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Effects of tibial tuberosity advancement and meniscal release on kinematics of the canine cranial cruciate deficient stifle during early, middle, and late stance

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EFFECTS OF TIBIAL TUBEROSITY ADVANCEMENT AND MENISCAL RELEASE ON KINEMATICS OF THE CANINE CRANIAL CRUCIATE DEFICIENT STIFLE DURING EARLY, MIDDLE, AND LATE STANCE

By

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Little research has been done to validate the biomechanical principles of tibial tuberosity advancement (TTA) throughout stance. The present study evaluates the effects of TTA on kinematics of the cranial cruciate (CrCL) deficient stifle during early, middle, and late stance. Cadaveric pelvic limbs were evaluated for the effects of TTA on kinematics under a load equivalent to 30% bodyweight and under the following treatment conditions: intact CrCL, CrCL deficient, TTA-treated, and TTA treated + meniscal release. Electromagnetic tracking sensors were used to determine tibial subluxation and rotation relative to the femur. Transection of the CrCL resulted in significant cranial tibial subluxation during early, middle, and late stance and significant internal rotation during early and middle stance. TTA normalized tibial subluxation in early, middle, and late stance but was unsuccessful in normalizing axial rotation in middle stance. Meniscal release had no effect on cranial/caudal or rotational displacement when performed following TTA.

DEDICATION

I would like to dedicate this research to my parents, Glenda and Jimmy Butler, for their faithful support of my pursuit of education.

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INTRODUCTION

Background

Rupture of the cranial cruciate ligament (CrCL) is one of the most common causes of pelvic limb lameness in dogs (1). The CrCL has three main functions within the joint: preventing cranial translation of the tibia, preventing hyperextension of the stifle, and limiting internal rotation of the tibia (2, 3). Because of the complex nature of the joint, a single effective technique that restores all functions of the CrCL has been elusive. Newer techniques that utilize tibial osteotomies were developed in an attempt to functionally stabilize the stifle during weight bearing by altering the geometry of the joint (4, 5). However, progressive osteoarthritis following these procedures is common (6-10). While this progression is likely multifactorial, potential causes include altered joint contact mechanics, abnormal joint biology, and altered joint kinematics (11).

Tibial osteotomy procedures alter the biomechanics of the stifle to provide functional craniocaudal stability during weight bearing. Due to the anatomy of the femorotibial joint surface, a cranially directed shear force is created between the two bones during weight bearing. In a CrCL deficient stifle, this shear force is unopposed, resulting in cranial translation of the tibia (12). Functional neutralization of this femorotibial shear force is the goal of tibial osteotomy procedures. Of the procedures described, the tibial plateau leveling osteotomy (TPLO) is the most commonly

performed. However, the tibial tuberosity advancement (TTA) is gaining in popularity as an alternative to TPLO.

TTA is based on a biomechanical theory that assumes the total femorotibial joint reaction force is parallel to the patellar tendon during ambulation (13). Because of the anatomy of the canine stifle, a shear force is created in a cranial direction during weight bearing. However, this shear force reaches zero when the patellar ligament is perpendicular to the tibial plateau and shifts caudally with further flexion of the stifle (13,14). In the dog, the cross-over point at which the cranially directed force is neutralized and directed caudally has been identified to be 90º of stifle flexion (14), which is below the normal weight-bearing angles of the canine stifle (15). The TTA procedure positions the patellar ligament perpendicular to the tibial plateau at a normal weight-bearing angle, resulting in a caudal shift of shear forces during ambulation (16). Because of the ability of TTA to neutralize and/or shift the femorotibial shear force caudally, it has been used clinically to stabilize CrCL deficient stifles with 90-95% of owners reporting an excellent functional outcome (10,17,18).

While TTA has proven to be a successful treatment for CrCL deficient stifles, complications such as continued progression of osteoarthritis and subsequent meniscal injury have been noted (17-19). Previous biomechanical studies have investigated the effect of the procedure on cranial tibial thrust at 135° of stifle flexion (16, 20-22), and only one paper has addressed the ability of TTA to control axial rotation at this weight-bearing angle (20). While 135° approximates the stifle angle at the middle of the stance phase of the gait, the stifle angle has been reported to range from approximately 145° to

125° during stance (15). One study evaluated the effects of TTA on eliminating cranial tibial thrust at multiple joint angles and axial loads (23, 24). However, the effects of TTA on three dimensional stifle kinematics throughout the entire stance phase of the gait have not been thoroughly evaluated.

The unopposed femorotibial shear force that is present in a CrCL deficient stifle not only allows for cranial translation of the tibia, but also predisposes the medial meniscus to injury (4). Because of the relatively high incidence of meniscal injury subsequent to TTA, concurrent meniscal release has been recommended as standard of care (18). However, this procedure is controversial, as the meniscus serves several important functions including energy absorption, stabilization of the joint by deepening the articular surfaces of the tibial plateau, joint lubrication and improvement of joint congruity between the femur and tibia (12). Furthermore, meniscal release has been shown to result in abnormal stress concentrations within the joint, which may predispose to osteoarthritis (25). The effects of the meniscal release on stifle stability when performed in conjunction with TTA have not been evaluated.

Canine Stifle Anatomy

The canine stifle is a complex, synovial, hinge joint (12). The principle bones that form the stifle articulations are the distal femur, proximal tibia, and proximal fibula (26). Other bony structures present include 4 sesamoid bones: popliteal, medial and lateral femoral fabellae, and the patella (26). The soft tissues supporting these bony structures include the joint capsule, medial and lateral collateral ligaments, cranial and caudal cruciate ligaments, medial and lateral

menisci, and various muscles (27). The complex geometry of the bony structures and the multiple soft tissue structures are crucial for overall joint function and stability of the articulations (12).

The distal femur and proximal tibia are divided into medial and lateral condyles. The femoral condyles are separated cranially by the femoral trochlear groove (26). The trochlear groove in combination with the patella forms the femoropatellar articulation and is responsible for lengthening the lever arm and improving the efficiency of the quadriceps musculature (28). The proximal tibial condyles are separated by medial and lateral intercondylar eminences. Collectively, the proximal aspect of the tibial condyles and intercondylar emininces are termed the tibial plateau. The femorotibial articulations form the principle weight-bearing surface of the joint. Axially, the femoral condyles articulate with the respective tibial condyle. However, abaxially, the femoral condyles articulate with the respective meniscus (29). Within these articulations, 3 distinct joint cavities that intercommunicate are present. The largest of these cavities is present between the femoropatellar articulation, and the remaining two cavities are found between the medial and lateral femorotibial articulations (26).

The lateral and medial menisci are semilunar fibrocartilage structures located between the femorotibial articulations (26). The menisci function to absorb energy across the joint, stabilize the joint by deepening the articular surface, lubricate the joint, and prevent synovial impingement between the femorotibial articulation (30-34). The meniscotibial ligaments attach the menisci to the tibia

cranially and caudally. The intermeniscal ligament joins the medial and lateral meniscotibial ligaments cranially. Additionally, the lateral meniscus is attached to the femur via the meniscofemoral ligament (26). The ligamentous attachments of the menisci are referred to as horns and the central portion of the menisci is referred to as the body (12). Structurally, the horns and body differ in that the collagen of the horns is organized into discrete bundles separated by loose connective tissue. However, the collagen of the body is organized in a herringbone pattern without separations (12). The meniscal horns have a rich nervous and vascular supply. However, the meniscal body is nearly devoid of blood supply with the exception of the peripheral 10-15% that is supplied by a vascular synovial plexus from the joint capsule (32). The remainder of the meniscal body is nourished by diffusion of synovial fluid (12). Because of the substantial innervation of the meniscal horns, they may also play an important role in joint proprioception and help protect the joint from excessive loading (33).

The medial and lateral collateral ligaments function primarily in limiting varus (lateral collateral ligament), valgus (medial collateral ligament) and axial rotation of the tibia, and their effect is most pronounced with the stifle in full extension (12, 35). The medial collateral ligament originates from the medial femoral epicondyle, blends with the joint capsule, and inserts over a large rectangular area of the proximal medial tibia (12, 36). The cranial border of the medial meniscus remains taught throughout range-of-motion; however, the caudal portion becomes relaxed with stifle flexion (12). The lateral collateral ligament

originates from the lateral femoral condyle and inserts on the fibular head. In contrast to the medial collateral ligament, the lateral ligament is only loosely connected to the joint capsule (36). The lateral collateral ligament is taught in extension but becomes completely lax upon flexion of the joint (12). This relaxation of the lateral collateral ligament allows the lateral femoral condyle to displace caudally during stifle flexion and result in relative tibial internal rotation. Furthermore, tightening of the lateral collateral ligament results in external rotation of the tibia during stile extension (12).

The cranial and caudal cruciate ligaments are named for their respective insertions on the cranial tibia. The caudal cruciate originates within the intercondyloid fossa from the lateral aspect of the medial femoral condyle and inserts on the lateral edge of the politeal notch on the caudal aspect of the proximal tibia (26). The cranial cruciate ligament originates within the intercondyloid fossa on the caudomedial aspect of the lateral femoral condyle and inserts on the cranial intercondyloid area of the tibia (26). The cranial cruciate ligament is the primary restraint against cranial tibial translation and hyperextension of the joint (2). The caudal cruciate ligament functions to prevent caudal translation of the tibia and as a secondary restraint against hyperextension (12). By twisting together during stifle flexion, the cranial and caudal cruciate also limit internal rotation of the tibia (2, 12).

