Effect of tibial insertion points for lateral suture stabilization on the kinematics of the cranial cruciate ligament deficient-stifle during early, middle and late stance: An *in vitro* study

By

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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

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March 8, 2013
Blacksburg, VA

Keywords: Cranial cruciate ligament, lateral suture stabilization, cranial tibial translation, kinematic, electromagnetic tracking sensors

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ABSTRACT

Objective: To evaluate the effect of two tibial attachment sites for lateral suture stabilization (LSS) on the kinematics of the cranial cruciate ligament-deficient (CrCL-D) canine stifle during early, middle and late stance.

Study design: *In vitro* biomechanical study: 32 hind limbs from 16 canine cadavers.

Methods: Limbs were mounted in a testing jig and an electromagnetic tracking system was used to determine 3-D stifle kinematics under 33% body weight load during early, middle and late stance in the following sequence: CrCL intact, CrCL-D and LSS with the distal anchor through the tibial tuberosity (LSS_{TT}) or through the cranial eminence of the extensor groove (LSS_{EG}). The proximal anchor point was the lateral femoro-fabellar ligament.

Results: Transection of the CrCL resulted in significant changes in stifle kinematics during early, middle and late stance. Post-LSS stifle kinematics were more comparable to normal than post-transection kinematics for both techniques. Both LSS techniques restored stifle kinematics in CrCL-D stifles to varying amounts but neither technique successfully restored normal 3-D stifle kinematics. LSS_{EG} improved kinematics of the CrCL-D stifle in the medial-lateral direction and axial rotation but performed poorly in restoring stifle kinematics in the cranial-caudal plane as compared to LSS_{TT}.

Clinical significance: LSS_{TT} and LSS_{EG} techniques failed to completely restore normal stifle kinematics in CrCL-D stifles *in vitro*.

DEDICATION

First of all I pay homage to Supreme Being, the merciful and compassionate for his benison that developed spirit and courage in me to make this attempt successful.

I would like to thank my parents, Karnail Singh Aulakh and Gurpal Kaur Aulakh, for their encouragement, love and support. You have taught me the meaning of hard work and sacrifice. Your prayers, good wishes, and interest are specially treasured in my heart.

Mere words of thanks would never express the sense of thanks to the person who is not only my best friend but my backbone and strength throughout this endeavor, my lovely wife, Harmeet Kaur Aulakh, who said 'okay' to everything and encouraged me through every step of this journey. I could not have done this without you.

I pay my sincere thanks to my brother, Gurwinder Paul Singh Aulakh, his wife Rajanjeet Kaur Aulakh, and their sons, Udayvir Singh Aulakh and Udaypaul Singh Aulakh, for their love and support.

I express my special thanks to my father-in-law Surjit Singh Saini and mother-in-law Chanchal Kaur Saini, my sister-in-laws; Loveleen Garg, Harkirat Kaur Saini and Brother-in-laws; Suresh Pal Garg and Satinder Mehru and my nices Kashish Garg and Shilpa Garg for their support, love and help. Their whole hearted devotion, care, espouse and never ending benediction that have brought prolific results.

I am also extremely thankful to my Uncle Gurjant Singh Sidhu and Aunt Jaswinder Kaur Sidhu and cousins Ramanpreet and Gagandeep for their heart full blessings, selfless affection and moral support that enabled me to pursue my studies with unfailing zeal.

I express my thanks to my friends "RUFFIANZ", Rubba Sadhu, Jassi, Saket, Joshi, Mann, D.P., Matharoo, Brar, Dham for their support and help.

I am highly thankful to my friends Laura, Julia, Kate and Dominique for their cooperation and best wishes for the success of this study.

To my loving daughter, Gurbani, who came into our lives towards the middle of this journey. You are the best thing that has ever happened to us and we are truly blessed.

All might not have been mentioned but none is forgotten.

Dated:

Signatures,
(Karanvir Singh Aulakh)

ACKNOWLEDGEMENTS

Any formal statement of acknowledgement can hardly express my gratitude to those who have helped me in one way or the other during my period of study here.

I would like to thank the following people for their invaluable contributions during this study:

Graduate committee – Dr. Otto I. Lanz (graduate advisor), Dr. Tisha A.M. Harper, and Dr. Ron M. McLaughlin for their ongoing support and assistance in project design and document preparation as well as guidance and encouragement throughout the duration of this program. I am truly honored to work with such a distinguished group of educators.

Assistance in project design and document preparation- Dr. Laura D'Amico and Dr. Ryan Butler

For providing the custom-built mounting bracket and loading frame- Dr. Steve Elder

Statistical analysis- Dr. Stephen Werre

Assistance with obtaining and storing specimens and use of the anatomy laboratory- Pam Arnold

Securos Inc. Veterinary Orthopedics, 443 Main Street Sturbridge, MA 01566 - for their donation of the equipment; PowerX Basic Cruciate Repair 80# Kit.

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INTRODUCTION

The canine cranial cruciate ligament (CrCL) is the most widely studied ligament in veterinary medicine. Rupture of the CrCL is frequently associated with pelvic limb lameness and is the most common condition to affect the canine stifle (1). Due to the frequency and economic impact (over $ 1billion) of this disorder it is understandable that a significant amount of interest is devoted to developing a technique that addresses the CrCL-deficient (CrCL-D) stifle (1). The stifle is a complex structure capable of translation and rotation in three planes (1, 2). The complex nature of the stifle has made the development of a consistently effective surgical technique challenging. None of the developed techniques have proven superior in the re-establishment of normal limb function or prevention of the progression of osteoarthritis (3). Potential causes for progression of osteoarthritis include altered joint kinematics, abnormal biological changes within the joint and altered joint contact mechanics (4).

The CrCL is one of the primary stabilizing structures of the canine stifle and its three main functions are: preventing cranial tibial translation (CTT), preventing hyperextension of the stifle and limiting tibial internal rotation (5). In the normal stifle, motion during the stance phase is mainly limited to flexion and extension, while in the swing phase motion occurs in three (X, Y, Z) planes (2, 6). Loss of the CrCL leads to changes in stifle motion such that motion in three planes is present during both the swing and stance phases (2). Additionally there is a significant internal tibial rotation and CTT that occurs throughout the stance phase in the CrCL-D stifle (2, 6). These changes in gait kinematics are thought to alter the load distribution across the articular surfaces of the femur and tibia promoting the development of osteoarthritis (7). The ideal surgical technique to address a CrCL-D stifle should restore normal stifle kinematics throughout the entire gait cycle and prevent the progression of osteoarthritis.

The various surgical techniques for stabilization of the CrCL-D stifle can be classified as intra-articular, extra-articular and corrective osteotomies (8-10). A survey showed that board certified veterinary surgeons preferred extra-articular techniques for repair of CrCL rupture in both small and larger dogs (11). Extra-articular procedures attempt to stabilize the stifle either by transposing autogenous structures or by using synthetic suture materials. (8, 10, 12). Outcomes after lateral suture stabilization (LSS) which is a type of extra-articular technique, have been suggested to be inferior to those seen after corrective tibial osteotomy procedures (13). However, many head-to-head prospective studies have shown no differences in outcomes between the LSS and the tibial osteotomies based on client- and veterinarian-based subjective assessments, force plate gait analysis, radiographic progression of osteoarthritis, or muscle mass measures (3, 14, 15). The LSS procedure has some potential advantages including relative technical ease, the safety profile and a low cost for surgical equipment (13). The traditional LSS procedure relies on placement of a suture around the lateral fabella and through a hole drilled in the cranial proximal aspect of the tibia. After passing the suture under the patellar tendon, the ends of the suture are secured with a knot or a crimp clamp on the lateral aspect of the joint (13). The purpose of this orientation is to prevent cranial translation and internal rotation of the tibia during weight-bearing. The LSS procedure transiently restrains abnormal stifle motion until sufficient joint adaptation (peri-articular fibrosis) occurs to provide functional stability and improved limb function.

Although most owners are satisfied with the results after LSS, most dogs do not regain normal function of the affected limb (8, 14, 16). LSS has also been shown to fail to mitigate the radiographic progression of osteoarthritis, similar to other stabilization techniques (1, 3, 15). LSS has evolved in recent years in an effort to improve the clinical outcome. Various technical modifications of the LSS have been reported including changes in suture material, methods of securing suture material, and the location of attachment sites (17-21). It has been suggested that the accurate identification and use of isometric attachment sites is critical to successful outcome for dogs treated with LSS as non-isometric positioning of suture attachment sites at the time of repair may lead to premature suture loosening and progression of osteoarthritis (13, 18). For example, if during joint range of motion the suture attachment sites move away from one another, the suture will tighten which may limit flexion and extension, or overload or break the suture (18). If, the attachment sites move closer to one another, the suture will become loose and may fail to fully control cranial translation of the tibia (18). For optimal tibial fixation, two recent studies suggested attaching the suture at a point just cranial or caudal to the long digital extensor groove (cranial or caudal eminence of the extensor groove) or just caudal to the tibial attachment of the patellar ligament (18, 19).

In theory, use of these 'near-isometric' fixation points in LSS procedures would allow for better re-establishment of stifle kinematics that could result in an improved functional outcome. There are few reports documenting the kinematic or biomechanical effects of sutures placed at these reported isometric points for repair of CrCL-D stifles. Two *in vivo* experimental studies described the three dimensional (3-D) kinematic changes that occur in the absence of the CrCL (2, 6). While each of these studies evaluated the stifle through a range of motion, none assessed the effect of the LSS. Two cadaveric biomechanical studies investigated CTT and axial tibial rotation after LSS at a standing stifle angle of 135^{o} (17, 22). The authors of these studies did not find any significant differences in CTT between stifles stabilized with the LSS technique and intact stifles. 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 120° to 145° during stance phase (23). Moreover, in these two studies loads were not applied to mimic weight bearing. In one of these studies tibial rotation exceeded instrumentation limits (22), whereas in the second study only the direction of tibial rotation was measured not the degree of rotation (17). Only one study in the veterinary literature has evaluated CTT and axial tibial rotation during the non-weight bearing phase (30 to 95 degrees of stifle flexion) after LSS (24). In that study, two lateral and one medial suture were used, and CTT was normally restored. However, this finding does not relate to the clinical situation as cranial tibial thrust is generated by weight bearing.

Stifle kinematics and biomechanics after tibial osteotomy procedures have been previously evaluated (25-28). However, to the best of our knowledge, the effect of the LSS technique on stifle kinematics during the stance phase of gait has not been thoroughly evaluated.

CHAPTER I: LITERATURE REVIEW

A. Canine stifle anatomy, structure and function

The canine stifle is the articulation between the femur and the tibia, and the femur and the patella. This joint is a complex diarthroidal joint capable of translation and rotation in three planes. While the primary movement of the stifle is flexion-extension the anatomy of the joint also allows for cranial-caudal translation, medial-lateral translation, internal-external rotation, varus-valgus angulation, and compression-distraction (2). The primary weight bearing occurs across the femoro-tibial articulation, which comprises the articulation between the thick, roller-like condyles of the femur and the flattened condyles of the tibia (1). The femoro-patellar articulation communicates freely with the femoro-tibial articulation. The femoro-patellar articulation greatly increases the mechanical efficiency of the quadriceps mechanism to maintain stifle extension (1). The quadriceps mechanism consists of the large rectus femoris muscle and three smaller muscles: the vastus lateralis, medialis, and intermedius.

The stifle is composed of three long bones: distal femur, proximal tibia, proximal fibula; four sesamoid bones: patella, popliteal, and medial and lateral fabellae; four major ligaments: cranial and caudal cruciate, medial and lateral collateral; two menisci; a joint capsule; and multiple muscles [14]. Each of these components works in concert to contribute to the dynamic stability of the stifle.

The distal end of the femur contains three articular areas, one each on the medial and lateral femoral condyles and the third within the femoral trochlea. The trochlea is a smooth, wide-grooved area located on the cranial surface on which the patella articulates. The medial ridge of the trochlear groove is slightly thickened compared to the lateral (29, 30). Axially the condyles each articulate directly with the tibial plateau, whereas abaxially the condyles articulate with the respective menisci (29). The medial and lateral femoral condyles are convex in both sagittal and frontal plane and are separated by the intercondyloid fossa. The medial condyle is smaller in size and less convex in shape as compared to the lateral condyle. This asymmetry assists in the relatively asymmetric motions of each condyle during range of motion; the lateral condyle is able to move more caudally with respect to the articular surface of the tibia as the stifle progresses through flexion. The lateral condyle contains a small depression cranially lateral to the lateral trochlear ridge known as the extensor fossa (29, 30).

The patella is a sesamoid bone located cranially and proximally in the stifle. There are three other sesamoid bones associated with the stifle, two of which are located in the tendons of origin of the medial and lateral gastrocnemius and the third is located in the tendon of origin of the popliteus muscle. The proximal tibia is relatively flat and is composed of medial and lateral condyles. The articular surface is often termed as the tibial plateau. The area between the tibial condyles is divided into a cranial intercondylar area, intercondylar eminence and caudal intercondylar area. The intercondylar eminence has a medial and lateral intercondylar tubercles that are articular on their abaxial surface. Cranial to the cranial intercondylar area is the tibial tuberosity where the patellar ligament inserts. The medial and lateral tibial condyles are the sites of articulation with the medial and lateral femoral condyles, respectively. Each condyle is concave transversely, convex sagitally and sloped in a caudal direction when viewed from the side. The caudo-lateral aspect of the tibial surface contains an oblique depression for articulation with the head of the fibula. The fibula is a long thin bone that contributes little to weight bearing and primarily functions as a site for ligamentous and muscular attachments.

The stifle joint capsule is formed by the patellar, lateral femoro-tibial, and medial femoro-tibial pouches (1, 30). The joint capsule of the stifle which is largest in the body is made up of two layers. The inner synovial layer contains vascular and nerve supply. This layer is a specialized connective tissue that produces synovial fluid and phagocytic synoviocytes. The outer fibrous layer is composed of dense, inelastic fibrous connective tissue. The normal canine stifle contains 0.2 to 2 ml of a viscous, transparent, colorless, fluid that is primarily composed of water and a strongly polymerized hyaluronic acid (1, 30).

In the stifle there are four femoro-tibial ligaments, the medial and lateral collaterals and cranial and caudal cruciates that provide primary ligamentous support. The cruciate ligaments are intra-articular but are considered extrasynovial as they are covered with synovium. These ligaments are called cruciate ligaments because they cross each other. The CrCL originates on the caudo-medial aspect of the lateral femoral condyle and extends cranio-distally and medially to insert on the cranial intercondylar region of the tibia (5). Functionally each cruciate ligament has two bands. The CrCL is composed of a smaller cranio-medial and a larger caudo-lateral band. During extension both bands are taut; in flexion only the cranio-medial band is taut. The CrCL prevents cranial displacement of the tibia in relation to the femur, limits internal tibial rotation in conjunction with the caudal cruciate ligament (CdCL) and prevents hyperextension of the stifle (31).

The canine CrCL has distinctive histologic features typical of dense collagenous connective tissue. The predominant cell type is the fibroblast and the extracellular matrix proteins are primarily composed of type I collagen (32). Histologically, the central region of a normal canine CrCL is formed of highly ordered, parallel arrays of collagen fibers. The central core regions of the cruciate ligaments are not as well vascularized as the proximal and distal portions. The most frequent degenerative changes occur in this central core region of the CrCL. Minor injury and stress resulting from twisting of the CrCL on the CdCL further reduces blood supply which limits the ability of the cruciate ligaments to be repaired and results in weakening of the ligaments. The synovial membrane covering the CrCL is more cellular than the ligament and is made up of fibroblasts and dense connective tissue (33). There is no differentiation between the cranio-medial and caudo-lateral bands of the CrCL based on histology (33).

The CdCL is longer and larger than the CrCL. It originates from the lateral aspect of the medial femoral condyle and extends caudo-distally crossing medial to the CrCL to insert on the medial aspect of the popliteal notch of the tibia (1, 31). There are two bands in the CdCL; cranial and caudal, these bands are much less distinct than the bands of the CrCL. The cranial band is taut in flexion and loose in extension and the caudal band is loose in flexion and taut in extension (1, 5). The CdCL prevents caudal translation of the tibia with respect to the femur and limits internal rotation through a twisting sequence with the CrCL (1, 5).

Mechanoreceptors (pressure-sensitive corpuscles) and free nerve endings have been identified in the canine CrCL (34). The presence of these mechanoreceptors indicates that the cruciate ligaments have a role in proprioception (35). The canine CrCL seems to have more mechanoreceptors than other species. The highest number of these receptors are present in the proximal third and the lowest numbers in the distal third of the CrCL (34). The free nerve endings are mainly nociceptive which react to pain stimuli and inflammation. The main innervation to the stifle peri-articular tissues is supplied by medial, caudal and lateral articular nerves which are branches of the saphenous, tibial and peroneal nerves respectively (36). The caudal articular nerve is variably present in the dog (36).

The medial collateral ligament originates from the medial epicondyle of the femur, fans out as it extends distally forming attachments with the joint capsule and medial meniscus and inserts on the proximal medial aspect of the tibia (37). This ligament has two parts; a cranial part that remains taut in both flexion and extension and a caudal part that is lax in flexion and taut in extension (37). The lateral collateral ligament originates on the lateral femoral epicondyle of the femur, extends caudo-distally forming a loose attachment to the joint capsule, and inserts on the head of the fibula. In flexion the ligament is loose but it is taut in extension (29, 37). Both collateral ligaments function to minimize varus and valgus motion of the tibia relative to the femur. The collateral ligaments are the primary restraint against tibial rotation in stifle extension whereas in stifle flexion the cruciate ligaments provide the primary check against rotation of the tibia (1, 37). The thin medial and lateral femoro-patellar ligaments, which are a continuation of the femoral fascia, run from the patella to the fabellae (29, 30).

The descending genicular artery is considered to be the predominant blood supply to the stifle; it originates from the femoral artery and courses disto-medially to supply the vastus medialis muscle and medial femoro-patellar and femoro-tibial joint capsule (38). The major blood supply to the cruciate ligaments originates from branches of the genicular arteries on the lateral and medial aspect of the stifle (38). These branches within the synovial tissues that ensheath the cruciate ligaments penetrate the ligament transversely and freely form anastomoses with longitudinal endoligamentous vessels (38). The infrapatellar fat pad and tissues caudal to the joint capsule also contribute. The lack of direct vascularization leaves the mid-section of the ligaments without a substantial blood supply. The veins important for stifle drainage include, descending and medial genicular veins, cranial and caudal tibial veins, popliteal vein, medial and lateral saphenous veins, distal and middle caudal femoral veins and femoral vein (39). The important lymphatic drainage of the stifle consists of the popliteal, femoral, and superficial inguinal lymph nodes and the superficial medial and lateral systems and the deep medial system (40).

Many muscles in the pelvic limb are important components of the stifle as they contribute to stifle motion. These muscles can be divided into two groups; flexors and extensors of the stifle. Some of these muscles can assist in both flexion and extension depending on the anatomical region of the muscle that is activated. Of the rump muscles the contraction of the tensor fasciae latae results in mild extension of the stifle. This muscle is innervated by the cranial gluteal nerve (29, 30). The quadriceps femoris is the primary extensor of the stifle. This muscle is composed of 4 separate muscles: the rectus femoris and vastus medialis, intermedius and lateralis. The quadriceps muscle group covers the cranial, medial, and lateral aspects of the femur. The rectus femoris originates from the ilium and the remaining three muscles arise from the proximal femur. All four muscles converge to insert on the tibial tuberosity via the patellar ligament. Contraction of this muscle group extends the stifle by forcing the patella into the trochlear groove. The quadriceps muscle group also imposes a cranio-proximal force on the tibia (41). This combination of forces acts as an antagonist to the function of the CrCL as one of the functions of the CrCL is to limit cranial translation of the tibia relative to the femur (30, 41).

Another muscle group that works in conjunction with the quadriceps to extend the stifle is the musculus articularis which originates on the cranial portion of the distal femur proximal to the patellar pouch of the femoro-patellar joint (30). Another muscle that contributes to stifle extension is the biceps femoris muscle. It has a cranial and caudal head. The cranial head originates from the sacrotuberous ligament and ischiatic tuberosity and the caudal head originates from the ischiatic tuberosity (29, 42). The cranial head inserts by means of an aponeurosis with the fascia lata onto the patella, patellar ligament, and the tibial tuberosity via the patellar ligament (42). This orientation allows the cranial head to function in both stifle flexion and extension (29, 30). The caudal head directly inserts on the cranial aspect of the tibial tuberosity. A portion of this insertion also continues distally to insert on the calcanean tuberosity(42). This arrangement allows contributions to stifle flexion and hock extension (29, 30). The caudal head does not assist in stifle extension. The biceps femoris muscle also imposes a caudal force on the tibia (41).

The sartorius muscle is located cranially and has cranial and caudal parts like the biceps femoris (42). The cranial portion which contributes to stifle extension originates at the iliac crest and inserts on the medial fascia of the femur proximal to the patella (42). The caudal portion contributes to stifle flexion (30). It originates from the cranio-ventral ilium and inserts on the medial aspect of the cranial tibia. The caudal portion imposes a caudal force on the tibia (41).

The semitendinosus muscle is present on the caudal aspect of the thigh and it contributes to stifle flexion (30, 42). It originates on the ischiatic tuberosity and runs medially and distally to insert on the medial aspect of the proximal tibia. The semimembranosus is another important flexor muscle of the stifle which is present on the caudal and medial aspect of the thigh (30, 42). This muscle has a cranial head which originates at the ischiatic tuberosity and inserts at the distal femur. This part of the muscle does not contribute to stifle flexion. The caudal head also originates on the ischiatic tuberosity and this part contributes to stifle flexion (30, 42). The caudal part inserts at the medial tibial condyle under the medial collateral ligament. The semimembranosus muscle imposes a caudal force on the tibia (41). The gracilis muscle also imposes a caudal force on the tibia and contributes to stifle flexion (30, 41). This muscle originates from the pelvic symphysis and inserts on the cranial tibial border and also plays an important role in limb adduction (42).

The anatomical position of the gastrocnemius muscle effects stifle stability but it does not play a direct role in stifle flexion and extension (30, 42). However, when the tarsus is fixed contraction of the gastrocnemius muscle can weakly flex the stifle (41). The muscle is located caudally and has medial and lateral heads which originate from the medial and lateral supracondylar tuberosities of the femur, respectively. The two heads fuse distally and insert on the calcanean tuberosity (30, 42). Contraction of the muscle causes hock extension. In addition, contraction imposes a caudo-distally directed force on the distal femur. This force is counteracted by the CrCL in the normal stifle whereas in the CrCL-D stifle this force contributes to the cranial translation of the tibia with respect to the femur (41).

The popliteus muscle plays a unique role in the stifle. It promotes internal rotation of the tibia relative to the femur, provides lateral stability to the stifle and extends the stifle (30, 42). It originates from the lateral condyle of the femur, spans the caudal aspect of the stifle and inserts on the caudal surface of the proximal tibia (42). Its origin contains the popliteal sesamoid.

The menisci are crescent-shaped fibrocartilaginous disks that are located between the condyles of the femur and the tibia (43). Both medial and lateral menisci are composed of a body and two horns, caudally and cranially. Peripherally the menisci are thick and convex but become thinner and concave as they slope centrally (29, 44). The medial meniscus is larger than lateral and more ovoid in shape (30, 43). Both lateral and medial menisci have attachments that influence their mobility and function. The cranial and caudal horns of each meniscus are firmly attached to the tibia via cranial and caudal menisco-tibial ligaments (1). The caudal horn of the lateral meniscus is also firmly attached to the femur by the menisco-femoral ligament whereas the medial meniscus lacks this attachment. Firm attachments are also present between the abaxial aspect of the medial meniscus and the medial collateral ligament and the joint capsule, but the lateral meniscus lacks these attachments (43). Additionally, the intermeniscal ligament attaches the two menisci cranially. The lateral meniscus is less firmly attached to the tibia than is the medial meniscus. During flexion of the stifle the caudal horns of the menisci move caudally. The increased mobility of the lateral meniscus allows for relatively more caudal transposition of the lateral femoral condyle during stifle flexion (1).The increased mobility of the lateral meniscus helps to explain the low incidence of severe lateral meniscal tears secondary to CrCL rupture when compared to the medial meniscus.

Menisci are primarily composed of water (60-70%) and also contain an interlacing network of collagen fibers interposed with cells and matrix of proteoglycans, and glycoproteins (1, 45). The collagen fibers in the menisci are type I and type II collagen, with type I collagen making up 90% of the collagen content (1). In the inner one-third of the menisci collagen fibrils are oriented radially, which may function to counteract compressive forces. In the outer two thirds of the menisci collagen fibrils are oriented circumferentially, which may function to counteract tensile forces (1, 45). Less frequently observed are radial fibers (or tie fibers) which can be present throughout the menisci, connect circumferential fibers and may function to counteract longitudinal splitting (1).

Both menisci demonstrate a common distribution and pattern of blood vessels that arise from branches of the medial and lateral genicular arteries. These branches supply the joint capsule which gives rise to vessels that penetrate the peripheral 15 – 25% of the width of the menisci (43, 46). This region is called the red-red zone because of the rich blood supply. The rest of the meniscus is divided into the intermediate zone called red-white zone and the axial zone called white-white (46). A large portion of the menisci rely to a large extent on the synovial source of nutrition.

The menisci improve stifle joint congruity due to their crescent shape (29, 44). The cranial and caudal poles of the menisci contain mechanoreceptors, which allow pressure to be detected and may also allow exchange of proprioceptive information (45). They also function in load transmission, deepen the articular surface of the tibial plateau to provide joint stability, aid in joint lubrication and the rich supply of nerve endings provides proprioceptive information to help coordinate muscular adaptations to joint load (1, 45). Menisci are able to absorb high loads across the joint by converting the compressive forces into radially directed forces. As the meniscus is loaded the cranial and caudal attachments of the meniscus are tensioned along with the circumferential fibers and this tension is called "hoop tension (1, 47)." Transection of the meniscus has been shown to disrupt the primary function of the meniscus because it eliminates the hoop tension (1, 47).

B. Cranial cruciate ligament disease

B.1. Progressive degeneration of the cranial cruciate ligament

The majority of the CrCL ruptures are due to the progressive degeneration of this ligament (1, 48). Deterioration of the cruciate ligament has been shown to be a natural process that occurs with age (49). The exact cause and pathogenesis of this extremely common disorder remains elusive. The CrCL is composed of 90% Type I collagen fibrils with the remainder being Type III collagen fibrils (32, 33). These fibrils are divided into smaller subfibrils and microfibrils and have a crimped arrangement (32, 33). Fibroblasts are the main cell type present supporting the collagen and consist of 3 separate types: fusiform, ovoid, and spheroid (32). With age the overall population of fibroblasts within the ligament decreases, some fibroblasts undergo metaplasia to form chondrocytes and the functional arrangement of the fibrils becomes disrupted (49). As ligament degeneration is a natural biologic process in dogs, failure of the ligament is likely attributable to other factors as not every aging dog develops cranial cruciate disease. Several factors have been implicated to play a role in the pathogenesis of progressive degeneration of CrCL. These factors include: abnormal conformation and gait, increased tibial plateau angle, neutering, body weight, obesity, genetics, breed, inactivity, lack of fitness, inflammatory factors and extracellular matrix (ECM) changes (1, 43, 49-57)

The variations in canine pelvic limb conformation such as narrow femoral intercondylar notch, straight stifle angle (i.e. prominent hyperextension of the stifle in a normal standing position) and steep tibial plateau slope have been associated with degeneration of the CrCL (58, 59). The presence of a straight stifle and narrow intercondylar notch may cause impingement and abnormal compression of the CrCL against the intercondylar notch (58). The resultant strain and micro-injury to this ligament can cause damage to the inter-ligamentous fibers even during normal activity. This damage is unable to adequately heal due to poor vascularity of the center of the CrCL and the ongoing strain on the ligament. The width of the intercondylar notch and its association with CrCL and anterior cruciate ligament (ACL) rupture in dogs and humans respectively has been well described (60, 61). The intercondylar notch width indices were significantly greater in the low-risk breed (Greyhounds) compared to high risk breeds (Labrador and Golden Retrievers) in a cadaveric study (58). The authors concluded that impingement by the intercondylar notch on the CrCL of high risk breeds may result in reduced structural integrity of the ligament, predisposing the ligament to increased laxity and degeneration. However, a radiographic comparison of other morphological features of the distal femur showed no anatomical differences in the distal portion of the femur between dogs with and without CrCL rupture (62).

