ($p=0.0597$). Though, TTA reduced CTT, there was a significant difference between the TTA and the iCCL, showing that the TTA did not eliminate CTT entirely ($p=0.0428$).

Results of the force/displacement data acquired from the materials testing machine revealed that an increased amount of force was necessary in the TTA as compared to the tCCL to create up to 6 mm of CTT (least squares mean difference in the force was 0.260 newtons, [95% CI: 0.078 to 0.533]) ($p<0.0001$) (Table 3). In the first hindlimb tested, test malfunction precluded accurate data collection, so only 11 legs were used for the force/displacement data. The end point of 6 mm was chosen as, it was the largest distance to which all 11 specimens moved. Additionally, as the axial load on the tibia increased, the force required to induce CTT showed some fluctuations in both tCCL and TTA but with an overall upward trend ($p=0.0002$) (Table 4).

Each stifle was explored following testing, and all caudal cruciate ligaments, menisci, and collateral ligaments were intact. The mean angle of the femur to the potting material was 89º +/-2, while the angle of the tibia in the pot was 91º +/-3. Preoperative mean tibial plateau angle was 19º +/-3. No association between these variables and the CTT was noted in the analysis (All p values were greater than 0.05)

D: DISCUSSION

Cranial tibial translational distance, as measured by the dial indicator, was shown to be statistically significantly reduced by the advancement of the tibial tuberosity 9 mm in this experimental model when TTA was compared to the tCCL. However, the 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 this model. The individual stifle tibial plateau angle and the angle of the bone to the potting material did not seem to relate to the variation seen, though due to 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 force/displacement data from this study demonstrated that a statistically significant increased amount of force was required to produce CTT in the TTA specimens when compared to the tCCL specimens. This suggests that the advancement of the tuberosity does modify the quadriceps mechanism's ability to restrain CTT. With the knowledge that the TTA increases the lever arm of the patellar ligament[97], it is assumed that the TTA does accomplish controlling CTT by increasing the effectiveness of the quadriceps force. At no time during loading of the stifle was there indication of caudal tibial translation as is described in the TPLO, though only a 9 mm advancement was performed.[54,151] Overall as the amount of axial load increased, in both TTA and tCCL, the amount of force necessary to cause CTT increased. As TTA did not seem to induce a caudal tibial translation in this study, the caudal cruciate ligament should not be subject to undue stress, unlike other repair methods, though a future study could include testing this specifically with a strain gauge.[54,151]

The quadriceps muscle force is integral to hindlimb weight bearing, providing not only extension of the stifle, but also aiding in stifle joint stability.[167] The TTA theory proposes that active contraction of the quadriceps muscle is necessary to achieve CTT control.[168] The actual force exerted by the quadriceps muscle is unknown in dogs, but logically varies with the body weight, phase of weight bearing, and speed of locomotion. Quadriceps force can be estimated experimentally[169-171] 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 newtons 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 used in this study may not, by itself, contribute to controlling CTT; though the p value approached significance.

The method of providing the quadriceps force in this experiment was to use a pin driven transversely through the patella attached to strong braided wire to a pulley system on which know masses were placed. This was an economical method, however the precision of a tensioning device set to a specific force would have been useful. The potentiometer used in another recent canine study may have allowed for more precision and control as well.[152] The method of applying the force to elicit CTT from the material testing machine could be improved. The force was applied via a long, thin, metal rod with a tapered end. This tapered end at times would slide distally along the caudal tibia and may have also induced some rotation during force application. An alternate method would be to use a flat plate on the end of the testing machine or apply the force through a metal implant placed in the bone. This force could also be adjusted to pull cranially instead of push caudally to measure the CTT from the machine.

The stifles used in this study were tested after being thawed from -70ºC. Ideally, specimens would be tested immediately after euthanasia, but this is often not feasible. Storage temperatures ranging from 4ºC to -80ºC have been shown to have minimal negative biomechanical effects on ligaments, but no ideal storage temperature has been identifed.[172-174] 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.

The tibial tuberosity advancement was first described in humans for the treatment of pain originating from increased pressure between the patella and the femoral trochlear.[97] By advancing the tuberosity, the patellofemoral compressive force should be reduced, and the anteriorly displaced patella ligament acts as a longer lever arm, thereby increasing the efficiency of the quadriceps muscle.[97] Biomechanical analysis of the procedure revealed that TTA did reduce patellofemoral forces in humans, while the patella was routinely moved into more of a patella baja position.[157] Improvement in pain was noted by most (80-95%) people[97,175], but long term results were not as satisfactory in some follow up studies.[98,106]

In canines, the TTA is used for an entirely different pathologic condition. In CCL deficient dogs, the TTA is used to control CTT by reducing the cranial portion of the force vector sum, by moving the total joint force into a neutral position. Another method by which TTA could accomplish this is by increasing the lever arm of the quadriceps mechanism. A reduction in patellofemoral pressure was described to be a benefit of the TTA in dogs thereby reducing articular chondromalacia, but this has yet to be proven.[94] If this reduced pressure and shifting the patella distally occurs in dogs as in humans, it may be of detriment by predisposing these dogs to patella luxation (which has been reported[176]), though, conversely it was reported that a majority of large breed dogs with patella luxation actually have patella alta.[177]

Limitations to this study included the inability of this model to mimic the 10mm amount of CTT noted in live dog experimental studies.[3,150] The magnitude of CTT elicited in the first part of this study was lower than some other in vitro studies testing CTT (4.7 to 19mm).[41,142,151,152] This may be due to 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 tibial plateau angle or perpendicularity of the bone in the potted material could be found. The tibial plateau was measured using the center of the proximal tibial shaft because measurements were taken after potting in most specimens.[58] This precluded the Slocum method of measurement where the talus is used as a distal reference point. This difference may have contributed a minor change in mean pre-testing tibial plateau angles, and radiographs made prior to soft tissue removal and any bony osteotomies could be used in future projects. Rotation of the tibia may have played a role in the measurements obtained for CTT, but since recording of the degree of this motion was not performed, its significance in not known. Future studies could examine the change in tibial rotation with the CCL intact, severed, and after a TTA during loading of the tibia as in Harper, et al.[41]

Shifting of the tibia within the potting material was not noted either during specimen loading or on sample post-testing radiographs. Polyester resin was used in the current protocol for securing the bone to the testing jig. Many previous studies have used polymethylmethacrylate as a potting material.[28,147] Expense has led investigators to use alternate potting materials such as browncast[136], or elimination of potting and use of screws, pins, or other structures.[89,129,153,166] A previous group from the institution where the current experiment was performed used a similar resin with a good outcome.[41]

The removal of all structures superficial to the stifle joint capsule and collateral ligaments was performed to reduce sources of variation in the data obtained. An alternate method of testing could utilize the entire hind limb with the foot and calcaneal mechanism intact, which may portray in vivo anatomy better, but also, may introduce more uncontrolled variables.[88,89,153] The TTA theory suggests that the forces beyond the quadriceps can be fully accounted for by using the total force acting around the stifle, and this is how testing was done in this experimental model. None of the cadavers used in this study had any 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 utilized 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.

E: CONCLUSIONS

In conclusion, the ability of TTA to limit CTT during simulated weight bearing in this in vitro model was documented, although with amounts of CTT less than that seen in clinical cases. 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.

REFERENCES

1. Hayashi K, Manley PA, Muir P. Cranial cruciate ligament pathophysiology in dogs with cruciate disease: a review. *J Am Anim Hosp Assoc* 2004;40:385-390.

2. Wilke VL, Robinson DA, Evans RB, et al. Estimate of the annual economic impact of treatment of cranial cruciate ligament injury in dogs in the United States. *J Am Vet Med Assoc* 2005;227:1604-1607.

3. Korvick DL, Pijanowski GJ, Schaeffer DJ. Three-dimensional kinematics of the intact and cranial cruciate ligament-deficient stifle of dogs. *J Biomech* 1994;27:77-87.

4. Briggs KK. The canine meniscus: Injury and treatment. *Compend Contin Educ Pract Vet* 2004;26:687-696.

5. Vasseur PB. Clinical results following nonoperative management for rupture of the cranial cruciate ligament in dogs. *Vet Surg* 1984;13:243-246.

6. Chauvet AE, Johnson AL, Pijanowski GJ, et al. Evaluation of fibular head transposition, lateral fabellar suture, and conservative treatment of cranial cruciate ligament rupture in large dogs: a retrospective study. *J Am Anim Hosp Assoc* 1996;32:247-255.

7. Hoelzler MG, Millis DL, Francis DA, et al. Results of arthroscopic versus open arthrotomy for surgical management of cranial cruciate ligament deficiency in dogs. *Vet Surg* 2004;33:146-153.

8. Dupuis J, Harari J. Cruciate ligament and meniscal injuries in dogs. *Compend Contin Educ Pract Vet* 1993;15:215-232.

9. Elkins AD, Pechman R, Kearney MT, et al. A retrospective study evaluating the degree of degenerative joint disease in the stifle joint of dogs following surgical repair of anterior cruciate ligament rupture. *J Am Anim Hosp Assoc* 1991;27:533-540.

10. Innes JF, Costello M, Barr FJ, et al. Radiographic progression of osteoarthritis of the canine stifle joint: a prospective study. *Vet Radiol Ultrasound* 2004;45:143-148.

11. Marsolais GS, McLean S, Derrick T, et al. Kinematic analysis of the hind limb during swimming and walking in healthy dogs and dogs with surgically corrected cranial cruciate ligament rupture. *J Am Vet Med Assoc* 2003;222:739-743.