The cruciate ligaments are composed of multiple collagen fascicles that extend from origin to insertion without crossing (12). The entire cranial cruciate ligament laterally twists approximately 90° from origin to insertion. This spiraling

of the ligament results in the gross appearance of two distinct bands of tissue: the craniomedial band and the caudolateral band (12). Generally speaking, the craniomedial band is taught throughout range-of-motion and the caudolateral band is taught in extension but relaxed in flexion (37). However, the structure and function of the cranial cruciate ligament is more complex and best described on a more microscopic level (12). As the stifle is flexed and extended, the multiple fascicles that form the cranial cruciate ligament sequentially tighten and relax and result in joint stability throughout the range-of-motion (38).

Because of the complex anatomy of the cruciate ligaments, femoral and tibial condyles, menisci, and collateral ligaments, the stifle joint does not rotate in a single plane when flexed (12). Upon flexion of the stifle, the lateral collateral ligament relaxes, the medial collateral ligament remains taught, the lateral femoral condyle displaces caudally resulting in relative tibial internal rotation, and the cranial and caudal cruciate ligaments twist together to help limit this internal rotation of the tibia (12, 35). During extension of the joint, the lateral collateral ligament tightens, the lateral femoral condyle shifts cranially, and the tibia externally rotates. This dynamic process allows the tibia to internally rotate during ambulation and is termed the "screw-home mechanism" in humans (30).

The cruciate ligaments invaginate the joint capsule from the caudal aspect of the joint and are covered by synovial tissue. Therefore, the cruciate ligaments are intra-articular but extra-synovial (12). The blood supply to the cruciate ligaments is primarily from the adjacent and surrounding soft tissues rather than osseous

attachments. Most of the vascular structures originate from the middle genicular artery, which is a branch of the popliteal artery (35). The principle blood supply and source of nourishment for the cruciate ligaments originates from the synovial tissues that surround the ligaments (35, 39).

The cruciate ligaments receive innervation from branches of the saphenous, tibial and fibular nerves. However, in the dog the medial articular branch of the saphenous nerve is the major nervous supply to the structures of the stifle joint (35). The cruciate ligaments are innervated by a rich nervous network present within the synovial tissue surrounding the ligaments. The nervous tissue within the cruciate ligaments functions primarily in autonomic regulation of blood flow and sensory perception (35). Mechanoreceptors within the ligaments in addition to these nerve fibers serve an important function in activating local reflex arcs to protect the ligament from tearing and contribute to the functional stability of the joint by activating or inhibiting periarticular musculature (12, 35).

Several pelvic limb muscles contribute to the stability and function of the stifle joint (12). The primary extensor of the joint is the quadriceps, which is composed of the vastus lateralis, vastus medialis, vastus intermedius, and rectus femoris (26). The biceps femoris is composed of a cranial and caudal head and functions to both flex and extend the stifle. The sartorius muscle is also composed of two distinct muscle bellies. The cranial sartorius functions to extend the stifle joint, and the caudal sartorius functions to flex the stifle (26). The semitendinosus and semimebranosus muscles function to flex the stifle. The gastrocnemius does

not play a direct role in stifle positioning, but it is an important factor when considering stifle stability in the cranial cruciate disease. Contraction of the gastrocnemius muscle creates a caudodistally directed shear force that leads to cranial displacement of the tibia relative to the femur (cranial tibial thrust) in the cranial cruciate deficient stifle (27).

Kinematics of the Canine Pelvic Limb

Kinematics is defined as the temporal and geometric characteristics of motion and has been used to describe the gait patterns of the dog (40). Studies evaluating the gait in the normal dog have been evaluated with the dog at a walk (15) and at a trot (41). At a walk, the canine pelvic limb progresses through the stance phase of gait by extension of the hip from 100 to 140°, flexion of the stifle from 145 to 120°, and initial flexion followed by extension of the tarsus from 130 to 170° (15). The swing phase is characterized by flexion of the hip from 90 to 130°, extension of the stifle from 110 to 150°, and continued flexion followed by rapid extension of the tarsus from 120 to 150° (15). More recent studies, however, have documented stifle angles of 150° during early stance (42). When comparing dogs at a walk to dogs at a trot, at least two differences have been documented: during stance phase while walking there tends to be more points of inflection indicating a more complete gait pattern and the angles of excursion noted at the walk tend to be less than those noted at the trot (15).

In addition to normal dogs, the kinematics of the pelvic limb in dogs with cruciate pathology has also been evaluated. As compared to the intact condition, the stifle tends to be carried in a more flexed position throughout stance and early swing phase (43, 44). However, relative hip and tarsus extension is present indicating compensation from the increased stifle flexion (43). In addition to increased stifle flexion, changes in internal rotation and tibial translation have been documented in dogs with cranial cruciate deficiency (44). Korvick et al. reported that internal rotation of the tibia was present in dogs 7 weeks after transection of the cranial cruciate ligament, and this alteration was most pronounced at midstance (44). However, a study by Tashman et al. found a non-significant trend towards reduced internal rotation up to 24 months after transection of the cruciate ligament in dogs evaluated at a walk (45). In addition to altered kinematic variables, dogs with cranial cruciate ligament rupture have been found to have decreases in paw velocity, stifle angular velocity, and stride length (46). Additionally, Ragetly et al. found that cranial cruciate deficient limbs had reduced vertical and braking ground reaction and joint reaction forces, decreased extensor moments at the hock and hip, and decreased flexor moments at the stifle when compared to normal limbs (42).

Pathophysiology of Cruciate Disease

Although rupture of the cranial cruciate ligament is one of the most common causes of lameness in the canine (1), the exact etiopathogenesis of cruciate disease is unclear (47). Purely traumatic ruptures do occur, but canine cruciate disease is

generally thought to be secondary to gradual degeneration of the ligament extracellular matrix leading to ligament rupture. Factors thought to be associated with cruciate disease include breed, bodyweight, gender, conformation, ligament extracellular matrix metabolism, and inflammation associated with immune-mediated disease within the stifle joint (11, 47).

A purely traumatic tear of the canine cranial cruciate ligament is uncommon. When this does occur, it is generally in dogs that are less than 4 years of age and typically occurs with additional injuries to the collateral and caudal cruciate ligaments (12). In puppies, traumatic ruptures of the cranial cruciate ligament typically occur secondary to avulsion of the ligament at its tibial insertion (48). The most common mechanism of cranial cruciate ligament injury is a sudden change in axial rotation of the joint when the stifle is in 20-50° of flexion. With excessive internal rotation of the stifle, the cranial cruciate ligament twists, becomes taught, and is subject to damage from the lateral femoral condyle. This type of injury usually occurs when an animal makes a sudden turn on a weight-bearing rear limb (30). Furthermore, because the cranial cruciate ligament functions to limit hyperextension of the joint, sudden hyperextension injuries can lead to ligamentous damage. Hyperextension injuries to the stifle typically occur when an animal steps into a hole while running (30).

Age has been reported to be a risk factor for the development of cranial cruciate disease, with dogs >4 years of at a higher relative risk (49) and a peak prevalence of disease occurring in dogs 7-10 years of age (49). As the dog ages,

changes occur within the structure of the cruciate ligaments that results in ligament weakening (50). The cruciate ligament is composed of type I and type III collagen fibrils and fibroblasts. With age, the structural arrangement of the collagen fibrils become disrupted and the cell population of fibroblasts decrease or undergo metaplasia to chondrocytes (50). This degenerative process typically occurs in the relatively avascular central core of the ligament and can lead to overall weakening of the structure (47). However, because all dogs do not develop clinically significant cruciate disease, other factors are likely more important in the etiopathogenesis.

The overall prevalence of cranial cruciate ligament rupture is higher in neutered dogs, particularly females (50-53). In women, increased incidence of anterior cruciate rupture is noted during times of elevated estrogen levels (47). However, low estrogen levels in the canine appear to have adverse affects on the ligament and estrogen may be protective for cruciate disease in dogs (54). The exact effect of hypogonadism on the development of cruciate disease in the dogs is unclear, and further research is needed to clarify this relationship.

Breed-specific variation in incidence of cruciate disease has been considered evidence for a cranial cruciate disease being a heritable trait (49, 55). A heritable basis of disease has been proposed in Newfoundlands (56) and boxers (57). In Newfoundlands, a recessive mode of inheritance is suggested, but environmental factors could not be ruled out as a cause for disease (56). Additionally, no statistical association with certain candidate genes has been identified in Newfoundlands or Boxers to date (47). Further research is needed in this area to identify causative

genes. Furthermore, conformational characteristics from breeds considered at-risk may play a role in the pathogenesis.

Certain conformational and dog-dependent variations of the canine pelvic limb have been suggested to predispose certain patients in cruciate disease. These include a straight stifle joint angle, narrow distal femoral intercondylar notch, and a steep tibial plateau slope (47). Intercondylar notch stenosis and its association with cruciate ligament pathology in dogs and humans have been well described (58-60). In one study, breeds at risk for developing cruciate disease (Labradors) were found to have lower intercondylar width indices when compared to breeds having a low risk of cruciate disease (greyhounds) (61). The authors concluded that impingement of the cruciate ligaments resulted in structural changes that predisposed the ligament to degeneration (61). Numerous studies have evaluated the role of the tibial plateau angle on the development of cruciate disease, but the role of this angle remains unclear (47). Studies comparing the tibial plateau angle between dogs with/without cruciate disease and dogs predisposed/at low risk have failed to find a significant difference in plateau angle between groups (62, 63). However, the association between cruciate disease and dogs with excessive tibial slopes (>35°) is well established (47). Furthermore, obesity can result in excessive loading of already compromised ligaments and is likely a contributing factor in many dogs (12).