Many studies have evaluated the association of the tibial plateau angle (TPA) and CrCL rupture (56, 59, 63, 64). The relationship between the CrCL pathogenesis and the conformation of the proximal aspect of the tibia originates from a biomechanical model developed by Slocum (44). This model explains that the ground reaction forces generated during the stance phase are transmitted along the longitudinal axis of the tibia and a cranially directed femoro-tibial shear force also called cranial tibial thrust is generated by the compression of the femur against the caudally oriented slope of the tibial plateau. Although differences in anatomic shape of the proximal tibia have been shown in dogs with CrCL rupture, the correlation between the steepness of the tibial plateau and the development of CrCL degeneration remains controversial (63, 64). A wide range of TPA has been reported in the dog with a mean of approximately 24^{o} (65). Greater TPAs were present in diseased stifles in a mixed population of dogs with CrCL deficiency compared with a normal group (57). This was further strengthened by similar findings in contralateral stifles of the affected dogs (66). However, other studies have shown that the TPA is not significantly different in Labrador Retrievers with and without CrCL degeneration (63, 64). TPA measured on dogs in a standing position was similar between Greyhounds and normal Labradors, eliminating differences in standing angles as a confounding factor (64). Recently, a study describing the morphometric characteristics of the pelvic limbs of Labrador retrievers with and without CrCL deficiency suggested that excessive TPA, cranial angulation of the proximal tibia, and distal femoral torsion appeared more likely to be associated with CrCL degeneration than tibial torsion, femoral angulation, and intercondylar notch stenosis (67). The true effect of TPA on CrCL stresses *in vivo* is currently unknown, because other factors discussed in this section of the chapter (see above and below) can influence the amount of stress sustained by the CrCL, in addition to the TPA.

Neutering also increases the risk for cruciate disease, although the cause of this effect is not understood (48, 50). Female dogs have an increased prevalence of CrCL disease compared with male dogs (48, 50). Age at the time of ovariohysterectomy was not associated with the prevalence of cruciate disease (50). Elevated estrogen levels are associated with increased incidence of ACL in women (68). However, as ovariohysterectomy in dogs is associated with persistent hypoestrogenemia, estrogen may reduce the incidence of CrCL degeneration in the dog (69). To date, there are no conclusive studies, in dogs as well as humans, determining the effect of hormonal status on CrCL degeneration.

Larger dogs weighing >22 kg are at greater risk of cruciate disease and tend to develop CrCL rupture at younger ages as opposed to smaller dogs (< 22kg) who tend to be affected later in life (48, 50). This epidemiological finding is consistent with histologic analysis showing that the CrCL of dogs weighing >15kg generally showed more severe degeneration of the ligament than those of smaller dogs, and the onset of degenerative processes started several years earlier in larger dogs. It remains unclear whether obesity is also a risk factor for the disease independent of the dog's size. Repetitive loading has been suggested to result in overuse and fatigue failure of the CrCL (32, 43). Obesity could increase the magnitude of forces applied to the CrCL and could contribute to the incidence of CrCL deficiency.

A breed predisposition has been reported in certain breeds including the Newfoundland, Labrador retriever, Chesapeake Bay retriever, Rottweiler, Akita, Boxers, Neapolitan mastiff, Saint Bernard and Staffordshire bull terrier(48, 50). This breed-specific variation in incidence of

CrCL rupture has been considered strong evidence for a genetic component to the condition (48, 50). It has been shown that Rottweiler breed requires half the load per unit body weight for CrCL rupture to occur as compared to the greyhound suggesting that different breeds have different biomechanical properties of the CrCL (70). Heritability indices for CrCL disease have been reported in different breeds (1, 71). In the Newfoundland breed a prevalence for CrCL deficiency of 22% has been reported (71). The authors proposed a recessive mode of inheritance with 51% penetrance. In another study, 4 microsatellite markers associated with CrCL rupture were found in chromosomes 3, 5, 13 and 24 in the Newfoundland breed (51). Even though there was high prevalence and odds ratio in that breed, 73% of the phenotypic expression of CrCL disease was attributable to environmental factors and only 27% was linked to genetics (71). It still remains unclear whether genetics exert a direct influence on the structural properties of the CrCL. Future research is warranted to identify gene markers for CrCL disease which may contribute to the control of CrCL deficiency in the dog.

There is a paucity of studies determining the effect of exercise on the properties of the canine CrCL. Simultaneous contraction of the caudal thigh muscles and relaxation of the quadriceps muscles has been noted with any joint load that causes increased strain in the CrCL (72). This is suggested to be mediated by mechanoreceptors and proprioceptors in the CrCL and this mechanism is protective of the CrCL (34, 35). Therefore it is possible that poor physical condition and/or inactivity may mitigate this protective mechanism leading to repetitive stress to the CrCL and eventually rupture. The results of an experimental study showed that a lifetime of regular weight-bearing exercise in dogs did not cause alterations in the stifle joints, its associated ligaments and cartilage structures (73). There was also no evidence of erosions or osteophytes in the joint. It has also been suggested that inactivity may predispose ligaments and tendons to altered and detrimental homeostasis. Controlled and regular exercise should be recommended as it does not appear to cause any articular and peri-articular tissue damage of canine stifle joints.

Both humoral and cellular immune mechanisms may result in CrCL disease but their exact role remains unclear (74). Type I and II anticollagen antibodies have been detected in dogs with CrCL disease (75, 76). Antibodies are present in both the synovial fluid aspirates from the affected joint and the serum, with a higher incidence in the synovial fluid indicating local antibody production (75). The specificity of these antibodies to CrCL disease however has not been documented. Antibodies to collagen type-I in the synovial fluid were quantified in dogs with diseased stifles, contralateral stifle and left shoulder over 12–18 months (77). The study showed that the antibody titers were higher in dogs with partial compared with complete CrCL tears (77). This finding may reflect continued antigenic stimulation and release of collagen type I from the damaged ligament into the articular space. In general titers were higher in contralateral stifles that eventually sustained a CrCL rupture (77). However, not all dogs with high antibodies developed a contralateral CrCL rupture. The Type I and Type II anticollagen antibodies are not specific to the type of joint disease process as these antibodies were also detected in the synovial fluid of stifles with arthritis secondary to arthropathies other than CrCL disease (75, 76).

Lymphocytic–plasmacytic synovitis in the CrCL-D stifle has been identified in about 67% of cases (78). The significance of the inflammatory cells is still poorly understood. It has been suggested that they are indicative of a cell-mediated immune response. Variable amounts of synovitis and inflammatory cells including B and T-lymphocytes, IgG+, IgM+ and IgA+ plasma cells, , CD1c^{+} MHC class II^{+} dendritic cells and tartrate-resistant acid phosphatase-positive or TRAP^{+} (activated) macrophages are present in CrCL-D stifles (78, 79). Antigen stimulated dendritic cells are believed to activate T-lymphocytes which engage other inflammatory cells and activate an inflammatory cascade leading to synovitis and ligament degeneration. Recently sequestration of bacteria has been proposed as a causative factor for the inflammatory component of CrCL deficiency. Bacterial DNA was isolated from synovial biopsies in 7% of 16 stifles with experimental transection of the CrCL, 37% of 43 dogs with naturally occurring CrCL deficiency, and none of 12 normal dogs (80).

Recent studies have investigated the ECM metabolic changes in ruptured CrCL (32, 79). Compared with the intact CrCL, ruptured CrCL has significantly higher levels of water content, glycosaminoglycans (GAG) and immature cross links (81). The levels of pro-matrix metalloproteinase-2 (inactive MMP-2) were also significantly higher in ruptured CrCL. The authors suggested that the ECM of ruptured CrCL had an increased matrix turnover indicated by higher GAG and collagen synthesis. It still remains undetermined if these changes occur before ligament rupture or if these observed changes are part of a reparative process after rupture. Another study comparing the ECM properties of grossly normal CrCL between high risk breeds (Labrador Retrievers) and low risk breeds (Greyhounds) showed that there were increased concentrations of pro-MMP-2 and decreased integrity of the collagen triple helices in the CrCL from high risk breeds (82). This altered collagen matrix metabolism may be responsible for decreased ligament strength and eventual rupture of CrCL. Ruptured CrCL also contains higher concentrations of tartrate-resistant acid phosphatase (TRAP), interleukin-1β, cathepsin S and cathepsin K, MMP-2, MMP-9, MMP-6 (54, 55). The author suggested that the collagenolytic activity of these proteinases contributes to the remodeling within the CrCL. A follow up study documented a higher concentration of cathepsin K and TRAP in the synovium as well as in the CrCL in stifles with ruptured CrCL (79).

The pathologic changes present in the ruptured CrCL have been described. The changes were characterized by loss of ligamentocytes, failure to maintain collagen fiber bundles and metaplasia of surviving ligamentocytes to chondrocytes (49, 83). The population of fibroblast subtypes was also altered with increased numbers of spheroid cells in the core region, and decreased fusiform and ovoid cells (83). The study also reported loss of normal crimping arrangement and loss of birefringence, important biomechanical features of the CrCL microstructure. These observations suggest that micro-injury to the ligament from mechanical loading may cause CrCL remodeling and eventual rupture.

The CrCL has poor healing capacity (84, 85). It has been shown that canine CrCL wounds had lower filling of the wound site and decreased presence of fibrinogen, fibronectin, TGFβ1 (transforming growth factor), FGF-2 (fibroblast growth factor), PDGF-A (platelet-derived growth factor) and von Willebrand’s factor when compared to extra-articular ligament wounds (patellar and medial collateral ligament) (86). The same study showed that treatment with a collagen-platelet-rich-plasma hydrogel resulted in increased filling of the CrCL wound site and increased amount of growth factors. This study suggested that lack of provisional scaffold (fibrin-platelet plug) formation may play a key role in failure of the CrCL to heal after injury. High levels of nitric oxide (NO) in CrCL has been proposed to be one of the reasons for the insufficient healing capacities of ruptured CrCL, however, its role in CrCL physiology largely remains unknown (87). In summary, as evidenced by the multitude of studies investigating the predisposing, primary and perpetuating factors of CrCL disease, the aetiopathogenesis of this disease is unknown, but it certainly appears to be a multifactorial condition.

B.2. Acute or traumatic rupture of the CrCL

Acute or traumatic rupture of the CrCL is a rare injury. However, acute CrCL rupture is usually extremely painful, causes joint effusion and instability of the stifle, resulting in severe lameness. Traumatic injury to the CrCL is usually associated with multiple ligamentous injury of the stifle and leads to joint luxation (1, 88). Such injuries are usually unilateral in contrast to most pathological CrCL disease in mature dogs where bilateral disease is often seen. Acute or traumatic CrCL is caused by a twisting injury to the stifle. This occurs most often when the dog is running and suddenly changes direction. This leads to stifle hyperextension, excessive limb loading and excessive internal tibial rotation which places the majority of the body weight on the stifle and may overload the CrCL, resulting in acute rupture. Stifle radiographs of dogs with acute CrCL rupture show cranial displacement of the infrapatellar fat pad, consistent with joint effusion, without any evidence of arthritis. Treatment options for isolated CrCL traumatic rupture are similar to treatment options for progressive degeneration of the CrCL.

B.3. Avulsion of the cranial cruciate ligament

Mineralized opacities in the stifle most commonly result from trauma, and are usually avulsed fragments or ligamentous calcifications. Avulsion of the CrCL is considered to occur rarely (1, 89). It has been suggested that avulsion, rather than tearing, of the CrCL is a disease primarily of skeletally immature patients (1). However, partial avulsion injury of the origin of the CrCL has been reported in a mature dog (90). In immature patients the ligamentous attachments to bone via Sharpey's fibers may be materially stronger than the bone itself (1, 91). Therefore an acute overload of the ligament allows avulsion to occur under a force insufficient to cause actual rupture of the ligament. The avulsion of the ligament with a small piece of bone occurs most commonly at its insertion site on the tibia but occasionally femoral avulsions have also been reported (1, 89, 90, 92). Treatment for acute avulsion cases may involve reattaching the bone fragment with internal fixation (1). In chronic cases the contracture of ligament fibers makes it impossible to anatomically reduce and stabilize the bone fragment. Several osteotomy procedures (see below) have been described for CrCL-D stifle repair, however, these procedures in immature dogs due to open growth plates are not appropriate. A proximal tibial epiphysiodesis technique has been described to treat CrCL avulsion fractures or to augment primary repair of CrCL avulsion fractures in young dogs (92).

C. Kinetic and Kinematics of the stifle

Kinetic and kinematic gait analyses are considered parts of kinesiology which is the science of motion (93, 94). Gait analysis can identify characteristic features of specific gait abnormalities. The main motion in the stifle occurs in the sagittal plane and it primarily consists of flexion and extension. Due to the stifle anatomy some motion occurs in other planes also (1, 2, 6). Canine joint and limb motion have been studied by various gait analysis studies (2, 6, 23, 93-96).

Kinetics is the study of the relationship of movement to the forces that generate it. Kinetic gait analysis can be performed using force plate to obtain objective measurements. Various studies have used force plate to measure ground reaction forces in normal healthy dogs. A study by Budsberg et al that measured ground reaction forces in healthy dogs at a walk is considered a founding study in the field of force plate analysis in dogs (97). For the hind limbs at a walk, the graph between vertical force and time has two characteristic peaks. The first peak corresponds to a rapid rise in vertical force at foot contact, then the force decreases as the stifle flexes during mid-stance. The second peak corresponds to increase in vertical force at push-off. After the second peak the vertical force finally drops down to zero as the opposite limb takes up the body weight. The first peak corresponds to early stance during which dogs bore about 45% of their body weight on the limb which decreased to about 33% during mid-stance and then increased to about 35% at late stance phase. As opposed to walking, during trotting the vertical ground reaction force of dogs were graphed as single, sharper peaks for both hind limbs and fore limbs (93). During early stance there is a braking phase due to friction between the foot and the floor. The cranio-caudal force during this phase becomes negative. During the mid-phase the foot pushes back on the floor and the cranio-caudal force becomes positive and this corresponds to the propulsive phase.

Kinematics is the study of motion of a body or a system of bodies without reference to its mass or the forces acting on it. It describes the geometric and temporal characteristics of motion (94). The combination of force plate and computer-assisted kinematic data can be used to correlate 3 dimensional limb motion with ground-reaction force measurement (93). Three-dimensional gait analysis in veterinary medicine has been performed by using stereoradiographic methods, invasive external fixators, or cadaveric models (1, 2, 6). Use of retro-reflective markers and infrared cameras has also been described for kinematic measurements of the sagittal (flexion-extension), transverse (internal and external rotation), and frontal (abduction-adduction) plane during a walk and a trot for the stifle, hip and tarsal joints in six healthy dogs (98). Kinematic gait analysis has been used to describe dynamic extension and flexion movement patterns for Labrador Retrievers, Greyhounds and mixed large-breed dogs during walk and trot (23, 53, 99-101). In the stifle of healthy greyhounds during trot there were two peaks of extension, one at the end of swing phase and one of decreased magnitude at nearing the end of stance (101). Motion ranged from 95^o to 150^o during the swing phase and was limited between 130^o and 140° during the stance phase. They also described flexion and extension movements in hip and tarsal joints. Similar gait patterns were identified in the hind limb in a follow up study by Allen et al using mixed breed dogs (95).

A noninvasive kinematic analysis was described by Hottinger et al in healthy large breed dogs at a walk (23). During a complete gait cycle, approximately 50% of the cycle was spent in 2-limb support and 50% in 3-limb support. There was gradual stifle flexion throughout the stance phase with two periods of slowed flexion during the early and late stages. During the late stance rapid flexion was then noted followed by rapid extension during the latter two-thirds of the swing phase. Stifle range of motion ranged from approximately 110^0 to 150^0 during the swing phase and 120^0 to 145^0 during the stance phase. There tends to be more points of inflection during the stance phase of the walk indicating a more complete gait pattern during a walk as compared to the trot (23, 95, 101). However, this difference was not noted by Korvick et al (2). Trotting and walking gaits display similar joint excursions, with a greater range of motion for all joints while trotting (23, 95, 101). Kinematic patterns of movements are similar between dogs of similar morphology (23).

Stifle range of motion has been reported to be 120^o measured by goniometry in healthy adult Labrador Retrievers (102). The mean flexion angle was 42^o and mean extension angle was 162^o. In the frontal plane the stifle is capable of slight varus and valgus angulation (1). At 90^o of flexion, valgus angulation is limited by all four femoro-tibial ligaments whereas varus angulation is limited by lateral collateral and both cruciate ligaments (1). In extension, lateral collateral and CrCL limit varus angulation and medial collateral limits valgus angulation (1). The asymmetry in size between the femoral condyles in combination with the shorter, tighter medial collateral ligament, the more caudal articulation of the lateral condyle, the increased mobility of the lateral meniscus, and the longer, looser lateral collateral ligament results in the passive internal rotation of the tibia during flexion (29, 30, 37, 44). The contraction of the popliteus muscle also plays an active role in this internal rotation (29, 30). Conversely during extension there is passive external rotation of the tibia and relaxation of the popliteus which is commonly referred to as the `screw home' mechanism in human (31, 37).

Multiple studies have described alterations in gait after CrCL rupture by measuring ground reaction forces and kinematic data in dogs (2, 96, 103-108). The kinematic analysis of dogs with ruptured CrCL has revealed decrease in peak vertical forces and impulses as well as braking and propulsion impulses. In one experimental study in adult dogs, the peak vertical force on the normal hind limb was 70% of the static body weight of the dog (109). Following transection of CrCL, the peak vertical force was 25% of body weight at 2 weeks, 32% at 6 weeks, and 37% at 12 weeks. The authors stated that these observations indicate that the osteoarthritic changes seen in the unstable limb are associated with decreased loading of that limb. In addition to these kinetic changes, kinematic analysis has revealed that CrCL-D stifle remains more flexed throughout the stance phase and early swing phase (96). The coxofemoral and tarsal joints remained more extended during the stance phase than in the normal gait cycle likely as compensation for the increased stifle flexion (96). This stifle flexion led to reduced contribution of the stifle to propulsion. A study by Korvick et al also noted this compensatory change in stifle flexion in which a 5-14° increase in flexion was noted at a walk 7 weeks after CrCL transection (2). However, the magnitude of this change was less pronounced during trotting. A third study evaluating kinematics of the canine stifle at a walk up to 24 months post CrCL-transection also noted a similar increase in stifle flexion in CrCL-D stifles (6).

In dogs with CrCL-D stifles, a cyclical pattern of cranial tibial subluxation during the stance phase and cranial tibial reduction during the swing phase occurs (2, 6). This type of dynamic imbalance in CrCL-D dogs is similar to that described in humans (2, 6). A study by Korvick et al utilized an instrumented spatial linkage directly attached to bones via modified bone plates to measure stifle joint motion in five large dogs (2). Kinematic data was obtained both during walking and trotting the dogs on a leash. This study demonstrated approximately 8 to 12mm of cranial tibial subluxation after CrCL transection during the stance phase of the gait. In most dogs in the study, cranial tibial subluxation was unchanged during the swing phase. These finding suggest that the swing phase is CrCL-independent, while the stance phase is CrCL-dependent. In this study tibial internal rotation increased after CrCL transection and this was most pronounced during the mid-stance. All dogs also demonstrated medial displacement of the stifle through swing and stance following CrCL transection. The distance between the femur and tibia (compression) decreased after CrCL transection in all dogs at the beginning of the stance phase which shifted back towards the baseline at the end of stance. Stifle valgus was also noted after transection of the CrCL.

Canine stifle joint kinematics were serially assessed by using dynamic radiostereometric analysis utilizing dual digital video radiographic capture, during treadmill gait before and serially for two years after CrCL transection in a cohort of 18 dogs, with a cohort of 5 dogs acting as sham controls (6). CrCL loss in the dog led to a consistent pattern of CTT during the stance phase of gait. Peak CTT displacement increased on average by 10 mm compared with the CrCL-intact condition. Two years following transection, an average of 5 mm of CTT was present at the terminal swing phase (pre-paw strike). This reduction in CTT was an indication of more persistent tibial subluxation throughout the gait cycle, rather than a return towards normal stifle kinematics. This persistent CTT throughout the gait cycle was speculated to be due to subsequent meniscal damage which may have prevented the stifle from springing back to the normal position during the swing phase. In this study significant changes in internal tibial rotation were not noted. The range of abduction and adduction of the stifle was nearly doubled by two months after CrCL transection and remained high for the duration of the study. A medial translation of the tibia relative to the femur in the CrCL-D was noted. This persisted until final follow up 2 years after surgery. As the medial/lateral motion range was significantly greater only during the first year, authors suggested that some degree of stabilization occurred over time most likely due to capsular thickening and/or osteophyte development in the joint. In addition, stifle joint flexion increased significantly until 6 months following CrCL-transection.

Kinematics have been evaluated after repair techniques for CrCL-D stifles. Kinematic gait differences were compared at a trot preoperatively and at 8 and 12 weeks after surgery between dogs treated with a TPLO or a cranial closing wedge osteotomy (CCWO) (110). In the TPLO group there was significant reduction in stifle angle (i.e. greater stifle flexion) at the end of stance and beginning of swing phase. This change was only significant at 8 weeks after transection. After CCWO dogs were found to carry their stifle in 10 to 20° more extension as compared to the preoperative values during the swing phase. While this study compared pre and post-operative changes, no sham control group was included so conclusions with respect to the specific procedural effects on gait were not determined. The results of this study showed that dogs that received CCWO procedure were more likely to have significantly hyperextended gait patterns of the swing phase postoperatively than the dogs that had a TPLO procedure for repair of a ruptured CrCL.

In vitro stifle kinematics were evaluated after TPLO with stifle angle adjusted at 135±5°, corresponding to the mid-point of stance phase of gait during walking (27). TPLO completely eliminated the marked CTT (15.2 mm) in CrCL-D stifles. These findings were in agreement with the results of previous studies (111, 112). Significant medial and proximal translation of the tibia relative to the femur was noted after TPLO. The study also measured rotations of the tibia relative to the femur after TPLO. The TPLO procedure resulted in significant stifle valgus. The excessive internal tibial rotation in CrCL-D stifles was eliminated by TPLO. The same group of researchers evaluated *in vitro* stifle kinematics after TTA under similar experimental conditions (28). The TTA procedure also completely eliminated CTT in CrCL-D stifles. As opposed to TPLO, significant medial and proximal translation of the tibia relative to the femur was not present. TTA also eliminated excessive internal tibial rotation in CrCL-D stifles. Significant stifle varus or valgus angulation were not present after TTA.

Whereas in these studies the stifle angle was fixed at mid stance, other studies have evaluated stifle biomechanics and kinematics at different stifle angles. An *in vitro* biomechanical study evaluated CTT in TPLO-repaired stifles through a weight-bearing range of motion from 120^0 stifle angle to maximal extension (26). CTT values in the TPLO group trended towards the intact CrCL condition. The values in the TPLO group tended to increase with increasing stifle extension. No significant difference was noted between the intact and TPLO repaired stifle from 120° to 130°. However the difference was significant from 131° to 145°. Despite the continued presence of detectable CTT in the TPLO group throughout the entire range of motion, TPLO values were significantly lower than CrCL-D values at all angles excluding 145°. The authors concluded that the TPLO is more effective at normalizing CTT at angles of greater flexion than extension *in vitro*. Another *in vitro* kinematic study investigated the effects of TTA, specifically on CTT and axial tibial rotation during early, middle and late stance phases of canine gait (25). To simulate *in vivo* conditions, a load of 30% of the dog's body weight was applied to the pelvic limb during testing. The results indicate that treatment with TTA was effective in eliminating CTT during all phases of stance tested; however, the TTA was unsuccessful in normalizing axial tibial rotation in mid stance. The authors also evaluated the effect of meniscal release on stifle stability and concluded that it had no effect on cranial-caudal or rotational displacement when performed in conjunction with the TTA.

Stifle kinematics have also been documented after the modified retinacular imbrication technique (MRIT) (24), however this study was performed on cadaveric limbs in an unloaded state. The authors also compared effects of MRIT and tibial plateau leveling osteotomy — Montavon (TPLO-M) on stifle kinematics. Post-procedural TPA ranging from 8 to 15° were achieved using the TPLO-M. The range of motion was analyzed between 95^0 and 30^0 of flexion. After CrCL transection no significant difference was noted in stifle range of motion, CTT, tibial rotation and tibial adduction and abduction. No difference in CTT was noted after repair using the MRIT. Significant caudal tibial translation was noted after repair using the TPLO-M. This finding is interesting in that the limbs were not loaded and tibial translation is a load generated phenomenon. After MRIT range of motion of stifle was significantly decreased whereas it was unchanged after TPLO-M. Relative external rotation and tibial abduction was noted after MRIT. External rotation was also noted after TPLO-M with significant increase in tibial adduction. With respect to the TPLO-M the authors suggest external rotation may have been created during fragment fixation.

Two cadaveric studies investigated effects of LSS on CTT and axial tibial rotation at a standing stifle angle with limbs in a loaded state (17, 22). In one study these parameters were evaluated using a mechanical testing system (17). The study also investigated if changing placement of the tibial anchor point for the LSS from the tibial crest to either the cranial or caudal eminence of the extensor groove would result in improved stiffness or enhanced stabilization of the CrCL-D stifle. The proximal suture anchor point was around the lateral fabella. Cranio-caudal displacement was defined as the femoral displacement between −25 and 25 N relative to a stable tibia. Femoral displacement in stifles stabilized with the suture placed in the standard position through the tibial crest was not significantly different from intact stifles. The standard tibial crest position resulted in less displacement than anchor points at the cranial or caudal eminence of the extensor groove. External tibial rotation was noted in all legs tested after standard LSS repair. Only the direction of tibial rotation was measured not the degree of rotation. The second study investigated cranio-caudal tibial translation and axial tibial rotation in CrCL-D stifle after LSS procedure using a custom designed device to apply a set force to the tibia while the femur was held immobilized. Radiographs taken before and after application of force were used to measure cranial-caudal tibial translation. For tibial rotation quantification, a torque of 2 Nm was applied first in the lateral direction and then in the medial direction. Axial rotation was defined as the rotation of the tibia in the x–z plane relative to the femur that occurred between the limits of the 2 Nm medial and lateral loads. The CTT in the LSS was not significantly different after stabilization when compared with intact CrCL group. During rotational testing, the limits of the instrument were met, in 20% LSS limbs precluding quantification of tibial rotation. The rotation in limbs that did not reach the instrumentation limits was 12.4° in the LSS group.

D. Treatment Options for stifle with cranial cruciate ligament rupture

D.1 Conservative management

Conservative management of CrCL disease consists of exercise restriction/modification, weight management, anti-inflammatory medications and pain medications as necessary (1, 113). The success rate with conservative management in large dogs is significantly less than small dogs (113). In one study only 6 of 20 large dogs with CrCL rupture became sound with conservative management (114). Conservative management by splint application has also been advocated (113). In majority of small dogs (< 20 kg), close confinement for 4 to 8 weeks was reported to yield satisfactory function (113). Vasseur et al found similar results for small dogs in their study, in which 86 - 90% of dogs weighing < 15 kg had improved at four months after conservative management, while 81% of large breed dogs treated conservatively were still lame after 10 months (115). In some of these cases the lameness progressed with time. All animals had evidence of osteoarthritis, and one must speculate how well they would function several years later. In another conservative management study, 10 dogs (11 stifles with CrCL rupture) with mean body weight of about 45kg were evaluated (116). The median follow up time was three months. Conservative treatment was defined as strict rest for a minimum of 4 to 6 weeks with NSAIDs. Conservative treatment was chosen by clients due to financial factors. Based on owner evaluation 73% stifles were graded as good to excellent whereas based on investigator evaluation of lameness about 58% of stifles were graded as good to excellent. Only two dogs were evaluated with force plate analysis and both were reported to have had peak vertical force values below normal range. Based on results of these studies, surgical intervention is typically recommended for large dogs as well as small dogs that fail to respond to conservative management. The intent of surgical intervention is to provide stability to the stifle, address concurrent meniscal injury and slow down the progression of osteoarthritis. The main aim of intra-articular and extra-articular surgical techniques is to eliminate CTT and stabilize the stifle for long enough to allow for peri-articular fibrosis which then becomes the primary restraint against CTT (1). Newer tibial osteotomy techniques do not rely on peri-articular fibrosis, instead they aim to dynamically stabilize the stifle during weight bearing (9).

D.2 Surgical management

D.2a. Intra-articular stabilization

Intra-articular techniques can be divided into three categories, intra-articular primary ligament repair, intra-articular ligament replacement and intra-articular augmentation and repair.