12. Lampman T, Lund E, Lipowitz A. Cranial cruciate disease: current status of diagnosis, surgery, and risk of disease. *Vet Comp Orthop Traumatol* 2003;16:122-126.

13. Jerram RM, Walker AM. Cranial cruciate ligament injury in the dog: pathophysiology, diagnosis and treatment. *N Z Vet J* 2003;51:149-158.

14. Paatsama S. Ligament injuries of the canine stifle joint: A clinical and experimental study. Helsinki, 1952.

15. Moore KW, Read RA. Rupture of the cranial cruciate ligament in dogs. Part I. *Compend Contin Educ Pract Vet* 1996;18:223-233.

16. Dueland R. A recent technique for reconstruction of the anterior cruciate ligament. *J Am Anim Hosp Assoc* 1966;2:1-5.

17. Dickinson CR, Nunamaker DM. Repair of ruptured anterior cruciate ligament in the dog: Experience of 101 cases using a modified fascia strip technique. *J Am Vet Med Assoc* 1977;170:827-830.

18. Arnoczky SP, Tarvin GB, Marshall JL, et al. The over-the-top procedure: A technique for anterior cruciate ligament stabilization in the dog. *J Am Anim Hosp Assoc* 1979;15:283-290.

19. Shires PK, Hulse DA, Liu W. The under-and-over fascial replacement technique for anterior cruciate ligament rupture in dogs: A retrospective study. *J Am Anim Hosp Assoc* 1984;20:69-77.

20. Hulse DA, Michaelson F, Johnson C, et al. A technique for reconstruction of the anterior cruciate ligament in the dog: Preliminary report. *Vet Surg* 1980;9:135-140.

21. Lopez MJ, Markel MD, Kalscheur V, et al. Hamstring graft technique for stabilization of canine cranial cruciate ligament deficient stifles. *Vet Surg* 2003;32:390-401.

22. Butler HC. Teflon as a prosthetic ligament in repair of anterior cruciate ligaments. *Am J Vet Res* 1964;25:55-60.

23. Denny HR, Goodship AE. Replacement of the anterior cruciate ligament with carbon fiber in the dog. *J Small Anim Pract* 1980;21.

24. Vaughan LC, Bowden NLR. The use of skin for replacement of the anterior cruciate ligament in the dog: A review of 30 cases. *J Small Anim Pract* 1964;5:167-171.

25. Hinko PJ. The use of prosthetic ligament in repair of torn anterior cruciate ligament in a dog. *J Am Anim Hosp Assoc* 1981;17.

26. Arnoczky SP, Warren RF, Ashlock MA. Replacement of the anterior cruciate ligament using a patellar tendon allograft. An experimental study. *J Bone Joint Surg Am* 1986;68:376-385.

27. Hulse DA, Butler DL, Kay MD. Biomechanics of cranial cruciate ligament reconstruction in the dog: I. In vitro laxity testing. *Vet Surg* 1983;12:109-112.

28. Butler DL, Hulse DA, Kay MD. Biomechanics of cranial cruciate ligament reconstruction in the dog: II. Mechanical properties. *Vet Surg* 1983;12:113-118.

29. Moore KW, Read RA. Rupture of the cranial cruciate ligament in dogs. Part II: Diagnosis and management. *Compend Contin Educ Pract Vet* 1996;18:381-391.

30. Innes JF, Bacon D, Lynch C, et al. Long-term outcome of surgery for dogs with cranial cruciate ligament deficiency. *Vet Rec* 2000;147:325-328.

31. Conzemius MG, Evans RB, Besancon MF, et al. Effect of surgical technique on limb function after surgery for rupture of the cranial cruciate ligament in dogs. *J Am Vet Med Assoc* 2005;226:232-236.

32. Hohn RB, Miller JM. Surgical correction of rupture of the anterior cruciate ligament in the dog. *J Am Vet Med Assoc* 1967;150:1133-1141.

33. DeAngelis M, Lau RE. A lateral retinacular imbrication technique for the surgical correction of anterior cruciate ligament rupture in the dog. *J Am Vet Med Assoc* 1970;157:79-84.

34. Flo GL. Modification of the lateral retinacular imbrication technique for stabilizing cruciate ligament injuries. *J Am Anim Hosp Assoc* 1975;11:570-576.

35. Gambardella PC, Wallace LJ, Cassidy F. Lateral suture technique for management of anterior cruciate ligament rupture in dogs: A retrospective study. *J Am Anim Hosp Assoc* 1981;17:33-38.

36. Dulisch ML. Suture reaction following extra-articular stifle stabilization in the dog: I. A retrospective study of 161 cases. *J Am Anim Hosp Assoc* 1981;17:569-571.

37. Vasseur PB. Stifle joint In: Slatter D, ed. *Textbook of Small Animal Surgery*. 3rd ed. Philadelphia: Saunders, 2003;2090-2133.

38. Sicard GK, Meinen J, Phillips T, et al. Comparison of fishing line for repair of the cruciate deficient stifle. *Vet Comp Orthop Traumatol* 1999;12:138-141.

39. Stork CK, Gibson NR, Owen MR, et al. Radiographic features of a lateral extracapsular wire suture in the canine cranial cruciate deficient stifle. *J Small Anim Pract* 2001;42:487-490.

40. Banwell MN, Kerwin SC, Hosgood G, et al. In vitro evaluation of the 18 and 36 kg Securos Cranial Cruciate Ligament Repair System. *Vet Surg* 2005;34:283-288.

41. Harper TA, Martin RA, Ward DL, et al. An in vitro study to determine the effectiveness of a patellar ligament/fascia lata graft and new tibial suture anchor points

for extracapsular stabilization of the cranial cruciate ligament-deficient stifle in the dog. *Vet Surg* 2004;33:531-541.

42. Prostrendy JM, Bauer MS, Blevins WE, et al. Effect of suture type on stifle joint biomechanics after extra-articular repair of cranial cruciate ligament transection in the dog. *Vet Comp Orthop Traumatol* 1991;4:144-149.

43. Guenego L, Zahra A, Madelenat A, et al. Cranial cruciate ligament rupture in large and giant dogs. A retrospective evaluation of a modified lateral extracapsular stabilization. *Vet Comp Orthop Traumatol* 2007;20:43-50.

44. Singer MJ, Pijanowski G, Wiley R, et al. Biomechanical evaluation of a veterinary suture anchor in the canine cadaver pelvis and femur. *Vet Comp Orthop Traumatol* 2005;18:31-36.

45. Vianna ML, Roe SC. Mechanical comparison of two knots and two crimp systems for securing nylon line used for extra-articular stabilization of the canine stifle. *Vet Surg* 2006;35:567-572.

46. Moores AP, Beck AL, Jespers KJ, et al. Mechanical evaluation of two crimp clamp systems for extracapsular stabilization of the cranial cruciate ligament-deficient canine stifle. *Vet Surg* 2006;35:470-475.

47. Pearson PT, McCurnin DM, Carter JD, et al. Lembert suture technique to surgically correct ruptured cruciate ligaments. *J Am Anim Hosp Assoc* 1971;7:1-13.

48. Hohn RB, Newton CD. Surgical repair of ligamentous structures of the stifle joint. In: Bojrab MJ, ed. *Current Techniques in Small Animal Surgery*. Philadelphia: Lea & Febiger, 1975.

49. Smith GK, Torg JS. Fibular head transposition for repair of cruciate-deficient stifle in the dog. *J Am Vet Med Assoc* 1985;187:375-383.

50. Mullen HS, Matthiesen DT. Complications of transposition of the fibular head for stabilization of the cranial cruciate-deficient stifle in dogs: 80 cases (1982-1986). *J Am Vet Med Assoc* 1989;195:1267-1271.

51. Slocum B, Slocum TD. Tibial plateau leveling osteotomy for repair of cranial cruciate ligament rupture in the canine. *Vet Clin North Am Small Anim Pract* 1993;23:777-795.

52. Slocum B, Devine T. Cranial tibial wedge osteotomy: a technique for eliminating cranial tibial thrust in cranial cruciate ligament repair. *J Am Vet Med Assoc* 1984;184:564-569.

53. Morris E, Lipowitz AJ. Comparison of tibial plateau angles in dogs with and without cranial cruciate ligament injuries. *J Am Vet Med Assoc* 2001;218:363-366.

54. Reif U, Probst CW. Comparison of tibial plateau angles in normal and cranial cruciate deficient stifles of Labrador retrievers. *Vet Surg* 2003;32:385-389.

55. Venzin C, Howard J, Rytz U, et al. Tibial plateau angles with and without cranial cruciate ligament rupture: Comparison between different dog populations and a wolf population. *Vet Comp Orthop Traumatol* 2004;17:232-236.

56. Caylor KB, Zumpano CA, Evans LM, et al. Intra- and interobserver measurement variability of tibial plateau slope from lateral radiographs in dogs. *J Am Anim Hosp Assoc* 2001;37:263-268.

57. Baroni E, Matthias RR, Marcellin-Little DJ, et al. Comparison of radiographic assessments of the tibial plateau slope in dogs. *Am J Vet Res* 2003;64:586-589.

58. Abel SB, Hammer DL, Shott S. Use of the proximal portion of the tibia for measurement of the tibial plateau angle in dogs. *Am J Vet Res* 2003;64:1117-1123.

59. Fettig AA, Rand WM, Sato AF, et al. Observer variability of tibial plateau slope measurement in 40 dogs with cranial cruciate ligament-deficient stifle joints. *Vet Surg* 2003;32:471-478.