Altered intra-articular environment secondary to inflammatory processes such as immune-mediated arthritis, immune synovitis, and joint sepsis may result in

cranial cruciate ligament rupture (64-67). Additionally, an immunologic component to cruciate disease has been suggested because of the identification of immune-complexes in synovial fluid and immunoglobulin in the synovial membrane (66-68). Collagenolytic enzyme expression in synovial fluid and synovial macrophage-like cells that are capable of producing degrative enzymes have been identified within joints affected by cranial cruciate disease. These findings suggest that the inflammatory process may predispose the cruciate ligaments to damage by the release of inflammatory mediators and proteolytic enzymes (69-71). Finally, antigenic stimulation secondary to bacterial DNA within the synovium has been suggested as a causative factor in naturally occurring cruciate disease (65). Despite multiple studies alluding to the fact that multiple inflammatory processes are present within joints affected by cruciate disease, a precise cause and effect relationship has yet to be identified, and it remains unclear whether these inflammatory changes are primary or secondary to rupture of the cruciate ligament (47).

Diagnosis and Treatment of Cruciate Disease

The diagnosis of cranial cruciate ligament rupture is based on history and physical exam findings. Most dogs present with a history that varies from an acute, completely non-weight bearing lameness with acute, complete tears to a more insidious history of intermittent lameness that worsens with exercise in chronic cases (12). It is not uncommon for dogs with a history of bilateral hip dysplasia to

present with a non-weight bearing lameness that, at least by the novice orthopedist, is attributed to the historical hip disease rather than stifle pathology. It is important to pay close attention to stifle palpation in all animals presenting for evaluation of pelvic limb lameness as cruciate disease is the most common cause of lameness in the dog (1).

Physical examination of dogs with cruciate disease can reveal alterations in gait. In addition to lameness or unwillingness to bear weight on the limb, most dogs with cruciate disease tend to carry the limb in a greater degree of flexion throughout the gait cycle (12). As the dog progresses throughout the gait cycle, the tibia tends to subluxate in the cruciate deficient stifle at the beginning of stance. As the dog progresses through stance, the stifle progressively flexes until the dog enters the swing phase of the gait and the stifle returns to a normal position. This cycle of subluxation during ambulation places significantly abnormal stresses on the articular structures and likely leads to changes within the joint such as osteoarthritis and meniscal injury (12).

Palpation of the stifle is the most common method used to diagnose cruciate disease. Several alterations can be noted during stifle palpation including: asymmetry in muscle mass, thickening of the periarticular structures (i.e. medial buttress) secondary to instability, effusion within the stifle joint, pain upon manipulation of the joint, and joint instability (12). Joint instability can be appreciated as "cranial drawer" motion or cranial tibial thrust. A positive cranial drawer test is present when the examiner is capable of translating the tibia cranially

relative to the femur by bone manipulation. Care must be taken when palpating the stifle of a young animal, as a short cranial drawer motion of up to 3mm with an abrupt stop can be present in the normal, immature stifle. In cases of complete cruciate tears, instability will be appreciated in both flexion and extension of the stifle. However, with partial tears involving only the craniomedial band of the cranial cruciate ligament, the stifle will be stable in extension and lax in flexion. The situation is further confused when only the caudolateral band is torn as the craniomedial band prevents cranial drawer regardless of limb position (12). Cranial tibial thrust is elicited by the tibial compression test (also known as the Henderson-Milton compression test) (72). The tibial compression test is performed with the limb in a normal standing angle. One hand supports the femur and stifle while the other places an axial load on the tibia by flexing the tarsus. This force mimics normal loading conditions during stance and leads to cranial displacement of the tibia relative to the femur (i.e. cranial tibial thrust) in the cranial cruciate deficient stifle (12).

Magnetic resonance imaging is commonly used in humans to detect cruciate injuries that are present despite inconclusive stifle palpation (73). However, this is usually not feasible or practical for the canine patient. In cases of a suspected partial cruciate tear in which the physical exam findings are inconclusive, radiographic examination of the limb can reveal non-specific changes that are suggestive of cruciate pathology. Characteristic radiographic findings of cruciate disease include joint effusion as noted by compression of the infrapatellar fat pad,

signs of degenerative joint disease, and, occasionally, an abnormal cranial drawer position of the tibia (12). In most cases characteristic radiographic findings within the stifle joint in combination with stifle pain, despite inconclusive palpation of cranial drawer or tibial thrust, is enough evidence to support surgical exploration of the joint with arthroscopy or arthrotomy to confirm the diagnosis. Additionally, sonography has been reported as one method of diagnosing cruciate disease. However, sensitivity of this modality is highly user dependent and not practical in most clinical scenarios (74).

Decisions about the most appropriate treatment for cruciate disease are influenced by the animal's size, activity level, concurrent orthopedic or systemic conditions, owner compliance, and economic considerations (12). Treatment is typically divided into two categories: medical/conservative treatment and surgical stabilization. Conservative therapy can be successful in many patients, but is typically reserved for small-breed dogs that are <15 kg as up to 86% of these animals will respond favorably to medical management (75). Conservative/medical management consists primarily of activity restriction, weight reduction, analgesics, anti-inflammatory medications, chondroprotectant medications, and specific physical therapy to improve joint range-of-motion and maintain/build muscle mass (12). Dogs >15 kg typically benefit most from surgical therapy as 81% of these dogs have been shown to have a persistent or worsening lameness without surgical intervention (75).

The intent of surgical therapy for cruciate disease is to provide stability to the stifle joint through restoration of joint mechanics, addressment of meniscal damage, and prevention of osteoarthritis progression. Numerous surgical procedures have been described to treat cruciate disease and are typically divided into intracapsular techniques, extracapsular techniques, and tibial osteotomy procedures (12). Intracapsular and extracapsular techniques aim to temporarily stabilize the joint to allow for periarticular fibrosis, which is responsible for stifle stability long-term (12). The newer osteotomy procedures result in dynamic stability of the joint during ambulation and are less reliant on periarticular fibrosis (76).

Intracapsular stabilization of the cruciate deficient stifle can be achieved by replacing the damaged cruciate ligament with a variety of materials. The most common repair methods in the canine patient utilize a portion of the patellar tendon, fascia lata, or both (12). The classic intraarticular technique described by Paatsama (77) utilizes a portion of the fascia lata that is routed through holes drilled in the femur and tibia. Modifications of this procedure, known as the "over the top procedures," place the graft over the caudolateral aspect of the lateral femoral condyle without the need to drill femoral bone tunnels (12). The original "over the top procedure" described by Arnocky (78) utilizes a portion of the patellar tendon and an osteotomized portion of the patella. Arnozky's procedure was further modified to the "Four-in-one" procedure that uses the fascia lata instead of the patellar tendon and incorporates advancement of the sartorius and biceps femoris

muscles and imbrication of the lateral soft tissues to further augment the repair (79). The "Hulse procedure" utilizes a portion of the patellar ligament and fascia lata and places the graft under the intermeniscal ligament and over the top of the lateral femoral condyle (80).

In addition to autogenic grafts, various allogenic grafts and synthetic intra-articular prostheses have been described. Allografts, although readily available, have been shown to incite an immune response despite storage methods before implantation, and their use is not widely accepted (81, 82). Synthetic materials such as Gore-tex and Dacron prostheses offer the convenience of off-the-shelf availability. However, their use has not been thoroughly evaluated (12).

While intracapsular repairs offer the theoretical advantage of placement of the graft in the most anatomical location, numerous studies have document unsatisfactory outcomes secondary to inferior graft strength, persistent instability, sub-optimal patient function, and progression of osteoarthritis (83-86). Because of the unsatisfactory outcomes with these procedures, most surgeons have abandoned these procedures in favor of extracapsular repairs or osteotomy procedures.

Extracapsular suture repairs aim to mimic the action of the cranial cruciate ligament and eliminate cranial drawer motion by placement of synthetic suture material extra-articularly from the distal femur to the proximal tibia or patellar ligament (12). Non-absorbable suture material is generally used, and monofilament nylon leader line is reported to have superior mechanical properties (87). However, newer braided suture materials, such as Fiber wire or Fiber tape, are gaining in

popularity. The most commonly performed extracapsular repair involves placement of a suture(s) around the lateral femoral fabellae and through a hole drilled transversely in the tibial tuberosity (12). Because this surgical technique is fundamentally flawed in that it fails to adequately restore the normal biomechanics of the intact cruciate ligament, numerous modifications to the procedure have occurred. The most notable modification in recent years is the issue of isometric suture placement (88). Placement of the suture in femoral and tibial insertions that are non-isometric results in excessive tightening or loosening of the suture as the stifle flexes and extends (88). This has the effect of either placing excessive force on the suture and potentially breaking or stretching the repair, or failing to eliminate cranial drawer throughout the range of motion. Various femoral and tibial bony landmarks have been reported to be isometric and their use is recommended when placing extracapsular sutures (88). To aid in placement of sutures in the most isometric location and help prevent the complication of avulsion of the femoral fabellae, screw and washer constructs as well as bone anchors have been used to facilitate anchorage of the suture in the distal femur. Additionally, a recent modification of the extracapsular suture method called the "Tightrope" procedure reportedly has the advantage of consistent isometric suture placement through bone tunnels that can be done in a minimally invasive manner (89).

Because extracapsular repairs place suture material on the lateral aspect of the joint, the normal internal rotation that occurs with flexion of the stifle joint is disrupted and results in abnormal motion at the articular surface (90). Disruption

of this joint orientation and alterations in load results in the progression of osteoarthritis (91) and likely explains the suboptimal outcomes seen with most of the extracapsular procedures (85, 92-94). Because of poor success in extra and intracapsular procedures in restoring normal joint biomechanics and preventing progression of osteoarthritis, osteotomy procedures that aim to create "dynamic" stability of the joint during ambulation have been developed (76).