The biological environment of the CrCL is a major factor that complicates healing. The synovial sheath and its vessels are also injured during any CrCL injury and result in hemorrhage. The blood does not stay at the site of injury to form an organized hematoma but instead it dissipates throughout the joint (117). The presence of circulating inflammatory mediators and proteins and constant flow of the synovial fluid flow prevent the formation of a hematoma necessary for normal ligament repair (118). Surgically apposing the ends of the CrCL after acute transection followed by stifle immobilization demonstrated some ability to heal in dogs (119). But the repaired ligaments were mechanically significantly weaker than control ligaments even after 10 weeks of immobilization. As constant mechanical load applied to the ligament is an additional complicating factor in CrCL healing. Spindler et al evaluated healing of the ligament by creating a central defect (with a 3.5mm beaver blade) in the ligament which did not destabilize the stifle (120). The defect resulted in maximum tolerated load of about 57% of control. After 6-weeks of *in vivo* healing periods, the maximum load was 65% compared to control with no significant gains in biomechanical parameters from defects tested immediately after the creation of injury. This study showed that the centrally placed CrCL defect in dogs failed to mechanically heal in 6 weeks. Collagen-platelet-rich plasma scaffold placed into CrCL defect has been shown to allow formation and organization of a fibrin clot with histologic evidence of ligament healing at 6 weeks (121).

The ligament may be reconstructed with other biologic tissues (autograft, allograft, xenograft), synthetic materials, or a combination of synthetic and biologic materials (composite grafts) (1). In 1917, in a human patient an iliotibial band was utilized to replace a torn anterior cruciate ligament by Hey Groves (117). In dogs the first technique reported for repair of the CrCL was the intra-articular procedure described by Saki Paatsama (122). This technique was a modification of Grove’s technique in which a strip of lateral fascia lata is harvested but the most distal aspect of graft remains attached. The graft is passed through tunnels drilled in the femur and tibia to replicate the position of the original CrCL. The graft is tightened with enough tension to eliminate CTT and then either sutured to the patellar insertion on the tibial tuberosity or passed through a hole in the tibial tuberosity to the lateral side of the tibia and sutured to the fascia. This procedure is technically demanding, as it is very difficult to position the drill holes such that they exit exactly at the origin and insertion of the CrCL. Additional disadvantages of this procedure are insufficient strength of the graft and possible abrasion at the graft-tunnel interface. In a study comparing this technique with an extra-articular procedure, rupture of the graft was noted in 10 of 26 stifles (123). However, only 4 of the 10 dogs were lame, peri-articular fibrosis may have been the primary source of stabilization in those dogs where an appreciable lameness was not detected. A modification of this technique was described by Dickinson and Nunamaker (124). Dueland described a similar technique using the middle third of the patella and patellar tendon (125). The classic Paatsama procedure and its modification techniques rarely are used because of the above mentioned weaknesses and the relative ease of other types of procedures i.e. extra-articular procedures.

Arnoczky et al described the over-the-top technique which uses the medial third of the patella, patellar ligament and quadriceps tendon (126). In this procedure the graft is placed over the proximal and caudal aspect of the lateral femoral condyle which results in consistent graft placement and is technically easier than creating bone tunnels. The same technique was also described by Hulse who used fascia lata, lateral retinacular fascia and lateral one third of the patellar ligament (127). Patellar ligament provides a graft of better strength as compared to the fascia lata however strength still does not approach that of the intact CrCL. Medial, lateral and central patellar tendon graft only tolerated 10%, 29% and 29% of the load tolerated by the intact cruciate, respectively (128). Similar results were reported in an experimental canine study in which grafts only achieved 31% of the stiffness of a normal CrCL 26 weeks after surgery (129). Using an instant center of motion analysis a study showed that over the top technique places the graft in an anatomically correct orientation (130). However, despite a decrease in the severity of lameness, all 21 dogs evaluated clinically and radiographically, continued to have crepitus, joint swelling, palpable instability, as well as progressive osteoarthritis at a mean of ~15 months after this procedure (131). Other tissue grafts have been reported in dogs including tendon of the peroneus longus, the flexor digitalis pedis longus, the long digital extensor, skin, and a semitendinosus-gracilis combination (117). A modification of Arnoczky's procedure is four-in-one over-the-top procedure that combines both intra-articular and extra-articular repair techniques (132). In this procedure a fascia lata graft is used in place of the patellar tendon in order to avoid the difficulties associated with harvesting the small wedge of the patella. Shires et al described under-and-over technique in which a fascia lata graft along with the lateral one third of the patellar ligament are passed under the intermeniscal ligament, through the joint and up and over the top of the lateral femoral condyle (133). A screw and spiked washer are used to secure the graft to the lateral femoral condyle. In the same study Shires et al reported that by 3 months post-op 93% of 35 dogs showed significant improvement in lameness scores with no more than a slight limp noticed after exercise in some dogs. At final follow-up which varied from 3 to 27 months, about 96% of dogs had less than 1-3 mm of cranial caudal laxity, a significant improvement over pre-op values. However, progression of varying degree of osteoarthritis was seen on radiographs in all but 2 stifles. Despite owner satisfaction in about 82% cases (134), force plate analysis showed significant decrease in limb function in dogs with experimentally transected CrCL treated with under-and-over technique (106).

A recent experimental report by Lopez et al proposed the use of a hamstring graft (combined tendinous insertion of the semitendinosus and gracilis muscles and the associated fascia) with encouraging results (135). Arthroscopy was performed to evaluate graft integrity at 12 weeks and gait analysis and stifle radiographs were performed preoperatively and up to 52 weeks after graft placement. Two dogs were euthanized at 12 weeks and further two at 52 weeks. The grafts were highly vascular and undergoing active remodeling by 12 weeks. By 52 weeks the grafts were well vascularized and encapsulated by synovium and there was only mild osteoarthritis based on radiographs and lameness had completely resolved. Based on these results the authors suggested that hamstring grafts may be an alternative technique for canine CrCL reconstruction but further studies are needed before clinical application.

Experimental use of allografts (patellar ligament and fascia lata) in the dog has shown good success (136, 137). However, allografts have not gained popularity due to potential for disease transmission, practicality of fresh graft collection, issues related to graft procurement and storage, their tendency to incite an immune response even when preserved by deep freezing or freeze drying before implantation (1, 117). In dogs, bovine xenograft was investigated as a possible CrCL graft with a follow up of 1 year with subsequent histological examination (138). The result of the study revealed that the grafts revascularized and were replaced by collagen, thus acting as a temporary scaffold while a new ligament was formed. But similar histologic studies in rabbits and humans failed to demonstrate that graft survived and furthermore the mechanical testing showed that these grafts were non-functional (139).

Synthetic grafts have been described as either primary replacement for CrCL or as augmentation devices for biologic grafts (117). Various synthetic materials have been used as prosthesis to replace the CrCL, including braided nylon, Dacron (polyethylene terephthalate, a polyester fiber), carbon fiber and Gore-Tex (1, 117). Failure of Gore-Tex and Dacron led to their withdrawal from the market in 1993 and 1994 respectively(1). Although there are theoretical advantages of intra-articular techniques, because of their failure these techniques have fallen out of favor, having been replaced by extra-articular repair and tibial osteotomy procedures.

D.2b. Tibial osteotomy procedures

The tibial osteotomy procedures that have been reported for surgical management of CrCL disease include cranial closing wedge ostectomy (CCWO), tibial plateau leveling osteotomy (TPLO), combined TPLO/CCWO, triple tibial ostectomy (TTO), chevron wedge ostectomy (CVWO), proximal tibial intra-articular ostectomy (PTIO), and tibial tuberosity advancement (TTA) (1, 9). These procedures are based on the concept of creating dynamic stability in the CrCL-D stifle by altering bone geometry. As CrCL-transection *in vivo* causes predominant cranio-caudal instability, it is reasonable to conclude that the most likely important function of the CrCL is to neutralize CTT (1, 9). Accordingly, the osteotomy procedures aim to address the sagittal plane instability that occurs as a result of weight-bearing. By establishing dynamic stability of the CrCL-D stifle, passive restraint against laxity is not required (1, 9). Although, long-term functional outcomes have been evaluated in only few studies after these osteotomy procedures, most have been associated with favorable clinical results (1, 9).

This approach of tibial osteotomy to CrCL disease to eliminate cranial subluxation during weight bearing was first proposed by Slocum in 1984 who described the cranial CCWO (140). Slocum's theoretical model states that the magnitude of CTT that occurs during weight-bearing in the CrCL-D stifle is dependent on the degree of the caudo-distally directed slope of the tibial plateau (44). CCWO reduces the tibial plateau angle and therefore mitigates the cranially directed femoro-tibial shear force responsible for CTT. The CCWO was initially recommended as an adjunct to procedures that provide passive stabilization (e.g. fascial imbrication) (140). The procedure involves removing a cranially based wedge of bone from the proximal tibia to level the tibial plateau angle. The osteotomy is made as proximally as feasible to still allow for placement of at least 3 screws in the proximal segment. The wedge angle to be excised would intuitively be equal to the angle of the tibial plateau minus the desired post-operative angle of 5°. However after this procedure, the functional axis of the tibia shifts cranially leading to inadequate leveling of the tibial plateau. To compensate for this change in position, ''over-rotation'' of the tibial plateau is necessary to achieve the expected TPA of about 5^{o} (9). If calculations do not account for the shift of the longitudinal axis of the tibia the postoperative TPA will be larger than anticipated. A study by Macias et al demonstrated this variability in achieving the appropriate post-operative angle with post-operative angle range from 7^{o} to 21^{o} (56). The variability in size and position of the ostectomy and tibial long axis shift may be associated with this difficulty in attaining the target TPA. Based on the results of a study, an ostectomy positioned at the distal extent of the tibial crest with an ostectomy wedge angle equal to tibial plateau angle + 5 degrees results in approximate TPA of approximately 6 degrees with a stable stifle joint; however cranial cortices were not aligned instead caudal cortices were aligned in this study (85). Clinical outcome reports after CCWO are rare. An 86% good to excellent limb function have been reported based on client survey and physical examination analysis (141). Advantages of this procedure include: no need for specialized equipment, ability to concurrently correct tibial angular limb deformity, ability to correct concurrent patella alta, can be performed in dogs with open tibial growth plates (1). Complications reported after CCWO primarily include; tibial fracture and nonunion, implant failure with reported re-operation rate of about 12% (1). Disadvantages of this procedure include; potential to create patella baja, limb shortening, variability in postoperative TPA and induction of longitudinal tibial axis shift which may result in aesthetically undesirable cranio-caudal angulation of the tibia (1).

In 1993 Slocum proposed TPLO which is essentially a modifying osteotomy of the proximal tibia that aims to provide neutralization of CTT by reducing the slope of the tibial plateau (142). TPLO is currently the most common tibial osteotomy performed for surgical management of CrCL-D stifles. The biomechanical analysis and clinical outcome of this procedure have been extensively described in the literature (1). To decrease complications and maximize outcome, the procedure should be performed accurately and the basic concepts of osteotomy, including preoperative planning, accurate execution of the procedure, rigid fixation and early return to function must be strictly followed (1). In the TPLO a crescent-shaped osteotomy centered over the intercondylar tubercles is created in the proximal tibia using a biradial saw. The proximal tibial segment is rotated, based on the preoperative TPA and the radius of the osteotomy, to achieve a postoperative TPA of 5^{o} (1). The recommended postoperative TPA was defined as 0^{o} when the procedure was first described (140), then in the TPLO licensing course the recommended TPA was defined as 5^{o} (1), however, *in vitro* studies have demonstrated that CTT is effectively neutralized at a mean angle of 6.5^{o} (111). In a retrospective longitudinal clinical study in Labrador Retrievers, the postoperative TPA varied from 0 to 14^{0} and there was no statistically significant relationship between TPA and ground reaction forces after TPLO (143). The results were satisfactory even in cases with under-rotated tibial plateaus and these results were indistinguishable from the outcome of dogs that had optimal postoperative TPAs. This finding may indicate that *in vivo* complete elimination of the CTT occurs over a wide range of angles or alternatively, the presence of residual CTT may not result in lameness (142). Studies have shown encouraging results in dogs after TPLO with earlier return to weight-bearing based on subjective evaluation when compared with the intra- and extra-articular methods of CrCL-D stifle management (9). However, currently there is no convincing evidence that the TPLO results in superior limb function. Several studies have documented progression of stifle OA after TPLO. A prospective radiographic study reported a significant overall increase in OA score 6 months after TPLO (144). Forty percent of 40 dogs showed progressive OA changes, about 57% dogs did not show any progression of OA and 2.5%

showed decrease in radiographic OA. In an evaluation of 373 stifles in 295 dogs a small but significant increase in radiographic progression of OA was reported at 8 weeks after TPLO (145). Thus progression of osteoarthritis occurs in the majority of stifles after TPLO despite this being initially proposed as a benefit of this technique over other techniques. Numerous intra- and postoperative complications have been reported in dogs undergoing TPLO (1). The overall complication rate is reportedly 26–34%, including tibial tuberosity fracture, implant failure, intra-articular screw placement, significant hemorrhage (often related to laceration of the cranial tibial artery), fibular fracture, intra-articular jig pin placement, placement of bone screws into the osteotomy, patellar tendonitis, subsequent meniscal tear after TPLO, infection (septic arthritis, osteomyelitis, or superficial wound infection) and luxation of the tendon of the long digital extensor muscle (1). Development of neoplasia at the proximal tibia has been reported following TPLO; however, a direct causal relationship has not been proven (1). Disadvantages of TPLO include the requirement of specialized instruments, technical difficulty, iatrogenic angular and torsional deformities, complications of the procedure and the potential adverse effects on stifle biomechanics (9).

CrCL-D stifles with excessive TPAs (>34^{o}) can be treated with a combination of TPLO and CCWO (1). Concurrent use of both techniques is intended to reduce the risk of complications encountered when either technique is performed alone e.g. tibial tuberosity fracture and patella baja (9). Precise preoperative planning is important for accurate execution of this procedure. Typically, the CCWO is standardized to 10° or 15° and the remainder of the angle is addressed with the TPLO (1). The crescent shaped osteotomy for TPLO is positioned in the standard location, and the ostectomy for CCWO is placed such that the apex is at the caudal cortical margin of the TPLO (1). This combination was first described by Talaat et al in a case series of 15 dogs with excessive TPAs (range 35° to 53°) (146). No lameness was noted in 73% and only a mild lameness was noted in 27% of cases at a mean follow-up of 23 weeks. All owners were satisfied with the outcome; however, a high complication rate of ~78% was reported. Complications included implant failure, patellar tendon thickening, seroma formation and local irritation. One third of the cases required a second surgical procedure to address the complication.

In 2002, Montavon and Tepic introduced TTA that neutralizes the cranial tibial thrust force by advancing the tibial tuberosity so that the patellar ligament becomes perpendicular to the tibial plateau (147). This is based on the theoretical model of the stifle that predicts that the total joint forces are approximately parallel to the patellar ligament (1). Thus, if a 90^{o} angle is achieved between the tibial plateau and the patellar ligament, there is no cranial or caudal tibio-femoral shear force component of the total joint force (1). In TTA, titanium implants are placed to secure the advanced tibial crest to the remaining tibial shaft. It has been proposed that dogs with TPA >30^{o} probably are not good candidates for TTA but there is no published proof regarding the range of TPA in dogs with successful TTA (148). Good clinical results were reported by Montavon in 200 cases with only 0.04% failure rate which was secondary to poor surgical procedure (149). A prospective clinical study reported doubling of mean peak vertical force to 64% at final examination between 4 and 12 months after TTA in 40 CrCL-D stifles (150). This was still significantly lower than clinically normal dogs, although the results were comparable with the findings in a similar study evaluating function of hind limb before and after TPLO (9, 150). Three main studies reported outcome and complications after TTA (151-153).

These studies reported 249 cases with an overall complication rate of 20% to 59% with this technique. After eliminating minor complications like swelling and bruising the resultant major complication rate was ~12% to 38%, with a reoperation rate of 11% to 14%. Additionally, most of the cases showed progression of osteoarthritis during follow up. Long-term follow up was limited to an average of 8 to 9 months after surgery. However, despite these complications and limitations, a good to excellent outcome was reported by owners in about 90% of the cases. In a review article Kim et al described two purely speculative advantages of TTA over TPLO from a biomechanical perspective (9). First as the TTA preserves the natural tibio-femoral articulation, the natural load transmission across the stifle (and menisci) is less likely to be altered as opposed to TPLO. Second, the TTA reduces the forces acting along the patellar tendon by increasing the extensor moment arm of the stifle and subsequently increasing the mechanical advantage of the patellar tendon. On the other hand, TPLO appears to increase the strain on the extensor mechanism of the stifle, resulting in clinically relevant complications. The disadvantages of TTA include need for specialized instruments, the potential to cause iatrogenic patellar luxation, and potentially high rate of late meniscal injuries (9).

Several other tibial osteotomy techniques have been described for surgical management of CrCL-D stifles; however the available information regarding these procedures is limited (9). PTIO is an another tibial plateau leveling technique, which was described by Damur et al from University of Zurich in 2003 as an alternative to the TPLO (154). The technique requires a biaxial approach and a wedge ostectomy is made with the base of the wedge located between the bursa of the patellar ligament and the cranial aspect of the menisci. The pivot point of the wedge was defined as the caudo-distal end of the medial collateral ligament. At present, PTIO cannot be advocated as a valid alternative to conventional tibial osteotomies because of its disadvantages, complications and limited available literature (9, 154). CVWO was described by Hildreth et al in 2006 with the rationale that opposed V-shaped osteotomy surfaces resist the torsional and cranio-caudal shear forces better than conventional linear osteotomies (155). The planning for CVWO is similar to CCWO. At this time there are no clinical reports in dogs using CVWO for management of CrCL-D stifles. In 2006 Bruce reported TTO procedure, which similar to TTA aims to reduce the patellar ligament angle to 90^{o} when the stifle is at a weight-bearing angle (156). In this technique, a partial frontal plane osteotomy of the tibial tuberosity is made, leaving the distal cortex intact. Then a partial wedge ostectomy is performed caudal to the tibial tuberosity osteotomy, which is started at the tibial tuberosity osteotomy and the caudal cortex is left intact. Angle of the wedge is equal to two-thirds of angle between the patellar tendon and a line perpendicular to the tibial plateau slope. Reduction of the wedge ostectomy site simultaneously reduces the tibial plateau slope and shifts the tibial tuberosity in a cranial direction. Complications after TTO were encountered in 36% of cases with most common (23%) intra-operative complication being tibial tuberosity fracture requiring tension-band wire fixation (156). Potential disadvantages include variability of the postoperative tibial plateau to patellar tendon angle when using the recommended calculations, additional fixation with intraoperative fractures of partial osteotomies and questionable protective effects against medial meniscal injury (1, 9). The osteotomy procedures do not provide a passive restraint against internal tibial rotation and this rotational instability may potentially contribute to the subsequent development of OA and meniscal injury (1, 9).

D.2c. Extra-articular stabilization

D.2c.I. History

The extra-articular suture (EAS) techniques have been used for CrCL-D stifle stabilization since the 1960s (13). The basic technique involves the stabilization of the joint by transposition of peri-articular structures or placement of suture materials with femoral and tibial fixation points. The main concept of the procedure is to transiently restrain abnormal stifle motion until sufficient peri-articular fibrosis can be produced for long term functional stability and improved limb function (10). The EAS is a commonly performed surgery for CrCL-D stifles, both in referral veterinary practices and general veterinary practices (11, 157). EAS procedure has some potential advantages including relative technical ease, the safety profile, a low cost for surgical equipment and low cost to the owner. Outcomes after EAS have been suggested to be inferior to those seen after corrective osteotomy procedures (13). However, many head-to-head prospective studies have shown no differences in outcomes between the EAS and the osteotomies based on client- and veterinarian-based subjective assessments, force plate gait analysis, radiographic progression of osteoarthritis, or muscle mass measures (3, 14, 15).

Childers described the original extra-articular suture technique in which a longitudinal fold in the para-patellar fascia with several catgut sutures was made to achieve lateral retinaculum imbrication (158). Pearson strengthened the repair by overlapping the caudal and cranial edges of the fascia with a second layer of non-absorbable suture material on the lateral aspect (159). In this technique two layers of sutures are placed on the lateral aspect and one on the medial aspect of the stifle. The first layer is placed at the junction of the fascia lata and the biceps femoris beginning approximately 2cm above the patella and extending to the level of the tibial tuberosity. A second layer is superficial to these sutures and consists of 2-4 sutures in the region between the patella and tibial tuberosity. A third set of sutures is placed in the peri-articular tissues between the patella and tibial tuberosity on the medial aspect of the stifle. DeAngelis and Lau described a method of imbrication by placing one or two Dacron sutures or stainless steel wires in the lateral retinaculum (8). After lateral arthrotomy heavy, non-absorbable suture is passed around the lateral fabella and then through the lateral aspect of the distal patellar ligament forming a mattress pattern. In the report first describing this procedure, 42 stifles in 37 dogs were evaluated with a follow up time between 2 and 14 months after surgery. In about 88% of the stifles the results were considered satisfactory, defined as no more than a slight lameness. This technique was termed lateral retinacular imbrication and became the basis for many of the subsequent techniques. Hohn and Newton described a unique technique also involving medial and lateral support, termed a postero-lateral capsulorrhaphy (160). In this technique the caudal Sartorius muscle is transposed cranially by suturing it to the cranio-medial aspect of the patellar ligament. Two mattress sutures are placed laterally in the caudo-lateral joint capsule. A third suture is placed superficially around the lateral fabella and through the distal aspect of the lateral collateral ligament. Then the fascia lata and biceps tendon are plicated over the patellar tendon. This provides imbrication of the caudo-lateral aspect of the joint and also effectively advances the active constraints of the stifle.

In 1975, Flo described a modification that involved taking a non-absorbable suture around both fabellae and securing it to the tibial crest (12). A third suture which was used as an imbrication suture was passed around the lateral fabella and tied to the tissue just lateral to the patella. This was the first true lateral suture stabilization (LSS) technique and was called modified retinacular imbrication technique (MRIT) by Flo. Securing the suture in the tibial crest was a new concept, as all previous techniques described using the patellar ligament as the cranial suture anchorage point. The repair was augmented by an imbrication suture. A 95% success rate was reported for the MRIT. Another modification of the technique described the placement of three lateral sutures. Two sutures placed through the lateral collateral ligament along with one lateral circumfabellar suture with all sutures passing through the patellar ligament. About 94% stifles repaired with this technique had no lameness at a mean follow up of 20 months. In addition about 80% had no palpable drawer on physical exam. However, the majority of stifles had progressive osteoarthritis. Brinker et al described a modification of Flo's technique, the three-in-one technique, that is similar to Flo's technique except that it included medial and lateral fascial imbrication instead of placing the third suture around the lateral fabella and just lateral to patella (113). A very similar technique to the one described by Flo is still being used four decades later (1, 157) and new studies have shown it compares favorably with the newly introduced extra-articular tibial osteotomy procedures (1, 3, 14, 15).

D.2c.II. Isometric fixation points or Suture anchorage sites

It has been suggested that the accurate identification and use of attachment sites or isometric points is very critical to successful outcome for dogs treated with LSS (13, 18). It has also been suggested that progression of osteoarthritis after LSS and premature suture loosening may occur due to non-isometric positioning of suture attachment at the time of repair (13, 18). For example, if during motion the suture attachment sites move away from one another, the suture will tighten which may limit the joint range of motion, or overload the suture. If, the attachment sites move closer to one another, the suture will become lax, and cranial translation of the tibia may not be controlled. Ideally, the suture attachment sites on the femur and tibia would be isometric. However, as the stifle is flexed the axis of rotation of the femur relative to the tibia does not remain constant (31). This is because of the geometry (cam shape) of the femoral condyle, and the structure of the ligamentous and muscular constraints (1, 31). However, at any one instant in time in any position of the stifle flexion, there is an instantaneous center of motion defined as one point on the femur with zero velocity with respect to the tibia (31). Because of the lack of a true center of rotation and complex sliding, rolling, and rotational motion of the femur with respect to the tibia, a combination of femoral and tibial points that is truly isometric does not exist (1, 10). The term isometry should be used cautiously as the origin and insertion of the CrCL are not isometric, explaining why some of the fibers become lax at certain angles of stifle flexion and extension (1, 10, 13). However, certain pairs of points are closer to isometric than others. Therefore, it has been suggested that the anchorage points used for extra-articular stabilization may be better defined as 'near-isometric' because these points aim to be as close as possible to isometric (1, 10, 13).

In a radiographic study of the isometric points, Roe et al defined sagittal plane isometric lateral suture placement points for the distal femur and proximal tibia by measuring the change in distance between radio-opaque markers (19). The distance from a femur marker to tibial markers was determined in each of seven cadaveric stifles, as it progressed from 150° to 130°, 105°, 90°, 65° and 45° of flexion. The most ideal femoral location was on the very caudal edge of the lateral femoral condyle, immediately adjacent to the distal pole of the fabella. This location is present directly over the origin of the CrCL. The tibial anchorage points adjacent to the extensor groove and the tibial attachment of the patellar ligament were found to be reasonably isomertic when combined with this femoral point. Authors reported few issues that were not accounted for in their study model. The isometric sites were examined in a two-dimensional model with the limb on the table such that varus-valgus and axial rotation was semi-constrained. A 2-dimensional study does not take into account the fact that the suture does not take a direct path from the fabella to the tibial crest. As it curves over the femoral condyle and joint capsule, during range of motion suture must slide over these structures and may change isometry. This study also did not take into consideration tibial rotation as internal rotation of tibia occurs in stifles with CrCL deficiency and external tibial rotation occurs in stifles with LSS.

Hulse et al also showed various isometric points by measuring the change in strain of a suture attached to different femoral and tibial anchorage sites (19). In this study, a suture anchored as caudal as possible on the femoral condyle at the level of the distal pole of the fabella paired with tibial anchorage at the caudal eminence of the extensor groove was found to be the most favorable. However, the suture anchorage at the caudal eminence of extensor groove resulted in extreme flexion of the joint and restricted stifle extension and was therefore not considered to be a functional suture placement position in a previous *in vitro* study (17). Attachment of suture at non-isometric sites was shown to result in change in radio-graphically measured distance in the study by Roe et al and suture loosening and tightening during range of motion of stifle in the cadaveric study by Hulse et al (18, 19). Clinically, this may result in suture failure by suture breakage, knot or crimp slippage, suture elongation, or loss of anchorage in joint positions in which the suture tightens or lead to stifle instability at joint positions in which the suture loosens. Clinically, femoral locations recommended by Roe et al and Hulse et al could only be achieved using bone anchors or possibly by bone tunnels. In a retrospective study of extra-articular sutures in 48 stifles where a bone anchor was used in the lateral aspect of the femoral condyle, even though the functional outcome was good, a high percentage of bone anchors pulled out from the bone on follow-up radiographs when the anchor was not positioned in an isometric location (161). The anisometric placement could result in over-tensioning of the implant during range of motion with resultant suture breakage or bone anchor loosening. None of anchorage pair sites in these isometric site studies achieved perfect isometery (18, 19). A study analyzing the effects of attachment site and joint angles at the time of lateral suture fixation revealed that neither the circumfabellar suture nor the anchor techniques provided a constant suture tension while moving the stabilized stifle through a full range-of-motion confirming that none of the tested sites is truly isometric (162).

D.2c. III. Stabilization Methods

Extra-articular stabilization can be divided in to two broad categories based on the type of prosthetic material used: techniques using transposed autogenous structures (biologic materials) or suture (synthetic) materials (1, 13).

Biological stabilization:

Biologic stabilization techniques for stabilization of CrCL-D stifle involve the transfer or transposition of local tissues. Currently the most commonly used techniques for biologic extra-articular stabilization are fibular head transposition (FHT) and fascial/biceps tendon imbrication procedures, however the sartorius muscle, the popliteal tendon and fascial strips, have also been used (13, 123, 158, 163, 164). Fascial/biceps tendon imbrication procedures were the first reported means for extra-articular stabilization and currently, these techniques are typically combined with other extra-articular procedure/s with the intent of augmenting the initial stabilization and encouraging more optimal fibrosis around the stifle. Some anecdotal reports and subjective assessments in small number of cases have shown successful improvement in function when fascial/biceps tendon imbrication was used alone (123, 158, 164, 165). These techniques have not been directly compared with others for treatment of CrCL rupture in clinical cases.