60. Odders JW, Jessen CR, Lipowitz AJ. Sequential measurements of the tibial plateau angle in large-breed, growing dogs. *Am J Vet Res* 2004;65:513-518.

61. Reif U, Dejardin LM, Probst CW, et al. Influence of limb positioning and measurement method on the magnitude of the tibial plateau angle. *Vet Surg* 2004;33:368-375.

62. Zeltzman PA, Pare B, Johnson GM, et al. Relationship between age and tibial plateau angle in dogs with cranial cruciate rupture. *J Am Anim Hosp Assoc* 2005;41:117-120.

63. Grierson J, Sanders M, Guitan J, et al. Comparison of anatomical tibial plateau angle versus observer measurement from lateral radiographs in dogs. *Vet Comp Orthop Traumatol* 2005;18:215-219.

64. Ritter MJ, Perry RL, Olivier NB, et al. Tibial plateau symmetry and the effect of osteophytosis on tibial plateau angle measurements. *J Am Anim Hosp Assoc* 2007;43:93-98.

65. Wheeler JL, Cross AR, Gingrich W. In vitro effects of osteotomy angle and osteotomy reduction on tibial angulation and rotation during the tibial plateau-leveling osteotomy procedure. *Vet Surg* 2003;32:371-377.

66. Hildreth BE, Marcellin-Little DJ, Roe SC, et al. In vitro evaluation of five canine tibial plateau leveling methods. *Am J Vet Res* 2006;67:693-700.

67. Budsberg SC, Verstraete MC, Soutas-Little RW, et al. Force plate analyses before and after stabilization of canine stifles for cruciate injury. *Am J Vet Res* 1988;49:1522-1524.

68. Jevens DJ, DeCamp CE, Hauptman J, et al. Use of force-plate analysis of gait to compare two surgical techniques for treatment of cranial cruciate ligament rupture in dogs. *Am J Vet Res* 1996;57:389-393.

69. Evans R, Horstman C, Conzemius M. Accuracy and optimization of force platform gait analysis in Labradors with cranial cruciate disease evaluated at a walking gait. *Vet Surg* 2005;34:445-449.

70. Ballagas AJ, Montgomery RD, Henderson RA, et al. Pre- and postoperative force plate analysis of dogs with experimentally transected cranial cruciate ligaments treated using tibial plateau leveling osteotomy. *Vet Surg* 2004;33:187-190.

71. Robinson DA, Mason DR, Evans R, et al. The effect of tibial plateau angle on ground reaction forces 4-17 months after tibial plateau leveling osteotomy in Labrador Retrievers. *Vet Surg* 2006;35:294-299.

72. Lazar TP, Berry CR, deHaan JJ, et al. Long-term radiographic comparison of tibial plateau leveling osteotomy versus extracapsular stabilization for cranial cruciate ligament rupture in the dog. *Vet Surg* 2005;34:133-141.

73. Lineberger JA, Allen DA, Wilson ER, et al. Comparison of radiographic arthritic changes associated with two variations of tibial plateau leveling osteotomy. *Vet Comp Orthop Traumatol* 2005;18:13-17.

74. Rayward RM, Thomson DG, Davies JV, et al. Progression of osteoarthritis following TPLO surgery: a prospective radiographic study of 40 dogs. *J Small Anim Pract* 2004;45:92-97.

75. Stauffer KD, Tuttle TA, Elkins AD, et al. Complications associated with 696 tibial plateau leveling osteotomies (2001-2003). *J Am Anim Hosp Assoc* 2006;42:44-50.

76. Priddy NH, 2nd, Tomlinson JL, Dodam JR, et al. Complications with and owner assessment of the outcome of tibial plateau leveling osteotomy for treatment of cranial cruciate ligament rupture in dogs: 193 cases (1997-2001). *J Am Vet Med Assoc* 2003;222:1726-1732.

77. Pacchiana PD, Morris E, Gillings SL, et al. Surgical and postoperative complications associated with tibial plateau leveling osteotomy in dogs with cranial

cruciate ligament rupture: 397 cases (1998-2001). *J Am Vet Med Assoc* 2003;222:184-193.

78. Barnhart MD. Results of single-session bilateral tibial plateau leveling osteotomies as a treatment for bilaterally ruptured cranial cruciate ligaments in dogs: 25 cases (2000-2001). *J Am Anim Hosp Assoc* 2003;39:573-578.

79. Kergosien DH, Barnhart MD, Kees CE, et al. Radiographic and clinical changes of the tibial tuberosity after tibial plateau leveling osteotomy. *Vet Surg* 2004;33:468-474.

80. Mattern KL, Berry CR, Peck JN, et al. Radiographic and ultrasonographic evaluation of the patellar ligament following tibial plateau leveling osteotomy. *Vet Radiol Ultrasound* 2006;47:185-191.

81. Carey K, Aiken SW, DiResta GR, et al. Radiographic and clinical changes of the patellar tendon after tibial plateau leveling osteotomy 94 cases (2000-2003). *Vet Comp Orthop Traumatol* 2005;18:235-242.

82. Boudrieau RJ, McCarthy RJ, Sisson RD, Jr. Sarcoma of the proximal portion of the tibia in a dog 5.5 years after tibial plateau leveling osteotomy. *J Am Vet Med Assoc* 2005;227:1613-1617, 1591.

83. Charles AE, Ness MG. Crevice corrosion of implants recovered after tibial plateau leveling osteotomy in dogs. *Vet Surg* 2006;35:438-444.

84. Boudrieau RJ, McCarthy RJ, Sprecher CM, et al. Material properties of and tissue reaction to the Slocum TPLO plate. *Am J Vet Res* 2006;67:1258-1265.

85. Ralphs SC, Whitney WO. Arthroscopic evaluation of menisci in dogs with cranial cruciate ligament injuries: 100 cases (1999-2000). *J Am Vet Med Assoc* 2002;221:1601-1604.

86. Messmer M, Schmokel H, Schawalder P. Intraarticular measurement of forces acting on the canine medial meniscus during motion. *Vet Comp Orthop Traumatol* 2001;14:133-138.

87. Slocum B, Slocum TD. Meniscal release In: Bojrab MJ, ed. *Current Techniques in Small Animal Surgery*. 4th ed. Baltimore: Williams & Wilkins, 1998;1197-1199.

88. Kennedy SC, Dunning D, Bischoff MG, et al. The effect of axial and abaxial release on meniscal displacement in the dog. *Vet Comp Orthop Traumatol* 2005;18:227-234.

89. Pozzi A, Kowaleski MP, Apelt D, et al. Effect of medial meniscal release on tibial translation after tibial plateau leveling osteotomy. *Vet Surg* 2006;35:486-494.

90. Thieman KM, Tomlinson JL, Fox DB, et al. Effect of meniscal release on rate of subsequent meniscal tears and owner-assessed outcome in dogs with cruciate disease treated with tibial plateau leveling osteotomy. *Vet Surg* 2006;35:705-710.

91. Damur DM, Tepic S, Montavon PM. Proximal tibial osteotomy for the repair of cranial cruicate-deficient stifle joints in dogs. *Vet Comp Orthop Traumatol* 2003;16:211-216.

92. Tepic S, Damur D, Montavon P. Biomechanics of the stifle joint. Proceedings of the 1st World Orthopaedic Veterinary Congress 2002;189-190.

93. Jerram RM, Walker AM, Warman CG. Proximal tibial intraarticular ostectomy for treatment of canine cranial cruciate ligament injury. *Vet Surg* 2005;34:196-205.

94. Montavon PM, Damur DM, Tepic S. Advancement of the tibial tuberosity for the treatment of cranial cruciate deficient canine stifle. Proceedings of the 1st World Orth Vet Congress 2002;152.

95. Montavon P, Damur D, Tepic S. Tibial tuberosity advancement (TTA) for the treatment of cranial cruciate disease in dogs: evidences, technique and initial clinical results. Proceedings of the 12th ESVOT Congress 2004;254-255.

96. Hoffmann DE, Miller JM, Ober CP, et al. Tibial tuberosity advancement in 65 canine stifles. *Vet Comp Orthop Traumatol* 2006;19:219-227.

97. Maquet P. Advancement of the tibial tuberosity. *Clin Orthop* 1976:225-230.

98. Engebretsen L, Svenningsen S, Benum P. Advancement of the tibial tuberosity for patellar pain. A 5-year follow-up. *Acta Orthop Scand* 1989;60:20-22.

99. Maquet P. Un traitement de l'arthrose femoropatellaire. *Rev Rhum Mal Osteoartic* 1963;30:225-230.

100. Mendes DG, Soudry M, Iusim M. Clinical assessment of Maquet tibial tuberosity advancement. *Clin Orthop Relat Res* 1987:228-238.

101. Hirsh DM, Reddy DK. Experience with Maquet anterior tibial tubercle advancement for patellofemoral arthralgia. *Clin Orthop Relat Res* 1980:136-139.

102. Schmid F. The Maquet procedure in the treatment of patellofemoral osteoarthrosis. Long-term results. *Clin Orthop Relat Res* 1993:254-258.

103. Sudmann E, Salkowitsch B. Anterior displacement of the tibial tuberosity in the treatment of chondromalacia patellae. *Acta Orthop Scand* 1980;51:171-174.

104. Hejgaard N, Watt-Boolsen S. The effect of anterior displacement of the tibial tuberosity in idiopathic chondromalacia patellae: a prospective randomized study. *Acta Orthop Scand* 1982;53:135-139.