In 1984, Slocum described the first osteotomy procedure, the cranial tibial wedge osteotomy (CTWO), to repair cruciate deficient stifles (95). The premise behind this procedure and subsequent osteotomy procedures is that reducing the caudally directed slope of the tibial plateau will eliminate cranial tibial subluxation during weight bearing and result in dynamic stability of the joint (76). By providing dynamic stability, the passive restraints such as the cranial cruciate ligament are not needed (76). Recognition that stability could be achieved through dynamic stability led to the development of several osteotomy procedures: tibial plateau leveling osteotomy (TPLO), combined TPLO and CTWO, proximal intraarticular osteotomy (PITO), triple tibial osteotomy (TTO), and chevron wedge osteotomy (CVWO) (96-100). Of these procedures, the TPLO is the most widely performed and levels the tibial plateau by creating a radial osteotomy in the proximal tibia. The TPLO is based on the biomechanical theory that the compressive forces of weight bearing can be divided into a cranially directed component (cranial tibial thrust) and a joint compressive force (101). The TPLO and similar procedures aim to neutralize the cranial tibial thrust by leveling the tibial plateau until the compressive force of

weight bearing (assumed to be parallel to the long axis of the tibia) is parallel to the joint compressive force (101). The biomechanical theory behind this procedure has been validated in multiple experimental models (102, 103). Success with the TPLO is generally considered to be very good with acceptable long-term outcomes (96) and subjective evaluation of limb usage immediately following surgery better than following intra or extracapsular techniques (104). However, complications such as progression of osteoarthritis are common (105). A more recent osteotomy procedure, the TTA, attempts to dynamically stabilize the cruciate deficient stifle by altering the relative alignment of the patellar tendon to the tibial plateau (5).

Tibial Tuberosity Advancement

Advancement of the tibial tuberosity has been advocated in humans for the relief of pain in the patellofemoral compartment (106). The operation in humans is known as Bandi's or Maquet's procedure as it was described by both authors (107, 108). The biomechanical basis behind the operation is that advancement of the tuberosity increases the moment arm and, therefore, the mechanical advantage of the patellar ligament. This reduces the force along the patellar ligament and the resultant force across the patellofemoral joint (106). Montavon and Tepic modified the procedure in 2002 for the treatment of cruciate disease in dogs (5).

Biomechanics

In contrast to the theory proposed by Slocum that the compressive forces of weight bearing are parallel to the long axis of the tibia (101), Montavon and Tepic

proposed in 2000 that the total tibofemoral joint forces in vivo are directed parallel to the patellar tendon (5). Cranial tibial thrust is then, at least according to this model, dependent upon the angle between the tibial plateau and the patellar tendon (14). Montavon and Tepic used this theory to their advantage when modifying the Maquet procedure for the treatment of cruciate disease in dogs. They theorized that advancement of the tibial tuberosity until the angle between the patellar tendon and tibial plateau (also known as the patellar tendon angle or PTA) is 90° would effectively neutralize cranial tibial thrust in a similar manner as the TPLO procedure (5).

To better understand the principles of the TTA procedure, it helps to understand the effect of stifle flexion on the PTA. In 2006, Dennler et al. evaluated the effect of stifle flexion on the inclination of the patellar ligament in relationship to the tibial plateau and the common tangent to the tibiofemoral contact point (TFCP) (14). The common tangent method was evaluated because many feel it more accurately reflects the biomechanics of the stifle joint because it takes into consideration the actively articulating convex surfaces of the joint and the caudally rolling motion of the femoral condyles during flexion (14). The distance from the TFCP to the patellar ligament is the patellar ligament moment arm and the determinant of the magnitude of joint forces in the stifle (109). Dennler et al. found that the crossover point at which the cranially directed shear force is neutralized (i.e. the point at which the force parallel to the patellar ligament is 90° to a line perpendicular to the functional or mechanical axis of the limb) occurred at 90° of

stifle flexion when using the tibial plateau as a reference point and 110° when using the common tangent to the TFCP as a reference (14). In other words, as the stifle flexes, the PTA progressively becomes smaller until it is 90° to the tibial plateau and common tangent at 90° and 110° of stifle flexion, respectively (14). Furthermore, the same study found that the angle between the patellar ligament and common tangent varied less throughout joint range of motion when compared to the tibial plateau (14). This relationship between the patellar ligament and the "cross-over" point helps understand the principle of TTA as when the limb is flexed to either 110° or 90° (depending on the reference point used), the need for the cranial cruciate ligament is eliminated. However, 110° and 90° of stifle flexion are outside the normal weight bearing angles of stifle flexion (15). The TTA procedure positions the tibial tuberosity to a point where the patellar tendon is 90° to the tibial plateau or common tangent at a weight-bearing angle (5).

Theoretical reduction of tibiofemoral shear forces by advancement of the tibial tuberosity has been proven in both cadaver and computer-modeling studies (16, 20-24, 106, 110). In 2007, Apelt et al. demonstrated that neutralization of cranial tibial thrust using TTA occurred at a PTA of 90 ± 9° when the stifle was at 135° stifle flexion (16). Further studies in the canine demonstrated advancement was successful in eliminating cranial tibial thrust at 135° of stifle flexion (21, 22).

In 2008, Hoffman et al. evaluated the effects of TTA on eliminating cranial tibial thrust at varying stifle angles (135° and 145°) and under varying loads (30% and 50% of body weight) (23, 24). They found that TTA was successful in

eliminating cranial tibial thrust at varying angles of stifle flexion and this occurred at a PTA of 91.1° at 135° of stifle flexion and a PTA of 98.3° at 145° of stifle flexion. The same study also evaluated the common tangent and found that cranial tibial thrust was eliminated at a PTA (using the common tangent as a reference) of 88.4° and 91.1° at 135° and 145° of stifle flexion, respectively (23-24). Furthermore, they found that with increasing loads, further advancement of the tibial tuberosity to a point <90° was required to successfully neutralize cranial thrust (23-24). The same study found that TTA was effective in decreasing retropatellar pressure while increasing patellar tendon force (23-23).

In 2009, Kim et al. further evaluated the effects of TTA on contact mechanics and three-dimensional kinematics of the limb at 135° of stifle flexion. They found that the TTA was successful in returning the kinematics and contact mechanics of the cranial cruciate deficient stifle to normal (20). This was also the first study to evaluate the effects of TTA on axial rotation. While no significant differences were noted, the authors felt that this may reflect a type II error in study design as several specimens had significant alterations in rotational alignment following TTA (20). When comparing this study to a similar study performed for the TPLO (111), the authors felt that the TTA was biomechanically superior because it preserved the normal geometry of the articular surface of the proximal tibia, and, therefore, did not alter the contact mechanics of the joint as seen with the TPLO (111). A similar study evaluated both the TTA and TPLO on contact mechanics at 90° of stifle flexion and found that the TTA did not alter contact mechanics and joint alignment at this

limb position, while the TPLO caused a mild varus and subsequent increase in medial compartment loading (112).

Surgical Planning and Technique

Standard craniocaudal and lateral radiographic projections of the affected stifle are taken preoperatively to allow for appropriate surgical planning. The lateral radiograph is positioned with the limb positioned approximately at midstance (135° of stifle flexion), taking care to maintain a neutral tibia position without the presence of cranial or caudal drawer. A transparency template (Kyon) is utilized to determine the approximate degree of advancement required to position the patellar tendon perpendicular to the tibial plateau (or common tangent). Alternatively, this measurement can be performed using digital computer software. Finally, the appropriate size plate template is positioned over the tibial crest. The largest plate possible that will fit on the tibial crest and still allow room for positioning of the cage should be used. In most cases, the proximal extent of the plate will align with the insertion of the patellar tendon. However, this can be shifted proximally if dictated by plate size or tibial conformation. This may be necessary in cases with a less prominent distal tibial crest to ensure the plate is appropriately positioned. Prior to advancement the distal aspect of the plate should align near the cranial cortex of the tibia to ensure that advancement of the tibial tuberosity will not move the distal plate screw holes off the caudal aspect of the tibia (18).

The surgical procedure begins with surgical exploration of the joint with either arthroscopy or an arthrotomy. Cruciate pathology is confirmed and the menisci are thoroughly inspected to identify and treat pathology and/or perform a meniscal release (depending upon surgeon preference). Following joint exploration, exposure of the craniomedial aspect of the tibial crest is performed by incising the insertion of the caudal belly of the sartorius muscle and aponeurosis of the gracilis, semimembranosus, and semitendinosus muscle insertions. The incision is made a few millimeters caudal and parallel to the tibial crest and extended distally along the tibial diaphysis. The periosteum and muscular insertions are elevated cranially to expose the tibial crest. An 8-hole drill guide (Kyon) for the TTA plate is positioned along the cranial aspect of the tibial crest at the location determined preoperatively for the plate to be positioned. The appropriate numbers of holes are drilled (depending on the plate size) using the drill guide and either a 1.8 mm or 2.0 mm drill bit. The position of the osteotomy is identified along the tibial crest and typically marked with electrocautery. The osteotomy should be positioned in the sagital plane from a point immediately cranial to the intermeniscal ligament to the distal extent of the tibial crest (or at least distal to the last fork prong). Care should be taken to ensure the distal extent of the osteotomy does not extend to or beyond the level of the distal plate screws. The osteotomy is then begun bicortically distally (under the area where the plate will be placed) and monocortically for approximately 50% of the proximal osteotomy. An appropriately sized TTA plate that has been pre-contoured to match the shape of the tibial crest and proximal tibia

is then attached to the tibial crest using a fork designed to fit within the tension-band TTA plate. The plate/fork construct is then secured within the tibial crest using a mallet and custom fork impactor (Kyon). Care is taken to ensure the fork/plate construct is securely seated along the tibial crest. The remainder of the osteotomy is then completed. An appropriately sized spacer is positioned within the osteotomy to allow the surgeon to ensure cranial tibial thrust has been eliminated. A cage of appropriate length is then selected. The ears of the cage are contoured to match the tibial contours, and the cage is positioned within the proximal portion of the osteotomy, 2-3mm distal to the tibial plateau. The cage is secured in position with two 2.4 mm screws placed through the cage ears. The plate is then secured distally to the tibia with either 2.7mm or 3.5mm screws, depending on plate and patient size. An autogenous bone graft, frozen allograft, or synthetic bone graft material is used to fill the osteotomy gap. The surgical site is closed in a routine manner, ensuring the tibial implants are covered by apposition of the aponeurosis of the medial thigh musculature (18). This procedure has been modified to treat dogs requiring excessive tuberosity advancement (113) and for the concurrent treatment of patellar luxation (114).