FHT helps to stabilize the CrCL-D stifle by reorienting the lateral collateral ligament such that it helps to counteract CTT and internal rotation (163). In an experimental study in dogs, elongation and remodeling of the lateral collateral ligament occurred after FHT such that stifle instability recurs, secondary meniscal pathology is seen, and osteoarthritis progresses (166, 167). However, in 64%-90% of patients, clinical efficacy has been shown with some reporting FHT being superior (163) and others reporting FHT being inferior (116) in direct comparison with other CrCL stabilization procedures. Complication rate range from 17% to 50% and reported complications include iatrogenic fracture of the fibular head, post-operative seroma formation, infection, continued instability and progressive osteoarthritis (13, 163, 168). Patternson et al compared FHT with three other techniques: the over-the-top as described by Arnoczky, the under-and-over as described by Shires, Flo's lateral retinacular technique with the exclusion of the medial fabellar-tibial suture, by measuring immediate post-operative cranial laxity and stiffness (169). FHT resulted in a significantly stiffer repair with less laxity and this technique was concluded to be biomechanically superior to the other techniques. In the first report describing this technique four cadaver limbs were evaluated which demonstrated that the FHT normalizes cranio-caudal instability, stifle range of motion and inhibits normal tibial internal rotation (163). In the same study 4 research dogs were also evaluated 6 months after surgery. In two of these dogs, mild cranio-caudal instability recurred and the 3 dogs evaluated at 6 months also had increased tibial internal rotation as compared to the assessment after surgery. The final group of dogs evaluated in this study included clinical cases. FHT was performed in seventy-one stifles while 25 were treated with the intra-articular fascia lata technique as previously described (124). Good to excellent outcome was reported in 90% after FHT whereas 88% had a good to excellent outcome with the fascial repair as assessed by the owners. Interestingly this difference was statistically significant with better outcome in dogs treated with FHT. Concurrent meniscal injury and higher body weights were found to adversely affect outcome. Another study reported good to excellent long term results in 90% of dogs as evaluated by the owners (168). However, a study evaluating outcome of FHT in 30 dogs reported recurrence of cranial drawer motion and tibial rotation by 3 weeks after surgery (166). Full function of the limb was not recovered in any dog as assessed by force plate analysis. Twenty five percent had meniscal damage by 4 months and 50% had evidence of meniscal injury by 10 months. Histological degenerative changes within the articular cartilage were noted in all dogs. Thus, although biomechanically this technique may be superior, outcome does not consistently appear to be significantly improved as compared to other techniques.

Another biological stabilization technique called the lateral graft technique was evaluated in two separate *in vitro* studies (17, 22). This technique is modified from the original MacIntosh procedure used in human orthopedics for anterior cruciate ligament reconstruction which involves the use of a combined patellar ligament and fascia lata graft placed in an extra-articular fashion from the tibial crest to the femoro-fabellar ligament (17). Results of the study by Harper et al showed no significant differences in cranial tibial displacement between the lateral graft technique and the intact CrCL (17). The authors suggested that this technique could be used for the treatment of CrCL ruptures in the dog. The technique was modified in another study by using a referencing instrument to find the relative isometric location for the fascia lata translocation technique (22). This study also showed that cranio-caudal tibial displacement was not significantly different between CrCL intact group and lateral fascia lata group. In both studies the static stability was evaluated immediately after the procedure. Future *in vitro* and *in vivo* studies are needed to evaluate this technique for cyclic stability of stifle and stifle biomechanics and kinematics.

Stabilization using synthetic implants:

Typically synthetic implants are placed on the lateral side of the stifle while some procedures also include a medially placed implant (1, 13). Currently most common procedures performed from this category include LSS, modified retinacular imbrication technique (MRIT), lateral suture anchor technique, TightRope CCL procedure (1, 13). Many studies have evaluated use of various types of suture materials for these procedures. It is assumed that all of these suture materials break or significantly elongate over time, but if suture maintains stifle stability for about 8 weeks then adequate fibrosis occurs around the joint to maintain long-term functional stability (1, 10, 13).

The biomechanical properties and clinical outcome of the LSS have been extensively described in the literature. These studies will be discussed in detail in the following section. The MRIT was evaluated using objective data measurements in an experimental study with force plate analysis (106). The results showed that normal healthy dogs which had a unilateral transection of the CrCL followed by MRIT did not have any significant difference in vertical impulses and peak vertical force at 20 weeks post-op as compared to preoperative values in the same limb. In this study, the preoperative CrCL-intact stifle was used to define normal limb function. MRIT was also used in a non-controlled prospective clinical study in 12 dogs with unilateral CrCL injuries (170). Force plate data was used as an objective measure of limb function. Peak vertical force, associated impulses, and weight distribution were significantly less in the affected hind limb before surgery than in the contralateral normal hind limb. At the time of follow-up evaluation at 7 to 10 months post-op, peak vertical force, vertical impulse and weight distribution had increased significantly in the operated limb in all dogs with no significant differences between the affected and normal hind limbs. Clinically no lameness was detected in any of the dogs at re-evaluation.

The lateral suture anchor technique uses a bone anchor in the femur and a bone tunnel or another anchor in the tibia (161). This technique uses new-generation synthetic material (multifilament polyester suture) as prosthesis. The technique was used in an attempt to use a femoral fixation point that may be more isometric and a fixation method that may prove stronger and less susceptible to creep. The use of lateral suture anchor technique has only been published in one study (161). This retrospective clinical and radiographic study evaluated use of this technique in 42 large and giant breed dogs with CrCL rupture. A bone anchor was placed in the lateral femoral condyle to secure the suture and other end of the suture was passed through two bone tunnels in the proximal tibia located just cranial to the groove of the long digital extensor tendon. The free ends of the suture were tightened using a self-locking knot with four additional square throws. A success rate of 91% was reported despite the fact that 21% of the anchors had pulled out of the femoral condyle at mean follow-up of 1.5 years. The likely reasons for this relatively high anchor failure rate were non-isometric anchorage of suture as well as fixation strength of the anchor. Anchor pullout was less frequent when it was placed in the caudo-lateral aspect of the lateral femoral condyle, just cranial to the fabella. None of the cases developed any draining tracts despite the use of braided suture material. A revision surgery was required in two dogs after the suture broke adjacent to the femoral anchor. Mild radiographic progression of osteoarthritis was noted during the study period.

Recently a new EAS technique termed TightRope CCL technique was developed by Cook et al (171). The technique utilizes one bone tunnel drilled in the femur starting 2 mm cranial and distal to the lateral fabella–femoral condyle junction and within the caudal portion of the lateral femoral condyle directed proximally to exit at the distal diaphysis of the femur on the medial side immediately caudal to the vastus medialis muscle at the level of the proximal pole of the patella with the stifle at a weight bearing angle (~140°). The tibial tunnel is drilled starting caudal and slightly distal to the tubercle of Gerdy directed cranio-distally to exit the proximal metaphysis of the tibia on the medial side midway between the caudal border and tibial crest. The suture is passed through the tunnels and anchored to the medial aspect of the femur and tibia using toggle buttons. The lateral fascia was imbricated using 2–3 modified Mayo mattress sutures. This technique also uses multifilament polyblend suture material (FiberTape) as prosthesis (171). The technique was recently reported in 24 canine stifles. In first part of the study the mechanical properties of FiberTape, were compared *in vitro* with 4 different commercially available extra-articular systems: a suture system using #5 fiber wire suture with knot fixation, 80# test monofilament NLL with crimp clamp fixation, XGEN CCR System with #5 OrthoFiber and the LigaFiba Iso Toggle System with braided polyethylene fiber. TR had superior mechanical properties for creep, stiffness, yield load, and load-at-failure when compared to other materials tested. In the second part of the study, TR was clinically compared with TPLO. The results of the study showed that the 6-month outcomes in terms of client-evaluated level of function and radiographic progression of osteoarthritis were not different between TR and TPLO. Total surgery time and duration of anesthesia were significantly shorter for the TR procedure. Major complication rates were not significantly different between TR (12.5%) and TPLO (17.4%). There was no significant difference between groups for cranial tibial thrust at all postoperative time points. Complications of TR included infection, implant failure, meniscal tear and seroma. The authors concluded that TR is an effective and safe technique and can be considered an appropriate surgical option for medium, large, and giant breed dogs with CrCL deficiency. A study reporting three months and three years follow up results involving 1,215 cases treated with TR at 31 centers has been recently presented (172). Overall success rate was about 94% with good to excellent outcomes. The major complication rate, defined as requiring further treatment, was 9.2%. The follow-up rechecks and outcomes were variable among dogs and generally based on the evaluation by the attending clinician, therefore the results should be interpreted with caution.

Kunkel et al described another similar transcondylar toggle system in small dogs and cats (173). A femoral transcondylar bone tunnel is drilled from lateral to medial. Then a bone tunnel is drilled in the tibial crest slightly caudal and distal to the proximal insertion of the straight patellar tendon. A toggle is threaded through nylon leader line (NLL) which is passed through the tunnels with the toggle seated on the medial aspect of femoral tunnel. The two ends of the NLL are hand-tied using either a surgeon's knot followed by 4 additional throws or a slip knot and 3 additional throws. The lateral fascia is imbricated using a modified Mayo mattress pattern.

A novel EAS technique called lateral extra-articular suture system (LESS) was recently evaluated in an *in vitro* study using 20 canine pelvic limbs (174). The technique utilizes a cortical bone screw in the femur and a cannulated cortical bone screw in the tibia to connect two strands of suture. The femoral screw is placed in the caudal aspect of the lateral femoral condyle 3 mm distal to the femoral-fabellar ligament and just cranial to the line of the caudal articular cartilage. The tibial screw is placed starting proximal and medial to the cranial aspect of the cranial eminence of the long digital extensor groove and directed 60° to the long axis of the tibia from lateral to medial. The suture is wrapped around the femoral screw as it exits providing additional length as the stifle moves from extension to flexion. The potential advantages of the system include bony fixation of the suture, near isometric placement of suture, and improved range of motion of the stifle due to lengthening of the suture during stifle flexion. Although limited results for these new techniques are positive, long-term *in vivo* studies evaluating these techniques still need to be investigated.

E. Lateral suture stabilization

E.1 Surgical technique

Most commonly, currently used LSS technique (1) is a modification of the extra-articular technique reported by Flo (12). Surgical approach can be performed by medial or lateral incisions and medial or lateral parapatellar arthrotomies (13). Because of the mobility of the skin over the stifle, access to relevant structures can be readily achieved via either approach for LSS. After parapatellar approach to the stifle the intra-articular structures are examined. The CrCL is inspected visually and by palpation with a small probe, and rupture and laxity are confirmed. Other structures in the joint including caudal cruciate, lateral and medial menisci are also evaluated. Generally, the remnants of the damaged CrCL are removed because they may act as source of continued inflammation (175), however, removal has not been definitively proven to reduce inflammation, and even careful excision invariably results in remaining remnants. The necessity of medial meniscal release is a topic of debate (1). The joint is thoroughly lavaged with sterile physiologic saline, and the joint capsule is closed with monofilament absorbable suture in a horizontal mattress pattern or a similar imbricating pattern. This closure leads to fibrosis of peri-articular tissue that ultimately helps to stabilize the joint. The lateral fabella (sesamoid of the gastrocnemius muscle tendon of origin) is identified, and a mattress suture of heavy, non-absorbable suture material is placed around it. Because the cranio-distal, aspect of the lateral fabella articulates with the femur, the suture is best placed slightly proximal to the fabella, in the strong fibrous origin of the lateral head of the gastrocnemius muscle (often referred to as the fabello-femoral ligament), rather than around the fabella itself, as placement around the fabella or distal to it, may result in suture slippage and failure due to pull-out through the gastrocnemius muscle. Typically a curved Securos J-needle loaded with monofilament NLL is used to pass the suture through the lateral tendon of origin of the gastrocnemius muscle in a proximal to distal direction (17). The suture placement is checked for security.

The lateral aspect of the proximal tibia is exposed by incising the fascia overlying the cranial tibial muscle. A hole is drilled in the tibial crest, several millimeters caudal to the tibial attachment of the patellar ligament. Typically a 3/32″ Steinmann pin is used to drill a hole through the tibial tuberosity. The distal end of the suture is passed caudal to the patellar ligament along the proximal aspect of the tibial plateau from lateral to medial and external to the joint capsule. The suture is then passed through the hole in the proximal tibia in a medial to lateral direction and then secured to the proximal strand in a figure-of-eight configuration. The suture is tensioned and secured either using the Securos crimp clamp system according to manufacturer recommendations or with knots (1, 176). With the stifle held at 135° of flexion the suture is tensioned until cranial drawer motion could no longer be elicited. The crimp clamp is then crimped with a crimper and the excess suture is removed. Suture is not over-tightened as this may result in decreased range of motion in the stifle and increased contact pressure in the joint (1). The stifle stability is confirmed by a negative cranial drawer test and a tibial compression test throughout stifle range of motion. If stifle instability persists or if range of motion is diminished once stability is achieved, it is likely that the suture anchor points are not positioned accurately; this may necessitate repositioning of the suture by drilling new tibial anchor holes (1). The biceps femoris fascia is closed in a modified Mayo mattress pattern (vest over pants) or with another imbrication technique, and the subcutaneous tissue and skin are closed routinely.

E.2 Prosthetic material and method of securing

Suggested ideal properties for a prosthetic material include: biocompatibility, excellent knot security, aseptic, easily handled, inexpensive, knot compactness and the ability to withstand cyclic and tensile loading (13, 177). Most commonly used synthetic suture for LSS are large diameter monofilament materials such as monofilament nylon fishing line (NFL) and monofilament nylon leader line (NLL) (1). Nylon leader line is composed of nylon 612, while the fishing lines are commonly copolymers of nylon 6 and nylon 66 (178). NFL has a higher melting point than the NLL, but is more hygroscopic and possesses inferior dimensional stability. A study evaluating mechanical comparison between NFL and NLL demonstrated higher tensile strength and stiffness of NLL (179). These materials are available in different sizes, for example 18 kg, 36 kg, and 45 kg test nylon (180). For monofilament nylon the strength of the line (pound test) is generally chosen to be at least equivalent to the body weight of the patient (1). The estimated load applied to the suture is 120 to 600N (179). Various brands of NFL and NLL are available in market, five brands of NFL including Ande, Sufix, Berkley, Hi-Seas and Maxima and three of NLL including Ande, Mason and Jinkai have been tested in veterinary studies (21, 181). One experimental study evaluating above mentioned first two brands of NFL and all three brands of NLL concluded that Mason NLL and Sufix NFL had the best mechanical properties for LSS of the canine stifle (21). A number of other materials have been proposed to use for extra-articular techniques including braided polyblend materials (e.g. braided ultrahigh-molecular weight polyethylene +/- polyester i.e. FiberWire, FiberTape, OrthoFiber, LigFiba), nylon band, orthopedic wire, ethylene tetrafluoroethylene tie, polyvinylidene fluoride, fluorocarbon leader line, silicone braided polyester suture, polybutilate-coated multifilament polyester; however, most of these materials are not routinely used (1, 21, 161, 171, 173, 176, 182-186).

Because of the availability of relatively smaller size monofilament suture material (USP #2 polypropylene and nylon are the largest diameter readily available) as compared to multifilament material (e.g. polyester, up to USP #5), multifilament polyesters were initially advocated for LSS for their perceived higher strength. However, multifilament material, specifically coated caprolactam, has been shown to be associated with post-operative infection and sinus tract formation in as many as 21% cases in one study (187). Removal of suture resulted in resolution of draining tracts in all cases; however, lameness was still present in 35% of the dogs after suture removal. On the other hand, monofilament suture materials are infrequently associated with the development of draining tracts (188). For these reasons, as mentioned above large diameter monofilament materials such as monofilament NFL and NLL are more commonly used for LSS (1, 21, 186). An *in vitro* mechanical study compared two routinely used suture material for LSS at that time (i.e. USP#2 polypropylene, USP #5 multifilament polyester) to monofilament NLL (186). The monofilament NLL was able to maintain a significantly greater percentage of static tensile loads than the other materials tested.

A new technique, using nylon band (nylon cable tie) with a locking mechanism, with the proposed advantages of being quickly and easily performed by an unassisted surgeon was described by Beckman et al (184). The authors reported that only one owner out of thirteen expressed dissatisfaction with the dog's lack of improvement. However, these nylon bands resulted in drainage and necrosis of the overlying soft tissues necessitating their removal within 1 to 3 years after surgery (189). In 1993, Olmstead first reported use of an orthopedic wire as prosthesis for LSS of the CrCL-D stifle (185). He recommended using 18 gauge, 20 gauge and 22 gauge wire for dogs weighing >40kg, 18 to 40kg and < 18 kg respectively. A retrospective radiographic study evaluating dogs that underwent LSS with orthopedic wire found that 92% of dogs had one or more breaks in the wire six month following surgery (190). In some cases wire had slipped off the fabella and was displaced distally along the gastrocnemius muscle. About 16% of wire implants were removed owing to complications, such as wire migration or seroma formation.

Various methods have been reported for securing the synthetic material used for LSS (12, 17, 20, 113, 161, 171, 178, 191-196). The two commonly used methods to secure LSS prosthesis are knotting the material or using a metallic crimp clamp. Knotting the material is the traditional method of securing the LSS prosthesis and several types of knots have been described or advocated for this use including square knots, a slip knot followed by a series of square knots, clamped square knots, sliding half-hitch knots, surgeon's knots, and a self-locking knot followed by a series of square knots (12, 17, 20, 113, 161, 171, 178, 191-195). The structural properties of some suture material loops can be influenced by the type of knot. This was demonstrated in a study comparing the mechanical performance of NLL and NFL with two different knot types (square knot and slip knot) (179). NLL secured with a slip knot recovered resting tension following cycling better than NLL with a square knot, whereas opposite was true for NFL. Therefore the authors suggested that NLL be secured with a slip knot, and NFL be secured with a square knot to optimize the mechanical performance of the loop. This study also showed that

NLL tolerated higher failure loads and deformed less than NFL for both knot types. In 1999, Huber et al also showed that knotting method does influence the structural properties of suture material and should be considered when tying knots under tension (193). In the study, a conventional square knot was compared with the surgeon's knot, sliding half-hitch, and clamped square knot for USP#2 nylon, #2 polybutester, #2 polypropylene, 27 kg test NFL, and 27 kg test NLL. For NLL surgeon's knot decreased stiffness by 27% and when the same material was knotted with a sliding half-hitch the suture's load-to-yield was reduced by 20%. For NFL also surgeon's knot decreased stiffness and sliding half-hitch decreased load to failure. Knot type, however, did not affect the stiffness or load-to-yield of nylon or polybutester. A clamped square knot did not have any detrimental effects to the structural properties of NFL, nylon and polybutester. Conversely, clamped square knot for NLL has been shown to increase failure load by 2% and stiffness by 7% to 16%, and to decrease elongation by 12% (178, 193). Thus according to the authors of the study, clamped square knot is preferable for NLL than sliding half-hitch to maximize the suture's structural properties. Unlike the results from Huber et al, there was no significant difference in failure load between clamped square and slip knot for NLL in a study by Burgess et al (176). The authors suggested that this may be because of difference in suture size, material manufacturer or use of an extra throw in the later study.

Large diameter nylon is a stiff prosthetic material that results in large, bulky knots with poor security (178, 179, 181, 187). Increased amount of foreign material resulting from large knots is a potential source for chronic irritation, infection and seroma formation (21, 195). Monofilament nylon deforms during knot formation and a bending moment is induced near the knot which leads to stress concentration points and breaking of knotted loops near their fixation (195). A crimp clamp system for securing prosthetic material for LSS reported by Anderson et al in 1998, offers an alternative to knot fixation (195). This system replaces the need for knots by securing the suture within a metallic tube (crimp). Their function is based on the friction developed between the crimp and the suture when it is compressed. Crimping has become a popular alternative to knot tying because of elimination of bulky knots, decreased retained foreign material, high initial stiffness, ease of maintaining loop tension during suture securing, decreased elongation of the construct, little permanent deformation after application of expected physiologic loads and increased strength of the repair (21, 178, 195). Crimp clamp system was biomechanically compared to sliding half-hitch knot using 27 kg test NLL in single load-to-failure and cyclic tests (195). During both type of testing, crimp-clamped loops were superior to knotted loops in all parameters including less elongation, greater load to failure and greater initial tension. In this study loop failure generally occurred by breaking within 3mm of the fixation in both clamped and knotted groups. A study by Peycke et al comparing crimp system to knot fixation found that the crimp system was as strong or stronger than square knot, half-hitch and Harris knotter fixation techniques using both 27 and 36 kg NLL, with less elongation as compared to the Harris knotter and self-locking loop and comparable elongation compared to the traditional knotting techniques (197). The peak force was lower for crimp clamp group when compared with the self-locking knot, but this is expected, as there are two strands of material resisting the applied load in self-locking knot group. Similarly another mechanical study comparing NLL crimped loops with clamped square knot loops showed that the crimped loops had less elongation and more stiffness in both static and cyclic loading (194). Initial tension was also greater in crimped loops but the clamped square knot resisted a greater peak load. Although square knots resist a greater load before failure, but it is important to realize that the elongation required before these loops failed after application of load exceeds the distance that a tibia would be able to move away from a femur because of secondary restraint tissues (177, 194).

Several newer sutures especially polyethylene-based non-absorbable, multifilament orthopedic sutures e.g. FiberWire, FiberTape, and OrthoFiber have been biomechanically compared with monofilament NLL *in vitro* using both knotting and crimping techniques (176, 198). Crimped polyethylene sutures were stiffer than crimped NLL and in general knotted polyethylene sutures were also stiffer than knotted NLL (176). Crimped polyethylene sutures were stiffer than knotted polyethylene sutures. Knotted polyethylene sutures had less elongation than knotted NLL under monotoic tensile and cyclical loading. Knotted FiberWire and OrthoFiber loops were weaker than knotted NLL and crimped FiberTape was stronger than crimped NLL. FiberTape was the strongest and stiffest material tested in the study. In general loops produced by square knots were stiffer, stronger and had less elongation compared with slip knots for polyethylene sutures. Failure mode for all crimped multifilament loops was slippage through the crimp. For crimped NLL failure mode contradictory information is available as some studies showed that NLL loops generally failed by slipping through the crimp (176, 196, 199) whereas other studies reported that crimped NLL failed commonly by means other than slipping through the crimp (e.g. breakage at the knot or away from the knot) (180, 197). Another mechanical testing study also showed that multifilament polyethylene sutures are stronger and elongate less than monofilament NLL in pure tension (198). This study also tested LigaFiba in addition to above mentioned polyblend sutures and showed that this suture had the greatest ultimate tensile strength. LigaFiba was also stiffest of all tested materials at 3mm of elongation. All of the materials tested exceeded the lowest estimated physiologic load (~120N) of the CrCL (estimated for dogs between 30 and 60 kg), but none exceeded the highest estimated minimum physiologic load (~600N). One of the limitations of this study was that only single strands of the suture material were tested in pure tension with no loops or knots as typically used in a clinical situation.

The suture can be tensioned by hand with a square knot, a sliding (slip) knot (179), a self-locking knot (192), clamping the first throw of a square knot (178), or a tensioning device (20). Clamped square knot loops are less tight because the 2nd throw of the knot must be tightened while simultaneously removing the clamp from the first throw (194). A slip knot decreased the yield of NLL (193) and a self-locking knot elongated more than other knots (197). An *in vitro* mechanical study compared monofilament NLL loops tightened by a hand tightening method with loops tightened in a standard manner with a tensioning device (196). In the hand tightening technique the crimp clamp was pinched once, with the jaws of the Securos crimping tool fully closed, in the middle of the tube and then each end of the suture was pulled tight, as tight as possible. The ends were released and the final 2 pinches were applied to the crimp clamp on either side of the 1st pinch. There was no significant difference noted in the mean ultimate load and mean stiffness of loops between the two techniques. The authors concluded that hand tightening method does not influence the mechanical properties of the loop. However, the hand tightening method described does require some strength to pull the loop tight after the crimp has been partially pinched. There could be two potential problems with this method. One if the first pinch is too tight then surgeon may not achieve required tightening and if the pinch is too loose then surgeon will have difficulty maintaining tension after complete tightening, whereas the tensioning devices allow incremental tightening of the loop and also maintain loop tension while the crimp clamp is secured.

Crimping systems are available from different manufacturers including Securos Universal crimping system designed around Mason Hard type monofilament NLL and Veterinary Instrumentation (Sheffield, UK) crimping system designed around Ande monofilament NLL (180, 194, 199). Third type of crimping system used in veterinary studies is called JorgVet crimping system from Jorgensen Laboratories (194). A new system designed by Securos is called PowerX crimping system (or wave pattern crimp device) (180). Different systems use different sized crimp clamps and there are differences in the tools used to pinch the crimp clamps. The tips of the jaws of the Securos Universal crimping tool are recessed and as long as the jaws are fully closed, this design feature should allow for a consistent pinch to be achieved (199). In contrast, the JorgVet and Veterinary Instrumentation crimping tools have no clearly defined end-point and therefore crimping is more likely to be operator dependent (199). The Securos tools provide mechanical advantage for the operator because of their double-action mechanism which makes it easier to fully pinch the crimp (180, 199). The jaws of the Veterinary Instrumentation crimper are thicker than those of the Securos crimper, so that the force applied to the crimp is applied over a larger area, or at a lesser pressure for identical forces (199). The jaws of the Securos universal crimper are narrow and create a single point of compression on the crimp (180). This requires three successive, equally-spaced pinches of the device to complete the crimp deformation. Whereas with the Power X (wave pattern) crimper only one pinch is required to completer the crimp deformation because it's jaws are designed with a continuous interlocking wave pattern, which is equal to the length of the crimp (180). When Securos universal crimping system was compared with JorgVet crimping system using 27 kg NLL loops, no significant difference was noted in initial tension and stiffness of loops, however peak elongation was significantly higher in Securos group when single loads were applied to failure (194). The loops secured with Securos system tended to resist higher repeated loads before failure. In the study, crimping methodology was different between the two systems and final crimp compression may vary between surgeons which may lead to differences in crimp/suture friction.

In another study mean stiffness and mean ultimate load were significantly higher for the Securos Universal system (36 kg NLL loops) than for the Veterinary Instrumentation system (45 kg NLL loops) (199). For both systems, there were significant differences between operators for ultimate load. A recent *in vitro* study compared two crimping devices from Securos using loops of monofilament NLL of 18 kg, 36 kg and 45 kg and multifilament polyethylene suture (180). The loops were tested to failure in quasistatic tensile loading. Results of the study showed that the loops secured with the PowerX crimper (waver pattern) were significantly stronger with a higher load yield, displacement yield, failure displacement, maximum load, and maximum displacement than the loops secured with three successive pinches on the crimp created by Universal single crimp device. The authors suggested that the increased strength of PowerX crimp construct is attributable to its improved holding power, because of the 'wave style' crimp pattern which produces more surface contact and friction between the suture material and the inner wall of the crimp (180). The PowerX device produces five contact points instead of the three separate crimp contact points produced with the Universal single compression device.

The loop configuration of the prosthetic material is also one of the many variables affecting its performance. Several configurations of double loops using 36 kg NLL secured with Securos crimping system have been mechanically tested *in vitro* including three configurations of double loop-single strand e.g. simple double loop, locking loop, interlocking loop and three configurations of double loop-double strand e.g. simple single loop, figure of eight loop, uneven loop (20). The uneven group had the same configuration as the simple single group except one of the loops was 2mm shorter than the other. The interlocking loop had the greatest load at yield but also the greatest elongation at yield. This elongation could lead to undesirable instability in the stifle. The simple double loop had an intermediate load at yield and the lowest elongation. Another significant finding of the study was the poor biomechanical performance of the double loop-double strand uneven group, with significantly lower mean stiffness and ultimate load than all of the single strand groups. A recent study by Choate et al evaluated mechanical properties of NLL, polyethylene cord, polyethylene tape secured to cadaveric canine femurs via various methods including knotting, crimping, bone anchors and toggle fixation (200). Some of the prostheses were also tested in single and double loop configuration. Single and double loop circumfabellar polyethylene tape constructs had the highest stiffness. Double loop BTPT (two strands of polyethylene tape secured to the femur via a toggle placed through a bone tunnel), double loop CPT (two strands of polyethylene tap secured around the lateral fabella and secured with knots) and BAPT (two strands of polyethylene tap secured to the femur via a bone anchor) constructs were mechanically superior to other constructs. Double NLL and single circumfabellar polyethylene cord constructs had the lowest loads at 3mm of displacement. Authors concluded that mechanical properties of constructs were dependent on mechanical properties and methods of anchorage and securing the suture materials. This is the first study that compared results of monotonic load to failure for isolated prosthetic loops to femoral constructs in which prostheses were secured around the lateral fabella. Authors suggested that based on the results of the study, the strength of femoro-fabellar ligaments seemed to exceed that of suture materials tested. The high incidence of prosthesis breakage or knot or crimp slippage in the circumfabellar femoral constructs supported this finding. Only 3 out of 20 polyethylene cord or polyethylene tape femoral constructs failed via pulling through peri-fabellar soft tissues and none of the NLL constructs failed by this manner. All NLL constructs failed via breakage or slippage at the crimp. Few studies have attributed early construct elongation and failure to properties of femoro-fabellar ligaments (185, 190) but based on above findings and the finding that circumfabellar polyethylene tape constructs had high stiffness Choate et al suggested that early construct elongation and failure in previous studies were more likely attributable to mechanical properties of prostheses or failure to properly secure prostheses around fabellae.