105. Sakai N, Koshino T, Okamoto R. Pain reduction after anteromedial displacement of the tibial tuberosity: 5-year follow-up in 21 knees with patellofemoral arthrosis. *Acta Orthop Scand* 1996;67:13-15.

106. Karlsson J, Sward L, Lansinger O. Bad results after anterior advancement of the tibial tubercle for patello-femoral pain syndrome. *Arch Orthop Trauma Surg* 1992;111:195-197.

107. Fulkerson JP, Becker GJ, Meaney JA, et al. Anteromedial tibial tubercle transfer without bone graft. *Am J Sports Med* 1990;18:490-496; discussion 496-497.

108. MacIntyre NJ, Hill NA, Fellows RA, et al. Patellofemoral joint kinematics in individuals with and without patellofemoral pain syndrome. *J Bone Joint Surg Am* 2006;88:2596-2605.

109. Dixit S, DiFiori JP, Burton M, et al. Management of patellofemoral pain syndrome. *Am Fam Physician* 2007;75:194-202.

110. Dejour D, Le Coultre B. Osteotomies in patello-femoral instabilities. *Sports Med Arthrosc* 2007;15:39-46.

111. Koeter S, Diks MJ, Anderson PG, et al. A modified tibial tubercle osteotomy for patellar maltracking: RESULTS AT TWO YEARS. *J Bone Joint Surg Br* 2007;89:180-185.

112. Kumar A, Jones S, Bickerstaff DR, et al. Functional evaluation of the modified Elmslie-Trillat procedure for patello-femoral dysfunction. *Knee* 2001;8:287-292.

113. Post WR. Patellofemoral pain: results of nonoperative treatment. *Clin Orthop Relat Res* 2005:55-59.

114. Jarvela T, Paakkala T, Kannus P, et al. The incidence of patellofemoral osteoarthritis and associated findings 7 years after anterior cruciate ligament reconstruction with a bone-patellar tendon-bone autograft. *Am J Sports Med* 2001;29:18-24.

115. Kartus J, Magnusson L, Stener S, et al. Complications following arthroscopic anterior cruciate ligament reconstruction. A 2-5-year follow-up of 604 patients with special emphasis on anterior knee pain. *Knee Surg Sports Traumatol Arthrosc* 1999;7:2-8.

116. Fulkerson JP. Diagnosis and treatment of patients with patellofemoral pain. *Am J Sports Med* 2002;30:447-456.

117. Woo SL, Thomas M, Chan Saw SS. Contribution of biomechanics, orthopaedics and rehabilitation: the past present and future. *Surgeon* 2004;2:125-136.

118. Beynnon BD, Johnson RJ, Abate JA, et al. Treatment of anterior cruciate ligament injuries, part I. *Am J Sports Med* 2005;33:1579-1602.

119. Takeda Y, Xerogeanes JW, Livesay GA, et al. Biomechanical function of the human anterior cruciate ligament. *Arthroscopy* 1994;10:140-147.

120. Ageberg E. Consequences of a ligament injury on neuromuscular function and relevance to rehabilitation - using the anterior cruciate ligament-injured knee as model. *J Electromyogr Kinesiol* 2002;12:205-212.

121. Griffin LY, Albohm MJ, Arendt EA, et al. Understanding and preventing noncontact anterior cruciate ligament injuries: a review of the Hunt Valley II meeting, January 2005. *Am J Sports Med* 2006;34:1512-1532.

122. Hewett TE, Paterno MV, Myer GD. Strategies for enhancing proprioception and neuromuscular control of the knee. *Clin Orthop Relat Res* 2002:76-94.

123. Segawa H, Omori G, Koga Y. Long-term results of non-operative treatment of anterior cruciate ligament injury. *Knee* 2001;8:5-11.

124. Casteleyn PP, Handelberg F. Non-operative management of anterior cruciate ligament injuries in the general population. *J Bone Joint Surg Br* 1996;78:446-451.

125. Fitzgerald GK, Axe MJ, Snyder-Mackler L. A decision-making scheme for returning patients to high-level activity with nonoperative treatment after anterior cruciate ligament rupture. *Knee Surg Sports Traumatol Arthrosc* 2000;8:76-82.

126. Delay BS, Smolinski RJ, Wind WM, et al. Current practices and opinions in ACL reconstruction and rehabilitation: results of a survey of the American Orthopaedic Society for Sports Medicine. *Am J Knee Surg* 2001;14:85-91.

127. Jaegger G, Marcellin-Little DJ, Levine D. Reliability of goniometry in Labrador Retrievers. *Am J Vet Res* 2002;63:979-986.

128. de Rooster H, de Bruin T, van Bree H. Morphologic and functional features of the canine cruciate ligaments. *Vet Surg* 2006;35:769-780.

129. Vasseur PB, Pool RR, Arnoczky SP, et al. Correlative biomechanical and histologic study of the cranial cruciate ligament in dogs. *Am J Vet Res* 1985;46:1842-1854.

130. Benjamin M, Toumi H, Ralphs JR, et al. Where tendons and ligaments meet bone: attachment sites ('entheses') in relation to exercise and/or mechanical load. *J Anat* 2006;208:471-490.

131. Huss BT, Lattimer JC. What is your diagnosis? Intra-articular avulsion fracture of the left tibia at the insertion of the cranial cruciate ligament. *J Am Vet Med Assoc* 1994;204:1017-1018.

132. Duval JM, Budsberg SC, Flo GL, et al. Breed, sex, and body weight as risk factors for rupture of the cranial cruciate ligament in young dogs. *J Am Vet Med Assoc* 1999;215:811-814.

133. Barrett JG, Hao Z, Graf BK, et al. Inflammatory changes in ruptured canine cranial and human anterior cruciate ligaments. *Am J Vet Res* 2005;66:2073-2080.

134. Danielsson F, Ekman S, Andersson M. Inflammatory response in dogs with spontaneous cranial cruciate ligament rupture. *Vet Comp Orthop Traumatol* 2004;17:237-240.

135. Hayashi K, Frank JD, Dubinsky C, et al. Histologic changes in ruptured canine cranial cruciate ligament. *Vet Surg* 2003;32:269-277.

136. Patterson RH, Smith GK, Gregor TP, et al. Biomechanical stability of four cranial cruciate ligament repair techniques in the dog. *Vet Surg* 1991;20:85-90.

137. Figgie HE, 3rd, Bahniuk EH, Heiple KG, et al. The effects of tibial-femoral angle on the failure mechanics of the canine anterior cruciate ligament. *J Biomech* 1986;19:89-91.

138. Budsberg SC, Verstraete MC, Soutas-Little RW. Force plate analysis of the walking gait in healthy dogs. *Am J Vet Res* 1987;48:915-918.

139. Fanchon L, Valette JP, Sanaa M, et al. The measurement of ground reaction force in dogs trotting on a treadmill: an investigation of habituation. *Vet Comp Orthop Traumatol* 2006;19:81-86.

140. Harari J. Caudal cruciate ligament injury. *Vet Clin North Am Small Anim Pract* 1993;23:821-829.

141. Hart R, Hulse DA, Slater M. Contribution of periarticular tissue to stabilization of the canine stifle joint after cranial cruciate ligament reconstruction. *Vet Comp Orthop Traumatol* 2003;16:21-25.

142. Lopez MJ, Kunz D, Vanderby R, Jr., et al. A comparison of joint stability between anterior cruciate intact and deficient knees: a new canine model of anterior cruciate ligament disruption. *J Orthop Res* 2003;21:224-230.

143. Hottinger HA, DeCamp CE, Olivier NB, et al. Noninvasive kinematic analysis of the walk in healthy large-breed dogs. *Am J Vet Res* 1996;57:381-388.

144. Shahar R, Banks-Sills L. A quasi-static three-dimensional, mathematical, three-body segment model of the canine knee. *J Biomech* 2004;37:1849-1859.

145. Shahar R, Milgram J. Biomechanics of tibial plateau leveling of the canine cruciate-deficient stifle joint: a theoretical model. *Vet Surg* 2006;35:144-149.

146. Yasuda K, Tomita F, Yamazaki S, et al. The effect of growth factors on biomechanical properties of the bone-patellar tendon-bone graft after anterior cruciate ligament reconstruction: a canine model study. *Am J Sports Med* 2004;32:870-880.

147. Tomita F, Yasuda K, Mikami S, et al. Comparisons of intraosseous graft healing between the doubled flexor tendon graft and the bone-patellar tendon-bone graft in anterior cruciate ligament reconstruction. *Arthroscopy* 2001;17:461-476.

148. Yamazaki S, Yasuda K, Tomita F, et al. The effect of intraosseous graft length on tendon-bone healing in anterior cruciate ligament reconstruction using flexor tendon. *Knee Surg Sports Traumatol Arthrosc* 2006;14:1086-1093.

149. Lopez MJ, Hagquist W, Jeffrey SL, et al. Instrumented measurement of in vivo anterior-posterior translation in the canine knee to assess anterior cruciate integrity. *J Orthop Res* 2004;22:949-954.

150. Tashman S, Anderst W, Kolowich P, et al. Kinematics of the ACL-deficient canine knee during gait: serial changes over two years. *J Orthop Res* 2004;22:931-941.

151. Warzee CC, Dejardin LM, Arnoczky SP, et al. Effect of tibial plateau leveling on cranial and caudal tibial thrusts in canine cranial cruciate-deficient stifles: an in vitro experimental study. *Vet Surg* 2001;30:278-286.