Outcome and Complications

Clinical outcomes following TTA are documented in a small number of clinical reports. Hoffman et al. in 2006 reported that owner satisfaction in 65 dogs treated with TTA was good or excellent in 90% of dogs (17). However, 67% of dogs

in this study demonstrated progression of osteoarthritis at study end (17). In 2007, Lafaver et al. compiled the largest clinical study to date on the TTA when they reported the early results and complications in 101 dogs that were treated with TTA. In this study, all but 2 owners were satisfied with the outcome and 83.1% reported a marked improvement or return to pre-injury status (18). In the same study, in hospital postoperative limb evaluation revealed 74.5% to have no detectable lameness, 23.5% to have a mild lameness, 2% to have a moderate lameness, and 1% to have a severe lameness (18).

More objective means of evaluating outcome following TTA using force plate gait analysis have been performed. Voss et al. found in 2008 that TTA significantly improved vertical ground reaction forces at follow up, but failed to return the limbs to normal (115). Based on this study, a return to normal of ~90% is expected with the TTA (19). A similar study in 2010 revealed that vertical ground reaction forces improved following surgery, despite progression of osteoarthritis in 55% of dogs (10). In that study no correlation between osteoarthritis progression and ground reaction forces was detected (10).

Overall complication rates following TTA range from 25% to 59% of dogs (17-19). However, most of these complications were minor and included incision infection, inconsequential implant failure, inconsequential tibial tuberosity chip fracture, post-operative limb swelling, incision dehiscence, chronic poor performance, inconsequential intra-operative tibia fracture, diarrhea, and inappetance (17-19). Major complications (those requiring surgical intervention)

occurred in 12.3% of cases and were usually attributed to surgeon error (18). Major complications seen include subsequent meniscal injury, patella luxation, implant failure, tibial fracture, lick granuloma, septic arthritis, and chronic poor performance (18).

Meniscal Release

Subsequent meniscal injury has been reported to occur in 21.7 % of cases following TTA. Consequentially, meniscal release by either transection of the meniscal body or caudal meniscotibial ligament is recommended by some surgeons when performing TTA for the treatment of cruciate disease (18). The menisci have several important functions within the joint, especially in cruciate deficient stifles (12). Multiple clinical and cadaveric studies have evaluated the role of the medial meniscus as a secondary stabilizer of the cruciate ligament-deficient knee in humans and dogs (25, 116-118). The posterior pole of the medial meniscus can act as a wedge, limiting the degree of posterior displacement of the medial femoral condyle (117, 119). Because of this role of the medial meniscus in the cruciate deficient stifle, it is subjected to increased shear forces that may predispose it to injury (117), and many surgeons recommend release of the medial meniscus through either the body or caudal horn to allow retraction of the caudal pole of the meniscus and prevent injury (18, 120, 121). However, meniscal release has been shown to increase stress concentration within the joint (25, 122) and result in lameness and medial compartment joint pathology 12 weeks after surgery (123). The wedge

shape of the meniscus and the orientation of the collagen fibers are important to give the meniscus great circumferential tensile strength and allow the menisci to equally distribute force across the tibial condyles (124). Loss of the integrity of the meniscus, either through release or partial meniscectomy, results in loss of this "hoop tension" and worsening of contact mechanics within the joint (124). Meniscal release has been shown to result in greater cranial tibial translation when performed in canine cruciate deficient stifles (25). However, the TPLO was shown to be protective of this increased instability by eliminating cranial tibial thrust (25). Additionally, tears of the posterior root of the medial meniscus have been shown to result in axial rotational instability in humans (125).

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CHAPTER II

EFFECTS OF TIBIAL TUBEROSITY ADVANCEMENT AND MENISCAL RELEASE ON KINEMATICS OF THE CANINE CRANIAL CRUCIATE DEFICIENT STIFLE DURING EARLY, MIDDLE, AND LATE STANCE

Objectives

The objectives of this study are to further investigate the biomechanical effects of TTA during the early, middle, and late stance phase of gait, specifically its effect on cranial tibial subluxation and axial rotation of the tibia when compared to normal limbs (cranial cruciate ligament intact). Additionally, the effects of a meniscal release on stifle stability when performed in conjunction with TTA are evaluated. Our hypotheses are: (1) significant differences in limb alignment, namely cranial/caudal subluxation and axial rotation, will be present in cranial cruciate deficient limbs treated with TTA at stifle angles greater than 135°, and (2) meniscal release following the TTA procedure will lead to significant changes in joint alignment when compared to limbs treated with TTA alone.

Materials and Methods

Specimen Preparation

Twenty-two pelvic limbs were collected via coxofemoral disarticulation from 11 adult dogs euthanized for reasons unrelated to this study. The mass of each dog was

recorded. Each limb was manually examined and radiographed to verify skeletal maturity and absence of pathology. Four limbs were used to establish experimental methods and 18 limbs were used for statistical analysis in the study. The tibial plateau angle (TPA) was measured from the lateral radiograph using previously described methods (26). The limbs were wrapped in saline (0.9% NaCl) solution-soaked towels and stored at -20°C until testing. Immediately prior to testing, the limbs were thawed to room temperature and stripped of their musculature. The medial and lateral collateral ligaments, cranial and caudal cruciate ligaments, joint capsule, and medial and lateral menisci were carefully preserved. Throughout the remainder of the testing the limbs were kept moist with saline solution.

The proximal aspect of the femur was osteotomized 5 cm distal to the most proximal aspect of the femoral head and potted using polymethylmethacrylate (PMMA)[a] within polyvinyl chloride (PVC) pipe (3.8 cm). The potted limb was loaded into a custom-built mounting bracket that was designed to allow attachment to the loading frame and adjustment of the hip angle during testing (Figure 1). The overall length of the osteotomized bone, potting material, and mounting bracket was the same as the unaltered limb. After potting, a 1.5 mm hole was drilled transversely through the widest portion of the patella. 36 kg test monofilament nylon was passed through the hole and tied to create a loop. A turnbuckle link extending from an eyelet attached to the most cranial and proximal aspect of the mounting bracket was attached to the patella nylon loop to mimic the quadriceps mechanism. Two 3.5 mm