The effects of commonly used sterilization methods on mechanical properties of suture material used for LSS have been evaluated in several studies (21, 178, 186). Ethylene oxide sterilization and steam-sterilization (including one cycle and five cycles in autoclave) have been the major methods studied (21, 186). Steam sterilization causes significant increase in elongation of NFL whereas ethylene oxide sterilization causes minimal effect on elongation of this material (21). There was no significant difference in effect on the strength of the fishing material between these two sterilization methods. NLL showed no significant change in strength or elongation regardless of sterilization method (21). Another study comparing ethylene oxide and steam sterilization methods on NLL showed that ethylene oxide had the least detrimental effects on the handling and material characteristics (186). Ethylene oxide did not significantly alter load at failure whereas one cycle of steam sterilization resulted in small, but significant decrease in the load at failure (186). Both ethylene oxide and one cycle of sterilization resulted in significant decrease in stiffness but the difference was not significant between the two techniques. The stiffness was significantly decreased further after five cycles of steam sterilization. A ~ 12% increase in elongation and ~10% decrease in stiffness of NLL after steam sterilization was also reported in a previous study (178). Although ethylene oxide has the least detrimental effects on monofilament nylon material characteristics, but some animal and human literature suggests that ethylene oxide could be carcinogenic and associated with breast and other cancers in those exposed (201, 202). In Canada regulations regarding ethylene oxide use has been modified which requires use of an appropriate aerator with an operating emission control system, which should be tested every year and this may be prohibitively expensive for some institutions (203). Above mentioned reason led to replacement of ethylene oxide by hydrogen peroxide gas plasma (HPGP) sterilization in a veterinary teaching hospital in Canada (203). Recently, HPGP sterilization of NLL and NFL was compared with ethylene oxide sterilization method (203). There was no significant difference for elongation and ultimate load of NLL between two sterilization techniques, however for NFL, HPGP sterilization led to slightly greater elongation than ethylene oxide technique. Authors concluded that effects of HPGP on NLL compared positively to EO, making HPGP an attractive alternative. Another study by the same author evaluated effects of sterilization method on bio-adhesive properties of NFL, NLL and polyethylene suture (204). The authors concluded that for most of the samples, bacterial adherence was lower or similar when HPGP sterilization was used, compared with results for sterilization via steam and EO respectively.

E.3 Clinical outcomes

LSS techniques have generally reported positive clinical results (1, 13-16, 205). Evaluation of the therapeutic efficacy of the technique has been determined based on both subjective (i.e. owner interviews, clinical assessment of gait, physical examination and radiography) and objective (i.e. force plate, thigh circumference measurement, goniometric measurement of stifle range of motion) data analysis (1, 13-16, 205). A retrospective study with a follow up time of about 20 months evaluated outcome after LSS technique in 40 dogs with CrCL deficiency (16). Prosthetic material used included No. 5 metric polypropylene or 36 kg test monofilament NLL. Ninety percent of the owners were satisfied with the treatment result based on telephone questionnaire. Follow up physical examination findings were available in only 11 dogs. Lameness improved in majority of the dogs following surgery with eighteen percent still lame at walk. There was about 12 degree loss of range of motion in the surgical stifle when compared with the contralateral un-operated stifle.

Limb function outcome and its relationship to tibial plateau slope were evaluated in 34 dogs with unilateral CrCL injury in a prospective clinical study (205). Survey stifle radiographs, lameness examination, and force plate analysis was performed in all dogs before and at 6, 12, 24 and 48 weeks after surgery. There was no significant correlation between tibial plateau angle and postoperative vertical impulse, peak vertical force, lameness score or radiographic osteoarthritis score. There was no change in tibial plateau angles after LSS procedure. All dogs had increased radiographic scores (more DJD) during the study period. The study also showed that lameness scores, peak vertical force and associated vertical impulse values improved from preoperative baseline level at serial postoperative measurements, however these data were not compared with data from clinically normal dogs.

Although most owners are satisfied with the results after LSS, progression of stifle osteoarthritis has been shown to occur similar to other stabilization techniques, which might be the likely reason that most dogs do not regain normal function of the affected stifle (3, 15, 134, 205). A retrospective radiographic study evaluated 58 dogs after LSS with a mean follow up of 23 months (range 6 – 132 months) (134). There were 18 dogs that weighted less than 15 kg and 40 weighted more than 15 kg. LSS did not mitigate progression of radiographic signs of osteoarthritis in either group. Dogs weighing < 15 kg had a lesser degree of DJD and returned to weight-bearing quicker than dogs weighing > 15 kg. Based on owner questionnaire there were 8 dogs that were consistently lame, 8 were lame only following exercise, 10 were lame only after resting and 32 were seldom to never lame.

Another long term retrospective study also showed radiographic progression of osteoarthritis after LSS in 27 stifles (3). Radiographs were taken immediately before surgery and ≥ 12 months later. The study also compared radiographic changes between LSS and TPLO (52 stifles) groups and showed that no significant difference in osteoarthritis was noted between the groups at the preoperative and final examination times. Based on their logistic regression analysis they were able to show that dogs with > 6 point change in osteoarthritis score were about 6 times more likely to have had LSS than TPLO; however because of limitations of the study, authors suggested further studies using a prospective, controlled, randomized clinical trial using these two techniques would be needed to confirm or refute their preliminary results. Around the same time Conzemius et al published a prospective clinical study using force plate to compare the effects of LSS and TPLO on limb function in Labrador retrievers with unilateral CrCL insufficiency and medial meniscal injuries (14). A control group composed of 17 healthy Labrador retrievers was also included in the study. The damaged meniscus was either partially or completely excised during surgery. The force plate evaluations performed two and six months following surgery showed no significant difference between the two surgical techniques. The only difference between the two groups was that dogs in LSS group underwent aggressive postoperative physical therapy whereas dogs in TPLO group did not. Although, the postoperative rehabilitation has been shown to affect clinical outcome (206) but in clinical case scenario, in general, rehabilitation is recommended soon after LSS procedure whereas exercise restriction is recommended for at least 6 weeks after TPLO (14). The results of the study by Conzemius et al showed that Peak vertical force and vertical impulse were both 96% of normal after TPLO and 93% and 96% respectively after LSS at six months after surgery. Authors defined a clinically important improvement as an improvement such that a dog in the treatment group was more clinically normal than abnormal at the time of re-evaluation (14, 207). Based on their definition, only 40% dogs in the LSS group and 34% dogs in the TPLO group were considered to have improved six months following surgery and only ~15% dogs in LSS and ~11% dogs in TPLO group considered to have had normal limb function. Thus the study showed that clinical limb function in dogs following LSS was not significantly different from clinical limb function in dogs which underwent a TPLO.

The argument that arose from Conzemius' study was that TPLO dogs may have had better outcome with physical rehabilitation or conversely LSS dogs may have had poorer outcome without post-operative physical rehabilitation. To address the above mentioned limitation a recent study used identical physical rehabilitation regimens in CrCL-D dogs treated with LSS or TPLO and compared short- and long-term functional and radiographic outcome (15). Radiographic DJD scores before surgery and at 24 months post-surgery were not significantly different between two groups, however in both groups these scores increased significantly during the study period. Force plate data was obtained at three, five and seven weeks, and at six and twenty four months after surgery to compare the functional outcome. Peak vertical force values were not significantly different between two treatment groups at any time point; however in both groups, these values increased significantly from 3 weeks to 24 months post-operatively. Comparison of stifle range of motion and thigh muscle mass circumference between two groups also showed no significant difference at any time point. The findings of this study were consistent with Conzemius et al. where similar limb function was observed in dogs after TPLO or LSS (14, 15). The results of this study should be interpreted with caution as the body weight of dogs in LSS group (mean 29kg) was significantly lower than dogs in the TPLO group (mean 35kg). So, it is possible that dogs of larger size treated with LSS would have had poorer outcome, however, regression analysis in the study showed that weight contributed no temporal variation in peak vertical force.

E.4 Complications

A retrospective study reported a 17.4% (63/363) complication rate for LSS procedure (208). In the study one complication occurred in 61 dogs and 2 complications occurred in 2 dogs (208). Complications rate in dogs that underwent simultaneous bilateral LSS was not different than the complication rate in dogs that had unilateral surgery. Twenty six of 363 patients (7.2%) required a second surgery to manage the complications. The factors that did not influence the complication rate included: implant material, breed of the dog, side on which surgery was performed, meniscal condition and treatment, percentage ligament tear, bandage use, perioperative antimicrobial use, and experience of the surgeon. Factors significantly associated with a higher rate of complications were high body weight and young age of the dog at the time of surgery. Intraoperative complication was reported in one dog (0.3%). In this case the suture tore through the attachment of the gastrocnemius muscle at the lateral fabella when the suture was tightened. Neurological deficits attributed to peroneal nerve damage were evident in one dog (0.3%) immediately after surgery. Significant improvement was noted at 1 week in this case so no additional treatment was performed. Neurological deficit resolved by 4 weeks after surgery. In the study the number of castrated and intact male dogs that developed complications was higher as compare to spayed and intact females. Surgical site infection, confirmed by use of positive bacteriologic cultures was reported in 14 of 363 cases (3.9%). Additional surgery was performed in all 14 dogs. Seven of 363 (1.9%) cases developed post-liminary meniscal tears resulting in lameness and requiring a second surgery. The post-liminary meniscal tear rate was 0% in cases in which a meniscal release was performed and 15.2% in cases in which a meniscal release was not performed. Other complications included, incisional complications (swelling and discharge, self-trauma and bandage-related complications) in 32 of 363 cases (8.8%), and implant-related complications (swelling and/or lameness) in 10 of 363 cases (2.8%). A retrospective study assessing risk factors for infection-inflammation in dogs undergoing surgery for rupture of the CrCL, the infection rate for the LSS procedure was 4.2% of 496 cases (209). Factors associated with a lower rate of infection-inflammation consisted of use of suture material other than skin staples for skin closure and postoperative oral administration of antimicrobials.

Following factors were found not to significantly affect the incidence of postoperative infection-inflammation; concurrent infection, experience of the primary surgeon, use of metal crimps, partial versus complete rupture of the CrCL, concurrent use of NSAIDs, and type and size of nylon or metal implants. Another study reported the incidence of infection and premature crimp failure in 110 cases of stifle stabilization using Veterinary Instrumentation crimp clamp system (210). There were 35 first-opinion and 75 second-opinion cases, of which 15 had had surgery already. Nine (8%) cases had slippage of NLL through the crimp, and in four cases there was a persistent infection. All these cases required exploratory surgery 2 to 6 weeks postoperatively. A further six cases developed minor postoperative infection and were successfully treated with antibiotic. Six out of 10 postoperative infection cases were the ones that were referred because of the failure of first lateral stabilization procedure. These results show that the infection rates were higher in such cases with overall infection rate at 9%. Crimp slippage was judged to have occurred by the presence of significant cranial drawer sign and an abnormal radiographic location of the crimp.

The overall reported complication rate of 17.4% for LSS procedure (208) is lower than the reported complication rate for other surgical techniques used for management of CrCL-D stifles. The complication rate following FHT is 17% to 50% (13, 163, 168). The complication rate associated with TPLO is between 19% and 28%, and that associated with TTA is 31% (152, 209, 211, 212).

CHAPTER II: Effect of tibial insertion points for lateral suture stabilization on the kinematics of the cranial cruciate ligament deficient-stifle during early, middle and late stance: An *in vitro* study

A. Objective

The purpose of our study was to evaluate and compare the effect of LSS_{TT} (distal anchor through the tibial tuberosity) and LSS_{EG} (distal anchor through the cranial eminence of the extensor groove) on kinematics of the canine CrCL-D stifle during early, middle and late stance. The proximal anchor point for both techniques was the lateral femoro-fabellar ligament.

B. Null Hypothesis

Our null hypothesis was that there would be no difference in stifle kinematics between the LSS techniques and the CrCL-intact stifle during early, middle and late stance phases of gait.

C. Materials and methods

C.1 Specimen preparation

Thirty two canine cadaver hind limbs were harvested from 16 skeletally mature medium size (weight range 22 – 36.4kg) dogs via disarticulation of the coxofemoral joint. The dogs were euthanized for reasons unrelated to this study. Various breeds of dogs were utilized for this study. Skin and soft tissues were removed from hind limbs to the level of the metatarsus. The patella, patellar ligament, medial and lateral collateral ligaments, joint capsule, cranial and caudal cruciate ligaments, fabellae, femoro-fabellar ligaments, proximal ~4cm of gastrocnemius muscle and medial and lateral menisci were preserved. Lateral radiographs were taken of all stifles to ensure skeletal maturity and that there was no radiographic evidence of orthopedic disease. The limbs were then wrapped in saline (0.9% NaCl) soaked towels and stored at -80°C until biomechanical testing. Immediately before testing the limbs were thawed at 4°C in a refrigerator for approximately 14-16 hours.

The proximal part of the femur was osteotomized transversely 5 cm distal to the most proximal aspect of the femoral head, and the femoral diaphysis was then potted using polymethylmethacrylate (PMMA; Technovit: Jorgensen Laboratories Inc, Loveland, CO, USA) within 3.8cm diameter polyvinyl chloride pipe. 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) (25). Once the limb was loaded in the mounting bracket the total length of the altered limb, PVC pipe and PMMA, and the mounting bracket approximated the length of the limb prior to performing the femoral ostectomy. A strand of 45.5 kg (100lbs) monofilament nylon leader line (Eagle Claw Fishing Tackle, Denver, CO, USA) was passed through a 2.0 mm diameter hole that was drilled in the center of the patella from medial to lateral. The two free strands of nylon were tied together to form a loop with its distal portion containing the patella. To simulate the quadriceps mechanism, the proximal portion of the nylon loop was attached to a turnbuckle link that was connected to an eyelet attached to the mounting bracket at its most cranial and proximal aspect (Figure 1). Another strand of 45.5 kg (100lb) monofilament nylon leader line was placed through a 2.0 mm diameter hole drilled through the proximal one third of the calcaneus. The two free strands were tied together to form a loop with its distal portion containing the calcaneus. The proximal portion of the nylon loop was attached to a turnbuckle link. At the approximate level of the origin of the gastrocnemius muscle one 6 mm eyelet was placed in the center of the caudal part of the femur. This eyelet was attached to the end of the turnbuckle link attached to the calcaneus. Together, the loop of nylon through the calcaneus, the turnbuckle link and the eyelet attached at the level of origin of the gastrocnemius muscle simulated the gastrocnemius muscle. Throughout testing isotonic saline was sprayed on the limbs intermittently to keep the tissues moist.

C.2 Cranial cruciate ligament rupture simulation

CrCL rupture simulation was performed as described previously (213). In order to simulate rupture of the CrCL a mini cranio-medial arthrotomy was performed to gain access into the stifle joint. The joint was inspected and both cranial and caudal cruciate ligaments were found to be intact in all specimens. The CrCL was isolated and transected using a nerve-hook (Miltex Instruments, York, PA, USA) and a beaver blade (376400 beaver blade: BD and Company, Franklin Lakes, NJ, USA). Transection was confirmed by visual inspection and presence of palpable CTT. The menisci were also inspected and were grossly normal in all specimens. The joint capsule was then closed with 0 (metric 3.5) polydioxanone (PDS; Ethicon, Inc., a Johnson & Johnson company, Somerville, NJ, USA) sutures in an interrupted cruciate pattern.

C.3 Lateral suture stabilization techniques

Lateral suture stabilization with distal anchor through tibial tuberosity (LSS_{TT}; Figure 2A): LSS_{TT} was performed as described previously (12, 17, 22). A curved Securos J-needle (Securos J-needle, Securos Veterinary Orthopedics, Sturbridge, MA, USA) loaded with 36.4 kg (80lb) test monofilament nylon leader line was passed through the lateral femoro-fabellar ligament in a proximal to distal direction. A 3/32″ Steinmann pin (IMEX Veterinary, Inc., Longview, Texas, USA) was used to drill a hole through the tibial tuberosity, from medial to lateral, 1 cm caudal to the attachment of the patellar ligament. The distal end of nylon was passed caudal to the patellar ligament along the proximal aspect of the tibial plateau from lateral to medial and external to the joint capsule. The suture was then passed through the hole in the proximal tibia in a medial to lateral direction and then secured to the proximal strand in a figure-of-eight configuration. The suture was tensioned and secured using the Securos crimp clamp system (Figure 3; PowerX Basic Cruciate Repair 80# Kit: Securos Inc., Fiskdale, MA, USA) according to manufacturer recommendations. With the stifle held at 135° of flexion the nylon leader line was tensioned until cranial drawer motion could no longer be elicited. The crimp clamp was then crimped with a crimper and the excess suture was removed.

Lateral suture stabilization with distal anchor through cranial eminence of extensor groove (LSS_{EG}; Figure 2B): A curved Securos J-needle loaded with 36.4 kg (80lb) test monofilament nylon leader line was passed through the lateral femoro-fabellar ligament in a proximal to distal direction. A 3/32″ Steinmann pin was used to drill a hole in a distal to proximal direction in the cranial prominence of the extensor groove (EG). The hole was centered on the cranial prominence of the extensor groove and exited just cranial to the tibial attachment of the intermeniscal ligament at the level of the caudal edge of the fat pad (17). The distal end of nylon was passed through this hole in a proximal to distal direction and secured to the proximal end in a figure of eight configuration in the same manner as for LSS_{TT}.

C.4 Biomechanical testing

Each limb was mounted in a custom-designed loading frame similar to the frame used in a study performed by Butler et al (Figure 1) (25). Custom hinges as described by Kim et al. and Butler et al were used to facilitate adjustment of the abduction-adduction angle and allow for unconstrained femoral axial rotation during testing (Figure 1) (25, 27). The stifle angle was adjusted using the previously placed turnbuckles to either 145°, 135° or 125°, approximating early, middle, and late stance phase during walking (23). The hip and tarsal angles were simultaneously adjusted during testing to correspond with the phase of gait being tested (23). In the early, middle and late stance phases the hip angles used were 148°, 160° and 178° respectively, while the tarsal angles were 140°, 145° and 155°, as previously described (23, 214). The values of hip angles were obtained from measurements described by Hottinger et al (23) taking into consideration the angle of inclination (approximately 45^{o}) of the pelvis (214). In our study hip angles were measured relative to the floor (i.e. relative to a vertical line drawn through the hip and perpendicular to the ground) since the pelvis was not intact. The abduction/adduction angle was set by visual inspection prior to testing to approximate even distribution of forces across the femoral condyles. The joint angles were measured with a plastic goniometer with each arm aligned along the central axis of the bone diaphysis proximal and distal to the joint (25, 27, 28, 215). The paw was allowed to rest on but was not fixed to the base of the loading frame as described previously (25). A textured surface (220 grit sandpaper) was fixed to the steel-loading frame to prevent cranio-caudal paw slippage during testing. One electromagnetic tracking sensor (FASTRAK, Polhemus; Colchester, VT, USA) was attached to the medial aspect of the distal femur and another to the proximal tibia using 3.18 mm end threaded pins (INTERFACE® Fixation Half-pins - Standard Threads, IMEX™ Veterinary, Inc., Longview, TX, USA) and nylon spacers to prevent direct contact with the stainless steel pin (Figure 1). We used positive threaded pins to mount sensors on the tibia and femur to prevent sliding or rotation of pins at the pin-bone interface as this was noticed in our test samples (not included in the study) where smooth Steinman pins were used. It is important to prevent any movement of pins or any external movement of sensors once they are mounted as this obviously will interfere with measurements.

C.5 Testing protocol

One hind limb from each canine cadaver was allotted to LSS_{TT} group and the other limb was allotted to LSS_{EG} group randomly. This resulted in total of 16 limbs (eight right and eight left) for each treatment. The limbs were tested in the following sequence: (1) Normal (CrCL intact, tibial hole drilled for LSS_{TT} or LSS_{EG} and lateral suture pre-placed just through lateral femoro-fabellar ligament); (2) CrCL-D (transected CrCL) with lateral suture pre-placed just through lateral femoro-fabellar ligament; (3) LSS_{TT} or LSS_{EG} (the suture was tensioned for these groups as described under LSS stabilization techniques). The limbs were evaluated for all conditions at all three stifle angles with corresponding hip and tarsal angles.

C.6 Data collection and analysis

The electromagnetic tracking system is designed to detect position and orientation of the sensors relative to the electromagnetic source transmitter (Figure 1) (216). It measures six degrees of freedom: X, Y, Z, Azimuth, Elevation and Roll (216) in a Cartesian coordinate system (Figure 4). In this study, cranial-caudal (X), proximal-distal (Y) and medial-lateral (Z) translations and internal-external (elevation) and varus-valgus (roll) rotations of the tibia relative to the femur between the 3 stifle conditions (CrCL intact, CrCL-D, and LSS_{EG} or LSS_{TT}) at each stifle angle (145°, 135° and 125°) were obtained using electromagnetic sensors. Stifle flexion-extension (azimuth) was a controlled variable, purposely kept consistent during early (145^0), mid (135^0) and late stance (125^0) for all groups. To simulate *in vivo* conditions, a load equivalent to 33% of the dog's body weight (27) was applied to the limb using the limb-press platform and appropriately sized weights for the limb being tested (Figure 1). The turnbuckles were adjusted throughout testing to maintain the appropriate hip, stifle and tarsal angles. During testing, the axial rotational hinge was left unconstrained. The cranial-caudal, medial-lateral and proximal-distal distance and internal-external and varus-valgus rotation difference between tibial and femoral sensor placement for intact CrCL group was considered base line (equal to zero) at each stifle angle to facilitate data interpretation. The values for all kinematic variables were determined as the relative difference between the coordinate position of the femoral and tibial tracking sensors between CrCL-D group or treatment group (LSS_{TT} or LSS_{EG}) and intact CrCL group (baseline) at that specific stifle angle. As demonstrated in Figure 5, corrections were made to tibial translation measurements ['XM' (measured X), 'ZM' (measured Z) and 'YM' (measured Y)] to account for translations occurring due to tibial rotations in different planes. Correction for 'XM' was determined as follows: X correction factor ('XCF') = sin (absolute value of elevation) x length of lever arm for internal-external rotation (lever arm = distance from center of sensor to center of bone). As demonstrated in Figure 5A, internal rotation of the tibia leads to less displacement of sensor in cranial-caudal plane ('XM') as compared to amount of displacement of tibia itself ('X') falsely decreasing the values for CTT. The magnitude of 'XCF' depends on the amount of elevation and the length of the lever arm. The correction factor was added to or subtracted from 'XM' depending on the direction of elevation (internal or external rotation). Correction for 'ZM' was determined as follows: Z correction factor ('ZCF') = length of lever arm - cos (absolute value of elevation) x length of lever arm. As demonstrated in Figure 5B, internal and external rotation of tibia falsely decreases the tibial translation measurements in Z plane. The correction factor was added to 'ZM' for internal and external rotation to obtain actual tibial translation value ('Z') in medial-lateral plane. Correction for 'YM' was determined as follows: Y correction factor ('YCF') = sin (absolute value of roll) x length of lever arm. As demonstrated in Figure 5C, valgus rotation of tibia falsely decreases the tibial translation measurements in Y plane. To obtain actual tibial translation value ('Y') in proximal-distal plane, the correction factor was added to or subtracted from 'YM' for valgus and varus rotation, respectively.

C.7 Statistical Analysis

Differences in X, Y, Z, Elevation and Roll between the 3 stifle conditions (CrCL intact, CrCL-D and LSS_{TT} or LSS_{EG}) at each stifle angle were evaluated. Normal probability plots showed that all measurements (X, Y, Z, Elevation and Roll) followed an approximately normal distribution. Subsequently, data were summarized as mean ± standard deviation (SD) for each combination of method (LSS_{TT} vs LSS_{EG}), stifle angle, (145^0, 135^0 and 125^0) and condition (CrCL-D vs LSS repaired). Each of these means was compared to 0 (CrCL-intact group baseline) using a one-sample t-test followed by Bonferroni's procedure for multiple comparisons. Comparisons between means (least squares means) were performed using mixed-model repeated-measures analysis of covariance.

To compare CrCL-D vs LSS repaired conditions for each of the treatment methods (LSS_{TT} vs LSS_{EG}), the linear model included condition (CrCL-D vs LSS repaired), stifle angle, condition*stifle angle, and dog weight (the covariate) as fixed effects. Limb ID within dog ID was specified as the random effect. To explicitly compare condition least squares means at each of the stile angles for each of the outcomes, the slicediff option of the glimmix procedure was applied to the Condition*Stifle interaction followed by Bonferroni's procedure for multiple comparisons.

To compare LSS_{TT} vs LSS_{EG} for each of the conditions (CrCL-D vs LSS repaired) the linear model included treatment method (i.e., LSS_{TT} vs LSS_{EG}), stifle angle (145^0, 135^0 and 125^0), treatment*stifle angle, and dog weight (the covariate) as fixed effects. Dog ID and limb ID within dog ID were specified as the random effects. To explicitly compare treatment least squares means at each of the stile angles for each of the outcomes, the slicediff option of the glimmix procedure was applied to the treatment *Stifle interaction followed by Bonferroni's procedure for multiple comparisons.

Common options across all models included Kenward-Roger for denominator degrees of freedom and the autoregressive order one (AR 1) covariance structure for measurements within each limb. Additionally, residual plots were inspected to verify model adequacy (i.e., the errors followed a normal distribution with constant variance). Statistical analyses were considered significant at $p<0.05$. All analyses were performed using SAS version 9.2 (SAS Institute Inc., Cary, NC, USA).

D. Results

The mean ± SD body weight of the dogs was 26.8 $\pm$ 4.6 kg. All dogs were skeletally mature and free from orthopedic disease of the stifle as determined by lateral radiographs. The results for translation of the tibia relative to the femur i.e. X, Y and Z for the 3 testing groups and the 3 testing stifle angles are presented in table 1. The results for rotation of the tibia relative to the femur i.e. elevation and roll are presented in table 2.

D.1 Cranial-caudal tibial translation

Transection of the CrCL resulted in significant CTT (increased X) relative to the femur at all stifle angles tested when compared to intact CrCL group (baseline). After LSS_{EG} repair statistically significant CTT was no longer present at 125^o stifle angle but it failed to restore CTT at 135^o and 145^o. Significant CTT was no longer present at any of the stifle angles evaluated after LSS_{TT} repair. The LSS_{TT} and LSS_{EG} CTT values were significantly lower than CrCL-D values at all stifle angles evaluated. There was no significant difference in CTT between LSS groups at 125^o and 145^o but it was significantly lower for LSS_{TT} at 135^o.

D.2 Proximal-distal tibial translation

Significant proximal translation (increased Y) of the tibia relative to the femur was present in CrCL-D group at stifle angles 135^0 and 145^0 when compared to intact CrCL group. This was no longer present after both LSS repairs. The values for proximal-distal tibial translation were significantly smaller (more distally located) for both LSS groups at all measured stifle angles when compared to values for CrCL-D stifle. There was no significant difference in proximal-distal tibial translation values between LSS_{TT} and LSS_{EG} at all stifle angles tested.

D.3 Medial-lateral tibial translation

Transection of the CrCL resulted in significant medial translation (decreased Z) of the tibia relative to the femur at all stifle angles tested. Significant lateral translation (increased Z) of the tibia occurred at stifle angle of 125^o after LSS_{EG} and at all measured stifle angles after LSS_{TT} when compared to CrCL-I stifle. The values for medial-lateral tibial translation were significantly higher (more laterally located) for both LSS groups at all measured stifle angles when compared to values for CrCL-D stifle. There was no significant difference for medial-lateral tibial translation between LSS_{TT} and LSS_{EG} groups at all stifle angles tested.