152. Reif U, Hulse DA, Hauptman JG. Effect of tibial plateau leveling on stability of the canine cranial cruciate-deficient stifle joint: an in vitro study. *Vet Surg* 2002;31:147-154.

153. Kowaleski MP, Apelt D, Mattoon JS, et al. The effect of tibial plateau leveling osteotomy position on cranial tibial subluxation: an in vitro study. *Vet Surg* 2005;34:332-336.

154. Watson C, Rochat M, Payton M. Effect of weight bearing on the joint angles of the fore- and hind limb of the dog. *Vet Comp Orthop Traumatol* 2003;16:250-254.

155. Milgram J, Slonim E, Kass P, et al. A radiographic study of joint angles in standing dogs. *Vet Comp Orthop Traumatol* 2004;17:82-90.

156. Nordgren B, Nordesjo LO, Rauschning W. Isokinetic knee extension strength and pain before and after advancement osteotomy of the tibial tuberosity. *Arch Orthop Trauma Surg* 1983;102:95-101.

157. Nakamura N, Ellis M, Seedhom BB. Advancement of the tibial tuberosity. A biomechanical study. *J Bone Joint Surg Br* 1985;67:255-260.

158. van Eijden TM, Kouwenhoven E, Weijs WA. The influence of anterior displacement of the tibial tuberosity on patellofemoral biomechanics. *Int Orthop* 1987;11:215-221.

159. Retaillaud JL, Darmana R, Devallet P, et al. [Anterior displacement of the tibial tuberosity. An experimental biomechanical study of its effects on the femoro-patellar joint]. *Chirurgie* 1991;117:737-744.

160. Koshino T. Changes in patellofemoral compressive force after anterior or anteromedial displacement of tibial tuberosity for chondromalacia patellae. *Clin Orthop Relat Res* 1991:133-138.

161. Cheng CK, Yao NK, Liu HC. Surgery simulation analysis of anterior advancement of the tibial tuberosity. *Clin Biomech (Bristol, Avon)* 1995;10:115-121.

162. Taskiran E, Celebi G, Kalayci T, et al. Effect of tibial tubercle elevation on patellofemoral compressive force in patellofemoral arthrosis. *Knee Surg Sports Traumatol Arthrosc* 1997;5:31-35.

163. Shirazi-Adl A, Mesfar W. Effect of tibial tubercle elevation on biomechanics of the entire knee joint under muscle loads. *Clin Biomech (Bristol, Avon)* 2007;22:344-351.

164. Dennler R, Kipfer NM, Tepic S, et al. Inclination of the patellar ligament in relation to flexion angle in stifle joints of dogs without degenerative joint disease. *Am J Vet Res* 2006;67:1849-1854.

165. Schwandt CS, Bohorquez-Vanelli A, Tepic S, et al. Angle between the patellar ligament and tibial plateau in dogs with partial rupture of the cranial cruciate ligament. *Am J Vet Res* 2006;67:1855-1860.

166. Apelt D, Kowaleski MP, Boudrieau RJ. Effect of tibial tuberosity advancement on cranial tibial subluxation in canine cranial cruciate-deficient stifle joints: an in vitro experimental study. *Vet Surg* 2007;36:170-177.

167. Hulse DA. Medial patellar luxation in the dog In: Bojrab M, ed. *Disease mechanisms in small animal surgery*. 2nd ed. Philadelphia: Lea&Febiger, 1993;809-810.

168. Tepic S, Montavon P. Is cranial tibial advancement relevant in the cruciate deficient stifle? . Proceedings of the 12th ESVOT Congress 2004;132-133.

169. Shahar R, Milgram J. Morphometric and anatomic study of the hind limb of a dog. *Am J Vet Res* 2001;62:928-933.

170. Yasuda K, Ohkoshi Y, Tanabe Y, et al. Quantitative evaluation of knee instability and muscle strength after anterior cruciate ligament reconstruction using patellar and quadriceps tendon. *Am J Sports Med* 1992;20:471-475.

171. Sakai N, Luo ZP, Rand JA, et al. The influence of weakness in the vastus medialis oblique muscle on the patellofemoral joint: an in vitro biomechanical study. *Clin Biomech (Bristol, Avon)* 2000;15:335-339.

172. Woo SL, Orlando CA, Camp JF, et al. Effects of postmortem storage by freezing on ligament tensile behavior. *J Biomech* 1986;19:399-404.

173. Nikolaou PK, Seaber AV, Glisson RR, et al. Anterior cruciate ligament allograft transplantation. Long-term function, histology, revascularization, and operative technique. *Am J Sports Med* 1986;14:348-360.

174. Barad S, Cabaud H, Rodrogo J. The effect of storage at -80 degrees C as compared to 4 degrees C on the strength of Rhesus monkey anterior cruciate ligament. *Trans Orthop Res Soc* 1982;378.

175. Mendes DG, Soudry M, Iusim M. Clinical assessment of Maquet tibial tuberosity advancement. *Clin Orthop* 1987:228-238.

176. Hoffman D, Miller J, Ober C, et al. Short term results of tibial tuberosity advancement performed in 57 dogs with cranial cruciate ruptures. Proceedings of the 33rd Veterinary orthopaedic society 2006.

177. Johnson AL, Broaddus KD, Hauptman JG, et al. Vertical patellar position in large-breed dogs with clinically normal stifles and large-breed dogs with medial patellar luxation. *Vet Surg* 2006;35:78-81.

APPENDIX I: Figures

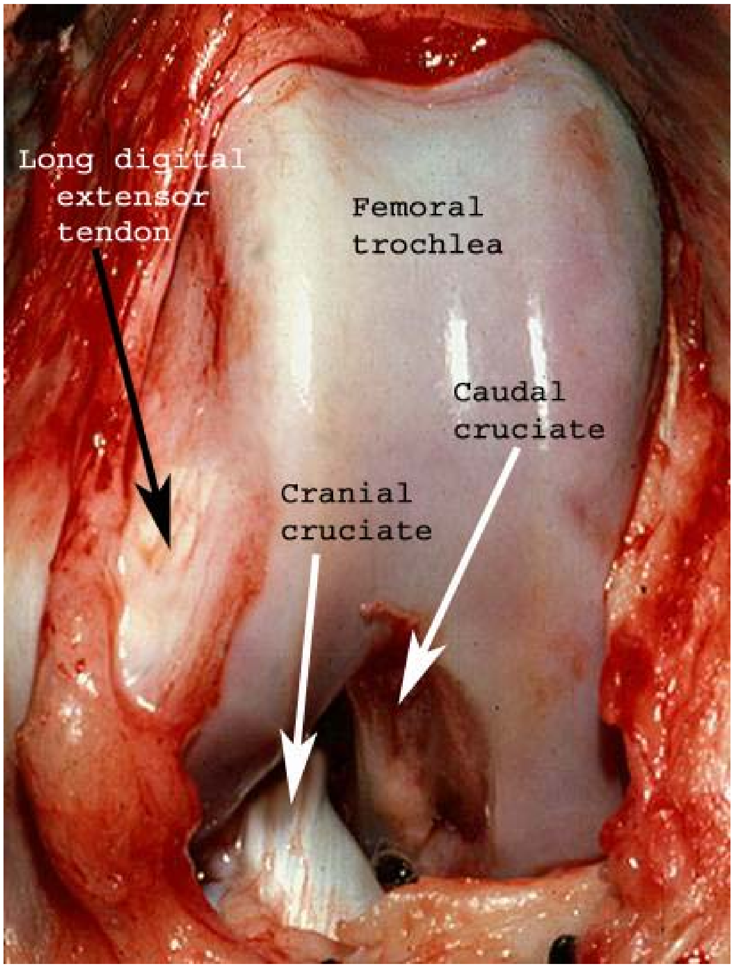

Figure 1: Canine Stifle Anatomy. View of the cranial surface of the canine stifle with the joint capsule and patella retracted out of view. The femur is at the top of the image, while the tibia is off the screen distally.