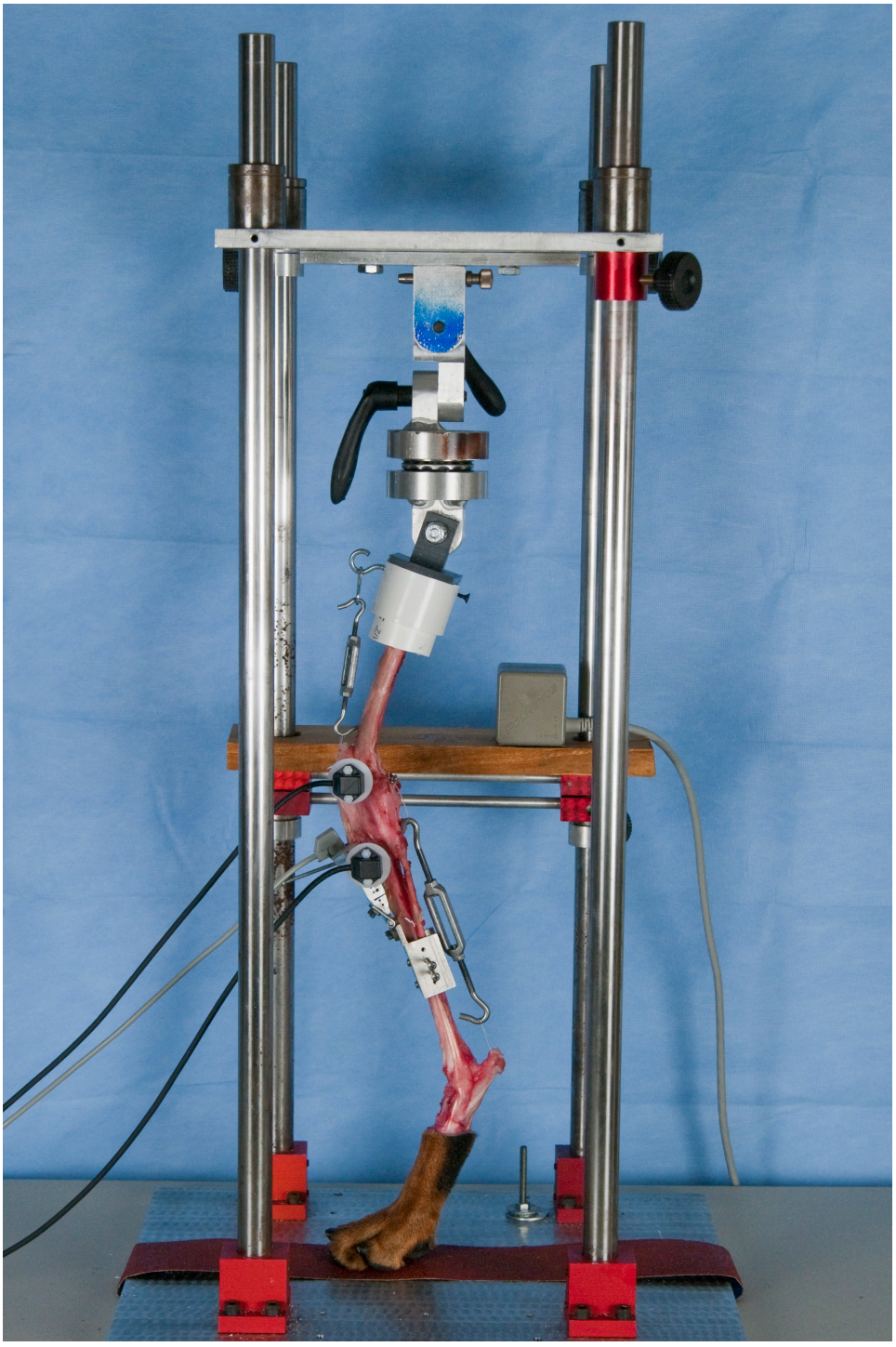

Figure 1

Prepared Cadaveric Limb Mounted in Limb Press

cortical bone screws were inserted at the level of the origin of the gastrocnemius muscle on the caudal femur, and another nylon loop was placed through a 1.5 mm diameter hole drilled transversely through the calcaneus. A turnbuckle link that extended from the calcaneus nylon loop to a nylon loop secured to the 3.5 mm screws was placed to mimic the gastrocnemius muscle.

A custom-designed hinge plate similar to that described by Apelt et al. (16) was secured to tibial diaphysis with two 3.5 mm cortical bone screws (Figure 2). The proximal aspect of the hinge plate was secured to the tibial tuberosity with two M3 x .05 machine screws. An osteotomy of the tibial crest was performed in the frontal plane from just cranial to the level of the long digital extensor tendon to the junction of the tibial crest and tibial body using an oscillating saw[b]. A M4 x .07 machine screw was placed through a threaded hole in the proximal aspect of the hinge plate in a craniocaudal direction and through a pre-drilled hole in the tibial tuberosity. To facilitate advancement of the tibial tuberosity during testing, this screw was tightened against a plastic "stopper plate" placed along the cut surface of the tibial body. Prior to testing, a medial mini-arthrotomy and a caudal mini-arthrotomy were performed to facilitate transection of the CrCL and the meniscotibial ligament during testing.

Each limb was mounted in a custom-designed loading frame similar to the frame used in the study performed by Warzee et al. (27). Custom hinges as described by Kim et al. (28) were used to facilitate adjustment of the abduction/adduction angle and allow for unconstrained femoral axial rotation during testing (Figure 1). The stifle angle was adjusted using the previously placed turnbuckles to either 145 °,

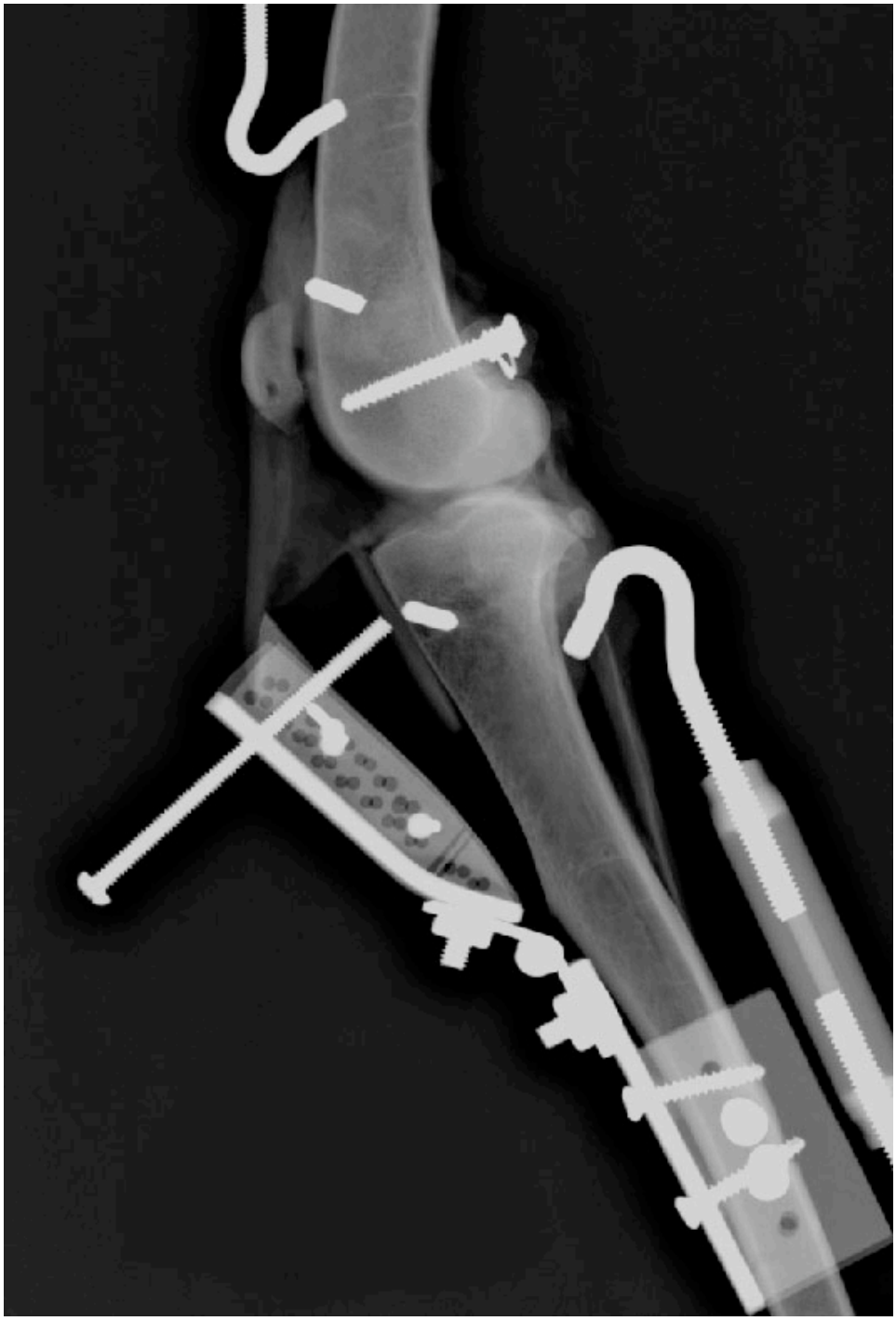

Figure 2

Radiograph Demonstrating Cadaveric Limb Preparation

135 °, or 125°, approximating early, middle, and late stance phase during walking (15). The hip and tarsal angles were simultaneously adjusted during testing to correspond with the phase of gait being tested (15). Hip and tarsal angles used to approximate early, middle and late stance were as follows: 113°, 120°, and 135°; 140°, 145°, and 160°, respectively (15). The joint angles were measured with a plastic goniometer with each arm aligned to the center of the bone diaphysis proximal and distal to the joint. The paw was allowed to rest on but was not fixed to the base of the loading frame. A textured surface (220 grit sandpaper) was fixed to the steel-loading frame to prevent craniocaudal paw slippage during testing. One electromagnetic tracking sensor[c] was attached to the lateral aspect of the distal femur and another to the proximal tibia using 4.8 mm Steinman pins and nylon spacers to prevent direct contact with the stainless steel pin. A third sensor was fixed to the cranial aspect of the proximal tibia using a custom plastic mounting bracket that attached to the proximal aspect of the hinge plate and was utilized in measurement of tibial subluxation. This electromagnetic tracking system[c] measures 6 degrees of freedom in a Cartesian coordinate system. The system software allows 3D measurement of the position of the receivers relative to a global coordinate system projected by the magnetic transmitter. This tracking system reportedly has a translational resolution and accuracy of 0.1 mm and 0.2 mm, respectively (29). Rotational resolution and accuracy has been reported to be 0.1° and 0.2°, respectively (29).

Testing Protocol

To reproduce in vivo conditions, a load of 30% of the body weight was applied to the limb during testing (28). The abduction/adduction angle was set by visual inspection

prior to testing to approximate even distribution of forces across the femoral condyles. During testing, the axial rotational hinge was left unconstrained.

The limbs were tested in the following sequence: (1) Normal (CrCL intact, sham TTA in which the osteotomy was performed but the tuberosity was not advanced) at all joint angles representing late, mid, and early stance (beginning with a stifle angle of 125°, then 135°, and then 145°, all with corresponding hip and tarsal angles); (2) CrCL deficient (transected CrCL, sham TTA) at all joint angles representing late, mid, and early stance; (3) TTA-treated (transected CrCL, TTA advanced to a PTA of 90°) at all joint angles representing late, mid, and early stance; (4) Meniscal release (transected CrCL, TTA, medial meniscal release) at all joint angles representing late, mid, and early stance. The CrCL was transected at its insertion on the proximal tibia through the previously made medial mini-arthrotomy. In the TTA treated groups, the tibial tuberosity was advanced and radiographed until the PTA was verified to be 90° at midstance when using the tibial plateau as a reference. The PTA and stifle angle were measured from a lateral radiograph as described by Dennler et al. (14). For comparison purposes, the PTA using the femorotibial tangent as a reference (PTA^{CT}) was also measured from the same lateral film as previously described (14). The advancement required to position the PTA to 90° was measured from the lateral radiographic projection along the caudal aspect of the osteotomy at a point 2 mm distal to the proximal extent of the tibia. This position correlates with correct placement of the TTA cage in clinical cases (18). The medial meniscal release was performed by transection of the meniscotibial ligament through the previously made caudal mini-arthrotomy.