D.4 Elevation (Axial tibial rotation)

Significant internal rotation (decreased elevation) of the tibia relative to the femur was present in CrCL-D group at all stifle angles evaluated when compared to intact CrCL group. Treatment with LSS_{EG} resulted in significant external rotation at all stifle angles when compared to CrCL-D stifles. When compared to CrCL intact stifles treatment with LSS_{EG} resulted in significant external tibial rotation at 125^o and 135^o stifle angles. Treatment with LSS_{TT} resulted in significant external rotation at all stifle angles when compared to CrCL-D limbs and CrCL intact limbs. The values for elevation were significantly higher (external rotation) for LSS_{TT} as compared to values for LSS_{EG} at 135^o and 145^o of stifle angles with no significant difference at stifle angle of 125^o.

D.5 Roll (Stifle varus-valgus)

Transection of the CrCL resulted in slight stifle valgus (increased roll) which was significant at stifle angles of 125^o and 135^o. Performance of both LSS_{EG} and LSS_{TT} resulted in significant stifle varus (decreased roll) at all stifle angles measured when compared to CrCL-intact and CrCL-D groups. There was no significant difference in values for roll (varus-valgus angulations) between LSS_{TT} and LSS_{EG} groups at all stifle angles measured.

E. Discussion

The LSS_{TT} is reportedly one of the most common extra-articular methods used to stabilize the CrCL-D stifle in the dog (17). Typically a single lateral (and occasionally a medial) suture is used. Studies by Roe et al and Hulse et al, showed various isometric points on distal femur and proximal tibia for lateral suture attachment sites in the un-loaded stifle (18, 19). In our study, we aimed to evaluate two suggested isometric points on the tibia (18, 19) during early, middle and late stance phase of canine gait. We achieved this by using an electromagnetic tracking system that tracked the position of the tibia relative to the femur in a Cartesian coordinate system. This and similar tracking systems have been successfully used in previous studies and have been reported to have a positional resolution of 0.1 mm and a rotational resolution of 0.1° (25, 217, 218). Positional and rotational errors have been reported to be 1.8% and 1.6 % respectively (217).

The significant increase in CTT after CrCL loss noted in this study during early, middle and late stance has also been reported in other studies at a single standing angle (17, 27, 111, 112). The results of the present study showed that treatment with LSS_{TT} as described was effective in eliminating CTT during early, mid and late stance suggesting that the LSS_{TT} is capable of restoring this aspect of stifle kinematics comparable to normal. This is consistent with previous reports which demonstrated the effectiveness of LSS_{TT} at eliminating CTT at mid stance (135° of stifle flexion) (17, 22). Failure of LSS_{EG} to consistently eliminate CTT during early and mid-stance is consistent with a previous report that demonstrated the failure of LSS_{EG} to eliminate CTT at mid stance (this was the only stance tested in that study) (17). Our results showed no significant difference in CTT between LSS_{EG} and LSS_{TT} during early and late stance whereas CTT values during mid-stance were significantly lower after LSS_{TT} as compared to LSS_{EG}. Overall better performance of LSS_{TT} in controlling CTT as compared to LSS_{EG} may be explained by a more centralized and cranial tibial anchor point for LSS_{TT} likely providing improved restraint of the tibia in the cranial-caudal plane.

Loss of CrCL resulted in significant proximal translation of the tibia during early and mid-stance. This finding is consistent with a previous study by Kim et al which reported significant proximal displacement of tibia after transection of CrCL when the limb was loaded at standing stifle angle (mid stance) (27). Two previous *in vivo* stifle kinematics studies have also reported the proximal translation of tibia relative to femur after CrCL transection (2, 6). The reason for the proximal displacement of the tibia was likely due to a decrease in the joint space that occurred as a result of combined cranial displacement of the tibia and compression of the stifle. Compression of the stifle occurs during the start of the stance phase of gait (2). The cranial displacement of the tibia causes the femur to be displaced both caudally and distally and thus the tibia became proximally oriented relative to the femur (2). Our study showed that both LSS techniques effectively eliminated proximal translation of the tibia. The proximal-distal tibial translation was not significantly different between LSS_{TT} and LSS_{EG} during all phases of stance tested.

Significant medial translation of the tibia in CrCL-D stifles was consistent with previous studies indicating that the CrCL may have an influence on preventing medial translation of the tibia (2, 6). No significant difference was present for medial-lateral tibial translation between two LSS techniques tested during all phases of stance tested. LSS_{EG} resulted in significant lateral translation of the tibia during only late phase of stance but LSS_{TT} resulted in significant lateral translation of the tibia during all phases of stance tested when compared to intact CrCL stifles. Therefore, LSS_{EG} was better than LSS_{TT} at restoring stifle kinematics in medial-lateral plane comparable to normal during early and mid-phases of stance.

Internal rotation demonstrated in the CrCL-D stifles was significantly greater than CrCL intact or LSS treated stifles during all phases of stance tested. Internal rotation of the tibia has been demonstrated in other studies (17, 25, 27) and is expected to occur in CrCL-D stifles because of the role the CrCL plays in limiting internal rotation (10). External tibial rotation noted after LSS repair during all phases of stance was consistent with previous reports that demonstrated this at mid-stance (17, 22). However, LSS_{EG} was better at normalizing tibial axial rotation when compared to LSS_{TT} and did not cause significant external tibial rotation during early stance. LSS_{TT} caused significantly higher external rotation as compared to LSS_{EG} during early and mid-stance but no significant difference was present during late stance. The increased lateral translation and external rotation of tibia relative to the femur after LSS_{TT} as compared to LSS_{EG} may be due to the longer lever arm and more cranial tibial anchor point for LSS_{TT} which may result in increased caudal pull and lateral tibial rotation. In our study maximum mean external tibial rotation after LSS (8.16±3.44 with LSS_{TT} and 5.95±2.51 with LSS_{EG}) was less than previously reported after extra-articular repair (22). In the previous study, 20% of the limbs in the extra-articular repair group exceeded the instrumentation limit for external rotation (>12.4°) (22).

Slight but significant stifle valgus after transection of the CrCL when compared to CrCL-intact stifle was consistent with previous studies (2, 6, 27, 28). In our study both LSS procedures caused slight stifle varus which was significant when compared to CrCL-I stifle during all phases of stance with no significant difference in between both procedures. The slight stifle varus created in the stifle after LSS is likely secondary to joint mal-alignment and in-congruency of the articular surface occurring due to simultaneous tibial rotations and translations. This joint mal-alignment may play a role in altering joint contact mechanics and lead to the progression of osteoarthritis.

Despite excellent clinical outcome (~94% dogs were sound at walk and trot) in a study evaluating 60 stifles after extra-articular stabilization, 65% of the stifles developed radiographic evidence of DJD (191). Recent studies in people have suggested that the progression of DJD is not only associated with the magnitude of tibial translation but also to alterations in the normal load-bearing regions of the knee due to changes in joint kinematics (219). It was suggested that this may be true in the dog as well due to progression of DJD that occur in dogs after CrCL stabilization (22). For example the present study showed that LSS_{TT} technique minimized CTT comparable to normal but caused significant external tibial rotation, lateral tibial translation and stifle varus. These changes may be responsible for abnormal stifle kinematics and hence progression of DJD. However, to our knowledge, the amount of these changes that are acceptable for an extra-articular repair is still unknown. Future studies for development of new techniques or improvement of the current extra-articular stabilization techniques are necessary to restore normal joint kinematics to reduce the propensity for DJD and improve clinical outcome.

This is an *in vitro* study that does not recreate all forces acting on the stifle during weight bearing. Stifle kinematics are complex in nature and therefore difficult to replicate in an *in vitro* testing environment. We evaluated the stifle at the angles145°, 135°, and 125°, instead of a continuum of stance phase as each of these angles previously have been reported to represent the early, middle, and late stance phases of the gait (23). Future *in vitro* models should consider evaluating stifle biomechanics through continuum of stance phase to more closely represent the *in vivo* stance phase. The study was performed on normal canine stifles and the parameters obtained from normal dogs may not be the same as those obtained from dogs with CrCL disease (6). Clinically, rupture of the CrCL leads to significant degenerative changes within the stifle and peri-articular fibrosis which can affect the kinematics of the stifle. This was demonstrated in the above referenced study (6) that evaluated the long-term effect of the transection of the CrCL.

With time it was found that CTT became progressively more abnormal as degenerative changes progressed in the stifle. This variable was not accounted for in our *in vitro* study. Cyclic loading of the LSS was not replicated in this model; only straight-line loading of the limb was taken into consideration (10) as the ex vivo cycling test would not accurately mimic the biological cycling the prosthesis undergoes because of many variables such as weight bearing, stifle conformation, degree of joint pathology, and activity level etc. The use of goniometry for angle measurements may have led to slight variation of joint positioning throughout testing which could potentially affect the results. However, a strong agreement has been shown between goniometric and radiographic angle measurements in a previous study (213), therefore the use of goniometric measurements in the current study is justified. The joint angle measurements have been described based on anatomic landmarks (23, 102, 220) and based on long bone axes (25, 27, 28, 215). A slight discrepancy exists between these two types of measurement methods for determining joint angles because the points of reference do not exactly coincide. For both treatment groups (LSS_{TT} and LSS_{EG}) in our study, we used long bone axes for measurement of joint angles as described by Dennler et al (215) and previous kinematic studies (25, 27, 28). For the hind limbs at walk, the vertical force has a very characteristic "M shape", with a rapid rise during early stance (at foot contact), followed by a drop as the stifle flexes during mid-stance, partially unloading the force plate (97). During late stance (at push-off), a second peak is recorded. During mid-stance vertical ground reaction force (axial load) of 33% body weight on hind limb has been reported, whereas during early and late stance these forces are ~43% and ~35% body weight respectively (93, 97). The difference in vertical reaction force during early, mid and late stance is due to the braking and propulsive phases of the stance in the hind limb (93, 97). For simplification of our model we used same loads (33% of body weight) during early, mid and late stance, which represents a limitation of our model. Taking this fact into consideration, there is a chance that the differences reported in kinematic values for two techniques (LSS_{TT} and LSS_{EG}) during early and late stance might have been slightly different. Similarly, other studies have used same loads during early, mid and late phases of stance for *in vitro* testing for model simplification (25, 26). Future studies are warranted to determine the effect of ~43% and ~35% body weight axial loads during early and late stance respectively on stifle 3D kinematics. A limitation of the method of correction to tibial translational measurements to account for translations occurring due to tibial rotations in different planes is that we performed a series of three corrections, each in a 2-dimensional Euclidean space and yet, the rotations and translations occur simultaneously in a 3-dimensional Euclidean space. We chose the 2-dimensional approach because it was easy to implement, and readily understandable. However, an appropriate alternative is to express all kinematic data in matrix form and perform the analysis using linear algebra (221-223).

Long-term stability of LSS relies on fibrosis of the peri-articular structures because the sutures eventually fail (10). Excessive suture tension may predispose to early failure of the suture, decrease range-of-motion and abnormal intra-articular pressure. After performing a modified retinacular imbrication technique a study showed a 30^{o} decrease in stifle flexion and increased external rotation raising concerns of increased lateral compartmental pressures (24). Recently, Tonks et al determined the effect of suture tension on contact pressures in the lateral compartment of the stifle for LSS_{TT} technique (224). This ex vivo biomechanical study found that excessive extra-articular prosthesis tension caused increased lateral compartmental pressures. The study also showed that axial compression eliminated any significant alteration of lateral compartment pressures and redistributed the abnormal pressure distribution to both compartments when the stifles were held at mid stance (135^0 angle). Even though LSS_{TT} minimizes CTT comparable to intact CrCL stifles, osteoarthritis still progresses which could be explained by alterations in joint kinematics, for example, change in intra-articular pressures due to external tibial rotation at other phases of weight bearing e.g. early and late phase, which were not tested in Tonks et al study. It would be interesting to measure intra-articular pressures and also to determine if axial loading will redistribute any abnormal pressures during early and late phases of stance. In our study significantly less external tibial rotation was noted in LSS_{EG} group as compared to LSS_{TT} group during early and mid-stance, which may indicate that the pressure generated in lateral compartment would be less after LSS_{EG} repair. Further studies would be needed to determine this and the significance of change in stifle intra-articular pressures after LSS in canine patients.

The most likely reason that both LSS techniques failed to completely restore normal stifle kinematics in CrCL-D stifles is failure of suture to maintain a constant tension at different joint angles with consequent joint instability and change in joint kinematics (162). Because of the caudal roll of the femoral condyles and internal rotation of the tibia during flexion of the stifle, the relative position between the femur and tibia does not stay constant throughout a range of motion (1). Moreover, the origin and insertion of the CrCL are not isometric, explaining why some of the fibers become lax at certain angles of stifle flexion and extension (1). Therefore, the term isometry should be used cautiously, and the anchor points used for extra-articular stabilization may be better defined as 'near-isometric' because a truly isometric suture is not achievable (1, 10). Because of the presence of variables such as mechanical properties of different suture materials and the options for fixation (i.e. soft tissue vs. bony anchorage), the guidelines regarding suture tension are not clear. Persistent stifle instability in different planes in the current study after repair may be a reflection of the type of prosthesis material or use of a soft tissue proximal anchor point. Better results may be seen using different types of prosthetic material or a bony proximal anchor point.

F. Conclusion

Our null hypothesis was rejected as both LSS techniques restored stifle kinematics in CrCL-D stifles to varying amounts with both techniques failing to restore normal 3-D stifle kinematics *in vitro*. From the current study we conclude that using the cranial eminence of the extensor groove as a tibial anchor site for LSS enhances stifle kinematics of CrCL-D stifle in the medial-lateral plane and also in axial rotation but performs poorly in normalizing stifle kinematics in the cranial-caudal plane as compared to standard LSS (LSS_{TT}) during the stance phase of gait in canine cadaveric hind limbs. To our knowledge, the clinical significance of tibial translational and rotational changes caused by two tested LSS techniques is unknown. Further *in vitro* and *in vivo* studies comparing extra-articular procedures as well as studies on the contact mechanics of the stifle are necessary to help determine the ideal extra-articular technique for CrCL repair in the dog.

REFERENCES

1. Kowaleski MP, Boudrieau RJ, Pozzi A. Stifle Joint. In: Tobias KM, Johnston SA, editors. Veterinary Surgery Small Animal. 1. St. Louis: Elsevier Saunders; 2012. p. 906-98.
2. Korvick DL, Pijanowski GJ, Schaeffer DJ. Three-dimensional kinematics of the intact and cranial cruciate ligament-deficient stifle of dogs. J Biomech. 1994 Jan;27(1):77-87. PubMed PMID: 8106538. Epub 1994/01/01. eng.
3. Lazar TP, Berry CR, deHaan JJ, Peck JN, Correa M. Long-term radiographic comparison of tibial plateau leveling osteotomy versus extracapsular stabilization for cranial cruciate ligament rupture in the dog. Vet Surg. 2005 Mar-Apr;34(2):133-41. PubMed PMID: 15860104. Epub 2005/04/30. eng.
4. Cook JL. Cranial cruciate ligament disease in dogs: biology versus biomechanics. Vet Surg. 2010 Apr;39(3):270-7. PubMed PMID: 20522208. Epub 2010/06/05. eng.
5. Arnoczky SP, Marshall JL. The cruciate ligaments of the canine stifle: an anatomical and functional analysis. Am J Vet Res. 1977 Nov;38(11):1807-14. PubMed PMID: 931164. Epub 1977/11/01. eng.
6. Tashman S, Anderst W, Kolowich P, Havstad S, Arnoczky S. Kinematics of the ACL-deficient canine knee during gait: serial changes over two years. J Orthop Res. 2004 Sep;22(5):931-41. PubMed PMID: 15304262. Epub 2004/08/12. eng.
7. Andriacchi TP, Koo S, Scanlan SF. Gait Mechanics Influence Healthy Cartilage Morphology and Osteoarthritis of the Knee. Journal of Bone and Joint Surgery-American Volume. 2009 Feb;91A:95-101. PubMed PMID: ISI:000263243000023. English.
8. 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 Jul 1;157(1):79-84. PubMed PMID: 5463398. Epub 1970/07/01. eng.
9. Kim SE, Pozzi A, Kowaleski MP, Lewis DD. Tibial osteotomies for cranial cruciate ligament insufficiency in dogs. Veterinary Surgery. 2008 Feb;37(2):111-25. PubMed PMID: ISI:000252776800001. English.
10. Tonks CA, Lewis DD, Pozzi A. A review of extra-articular prosthetic stabilization of the cranial cruciate ligament-deficient stifle. Vet Comp Orthop Traumatol. 2011;24(3):167-77. PubMed PMID: 21373714. Epub 2011/03/05. eng.
11. Leighton RL. Preferred method of repair of cranial cruciate ligament rupture in dogs: a survey of ACVS diplomates specializing in canine orthopedics. American College of Veterinary Surgery. Vet Surg. 1999 May-Jun;28(3):194. PubMed PMID: 10338165. Epub 1999/05/25. eng.
12. Flo GL. Modification of the latearl retinacular imbrication technique for stabilizing cruciate ligament injuries. . Journal of the American Animal Hospital Association. 1975;11:570-6.
13. Cook JL. Extracapsular stabilization. In: Muir P, editor. Advances in the canine cranial cruciate ligament. Ames, IA: Wiley-Blackwell; 2010. p. 163-8.
14. Conzemius MG, Evans RB, Besancon MF, Gordon WJ, Horstman CL, Hoefle WD, 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 Jan 15;226(2):232-6. PubMed PMID: 15706973. Epub 2005/02/15. eng.
15. Au KK, Gordon-Evans WJ, Dunning D, O'Dell-Anderson KJ, Knap KE, Griffon D, et al. Comparison of Short- and Long-term Function and Radiographic Osteoarthrosis in Dogs After Postoperative Physical Rehabilitation and Tibial Plateau Leveling Osteotomy or Lateral Fabellar Suture Stabilization. Veterinary Surgery. 2010 Feb;39(2):173-80. PubMed PMID: ISI:000274258600006. English.
16. Moore KW, Read RA. Cranial cruciate ligament rupture in the dog--a retrospective study comparing surgical techniques. Aust Vet J. 1995 Aug;72(8):281-5. PubMed PMID: 8579557.
17. Harper TA, Martin RA, Ward DL, Grant JW. 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 Sep-Oct;33(5):531-41. PubMed PMID: 15362993. Epub 2004/09/15. eng.
18. Roe SC, Kue J, Gemma J. Isometry of potential suture attachment sites for the cranial cruciate ligament deficient canine stifle. Veterinary and Comparative Orthopaedics and Traumatology. 2008;21(3):215-20. PubMed PMID: ISI:000257097200006. English.
19. Hulse D, Hyman W, Beale B, Saunders B, Hosgood G. Determination of isometric points for placement of a lateral suture in treatment of the cranial cruciate ligament deficient stifle (vol 23, pg 163, 2010). Veterinary and Comparative Orthopaedics and Traumatology. 2010;23(4):293-. PubMed PMID: ISI:000280904600015. English.
20. Wallace AM, Cutting ED, Sutcliffe MPF, Langley-Hobbs SJ. A biomechanical comparison of six different double loop configurations for use in the lateral fabella suture technique. Veterinary and Comparative Orthopaedics and Traumatology. 2008;21(5):391-9. PubMed PMID: ISI:000261824600003. English.
21. Sicard GK, Hayashi K, Manley PA. Evaluation of 5 types of fishing material, 2 sterilization methods, and a crimp-clamp system for extra-articular stabilization of the canine stifle joint. Vet Surg. 2002 Jan-Feb;31(1):78-84. PubMed PMID: 11778171. Epub 2002/01/05. eng.
22. Snow LA, White R, Gustafson S, Xie L, Hosgood G, Monroe WT, et al. Ex Vivo Comparison of Three Surgical Techniques to Stabilize Canine Cranial Cruciate Ligament Deficient Stifles. Veterinary Surgery. 2010 Feb;39(2):195-207. PubMed PMID: ISI:000274258600009. English.
23. Hottinger HA, DeCamp CE, Olivier NB, Hauptman JG, Soutas-Little RW. Noninvasive kinematic analysis of the walk in healthy large-breed dogs. Am J Vet Res. 1996 Mar;57(3):381-8. PubMed PMID: 8669773. Epub 1996/03/01. eng.
24. Chailleux N, Lussier B, De Guise J, Chevalier Y, Hagemeister N. In vitro 3-dimensional kinematic evaluation of 2 corrective operations for cranial cruciate ligament-deficient stifle. Can J Vet Res. 2007 Jul;71(3):175-80. PubMed PMID: 17695591. Pubmed Central PMCID: 1899862. Epub 2007/08/19. eng.
25. Butler JR, Syrcle JA, McLaughlin RM, Elder SH. The effect of tibial tuberosity advancement and meniscal release on kinematics of the cranial cruciate ligament-deficient stifle during early, middle, and late stance. Vet Comp Orthop Traumatol. 2011;24(5):342-9. PubMed PMID: 21792471. Epub 2011/07/28. eng.
26. Johnson K, Lanz O, Elder S, McLaughlin R, Werre S, Harper T. The effect of stifle angle on cranial tibial translation following tibial plateau leveling osteotomy: an in vitro experimental analysis. Can Vet J. 2011 Sep;52(9):961-6. Epub 2012/03/02.
27. Kim SE, Pozzi A, Banks SA, Conrad BP, Lewis DD. Effect of tibial plateau leveling osteotomy on femorotibial contact mechanics and stifle kinematics. Vet Surg. 2009 Jan;38(1):23-32. PubMed PMID: 19152614. Epub 2009/01/21. eng.
28. Kim SE, Pozzi A, Banks SA, Conrad BP, Lewis DD. Effect of tibial tuberosity advancement on femorotibial contact mechanics and stifle kinematics. Vet Surg. 2009 Jan;38(1):33-9. PubMed PMID: 19152615. Epub 2009/01/21. eng.
29. Robins GM. The canine stifle joint. Canine Orthopedics. Philadelphia: Lea & Febiger; 1990. p. 693-720.
30. Evans H, Hermanson JW. The skeleton, arthrology, the muscular system. In: HE E, editor. Miller's anatomy of the dog. philadelphia: W.B. Saunders; 1993. p. 122-384.
31. Arnoczky SP, Torzilli PA, Marshall JL. Biomechanical Evaluation of Anterior Cruciate Ligament Repair in Dog - Analysis of Instant Center of Motion. Journal of the American Animal Hospital Association. 1977;13(5):553-8. PubMed PMID: ISI:A1977DT37500003. English.
32. Hayashi K, Muir P. Histology of cranial cruciate ligament rupture. In: Muir P, editor. Advances In The Canine Cranial Cruciate Ligament. Ames, IA: Wiley-Blackwell; 2010. p. 45-51.

33. Heffron LE, Campbell JR. Morphology, histology and functional anatomy of the canine cranial cruciate ligament. Vet Rec. 1978 Apr 1;102(13):280-3. PubMed PMID: 654025.
34. Arcand MA, Rhalmi S, Rivard CH. Quantification of mechanoreceptors in the canine anterior cruciate ligament. Int Orthop. 2000;24(5):272-5. PubMed PMID: 11153457.
35. Yahia LH, Newman NM, St-Georges M. Innervation of the canine cruciate ligaments. A neurohistological study. Anat Histol Embryol. 1992 Mar;21(1):1-8. PubMed PMID: 1585987.
36. Oconnor BL, Woodbury P. The Primary Articular Nerves to the Dog Knee. Journal of Anatomy. 1982;134(May):563-72. PubMed PMID: WOS:A1982NS60500010. English.
37. Vasseur PB, Arnoczky SP. Collateral ligaments of the canine stifle joint: anatomic and functional analysis. Am J Vet Res. 1981 Jul;42(7):1133-7. PubMed PMID: 7271030.
38. Evans HE. The heart and arteries. . In: Evans HE, editor. Miller's anatomy of the dog. 3rd ed. Philadelphia, PA: W. B. Saunders; 1993. p. 586-681.
39. Evans HE. Veins. In: HE E, editor. Miller's anatomy of the dog. 3rd ed. Philadelphia: W.B. Saunders; 1993. p. 682-716.
40. Bezuidenhout AJ. The lymphatic system. In: HE E, editor. Miller's anatomy of the dog. 3rd ed. Philadelphia: W.B. Saunders; 1993. p. 717-57.
41. Henderson RA, Milton JL. The tibial compression mechanism: A diagnostic aid in stifle injuries. J Am Anim Hops Assoc. 1978 (14):474-9.
42. Hermanson JW, Evans HE. The muscular system. In: Evans HE, editor. Miller's anatomy of the dog. 3rd ed. Philadelphia: W.B. Saunders; 1993. p. 258-384.
43. Arnoczky SP. Pathomechanics of cruciate ligament and meniscal injuries. In: Bojrab MJ, editor. Disease Mechanisms in Small Animal Surgery Philadelphia: Lea & Febiger; 1993. p. 764-76.
44. Slocum B, Devine T. Cranial tibial thrust: a primary force in the canine stifle. J Am Vet Med Assoc. 1983 Aug 15;183(4):456-9. PubMed PMID: 6618973.
45. Hayashi K, Landsdowne JL, Dejardin LM. Cranial Cruciate Ligament and Meniscal Injuries in Dogs. In: Bojrab MJ, editor. Mechanisms of Disease in Small Animal Surgery. Jackson: Teton New Media; 2010. p. 646-54.
46. Arnoczky SP, Warren RF. The microvasculature of the meniscus and its response to injury. An experimental study in the dog. Am J Sports Med. 1983 May-Jun;11(3):131-41. PubMed PMID: 6688156.
47. Pozzi A, Tonks CA, Ling HY. Femorotibial contact mechanics and meniscal strain after serial meniscectomy. Vet Surg. 2010 Jun;39(4):482-8. PubMed PMID: 20345523.
48. Duval JM, Budsberg SC, Flo GL, Sammarco JL. Breed, sex, and body weight as risk factors for rupture of the cranial cruciate ligament in young dogs. Journal of the American Veterinary Medical Association. 1999 Sep 15;215(6):811-4. PubMed PMID: WOS:000082471400014. English.
49. Vasseur PB, Pool RR, Arnoczky SP, Lau RE. Correlative biomechanical and histologic study of the cranial cruciate ligament in dogs. Am J Vet Res. 1985 Sep;46(9):1842-54. PubMed PMID: 3901837.
50. Whitehair JG, Vasseur PB, Willits NH. Epidemiology of cranial cruciate ligament rupture in dogs. J Am Vet Med Assoc. 1993 Oct 1;203(7):1016-9. PubMed PMID: 8226247.
51. Wilke VL, Zhang S, Evans RB, Conzemius MG, Rothschild MF. Identification of chromosomal regions associated with cranial cruciate ligament rupture in a population of Newfoundlands. American Journal of Veterinary Research. 2009 Aug;70(8):1013-7. PubMed PMID: WOS:000268608600010. English.
52. Witsberger TH, Villamil JA, Schultz LG, Hahn AW, Cook JL. Prevalence of and risk factors for hip dysplasia and cranial cruciate ligament deficiency in dogs. J Am Vet Med Assoc. 2008 Jun 15;232(12):1818-24. PubMed PMID: 18598150.
53. Colborne GR, Innes JF, Comerford EJ, Owen MR, Fuller CJ. Distribution of power across the hind limb joints in Labrador Retrievers and Greyhounds. Am J Vet Res. 2005 Sep;66(9):1563-71. PubMed PMID: 16261830.