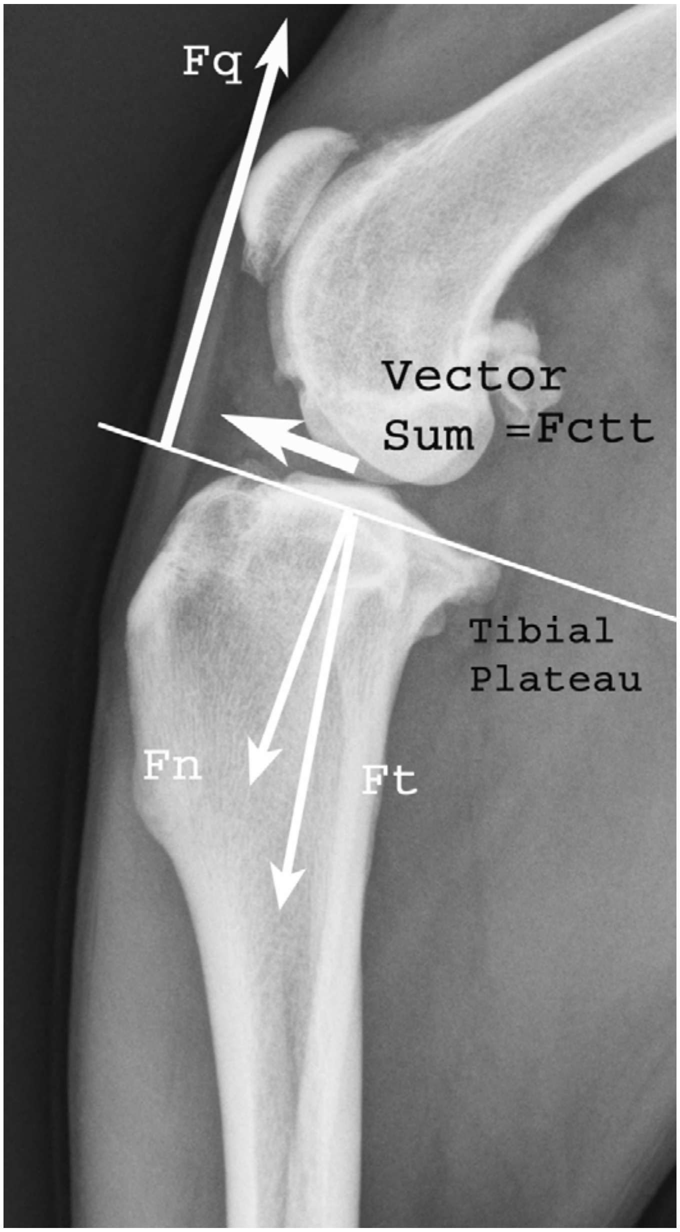

Figure 2: Force vectors of canine stifle. Radiograph of a diseased stifle demonstrating the forces acting on a cranial cruciate ligament deficient stifle. F_t = total force acting on the stifle. F_q = quadriceps force counteracting F_t. TPA = line of angle of tibial plateau. F_n = force in the neutral plane. Vector sum = the sum of all the forces revealing a force creating cranial tibial translation (F_{ctt}), because F_q does not balance F_t in the craniocaudal vector.

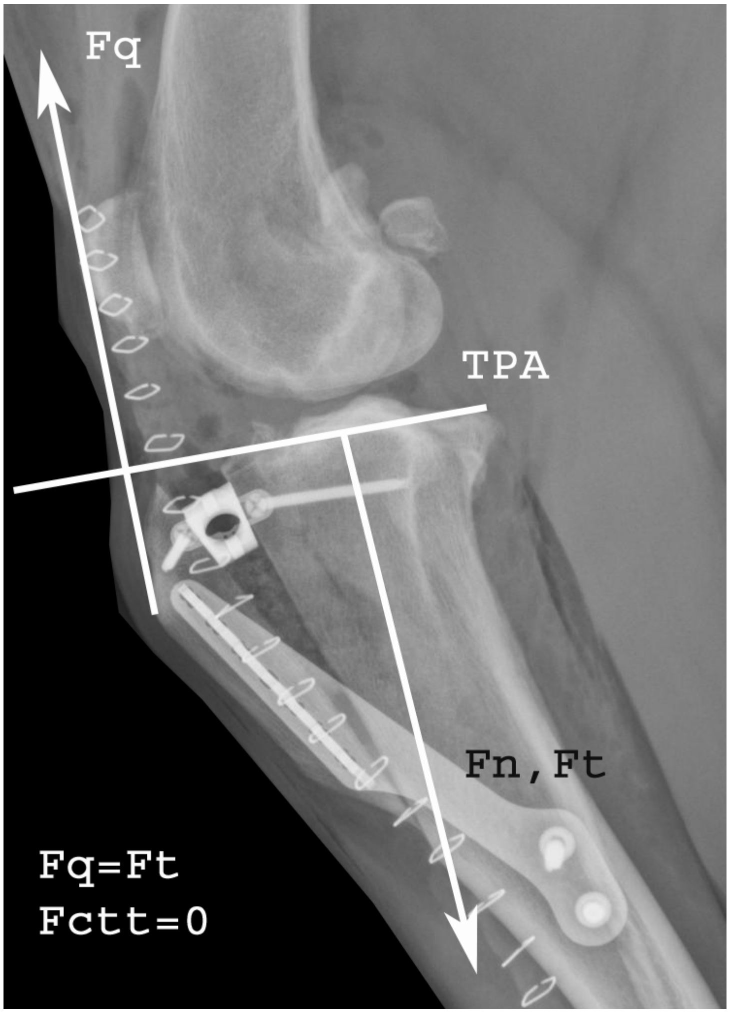

Figure 3: Force vectors following tibial tuberosity advancement. Radiograph of clinical tibial tuberosity advancement demonstrating the forces acting on the stifle after surgery. Note now F_t and F_q act in the same plane thereby eliminating the cranial translation force vector. F_q = quadriceps force counteracting F_t. TPA = line of angle of tibial plateau. F_t = total force acting on the stifle. F_n = neutral plane of force.

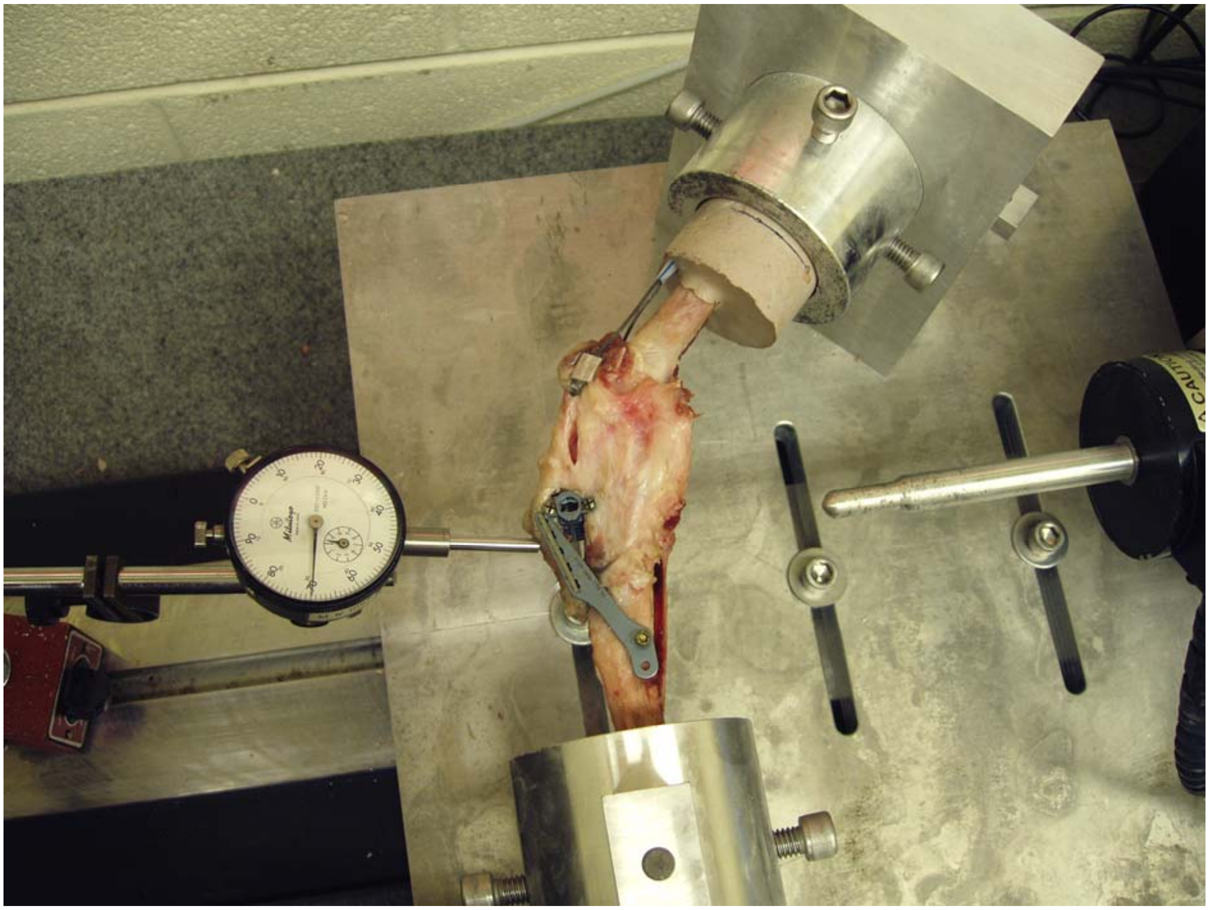

Figure 4: TTA specimen with dial indicator. Stifle secured to testing jig with tibial tuberosity advancement performed. Note the wire starting at the patella and extending proximally through a hole drilled in the potting material at the top and the dial indicator on the left.