Data for cranial/caudal displacement and internal/external rotation of the tibia in relationship to the femur was collected for each treatment group using the electromagnetic tracking sensors. The limbs were positioned in the loading frame, the appropriate joint angles set for the phase of stance being tested, and the coordinate position of the tracking sensors was acquired and recorded for the limb while unloaded. A load equivalent to 30% of the body weight was applied to the limb using the limb-press platform and appropriately sized weights for the limb being tested (Figure 1). The limb construct was allowed to settle under the load, and the joint angles were readjusted to account for settling of the limb. The coordinate position of the tracking sensors for the loaded limbs was acquired after adjusting these joint angles. This sequence of events was repeated three times for all joint angles evaluated and all treatment conditions, and the mean value for displacement and axial rotation calculated. The values for cranial/caudal displacement and axial rotation of the tibia in relationship to the femur were determined as the relative difference between the coordinate position of the femoral and tibial tracking sensors before and after application of the load.

Statistical Analysis

Data for cranial/caudal tibial displacement and internal/external rotation were statistically analyzed by using PROC MIXED (mixed procedure) with the REPEATED statement and the autoregressive order one (AR 1) covariance structure on the SAS/STAT software package[d]. Efficacy analyses were performed using analysis of variance (ANOVA) with the repeated-measures design. The limbs were evaluated for differences among the 4 treatment groups (normal, transected CrCL, TTA treated, and

meniscal release) and for differences between the stifle angles (145°, 135°, and 125°). The least square (LS) means were used when there was statistical significance to determine the differences between treatments. All statistical comparisons were two-sided using the 0.05 significance level.

Results

Mean (± SD) body mass of the dogs was 22.8 ± 4.6 kg; mean tibial plateau angle was 23.0 ± 1.9°; mean PTA was 89.8 ± 0.9° at a mean stifle angle of 137.3 ±2° following TTA; mean PTA^{CT} was 85.2 ± 1.5° following TTA; mean advancement of the tibial tuberosity required to obtain a patellar tendon-tibial plateau angle of 90° was 14.6 ± 2.4 mm.

The results for cranial/caudal tibial displacement and tibial rotation are presented in Table 1. Transection of the CrCL resulted in significant cranial displacement of the tibial sensor relative to the femoral sensor for all stifle angles (P<.0001). After advancement of the tibial tuberosity to a PTA of 90°, significant cranial tibial displacement was no longer present (P<.0001). Release of the medial meniscus had no effect on cranial/caudal stifle displacement when performed in conjunction with TTA.

Transection of the CrCL resulted in significant internal rotation of the tibia at stifle angles of 125° (P =.049) and 135° (P=.0006); however, no significant change in rotation was detected at a stifle angle of 145°. Treatment with TTA failed to result in a significant difference in rotation when compared to CrCL-deficient limbs at all stifle angles. At 135° of stifle flexion, TTA failed to realign axial rotation when compared to

Table 1

Tibial Subluxation and Tibial Rotation

	Stifle Angle	Normal (1)	CrCL-Deficient (2)	TTA-Treated (3)	TTA-Treated + Meniscal Release (4)
Subluxation (mm, ± SD)*	125°	-3.5 ± 2.8	10.3 ± 7.1 (**P_{1-2}=<.0001**)	-2.0 ± 3.3 (P_{1-3}=.890, **P_{2-3}=<.0001**)	-2.8 ± 5.6 (P_{3-4}=.898)
	135°	-2.4 ± 3.9	9.0 ± 3.1 (**P_{1-2}=<.0001**)	-1.5 ± 6.7 (P_{1-3}=.749, **P_{2-3}=<.0001**)	0.07 ± 4.9 (P_{3-4}=.159)
	145°	0.02 ± 2.0	7.6 ± 4.6 (**P_{1-2}=<.0001**)	0.2 ± 5.0 (P_{1-3}=.318, **P_{2-3}=<.0001**)	-0.5 ± 4.2 (P_{3-4}=.194)
Rotation (°, ± SD)**	125°	1.1 ± 1.5	-0.9 ± 3.0 (**P_{1-2}=.049**)	-0.14 ± 3.4 (P_{1-3}=.142, P_{2-3}=.600)	-0.8 ± 3.2 (P_{3-4}=.266)
	135°	1.5 ± 2.0	-2.4 ± 3.9 (**P_{1-2}=.0006**)	-0.5 ± 3.3 (**P_{1-3}=.030**, P_{2-3}=.138)	-1.1 ± 3.0 (P_{3-4}=.276)
	145°	0.7 ± 1.4	-0.5 ± 3.6 (P_{1-2}=.263)	0.3 ± 3.1 (P_{1-3}=.827, P_{2-3}=.365)	-0.14 ± 2.2 (P_{3-4}=.193)

* Positive values indicate cranial subluxation, and negative values indicate caudal subluxation.

** Positive values indicate external rotation, and negative values indicate internal rotation.

the intact limb (P=.030). Performing a meniscal release in the TTA treated limbs did not result in a significant change in axial rotation.

Discussion

The present study evaluated the effects of TTA on three-dimensional joint alignment at varying joint angles of the canine stifle that approximated early, middle, and late stance. The results indicate that treatment with TTA as described was effective in eliminating cranial tibial displacement at the stifle angles of 145°, 135°, and 125°, which correspond to early, middle, and late stance phase of the gait as described by Hottinger et al. (15). This is consistent with previous reports which demonstrated the effectiveness of TTA at eliminating cranial tibial displacement at 135° of stifle flexion (16, 20, 21) and at varying stifle angles and axial loads (23, 24). Additionally, the results of this study revealed that the TTA failed to consistently eliminate abnormal rotational alignment secondary to a transected cranial cruciate ligament. This is the first report to identify a statistically significant difference in rotational alignment when comparing TTA treated limbs to the intact stifle.

The PTA is highly dependent upon the degree of stifle flexion and a linear relationship between the stifle angle and PTA exists (14, 30). In the normal canine stifle, the patellar tendon becomes perpendicular to the tibial plateau at 90° of stifle flexion, which is a greater degree of stifle flexion than the normal weight-bearing stifle angle (14, 15). Advancement of the tibial tuberosity effectively positions the patellar tendon perpendicular to the tibial plateau at a lesser degree of stifle flexion. 135° of stifle flexion is chosen for surgical planning of TTA because it approximates the middle of the

stance phase of the gait (5). However, this fixed position does not take into consideration the stifle angle at any other point during ambulation. The effects of TTA on stifle stability at stifle angles less than 135° is supported by Dennler et al (14) as further flexion of the stifle beyond the point at which the patellar tendon is perpendicular to the tibial plateau results in caudal tibial thrust (16). This theory was further supported in a recent paper that evaluated the biomechanics of TTA and TPLO at 90° of stifle flexion (31). Therefore, the fact that the stifle was stable in a cranial-caudal plane at 125° of stifle flexion in the present study is not surprising. However, the effects of TTA on stifle stability with extension of the stifle beyond 135° is less clear.

At 145° of stifle flexion, the present study indicated that the TTA procedure as described effectively eliminated cranial tibial displacement. This finding is counterintuitive given the effects of the stifle angle on the PTA (14, 30) and the results of previous studies that report the critical point for neutralization of cranial/caudal tibial thrust as a PTA of 90° at 135 degrees of stifle flexion (16). One explanation for this finding may lie in a flaw in surgical planning for TTA, which uses the slope of the tibial plateau as a reference (5, 18). Recent reports have suggested determining PTA using the tangent to the femorotibial contact point rather than the tibial plateau may more accurately represent the forces acting across the stifle during ambulation and be less affected by stifle angle (14, 32, 24). The critical point for stifle neutrality has been reported to be 110° of stifle flexion when using the tangent method for determining the PTA (14). Therefore, a lesser degree of tuberosity advancement would be necessary to obtain a PTA^{CT} of 90° at any given stifle angle. The fact that the stifle remained stable at

145° of stifle flexion in the present study suggests that the advancement of the tibial tuberosity to a PTA of 90° at the stifle angle of 135° may represent over-advancement of the tibial tuberosity when using the tibial plateau as a reference. This theory has been supported by a recent study that reports craniocaudal stifle stability to occur following TTA to a PTA of 91.1° and 98.3°; and PTA^{CT} of 88.4° and 91.1° when the stifle is at 135° and 145° of flexion, respectively (24). The same study (24) also found that PTA^{CT} varied less during stifle flexion as previously suggested by Dennler (14) and that the PTA at 135° of stifle flexion was significantly greater that the PTA^{CT} at the same angle. The latter finding was supported in the current study and the work by done by Dennler (14). However, Hoffman et al. also suggest that surgical planning using the femorotibial tangent as a reference may require TTA with a target PTA of < 90° (24). Further biomechanical research is warranted to determine whether using the femorotibial tangent as a reference is more reliable and determine the most appropriate target angle when using this method.