54. Fujita Y, Hara Y, Nezu Y, Schulz KS, Tagawa M. Proinflammatory cytokine activities, matrix metalloproteinase-3 activity, and sulfated glycosaminoglycan content in synovial fluid of dogs with naturally acquired cranial cruciate ligament rupture. Vet Surg. 2006 Jun;35(4):369-76. PubMed PMID: 16756618.
55. Muir P, Danova NA, Argyle DJ, Manley PA, Hao Z. Collagenolytic protease expression in cranial cruciate ligament and stifle synovial fluid in dogs with cranial cruciate ligament rupture. Vet Surg. 2005 Sep-Oct;34(5):482-90. PubMed PMID: 16266341.
56. Macias C, McKee WM, May C. Caudal proximal tibial deformity and cranial cruciate ligament rupture in small-breed dogs. J Small Anim Pract. 2002 Oct;43(10):433-8. PubMed PMID: 12400640.
57. Morris E, Lipowitz AJ. Comparison of tibial plateau angles in dogs with and without cranial cruciate ligament injuries. Journal of the American Veterinary Medical Association. 2001 Feb 1;218(3):363-6. PubMed PMID: WOS:000166577300017. English.
58. Comerford EJ, Tarlton JF, Avery NC, Bailey AJ, Innes JF. Distal femoral intercondylar notch dimensions and their relationship to composition and metabolism of the canine anterior cruciate ligament. Osteoarthritis and Cartilage. 2006 Mar;14(3):273-8. PubMed PMID: WOS:000236277600010. English.
59. Duerr FM, Duncan CG, Savicky RS, Park RD, Egger EL, Palmer RH. Risk factors for excessive tibial plateau angle in large-breed dogs with cranial cruciate ligament disease. J Am Vet Med Assoc. 2007 Dec 1;231(11):1688-91. PubMed PMID: 18052804.
60. Aiken SW, Kass PH, Toombs JP. Intercondylar Notch Width in Dogs with and without Cranial Cruciate Ligament Injuries. Veterinary and Comparative Orthopaedics and Traumatology. 1995 Sep;8(3):128-32. PubMed PMID: WOS:A1995RZ91000002. English.
61. Stein V, Li L, Guermazi A, Zhang Y, Kent Kwoh C, Eaton CB, et al. The relation of femoral notch stenosis to ACL tears in persons with knee osteoarthritis. Osteoarthritis Cartilage. 2010 Feb;18(2):192-9. PubMed PMID: 19835830.
62. Guerrero TG, Geyer H, Hassig M, Montavon PM. Effect of conformation of the distal portion of the femur and proximal portion of the tibia on the pathogenesis of cranial cruciate ligament disease in dogs. Am J Vet Res. 2007 Dec;68(12):1332-7. PubMed PMID: 18052737.
63. Reif U, Probst CW. Comparison of tibial plateau angles in normal and cranial cruciate deficient stifles of Labrador retrievers. Vet Surg. 2003 Jul-Aug;32(4):385-9. PubMed PMID: 12866002.
64. Wilke VL, Conzemius MG, Besancon ME, Evans RB, Ritter M. Comparison of tibial plateau angle between clinically normal Greyhounds and Labrador Retrievers with and without rupture of the cranial cruciate ligament. Journal of the American Veterinary Medical Association. 2002 Nov 15;221(10):1426-9. PubMed PMID: WOS:000179237100025. English.
65. Comerford EJ, Smith K, Hayashi K. Update on the aetiopathogenesis of canine cranial cruciate ligament disease. Vet Comp Orthop Traumatol. 2011;24(2):91-8. PubMed PMID: 21243176.
66. Doverspike M, Vasseur PB, Harb MF, Walls CM. Contralateral Cranial Cruciate Ligament Rupture - Incidence in 114 Dogs. Journal of the American Animal Hospital Association. 1993 Mar-Apr;29(2):167-70. PubMed PMID: WOS:A1993KW29500012. English.
67. Mostafa AA, Griffon DJ, Thomas MW, Constable PD. Morphometric characteristics of the pelvic limbs of Labrador Retrievers with and without cranial cruciate ligament deficiency. American Journal of Veterinary Research. 2009 Apr;70(4):498-507. PubMed PMID: WOS:000264818300009. English.
68. Renstrom P, Ljungqvist A, Arendt E, Beynnon B, Fukubayashi T, Garrett W, et al. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. Br J Sports Med. 2008 Jun;42(6):394-412. PubMed PMID: 18539658.
69. Dannucci GA, Martin RB, Patterson-Buckendahl P. Ovariectomy and trabecular bone remodeling in the dog. Calcif Tissue Int. 1987 Apr;40(4):194-9. PubMed PMID: 3107772.

70. Wingfield C, Amis AA, Stead AC, Law HT. Comparison of the biomechanical properties of rottweiler and racing greyhound cranial cruciate ligaments. J Small Anim Pract. 2000 Jul;41(7):303-7. PubMed PMID: 10976625.
71. Wilke VL, Conzemius MG, Kinghorn BP, Macrossan PE, Cai W, Rothschild MF. Inheritance of rupture of the cranial cruciate ligament in Newfoundlands. J Am Vet Med Assoc. 2006 Jan 1;228(1):61-4. PubMed PMID: 16426167.
72. Solomonow M, Baratta R, Zhou BH, Shoji H, Bose W, Beck C, et al. The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. Am J Sports Med. 1987 May-Jun;15(3):207-13. PubMed PMID: 3618871.
73. Newton PM, Mow VC, Gardner TR, Buckwalter JA, Albright JP. Winner of the 1996 Cabaud Award. The effect of lifelong exercise on canine articular cartilage. Am J Sports Med. 1997 May-Jun;25(3):282-7. PubMed PMID: 9167804.
74. Doom M, de Bruin T, de Rooster H, van Bree H, Cox E. Immunopathological mechanisms in dogs with rupture of the cranial cruciate ligament. Vet Immunol Immunopathol. 2008 Sep 15;125(1-2):143-61. PubMed PMID: 18621423.
75. Niebauer GW, Wolf B, Bashey RI, Newton CD. Antibodies to canine collagen types I and II in dogs with spontaneous cruciate ligament rupture and osteoarthritis. Arthritis Rheum. 1987 Mar;30(3):319-27. PubMed PMID: 3566823.
76. de Rooster H, Cox E, van Bree H. Prevalence and relevance of antibodies to type-I and -II collagen in synovial fluid of dogs with cranial cruciate ligament damage. American Journal of Veterinary Research. 2000 Nov;61(11):1456-61. PubMed PMID: WOS:000165097000023. English.
77. de Bruin T, de Rooster H, van Bree H, Cox E. Evaluation of anticollagen type I antibody titers in synovial fluid of both stifle joints and the left shoulder joint of dogs with unilateral cranial cruciate disease. American Journal of Veterinary Research. 2007 Mar;68(3):283-9. PubMed PMID: WOS:000244625700011. English.
78. Galloway RH, Lester SJ. Histopathological Evaluation of Canine Stifle Joint Synovial-Membrane Collected at the Time of Repair of Cranial Cruciate Ligament Rupture. Journal of the American Animal Hospital Association. 1995 Jul-Aug;31(4):289-94. PubMed PMID: WOS:A1995RF56100004. English.
79. Muir P, Schamberger GM, Manley PA, Hao ZL. Localization of cathepsin K and tartrate-resistant acid phosphatase in synovium and cranial cruciate ligament in dogs with cruciate disease. Veterinary Surgery. 2005 May-Jun;34(3):239-46. PubMed PMID: WOS:000231452100008. English.
80. Muir P, Oldenhoff WE, Hudson AP, Manley PA, Schaefer SL, Markel MD, et al. Detection of DNA from a range of bacterial species in the knee joints of dogs with inflammatory knee arthritis and associated degenerative anterior cruciate ligament rupture. Microb Pathog. 2007 Feb-Mar;42(2-3):47-55. PubMed PMID: 17320342.
81. Comerford EJ, Innes JF, Tarlton JF, Bailey AJ. Investigation of the composition, turnover, and thermal properties of ruptured cranial cruciate ligaments of dogs. Am J Vet Res. 2004 Aug;65(8):1136-41. PubMed PMID: 15334849.
82. Comerford EJ, Tarlton JF, Innes JF, Johnson KA, Amis AA, Bailey AJ. Metabolism and composition of the canine anterior cruciate ligament relate to differences in knee joint mechanics and predisposition to ligament rupture. J Orthop Res. 2005 Jan;23(1):61-6. PubMed PMID: 15607876.
83. Hayashi K, Frank JD, Dubinsky C, Zhengling H, Markel MD, Manley PA, et al. Histologic changes in ruptured canine cranial cruciate ligament. Vet Surg. 2003 May-Jun;32(3):269-77. PubMed PMID: 12784204.
84. Frank C, Amiel D, Woo SL, Akeson W. Normal ligament properties and ligament healing. Clin Orthop Relat Res. 1985 Jun(196):15-25. PubMed PMID: 3995817.

85. 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 Feb;36(2):170-7. PubMed PMID: 17335425. Epub 2007/03/06. eng.
86. Murray MM, Spindler KP, Ballard P, Welch TP, Zurakowski D, Nanney LB. Enhanced histologic repair in a central wound in the anterior cruciate ligament with a collagen-platelet-rich plasma scaffold. J Orthop Res. 2007 Aug;25(8):1007-17. PubMed PMID: 17415785.
87. Spreng D, Sigrist N, Jungi T, Busato A, Lang JH, Pfister H, et al. Nitric oxide metabolite production in the cranial cruciate ligament, synovial membrane, and articular cartilage of dogs with cranial cruciate ligament rupture. American Journal of Veterinary Research. 2000 May;61(5):530-6. PubMed PMID: WOS:000086787300010. English.
88. Aron DN. Traumatic Dislocation of the Stifle Joint - Treatment of 12 Dogs and One Cat. Journal of the American Animal Hospital Association. 1988 May-Jun;24(3):333-40. PubMed PMID: WOS:A1988N408800017. English.
89. Reinke JD. Cruciate Ligament Avulsion Injury in the Dog. Journal of the American Animal Hospital Association. 1982;18(2):257-64. PubMed PMID: WOS:A1982NG22900005. English.
90. Williams J, Fitch RB, Lemarie RJ. Partial avulsion of the origin of the cranial cruciate ligament in a 4 year-old dog. Vet Radiol Ultrasound. 1997 Sep-Oct;38(5):380-3. PubMed PMID: 9335097.
91. Gupta BN, Brinker WO, Subramanian KN. Breaking strength of cruciate ligaments in the dog. J Am Vet Med Assoc. 1969 Nov 15;155(10):1586-8. PubMed PMID: 5388946.
92. Vezzoni A, Bohorquez Vanelli A, Modenato M, Dziezyc J, Devine Slocum T. Proximal tibial epiphysiodesis to reduce tibial plateau slope in young dogs with cranial cruciate ligament deficient stifle. Vet Comp Orthop Traumatol. 2008;21(4):343-8. PubMed PMID: 18704241.
93. DeCamp CE. Kinetic and kinematic gait analysis and the assessment of lameness in the dog. Vet Clin North Am Small Anim Pract. 1997 Jul;27(4):825-40. PubMed PMID: 9243783. Epub 1997/07/01. eng.
94. McLaughlin RM. Kinetic and kinematic gait analysis in dogs. Vet Clin North Am Small Anim Pract. 2001 Jan;31(1):193-201. PubMed PMID: 11787262.
95. Allen K, Decamp CE, Braden TD, Bahns M. Kinematic Gait Analysis of the Trot in Healthy Mixed Breed Dogs. Veterinary and Comparative Orthopaedics and Traumatology. 1994 Nov;7(4):148-53. PubMed PMID: WOS:A1994PT91900004. English.
96. DeCamp CE, Riggs CM, Olivier NB, Hauptman JG, Hottinger HA, SoutasLittle RW. Kinematic evaluation of gait in dogs with cranial cruciate ligament rupture. American Journal of Veterinary Research. 1996 Jan;57(1):120-6. PubMed PMID: WOS:A1996TN53800027. English.
97. Budsberg SC, Verstraete MC, Soutas-Little RW. Force plate analysis of the walking gait in healthy dogs. Am J Vet Res. 1987 Jun;48(6):915-8. PubMed PMID: 3605807. Epub 1987/06/01. eng.
98. Fu YC, Torres BT, Budsberg SC. Evaluation of a three-dimensional kinematic model for canine gait analysis. Am J Vet Res. 2010 Oct;71(10):1118-22. PubMed PMID: 20919895.
99. Clements DN, Owen MR, Carmichael S, Reid SW. Kinematic analysis of the gait of 10 labrador retrievers during treadmill locomotion. Vet Rec. 2005 Apr 9;156(15):478-81. PubMed PMID: 15828744.
100. Schaefer SL, DeCamp CE, Hauptman JG, Walton A. Kinematic gait analysis of hind limb symmetry in dogs at the trot. American Journal of Veterinary Research. 1998 Jun;59(6):680-5. PubMed PMID: WOS:000076814200008. English.
101. DeCamp CE, Soutas-Little RW, Hauptman J, Olivier B, Braden T, Walton A. Kinematic gait analysis of the trot in healthy greyhounds. Am J Vet Res. 1993 Apr;54(4):627-34. PubMed PMID: 8484586.
102. Jaegger G, Marcellin-Little DJ, Levine D. Reliability of goniometry in Labrador Retrievers. Am J Vet Res. 2002 Jul;63(7):979-86. PubMed PMID: 12118679. Epub 2002/07/18. eng.
103. Vilensky JA, O'Connor BL, Brandt KD, Dunn EA, Rogers PI, DeLong CA. Serial kinematic analysis of the unstable knee after transection of the anterior cruciate ligament: temporal and angular changes in a canine model of osteoarthritis. J Orthop Res. 1994 Mar;12(2):229-37. PubMed PMID: 8164096.

104. Marsolais GS, McLean S, Derrick T, Conzemius MG. Kinematic analysis of the hind limb during swimming and walking in healthy dogs and dogs with surgically corrected cranial cruciate ligament rupture. Journal of the American Veterinary Medical Association. 2003 Mar 15;222(6):739-43. PubMed PMID: WOS:000181600400014. English.
105. Budsberg SC. Long-term temporal evaluation of ground reaction forces during development of experimentally induced osteoarthritis in dogs. American Journal of Veterinary Research. 2001 Aug;62(8):1207-11. PubMed PMID: WOS:000170190900004. English.
106. Jevens DJ, DeCamp CE, Hauptman J, Braden TD, Richter M, Robinson R. Use of force-plate analysis of gait to compare two surgical techniques for treatment of cranial cruciate ligament rupture in dogs. American Journal of Veterinary Research. 1996 Mar;57(3):389-93. PubMed PMID: WOS:A1996TX19100027. English.
107. Sanchez-Bustinduy M, de Medeiros MA, Radke H, Langley-Hobbs S, McKinley T, Jeffery N. Comparison of kinematic variables in defining lameness caused by naturally occurring rupture of the cranial cruciate ligament in dogs. Vet Surg. 2010 Jun;39(4):523-30. PubMed PMID: 20345532.
108. Ballagas AJ, Montgomery RD, Henderson RA, Gillette R. Pre- and postoperative force plate analysis of dogs with experimentally transected cranial cruciate ligaments treated using tibial plateau leveling osteotomy. Veterinary Surgery. 2004 Mar-Apr;33(2):187-90. PubMed PMID: WOS:000220223300013. English.
109. O'Connor BL, Visco DM, Heck DA, Myers SL, Brandt KD. Gait alterations in dogs after transection of the anterior cruciate ligament. Arthritis Rheum. 1989 Sep;32(9):1142-7. PubMed PMID: 2775322.
110. Lee JY, Kim G, Kim JH, Choi SH. Kinematic gait analysis of the hind limb after tibial plateau levelling osteotomy and cranial tibial wedge osteotomy in ten dogs. J Vet Med A Physiol Pathol Clin Med. 2007 Dec;54(10):579-84. PubMed PMID: 18045343.
111. Warzee CC, Dejardin LM, Arnoczky SP, Perry RL. 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 May-Jun;30(3):278-86. PubMed PMID: 11340560. Epub 2001/05/08. eng.
112. 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 Mar-Apr;31(2):147-54. PubMed PMID: 11884960. Epub 2002/03/09. eng.
113. Brinker WO, Piermattei DL, Flo GL. The Stifle Joint. Handbook of Small Animal Orthopedics and Fracture Repair. 4th ed. Philadelphia: WB Saunders; 2006. p. 562-632.
114. Strande A. Repair of the ruptured cranial cruciate ligament in the dog. Baltimore: Williams & Williams; 1967.
115. Vasseur PB. Clinical results following nonoperated management for rupture of the cranial cruciate ligament in dogs. Veterinary Surgery. 1984;13:243-6.
116. Chauvet AE, Johnson AL, Pijanowski GJ, Homco L, Smith RD. 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 May-Jun;32(3):247-55. PubMed PMID: 8731140.
117. Manley PA. Intra-articular Stabilization. In: Muir P, editor. Advances in The Canine Cranial Cruciate Ligament. Ames, Iowa: Wiley-Blackwell; 2010. p. 189-93.
118. Fu FH, Bennett CH, Lattermann C, Ma CB. Current trends in anterior cruciate ligament reconstruction. Part 1: Biology and biomechanics of reconstruction. Am J Sports Med. 1999 Nov-Dec;27(6):821-30. PubMed PMID: 10569374.
119. O'Donoghue DH, Rockwood CA, Jr., Frank GR, Jack SC, Kenyon R. Repair of the anterior cruciate ligament in dogs. J Bone Joint Surg Am. 1966 Apr;48(3):503-19. PubMed PMID: 5929794.

120. Spindler KP, Devin C, Nanney LB, Davidson JM. The central ACL defect as a model for failure of intra-articular healing. Journal of Orthopaedic Research. 2006 Mar;24(3):401-6. PubMed PMID: WOS:000235919900013. English.
121. Murray MM, Spindler KP, Devin C, Snyder BS, Muller J, Takahashi M, et al. Use of a collagen-platelet rich plasma scaffold to stimulate healing of a central defect in the canine ACL. Journal of Orthopaedic Research. 2006 Apr;24(4):820-30. PubMed PMID: WOS:000236682700029. English.
122. Paatsama S. Ligament injuries of the canine stifle joint: A clinical and experimental study. Helsinki. 1952.
123. McCurin DM, Pearson PT, Wass WM. Clinical and pathologic evaluation of ruptured cranial cruciate ligament repair in the dog. Am J Vet Res. 1971;32:1517-24.
124. Dickinson CR, Nunamaker DM. Repair of ruptured anterior cruciate ligament in the dog: experience in 101 cases, using a modified fascia strip technique. J Am Vet Med Assoc. 1977 Apr 15;170(8):827-30. PubMed PMID: 853014.
125. Dueland R. A recent technique for reconstruction of the anterior cruciate ligament. J Am Anim Hops Assoc. 1966;2:1.
126. Arnoczky SP, Tarvin GB, Marshall JL, Saltzman B. Over-the-Top Procedure - Technique for Anterior Cruciate Ligament Substitution in the Dog. Journal of the American Animal Hospital Association. 1979;15(3):283-90. PubMed PMID: ISI:A1979GW20200008. English.
127. Hulse DA, Michaelson F, Johnson C, Abdelbaki YZ. A Technique for Reconstruction of the Anterior Cruciate Ligament in the Dog - Preliminary-Report. Veterinary Surgery. 1980;9(4):135-40. PubMed PMID: WOS:A1980LA88300004. English.
128. Johnson SG, Hulse DA, Hogan HA, Nelson JK, Boothe HW. System Behavior of Commonly Used Cranial Cruciate Ligament Reconstruction Autografts. Veterinary Surgery. 1989 Nov-Dec;18(6):459-65. PubMed PMID: WOS:A1989CF15300009. English.
129. Butler DL, Hulse DA, Kay MD, Grood ES, Shires PK, Dambrosia R, et al. Biomechanics of Cranial Cruciate Ligament Reconstruction in the Dog .2. Mechanical-Properties. Veterinary Surgery. 1983;12(3):113-8. PubMed PMID: WOS:A1983RH11700002. English.
130. Mitton GR, Ireland WP, Runyon CL. Evaluation of the Instantaneous Centers of Rotation of the Stifle before and after Repair of Torn Cruciate Ligament by Use of the over-the-Top Technique in Dogs. American Journal of Veterinary Research. 1991 Oct;52(10):1731-7. PubMed PMID: WOS:A1991GH64000032. English.
131. Vasseur PB, Berry CR. Progression of Stifle Osteoarthrosis Following Reconstruction of the Cranial Cruciate Ligament in 21 Dogs. Journal of the American Animal Hospital Association. 1992 Mar-Apr;28(2):129-36. PubMed PMID: WOS:A1992HL08700005. English.
132. Vasseur PB. Stifle joint, In: Slatter D (ed): . Textbook of Small Animal Surgery (ed 3). Philadelphia, PA, : Elsevier Science; 2003. p. 2090–133.
133. Shires PK, Hulse DA, Liu W. The under-and-over Fascial Replacement Technique for Anterior Cruciate Ligament Rupture in Dogs - a Retrospective Study. Journal of the American Animal Hospital Association. 1984;20(1):69-77. PubMed PMID: ISI:A1984SE72500008. English.
134. Elkins AD, Pechman R, Kearney MT, Herron M. A Retrospective Study Evaluating the Degree of Degenerative Joint Disease in the Stifle Joint of Dogs Following Surgical Repair of Anterior Cruciate Ligament Rupture. Journal of the American Animal Hospital Association. 1991 Sep-Oct;27(5):533-40. PubMed PMID: WOS:A1991GK02000009. English.
135. Lopez MJ, Markel MD, Kalscheur V, Lu Y, Manley PA. Hamstring graft technique for stabilization of canine cranial cruciate ligament deficient stifles. Vet Surg. 2003 Jul-Aug;32(4):390-401. PubMed PMID: 12866003. Pubmed Central PMCID: 1965501.

136. Curtis RJ, Delee JC, Drez DJ. Reconstruction of the Anterior Cruciate Ligament with Freeze-Dried Fascia Lata Allografts in Dogs - a Preliminary-Report. American Journal of Sports Medicine. 1985;13(6):408-14. PubMed PMID: WOS:A1985AVV3500008. English.
137. Arnoczky SP, Warren RF, Ashlock MA. Replacement of the Anterior Cruciate Ligament Using a Patellar Tendon Allograft - an Experimental-Study. Journal of Bone and Joint Surgery-American Volume. 1986 Mar;68A(3):376-85. PubMed PMID: WOS:A1986A593200010. English.
138. Mcmaster WC. A Histologic Assessment of Canine Anterior Cruciate Substitution with Bovine Xenograft. Clinical Orthopaedics and Related Research. 1985 (196):196-201. PubMed PMID: WOS:A1985AKA9500026. English.
139. Allen PR, Amis AA, Jones MM, Heatley FW. Evaluation of preserved bovine tendon xenografts: a histological, biomechanical and clinical study. Biomaterials. 1987 Mar;8(2):146-52. PubMed PMID: 3580474.
140. Slocum B, Devine T. Cranial Tibial Wedge Osteotomy - a Technique for Eliminating Cranial Tibial Thrust in Cranial Cruciate Ligament Repair. Journal of the American Veterinary Medical Association. 1984;184(5):564-9. PubMed PMID: WOS:A1984SE88700016. English.
141. Watt P, Sommerlad S, Robins GM, editors. Tibial wedge osteotomy for treatment of cranial cruciate rupture. American College of Veterinary Surgeons Symposium; 2000; Arlington, VA.
142. Slocum B, Slocum TD. Tibial Plateau Leveling Osteotomy for Repair of Cranial Cruciate Ligament Rupture in the Canine. Veterinary Clinics of North America-Small Animal Practice. 1993 Jul;23(4):777-95. PubMed PMID: ISI:A1993LH93100008. English.
143. Robinson DA, Mason DR, Evans R, Conzemius MG. The effect of tibial plateau angle on ground reaction forces 4-17 months after tibial plateau leveling osteotomy in Labrador Retrievers. Vet Surg. 2006 Apr;35(3):294-9. PubMed PMID: 16635011.
144. Rayward RM, Thomson DG, Davies JV, Innes JF, Whitelock RG. Progression of osteoarthritis following TPLO surgery: a prospective radiographic study of 40 dogs. J Small Anim Pract. 2004 Feb;45(2):92-7. PubMed PMID: 14984152.
145. Hurley CR, Hammer DL, Shott S. Progression of radiographic evidence of osteoarthritis following tibial plateau leveling osteotomy in dogs with cranial cruciate ligament rupture: 295 cases (2001-2005). J Am Vet Med Assoc. 2007 Jun 1;230(11):1674-9. PubMed PMID: 17542736.
146. Talaat MB, Kowaleski MP, Boudrieau RJ. Combination tibial plateau leveling osteotomy and cranial closing wedge osteotomy of the tibia for the treatment of cranial cruciate ligament-deficient stifles with excessive tibial plateau angle. Vet Surg. 2006 Dec;35(8):729-39. PubMed PMID: 17187635.
147. Montavon PM, Damur DM, Tepic S, editors. Advancement of the tibial tuberosity for the treatment of cranial cruciate deficient canine stifle. . In Proceedings of the 1st World Orthopedic Veterinary Congress; 2002 September 5–8; Munich, Germany.
148. Boudrieau RJ. Tibial plateau leveling osteotomy or tibial tuberosity advancement? Vet Surg. 2009 Jan;38(1):1-22. PubMed PMID: 19152613.
149. Montavon P, Damur D, Tepic S, editors. 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.
150. Damur DM, editor Tibial tuberosity advancement: clinical results. Proceedings of the 15th Annual American College of Veterinary Surgeons Symposium; 2005; San Diego, CA.
151. Hoffmann DE, Miller JM, Ober CP, Lanz OI, Martin RA, Shires PK. Tibial tuberosity advancement in 65 canine stifles. Vet Comp Orthop Traumatol. 2006;19(4):219-27. PubMed PMID: 17143394.
152. Lafaver S, Miller NA, Stubbs WP, Taylor RA, Boudrieau RJ. Tibial tuberosity advancement for stabilization of the canine cranial cruciate ligament-deficient stifle joint: surgical technique, early results, and complications in 101 dogs. Vet Surg. 2007 Aug;36(6):573-86. PubMed PMID: 17686132. Epub 2007/08/10. eng.

153. Stein S, Schmoekel H. Short-term and eight to 12 months results of a tibial tuberosity advancement as treatment of canine cranial cruciate ligament damage. Journal of Small Animal Practice. 2008 Aug;49(8):398-404. PubMed PMID: WOS:000257990600005. English.
154. Damur DM, Tepic S, Montavon PM. Proximal tibial osteotomy for the repair of cranial cruciate-deficient stifle joints in dogs. Veterinary and Comparative Orthopaedics and Traumatology. 2003;16(4):211-6. PubMed PMID: WOS:000187665600003. English.
155. Hildreth BE, Marcellin-Little DJ, Roe SC, Harrysson OLA. In vitro evaluation of five canine tibial plateau leveling methods. American Journal of Veterinary Research. 2006 Apr;67(4):693-700. PubMed PMID: WOS:000236635000020. English.
156. Bruce WJ, Rose A, Tuke. J, editors. Evaluation of the triple tibial osteotomy (TTO): a new technique for the management of the canine cruciate-deficient stifle. European Society of Veterinary Orthopaedics and Traumatology Congress; 2006; Munich, Germany.
157. Korvick DL, Johnson AL, Schaeffer DJ. Surgeons Preferences in Treating Cranial Cruciate Ligament Ruptures in Dogs. Journal of the American Veterinary Medical Association. 1994 Nov 1;205(9):1318-24. PubMed PMID: ISI:A1994PN64700017. English.
158. Childers HE. New methods for cruciate ligament repair. Repair by suture technique. Mod Vet Pract. 1966;47:59.
159. Pearson PT. Ligamentous and meniscal injuries of the stifle joint. Vet Clin North Am Small Anim Pract. 1967 (1):439.
160. Hohn RB, Newton CD. Surgical repair of ligamentous structures of the stifle joint. In: Bojrab MJ, editor. Current Techniques in Small Animal Surgery. Philadelphia: Lea and Febiger; 1975. p. 470-9.
161. Guenego L, Zahra A, Madelenat A, Gautier R, Marcellin-Little DJ, Hulse D. Cranial cruciate ligament rupture in large and giant dogs. A retrospective evaluation of a modified lateral extracapsular stabilization. Vet Comp Orthop Traumatol. 2007;20(1):43-50. PubMed PMID: 17364096. Epub 2007/03/17. eng.
162. Fischer C, Cherres M, Grevel V, Oechtering G, Bottcher P. Effects of Attachment Sites and Joint Angle at the Time of Lateral Suture Fixation on Tension in the Suture for Stabilization of the Cranial Cruciate Ligament Deficient Stifle in Dogs. Veterinary Surgery. 2010 Apr;39(3):334-42.
163. Smith GK, Torg JS. Fibular head transposition for repair of cruciate-deficient stifle in the dog. J Am Vet Med Assoc. 1985 Aug 15;187(4):375-83. PubMed PMID: 4030471. Epub 1985/08/15. eng.
164. Pearson PT, McCurin DM, Carter JD, Hoskins JD. Lembert suture technique to surgically correct ruptured cruciate ligaments. J Am Anim Hops Assoc. 1971;7:1-13.
165. Aiken SW, Bauer MS, Toombs JP. Extra Articular Fascial Strip Repair of the Cranial Cruciate Deficient Stifle: Technique and Results in Seven Dogs. Vet Comp Orthop Traumatol. 1992 (5):145-50.
166. Dupuis J, Harari J, Papageorges M, Gallina AM, Ratzlaff M. Evaluation of fibular head transposition for repair of experimental cranial cruciate ligament injury in dogs. Vet Surg. 1994 Jan-Feb;23(1):1-12. PubMed PMID: 8140733.
167. Dupuis J, Harari J, Blackketter DM, Gallina AM. Evaluation of the lateral collateral ligament after fibular head transposition in dogs. Vet Surg. 1994 Nov-Dec;23(6):456-65. PubMed PMID: 7871709.
168. 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 Nov 1;195(9):1267-71. PubMed PMID: 2584129.
169. Patterson RH, Smith GK, Gregor TP, Newton CD. Biomechanical stability of four cranial cruciate ligament repair techniques in the dog. Vet Surg. 1991 Mar-Apr;20(2):85-90. PubMed PMID: 2042286. Epub 1991/03/01. eng.
170. Budsberg SC, Verstraete MC, Soutaslittle RW, Flo GL, Probst CW. Force Plate Analyses before and after Stabilization of Canine Stifles for Cruciate Injury. American Journal of Veterinary Research. 1988 Sep;49(9):1522-4. PubMed PMID: WOS:A1988P941700017. English.