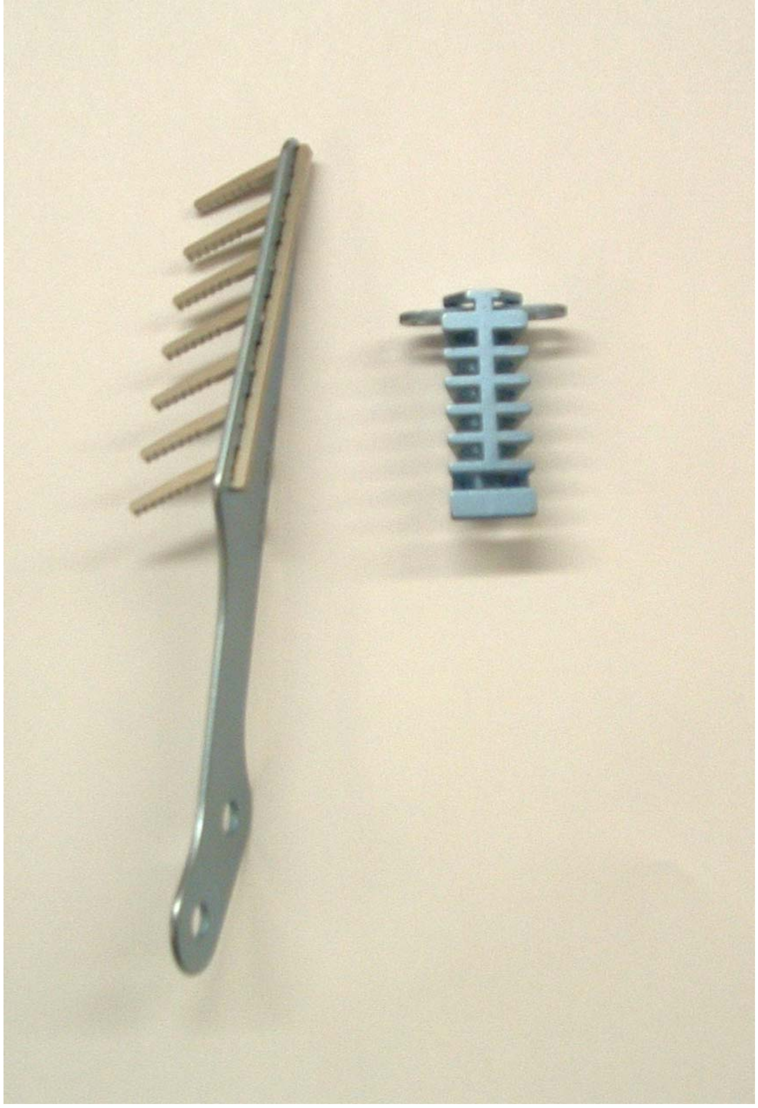

Figure 5: Commercially available TTA plate and 9 mm cage.

Figure 6: Radiograph of specimen after testing with TTA. Post tibial tuberosity advancement experimental specimen with a 9mm cage showing TTA and transpatellar Kirschner wire.

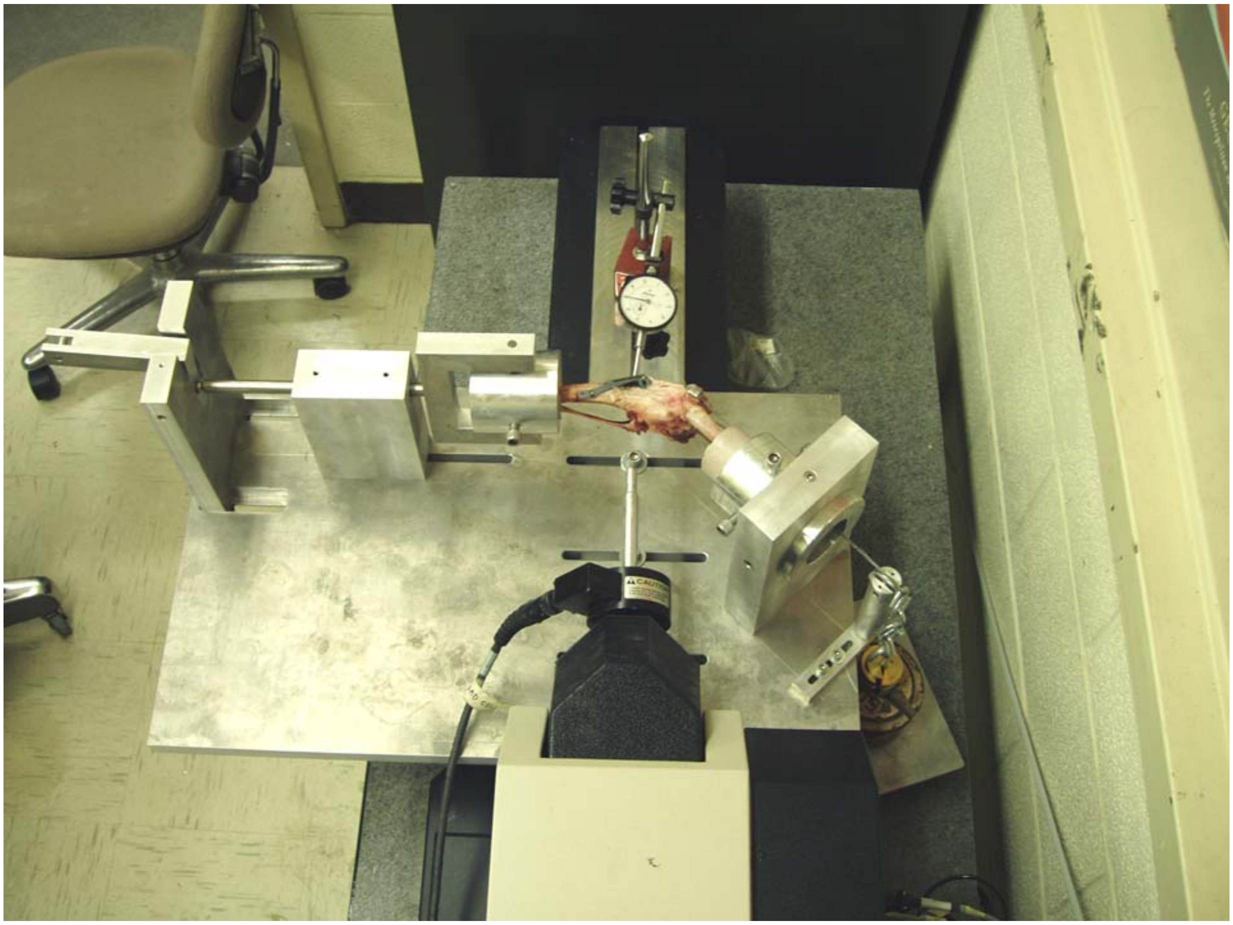

Figure 7: TTA stifle in testing jig. Specimen in testing jig with the material testing machine and load cell at the bottom, dial indicator on the top, simulated quadriceps force applied on the right, and the axial force applied to the tibia on the left.

APPENDIX II: Tables

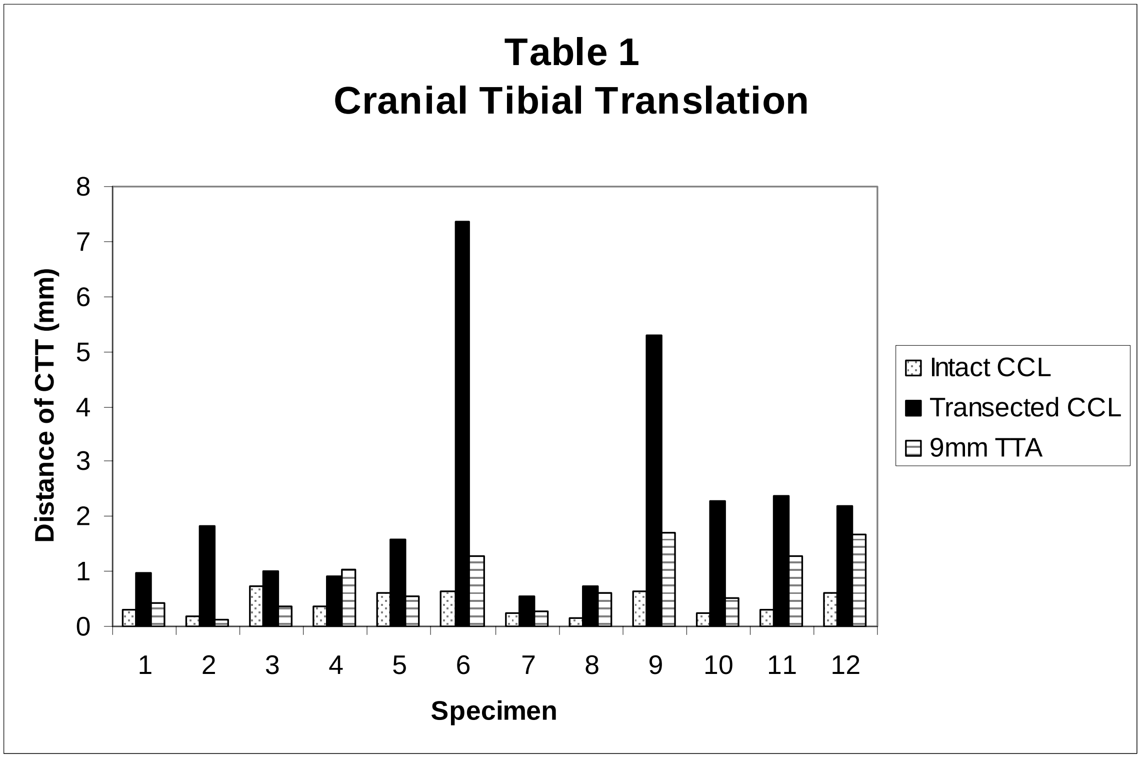

Table 1: Cranial tibial translation. Direct measurement of the distance in mm of cranial tibial translation (y axis) was recorded for the intact CCL, transected CCL with 10% body weight applied to the patella, and the TTA with a 9mm advancement.