In the present study, the tibial tuberosity was advanced a mean of 14.6 mm in order to obtain a PTA of 90° at a stifle angle of 135°. This is a greater magnitude of advancement than is typically performed clinically in similar size dogs with similar tibial conformations (5). However, in a comparable biomechanical study by Kim and others, an advancement of 13.5 mm was necessary (20). Advancement of the tibial tuberosity to the optimal PTA in some of these cases would not be possible with the currently available implants[e]. In some clinical cases, the TTA procedure may result in under-advancement of the tibial tuberosity which may result in suboptimal neutralization of

cranial tibial thrust. Continued craniocaudal instability may result in complications seen with TTA, particularly the high incidence of subsequent meniscal injury (18). Alternatively, the tibial tuberosity may be advanced to a lesser degree if the femorotibial tangent was used as a reference during surgical planning (20, 32) and may explain the apparent clinical success with the TTA procedure (10, 17, 18). However, as noted by Hoffman et al. (24), TTA using the femorotibial tangent as a reference may require advancement to a target PTA of < 90°. Apelt and others reported a lesser degree of advancement (10.2 ± 3.7°) required to reach the "critical point" in their study (16). While the exact reason for the discrepancy among studies is unclear, the authors feel this is likely multifactorial and may include conformational differences amongst the dogs (such as size of the tibia, shape of the tibial crest and tibial plateau slope) as well as methodology differences between studies. In the study by Kim as well as the current study, a predetermined PTA was set (PTA of 90° at 135° of stifle flexion) and the limbs were evaluated (20). The study by Apelt and others differed slightly in that the TTA was sequentially advanced under load until the stifle reached a "critical point" defined as the position one revolution before joint instability occurred. This occurred at a PTA of 90.3 ± 9° using the tibial plateau as a reference (16). Given the wide range of PTA reported (16) and because the PTA data in relationship to the femorotibial tangent was not reported, the relationship between the "critical point", tibial plateau, and femorotibial tangent is unclear. Further research is needed to determine the biomechanical effects of performing TTA utilizing the femorotibial tangent as a reference to define the PTA.

Only one previous study has documented the effects of TTA on rotational stability during weight bearing (20). In that study, transection of the CrCL resulted in significant internal rotation of the tibia and that rotational change was partially corrected following TTA (20). In the present study, significant internal tibial rotation after transection of the CrCL at 125° and 135° of stifle flexion was seen, but the values were of a small magnitude (<10°). Unlike the previous study, TTA in the current study did not consistently correct the internal rotation caused by CrCL transection. Considering the small magnitude of rotation seen in this study, we question the clinical significance of this finding. Similarly small values for internal rotation during ambulation following cranial cruciate loss were noted in an in vivo study by Tashman and others (33). These studies must be compared with caution due to the multiple active stabilizers of the joint that are present in vivo that are not present in the current cadaveric study. However, the findings of the current study bring into question the clinical significance of tibial rotation as a cause of instability after CrCL rupture and tibial osteotomy procedures, at least during straight-line walking.

Previous reports have documented subsequent meniscal injury rates as high 21.7% following TTA (18). For this reason, routine meniscal release at the time of initial surgery has been recommended (18). The medial meniscus has been shown to aid in stabilization of the CrCL deficient stifle via a wedge-effect of the caudal pole of the medial meniscus that helps prevent tibial subluxation (34, 35). A previous study evaluated the effects of medial meniscal release when performed in conjunction with TPLO (35). In that study, medial meniscal release resulted in a significant increase in

cranial tibial displacement in the CrCL deficient stifle, but TPLO appeared to negate this effect by neutralization of cranial tibial thrust (35). In the present study, performance of medial meniscal release via transection of the meniscotibial ligament failed to result in a significant increase in cranial tibial displacement when performed in limbs treated with TTA. While the effects of meniscal release on rotational alignment in the canine stifle have not been evaluated, rotational alignment consequences of tears of the posterior root of the medial meniscus in humans have been studied (36). In humans, a tear of the posterior root of the medial meniscus (analogous to the meniscotibial ligament in the canine stifle) has been reported to result in significant alterations in axial rotational alignment (36). The present study is the first to report the effects of medial meniscal release on stifle rotation in the canine stifle when performed in conjunction with a tibial osteotomy procedure performed for the treatment of CrCL deficiency. In the present study, medial meniscal release failed to result in significant internal rotation of the tibia when performed in conjunction with TTA. As with subluxation (35), this suggests that the TTA unloads the medial meniscus and at least partially eliminates its role as a secondary restraint within the joint. While releasing the meniscus did not result in statistically significant alterations in axial rotational alignment, the limbs did tend to be slightly more internally rotated following meniscal release, especially during early and mid stance. These results may reflect a type II error (i.e. low statistical power) and further studies with additional limbs may reveal a different result. Additionally, the increased stress and friction generated between articular surfaces following mensical release and elimination of its role as a spacer within the joint (37) may further limit

alterations in limb alignment as previously suggested by a similar study in human cadavers following meniscectomy (38).

Given the in vitro nature of this study and the inherent limitations associated with the methodology and complexity of the limb-press model, these results should be interpreted with caution. While a similar limb-press model has been used in previous studies to evaluate the biomechanical effectiveness of tibial osteotomy procedures for the treatment of CrCL deficient stifles (16, 20, 23, 27, 28), it does not accurately represent all forces acting upon the stifle during ambulation, and the effects of active stabilizers of the stifle are not evaluated. Furthermore, this model only takes into consideration straight-line loading of the limb. The effects of other activities such as turning, jumping, and sitting on stifle stability were not evaluated.

We evaluated the stifle at the angles 145°, 135°, and 125° which have previously been reported to represent the early, middle, late stance phase of the gait (15). However, more recent studies have suggested a degree of stifle extension exceeding 150° at the beginning of the stance phase (39) and postural variations among breeds (40). Furthermore, a slight discrepancy exists between the methodology of determining joint angles utilized in our study and those determined from in vivo kinematic measurements (15, 39). The joint angles in the current study were measured and set as described by Dennler and others (14) and the points of reference do not exactly coincide with those utilized by the in vivo kinematic studies (15, 39). Taking this fact into consideration, there is a chance that the present study slightly underestimated the degree of joint extension for the stifle and this represents one limitation to our study. While the effect of

this slight underestimation is uncertain, it is likely not significant as the difference is likely small and the stifle angle is not the only factor affecting limb position during ambulation (15). The hip and tarsal angles were simultaneously adjusted to approximate the forces acting across the joint during early, middle, and late stance. Future studies with the stifle in a greater degree of extension are warranted.

The current study sought to mimic the clinical scenario in which the limb was positioned at midstance and TTA performed to a PTA of 90° without regard to the effect of stifle flexion/extension on PTA. Because radiographic projections were not performed with the limb in extension and flexion beyond midstance, the actual PTA at these limb positions cannot be reported. This represents a limitation to the current study design as these values cannot be used to help explain the biomechanical results obtained. However, the clinical implications of the results remain unchanged and, as previously noted, the relationship between stifle flexion/extension and PTA has been previously described (14).

In this study, an electromagnetic tracking system was used to track the position of the tibia relative to the femur in a Cartesian coordinate system. This and similar tracking equipment has been validated for use in similar human studies (29, 41). Given the high sensitivity of the tracking equipment to positional change, its use in this study is justified. Furthermore, nylon leader line was used to attach the turnbuckles to the patella and femur/tibia to mimic the quadriceps and gastrocnemius musculature, respectively. This may represent one potential limitation to the study design as nylon can "creep" while under load and potentially affect the position of the limb construct. However, the

significance of creep in the current model is likely insignificant given the time required for data acquisition using the electromagnetic tracking system (0.004 seconds[c]). Additionally, joint angles were determined using goniometry. Goniometry has been proven to be as accurate as radiography in measuring joint angles (42). However, the use of goniometry may have lead to slight variation of joint positioning throughout testing which could potentially affect the results. Furthermore, because goniometry was used as the sole means to adjust the joint angles during testing, an actual value for the angles tested cannot be reported. However, given the results of the previous studies (42), a standard deviation of no more than ±3° was expected for joint angular adjustments using goniometry and its use in the current study is justified.

The effects of a medial meniscal release on cranial/caudal stifle stability when performed in the normal stifle, CrCL deficient stifle, and TPLO treated stifle have been previously reported (35). In this study we only evaluated the effects of a medial meniscal release on cranial/caudal and rotational stability only in stifles previously treated with a TTA. While this limits our ability to make conclusions regarding the contribution of the medial meniscus to stifle stability in the normal and CrCL deficient limbs, it does provide evidence of the effects of a meniscal release on stifle stability when performed in conjunction with TTA.

FOOTNOTES

[a] Technovit, Jorgensen Laboratories Inc; Loveland, CO, USA.

[b] Synthes Oscillating Bone Saw, Serial No. 5789; West Chester, PA, USA.

[c] FASTRAK, Polhemus; Colchester, VT, USA.

[d] SAS 9.2, SAS Institute; Cary, NC, USA.

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CHAPTER III

CONCLUSION

TTA effectively eliminates cranial tibial displacement during early, middle, and late stance as depicted in this in vitro model. TTA failed to consistently restore the rotational alignment of the stifle, although the magnitude of tibial rotation noted in this study is of questionable clinical significance. Release of the medial meniscus did not result in any significant cranial/caudal or axial rotational joint displacement when performed in conjunction with the TTA. Further in vivo and in vitro research is warranted to evaluate the biomechanical effects of TTA and meniscal release on tibial translation and axial rotational using the femorotibial tangent as a reference in surgical planning, with further extension of the stifle beyond 145°, and under loading conditions other than straight-line walking.