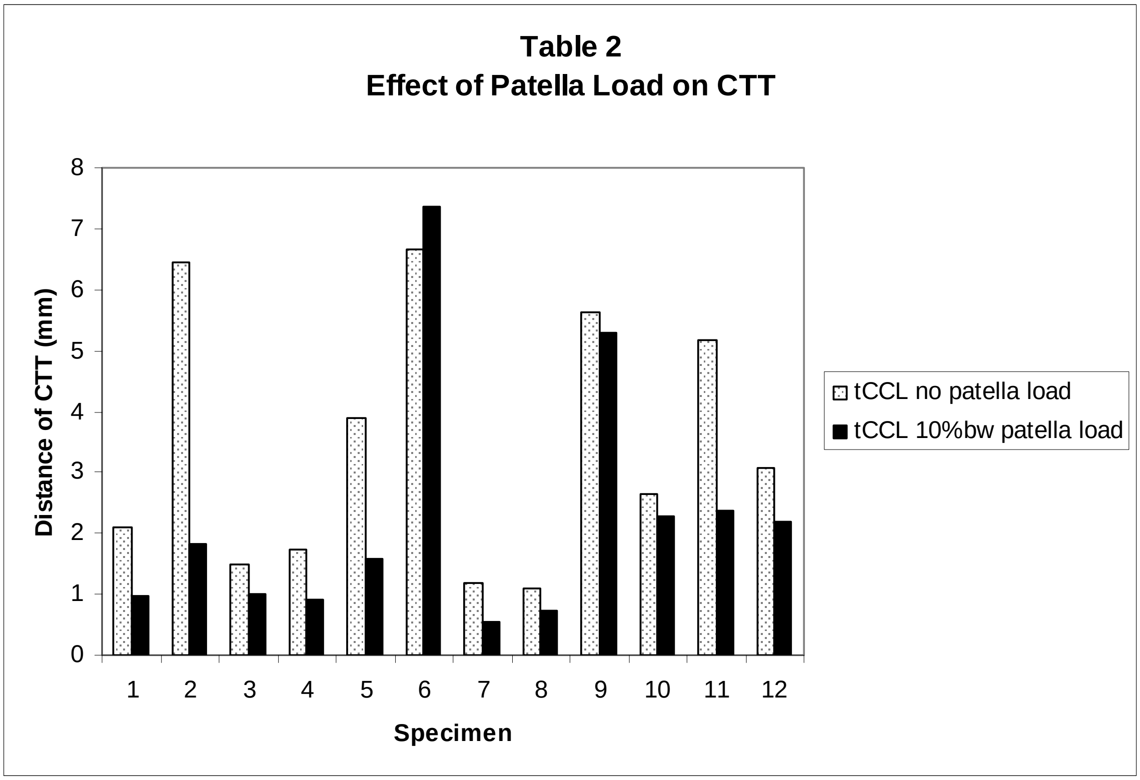

Table 2: Effect of force through the patella on CTT. Direct measurement using the dial indicator of the distance in mm (y axis) was recorded for the transected CCL with and without 10% body weight (bw) applied to the patella.

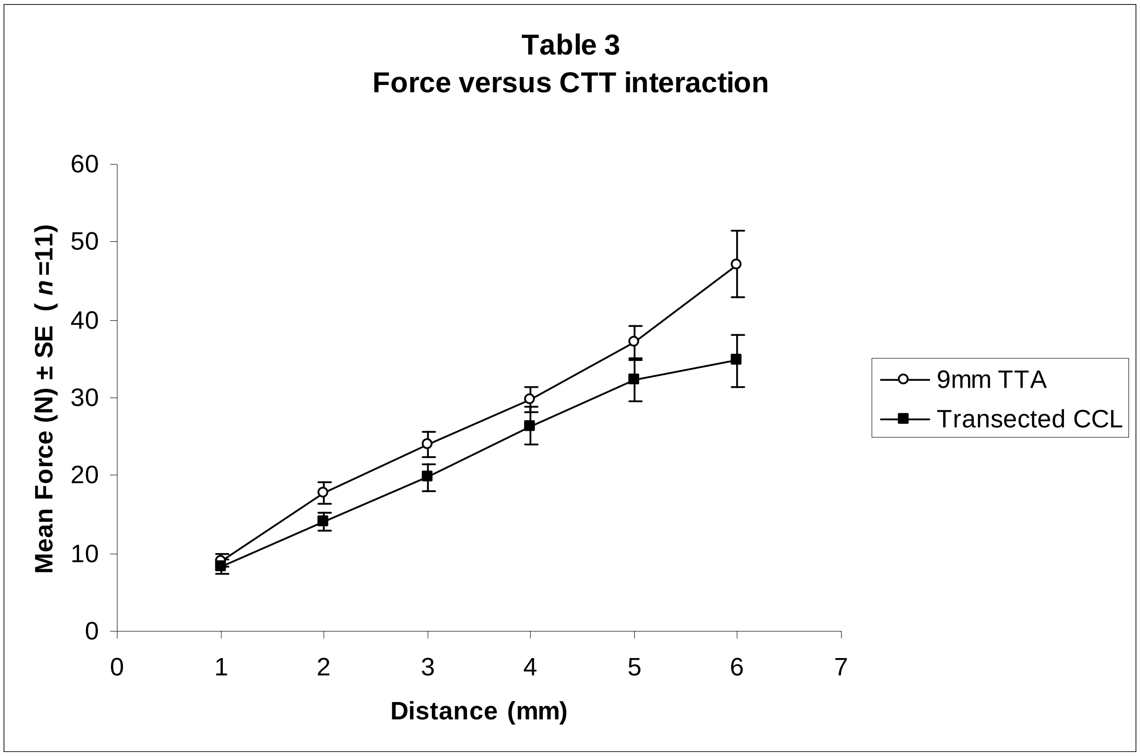

Table 3: Effect of force causing CTT on distance of CTT. Force required for cranial tibial translation (N) is on the y axis, while translation distance (mm) is present on the x axis calculated with a mean axial tibial load. The transected CCL is labeled as diamonds and the TTA as squares. Vertical bars represent the standard error.

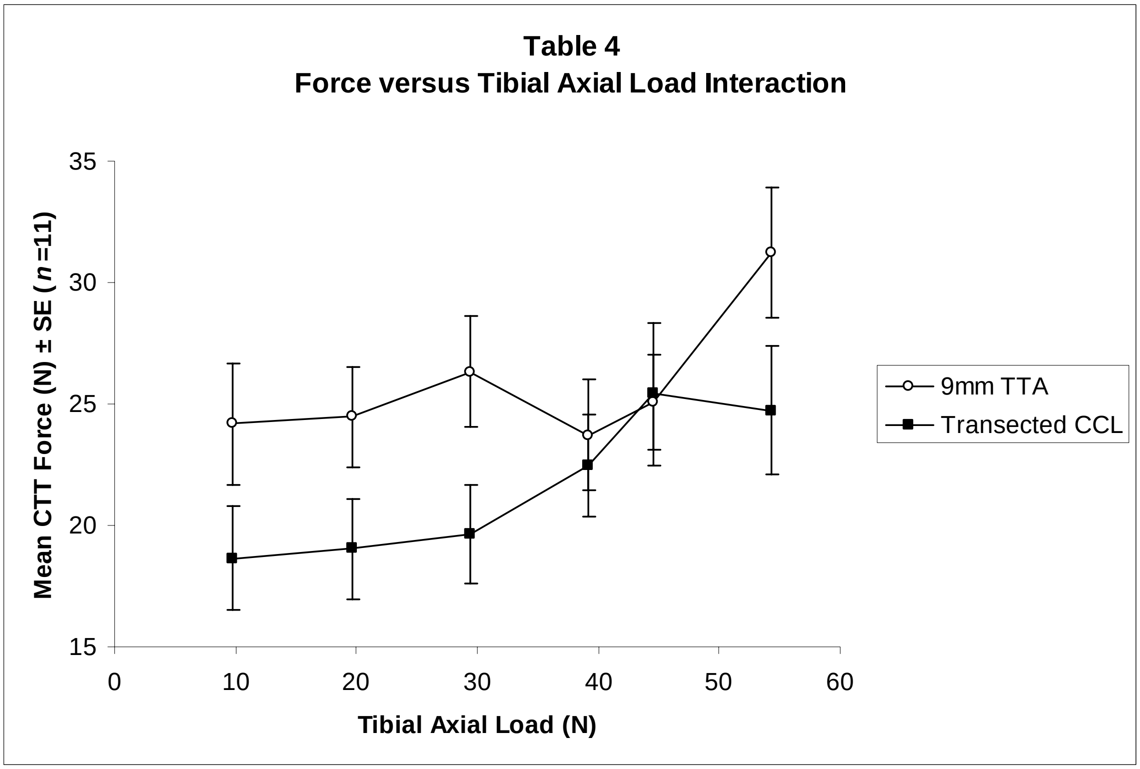

Table 4: Effect of axial tibial load on force required for CTT. Amount of tibial axial load (N) is presented on the x axis, while force (N) required to induce a mean cranial tibial translation is present on the y axis. The transected CCL is labeled as diamonds and the TTA as squares. Vertical bars represent the standard error.

VITA

Jonathan Miller was born in 1976 in Dekalb, Illinois to Paul and Sara Miller, and raised in Kankakee, Illinois. He received his Bachelor of Science degree from Illinois Wesleyan University in 1998 and his Doctor of Veterinary Medicine from the University of Illinois in 2002.

Jonathan completed his rotating small animal internship at the Atlantic Veterinary College at the University of Prince Edward Island and a surgical internship at Gulf Coast Veterinary Specialists in Houston, Texas. In 2004, he was accepted into the small animal surgery residency program at the Virginia-Maryland Regional College of Veterinary Medicine in Blacksburg, Virginia, and concurrently pursued a Master's of Science degree in Biomedical and Veterinary Sciences with anticipated completion in 2007.