171. Cook JL, Luther JK, Beetem J, Karnes J, Cook CR. Clinical comparison of a novel extracapsular stabilization procedure and tibial plateau leveling osteotomy for treatment of cranial cruciate ligament deficiency in dogs. Vet Surg. 2010 Apr;39(3):315-23. PubMed PMID: 20345535.
172. Cook JL, editor Multicenter outcomes study for evaluation of Tightrope CCL for treatment of cranial cruciate deficiency in dogs: the first 1000 cases. Proceedings of the 37th Annual Conference of the Veterinary Orthopedic Society; 2010 February 20-27; Breckenridge, CO, USA.
173. Kunkel KA, Basinger RR, Suber JT, Gerard PD. Evaluation of a transcondylar toggle system for stabilization of the cranial cruciate deficient stifle in small dogs and cats. Vet Surg. 2009 Dec;38(8):975-82. PubMed PMID: 20017856.
174. D'Amico L, Lanz OI, Aulakh K, Butler J, McLaughlin R, Harper T, et al. The effects of a novel extracapsular suture technique (lateral extracapsular suture system or LESSa) on the kinematics of the cranial cruciate deficient stifle. Kyon Symposium 2012, Zurich, Switzerland; April 25, 20122013.
175. Tang Z, Yang L, Wang Y, Xue R, Zhang J, Huang W, et al. Contributions of different intraarticular tissues to the acute phase elevation of synovial fluid MMP-2 following rat ACL rupture. J Orthop Res. 2009 Feb;27(2):243-8. PubMed PMID: 18846548.
176. Burgess R, Elder S, McLaughlin R, Constable P. In vitro biomechanical evaluation and comparison of FiberWire, FiberTape, OrthoFiber, and nylon leader line for potential use during extraarticular stabilization of canine cruciate deficient stifles. Vet Surg. 2010 Feb;39(2):208-15. PubMed PMID: 20210968.
177. Banwell MN, Kerwin SC, Hosgood G, Hedlund CS, Metcalf JB. In vitro evaluation of the 18 and 36 kg Securos Cranial Cruciate Ligament Repair System (TM). Veterinary Surgery. 2005 May-Jun;34(3):283-8. PubMed PMID: WOS:000231452100014. English.
178. Caporn TM, Roe SC. Biochemical evaluation of the suitability of monofilament nylon fishing and leader line for extra-articular stabilisation of the canine cruciate-deficient stifle. Veterinary and Comparative Orthopaedics and Traumatology. 1996 Aug;9(3):126-33. PubMed PMID: ISI:A1996VD71900008. English.
179. Nwadike BS, Roe SC. Mechanical comparison of suture material and knot type used for fabello-tibial sutures. Veterinary and Comparative Orthopaedics and Traumatology. 1998 Feb;11(1):52-7. PubMed PMID: WOS:000072075900009. English.
180. Maritato KC, Barnhart MD, Kazanovicz AJ, Naber SJ. In vitro mechanical evaluation and comparison of two crimping devices for securing monofilament nylon and multifilament polyethylene for use in extracapsular stabilization of the canine stifle. Vet Comp Orthop Traumatol. 2012 Nov 14;25(6):466-71. PubMed PMID: 22828974.
181. Sicard GK, Meinen J, Phillips T, Manley PA. Comparison of fishing line for repair of the cruciate deficient stifle. Veterinary and Comparative Orthopaedics and Traumatology. 1999 Aug;12(3):138-41. PubMed PMID: WOS:000082252000007. English.
182. Banwell MN, Hosgood G, Kerwin SC, Hedlund CS, Metcalf JB. In vitro evaluation of fluorocarbon leader line for use as a fabella-tibial suture. Veterinary and Comparative Orthopaedics and Traumatology. 2004;17(1):35-40. PubMed PMID: WOS:000220894100007. English.
183. Yamada E, Imayama Y, Katano S, Nagashima F, Shibata T. A new technique of extracapsular restoration with a tie made of ethylene tetrafluoroethylene (ETFE) for rupture of the cranial cruciate ligament in dogs. J Vet Med Sci. 1996 Jun;58(6):571-5. PubMed PMID: 8811630.
184. Beckman SL, Wadsworth PL, Hunt CA, Henry WB. Technique for Stabilizing the Stifle with Nylon Bands in Cases of Ruptured Anterior Cruciate Ligaments in Dogs. Journal of the American Animal Hospital Association. 1992 Nov-Dec;28(6):539-44. PubMed PMID: WOS:A1992KD12300009. English.
185. Olmstead ML. The Use of Orthopedic Wire as a Lateral Suture for Stifle Stabilization. Veterinary Clinics of North America-Small Animal Practice. 1993 Jul;23(4):735-53. PubMed PMID: WOS:A1993LH93100005. English.

186. Lewis DD, Milthorpe BK, Bellenger CR. Mechanical comparison of materials used for extra-capsular stabilisation of the stifle joint in dogs. Aust Vet J. 1997 Dec;75(12):890-6. PubMed PMID: 9469225. Epub 1998/02/20. eng.
187. Dulisch ML. Suture Reaction Following Extra-Articular Stifle Stabilization in the Dog .1. A Retrospective Study of 161 Stifles. Journal of the American Animal Hospital Association. 1981;17(4):569-71. PubMed PMID: WOS:A1981MC89100009. English.
188. Piermattei DL, Flo GL, DeCamp CE. The stifle joint. Brinker, Piermattei, and Flo's Handbook of Small Animal Orthopaedics and Fracture Repair. 4rth ed. St. Louis: Elsevier Inc; 2006. p. 586–92.
189. Henry WB. Nylon Bands. Journal of the American Animal Hospital Association. 1993 May-Jun;29(3):191-. PubMed PMID: WOS:A1993LF11000001. English.
190. Stork CK, Gibson NR, Owen MR, Li A, Schwarz T, Bennett D, et al. Radiographic features of a lateral extracapsular wire suture in the canine cranial cruciate deficient stifle. J Small Anim Pract. 2001 Oct;42(10):487-90. PubMed PMID: 11688523.
191. Gambardella PC, Wallace LJ, Cassidy F. Lateral Suture Technique for Management of Anterior Cruciate Ligament Rupture in Dogs - a Retrospective Study. Journal of the American Animal Hospital Association. 1981;17(1):33-8. PubMed PMID: ISI:A1981LB48400004. English.
192. McKee WM, Miller A. A self-locking knot for lateral fabellotibial suture stabilisation of the cranial cruciate ligament deficient stifle in the dog. Veterinary and Comparative Orthopaedics and Traumatology. 1999 May;12(2):78-80. PubMed PMID: WOS:000080708100007. English.
193. Huber DJ, Egger EL, James SP. The effect of knotting method on the structural properties of large diameter nonabsorbable monofilament sutures. Veterinary Surgery. 1999 Jul-Aug;28(4):260-7. PubMed PMID: WOS:000081353200006. English.
194. 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 Aug;35(6):567-72. PubMed PMID: 16911157.
195. Anderson CC, Tomlinson JL, Daly WR, Carson WL, Payne JT, Wagner-Mann CC. Biomechanical evaluation of a crimp clamp system for loop fixation of monofilament nylon leader material used for stabilization of the canine stifle joint. Veterinary Surgery. 1998 Nov-Dec;27(6):533-9. PubMed PMID: WOS:000077080500001. English.
196. Moores AP, Beck AL, Jespers KJ, Wilson AM. Mechanical evaluation of two loop tensioning methods for crimp clamp extracapsular stabilization of the cranial cruciate ligament-deficient canine stifle. Vet Surg. 2006 Jul;35(5):476-9. PubMed PMID: 16842294.
197. Peycke LE, Kerwin SC, Hosgood G, Metcalf JB. Mechanical comparison of six loop fixation methods with monofilament nylon leader line. Veterinary and Comparative Orthopaedics and Traumatology. 2002;15(4):210-4. PubMed PMID: WOS:000179538400005. English.
198. Rose ND, Goerke D, Evans RB, Conzemius MG. Mechanical Testing of Orthopedic Suture Material Used for Extra-Articular Stabilization of Canine Cruciate Ligament-Deficient Stifles. Vet Surg. 2011 Sep 23. PubMed PMID: 22092233.
199. Moores AP, Beck AL, Jespers KJ, Halfacree Z, Wilson AM. Mechanical evaluation of two crimp clamp systems for extracapsular stabilization of the cranial cruciate ligament-deficient canine stifle. Vet Surg. 2006 Jul;35(5):470-5. PubMed PMID: 16842293.
200. Choate CJ, Pozzi A, Lewis DD, Hudson CC, Conrad BP. Mechanical properties of isolated loops of nylon leader material, polyethylene cord, and polyethylene tape and mechanical properties of those materials secured to cadaveric canine femurs via lateral femoral fabellae, toggles placed through bone tunnels, or bone anchors. Am J Vet Res. 2012 Oct;73(10):1519-29. PubMed PMID: 23013177.
201. Steenland K, Whelan E, Deddens J, Stayner L, Ward E. Ethylene oxide and breast cancer incidence in a cohort study of 7576 women (United States). Cancer Causes Control. 2003 Aug;14(6):531-9. PubMed PMID: 12948284.

202. Steenland K, Stayner L, Deddens J. Mortality analyses in a cohort of 18 235 ethylene oxide exposed workers: follow up extended from 1987 to 1998. Occup Environ Med. 2004 Jan;61(1):2-7. PubMed PMID: 14691266. Pubmed Central PMCID: 1757803.
203. Gatineau M, Huneault L, Lussier B, Lefevre-Lavoie J. Mechanical evaluation of hydrogen peroxide gas plasma sterilization of nylon lines used for extra-articular stabilization of the canine stifle joint. Vet Surg. 2010 Jan;39(1):48-53. PubMed PMID: 20210944.
204. Gatineau M, El-Warrak AO, Bolliger C, Mourez M, Berthiaume F. Effects of sterilization with hydrogen peroxide gas plasma, ethylene oxide, and steam on bioadhesive properties of nylon and polyethylene lines used for stabilization of canine stifle joints. Am J Vet Res. 2012 Oct;73(10):1665-9. PubMed PMID: 23013195.
205. Havig ME, Dyce J, Kowaleski MP, Reynolds LR, Budsberg SC. Relationship of tibial plateau slope to limb function in dogs treated with a lateral suture technique for stabilization of cranial cruciate ligament deficient stifles. Vet Surg. 2007 Apr;36(3):245-51. PubMed PMID: 17461949.
206. Jerre S. Rehabilitation after extra-articular stabilisation of cranial cruciate ligament rupture in dogs. Vet Comp Orthop Traumatol. 2009;22(2):148-52. PubMed PMID: 19290397.
207. Jacobson NS, Truax P. Clinical significance: a statistical approach to defining meaningful change in psychotherapy research. J Consult Clin Psychol. 1991 Feb;59(1):12-9. PubMed PMID: 2002127.
208. Casale SA, McCarthy RJ. Complications associated with lateral fabellotibial suture surgery for cranial cruciate ligament injury in dogs: 363 cases (1997-2005). Javma-Journal of the American Veterinary Medical Association. 2009 Jan 15;234(2):229-35. PubMed PMID: WOS:000262483300015. English.
209. Frey TN, Hoelzler MG, Scavelli TD, Fulcher RP, Bastian RP. Risk factors for surgical site infection-inflammation in dogs undergoing surgery for rupture of the cranial cruciate ligament: 902 cases (2005-2006). J Am Vet Med Assoc. 2010 Jan 1;236(1):88-94. PubMed PMID: 20043807.
210. McCartney WT, O'Connor JV, McCann WM. Incidence of infection and premature crimp failure after repair of cranial cruciate ligament-deficient stifles in 110 dogs. Vet Rec. 2007 Aug 18;161(7):232-3. PubMed PMID: 17704468.
211. Stauffer KD, Tuttle TA, Elkins AD, Wehrenberg AP, Character BJ. Complications associated with 696 tibial plateau leveling osteotomies (2001-2003). J Am Anim Hosp Assoc. 2006 Jan-Feb;42(1):44-50. PubMed PMID: 16397194.
212. Pacchiana PD, Morris E, Gillings SL, Jessen CR, Lipowitz AJ. Surgical and postoperative complications associated with tibial plateau leveling osteotomy in dogs with cranial cruciate ligament rupture: 397 cases (1998-2001). Journal of the American Veterinary Medical Association. 2003 Jan 15;222(2):184-93. PubMed PMID: WOS:000180350500022. English.
213. Aulakh KS, Harper TA, Lanz OI, Daniel GB, Werre SR. Effect of stifle angle on the magnitude of the tibial plateau angle measurement in dogs with intact and transected cranial cruciate ligament. A cadaveric study. Vet Comp Orthop Traumatol. 2011;24(4):272-8. PubMed PMID: 21451886. Epub 2011/04/01. eng.
214. Newton CD. Fractures of the pelvis. In: Newton CD, Nunamaker DM, editors. Textbook of Small Animal Orthopaedics. Philadelphia: J.B. Lippincott Company; 1985. p. 393-402.
215. Dennler R, Kipfer NM, Tepic S, Hassig M, Montavon PM. Inclination of the patellar ligament in relation to flexion angle in stifle joints of dogs without degenerative joint disease. Am J Vet Res. 2006 Nov;67(11):1849-54. PubMed PMID: 17078745. Epub 2006/11/03. eng.
216. Jordan K, Dziedzic K, Jones PW, Ong BN, Dawes PT. The reliability of the three-dimensional FASTRAK measurement system in measuring cervical spine and shoulder range of motion in healthy subjects. Rheumatology (Oxford). 2000 Apr;39(4):382-8. PubMed PMID: 10817770. Epub 2000/05/19. eng.

217. Milne AD, Chess DG, Johnson JA, King GJ. Accuracy of an electromagnetic tracking device: a study of the optimal range and metal interference. J Biomech. 1996 Jun;29(6):791-3. PubMed PMID: 9147976. Epub 1996/06/01. eng.
218. Magit DP, McGarry M, Tibone JE, Lee TQ. Comparison of cutaneous and transosseous electromagnetic position sensors in the assessment of tibial rotation in a cadaveric model. Am J Sports Med. 2008 May;36(5):971-7. PubMed PMID: 18272792. Epub 2008/02/15. eng.
219. Defrate LE, Papannagari R, Gill TJ, Moses JM, Pathare NP, Li G. The 6 degrees of freedom kinematics of the knee after anterior cruciate ligament deficiency: an in vivo imaging analysis. Am J Sports Med. 2006 Aug;34(8):1240-6. PubMed PMID: 16636348. Epub 2006/04/26. eng.
220. Ragetly CA, Griffon DJ, Mostafa AA, Thomas JE, Hsiao-Wecksler ET. Inverse dynamics analysis of the pelvic limbs in Labrador Retrievers with and without cranial cruciate ligament disease. Vet Surg. 2010 Jun;39(4):513-22. PubMed PMID: 20345530. Epub 2010/03/30. eng.
221. Duck TR, Dunning CE, King GJW, Johnson JA. Variability and repeatability of the flexion axis at the ulnohumeral joint. Journal of Orthopaedic Research. 2003 May;21(3):399-404. PubMed PMID: ISI:000182683000004. English.
222. Crawford NR, Yamaguchi GT, Dickman CA. Methods for determining spinal flexion/extension, lateral bending, and axial rotation from marker coordinate data: Analysis and refinement. Human Movement Science. 1996 Feb;15(1):55-78. PubMed PMID: ISI:A1996VD45000004. English.
223. Grood ES, Suntay WJ. A joint coordinate system for the clinical description of three-dimensional motions: application to the knee. J Biomech Eng. 1983 May;105(2):136-44. PubMed PMID: 6865355. Epub 1983/05/01. eng.
224. Tonks CA, Pozzi A, Ling HY, Lewis DD. The Effects of Extra-Articular Suture Tension on Contact Mechanics of the Lateral Compartment of Cadaveric Stifles Treated with the TightRope CCL (R) or Lateral Suture Technique. Veterinary Surgery. 2010 Apr;39(3):343-9. PubMed PMID: ISI:000276619000010. English.

APPENDIX I: Figures

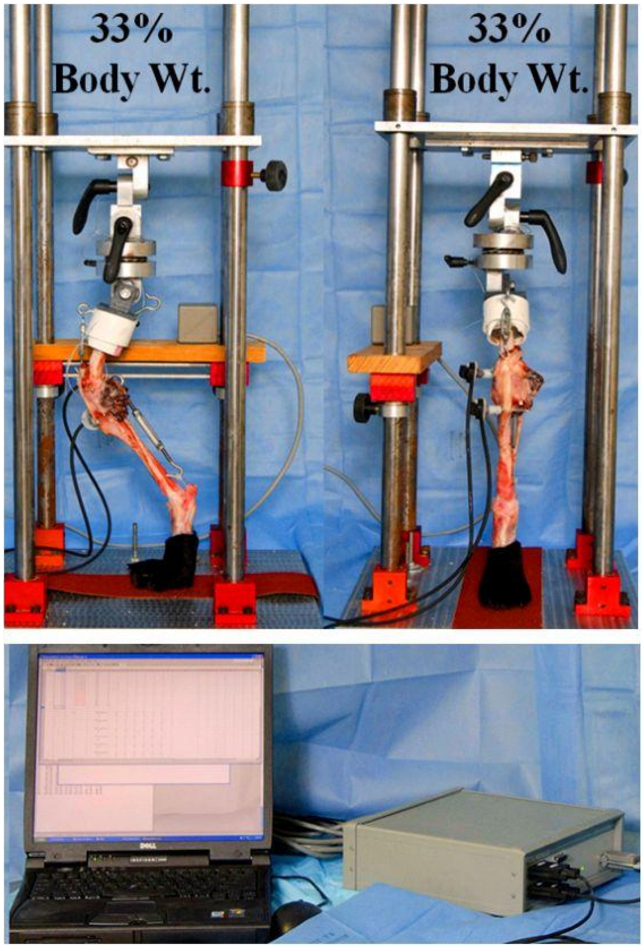

Figure 1: Custom designed loading jig (lateral and cranio-caudal view) with electromagnetic tracking system. The tracking sensors can be seen mounted to the distal femur and proximal tibia. The electromagnetic transmitter can be seen caudal and medial to the stifle. Adjustable turnbuckles from the proximal femur to the patella and from the distal femur to calcaneus mimic the quadriceps and gastrocnemius muscles, respectively. The platform proximal to the limb allowed application of a load equivalent to 33% of the dog's body weight.

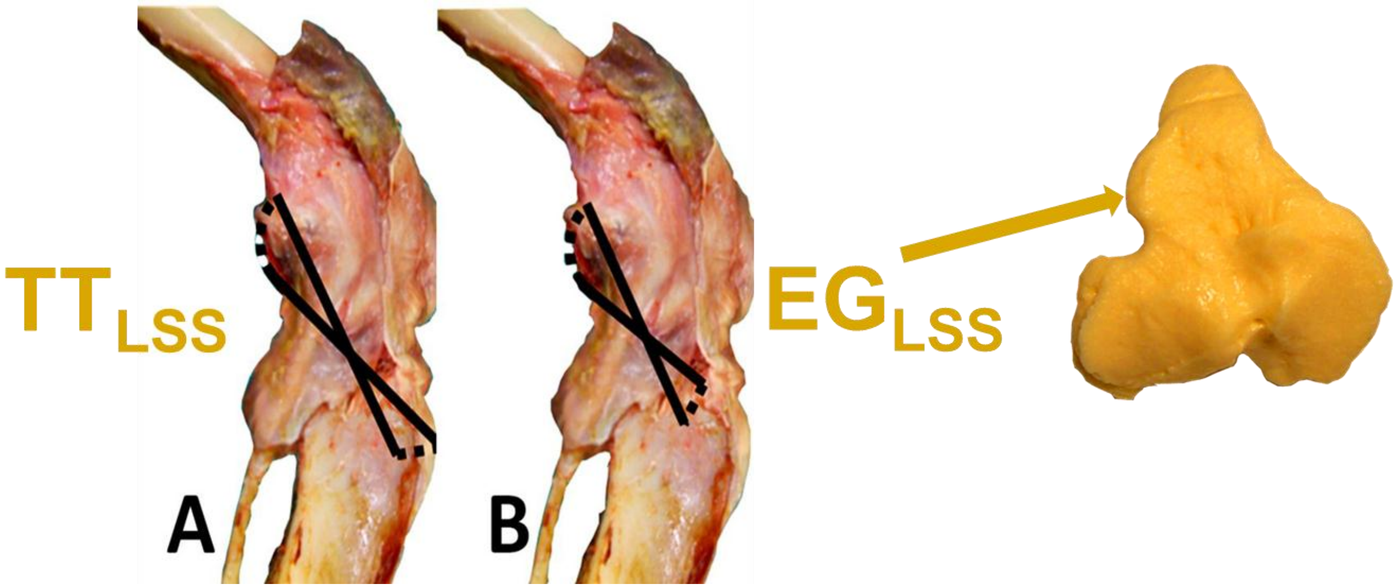

Figure 2: Location of suture anchor points for the LSS techniques. Lateral views (A): LSS_{TT}, (B): $LSS_{EG.}$ The arrow is pointing at the cranial eminence of the extensor groove in this proximodistal view of the tibia.

Figure 3: Instrumentation for LSS techniques

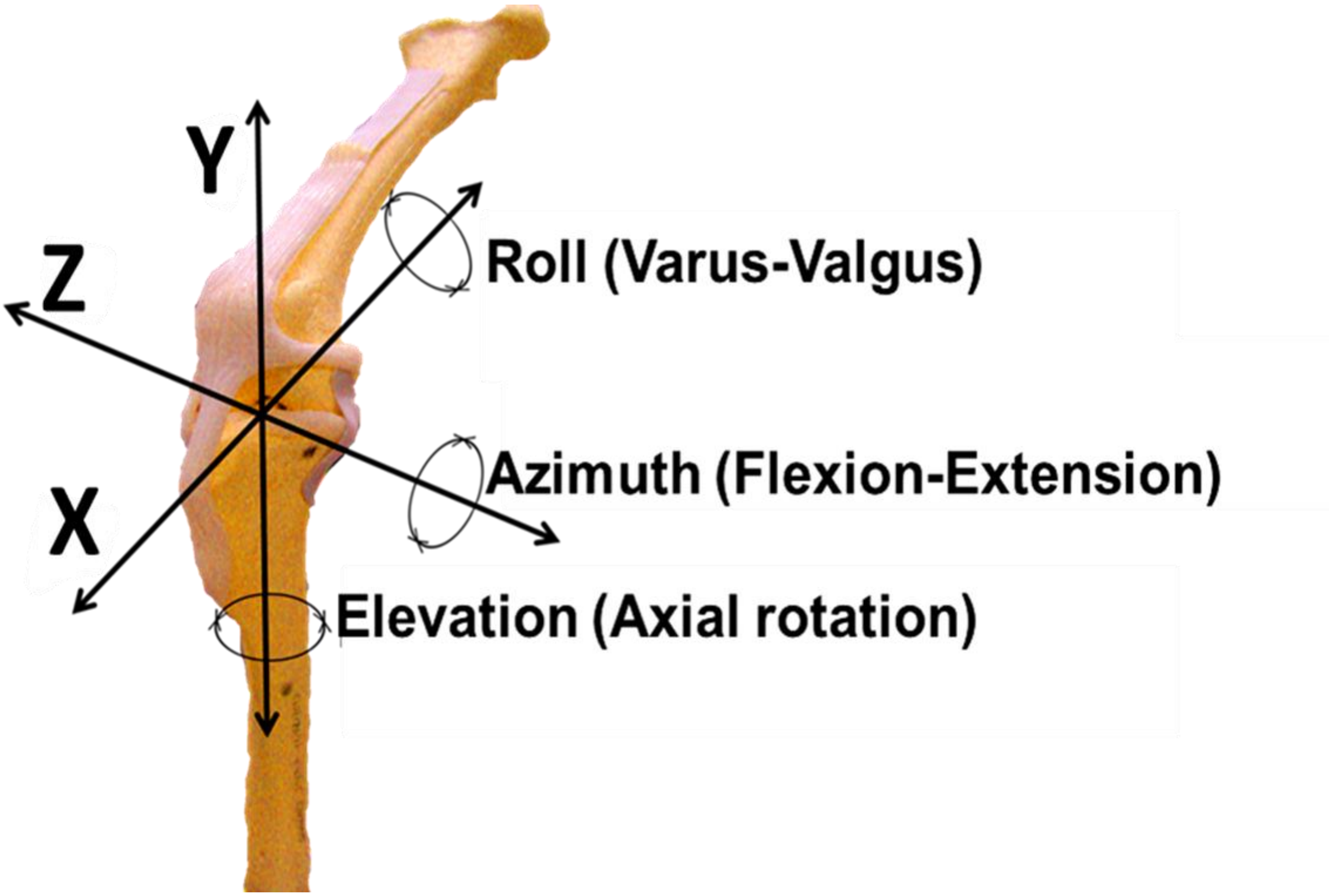

Figure 4: Cranial-caudal (X), proximal-distal (Y) and medial-lateral (Z) translations and internal-external (elevation) and varus-valgus (roll) rotations of the tibia relative to the femur. Stifle flexion-extension (azimuth) was a controlled variable, purposely kept consistent during early (145^0), mid (135^0) and late stance (125^0) for all groups.

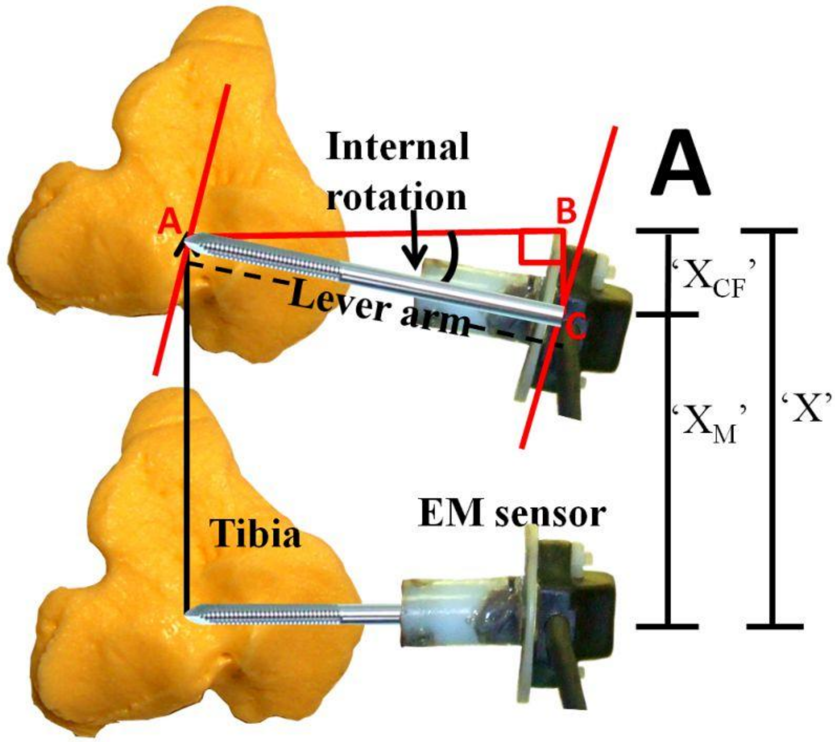

Figure 5: Effect of tibial rotation on tibial translations (The images are not drawn to scale).

Figure 5A: Effect of tibial rotation on cranial-caudal tibial translation.
Dorso-ventral view of tibia and sensors showing the effect of internal-external rotation (elevation) due to lever arm between the bone and the sensor on measurement of cranial-caudal displacement of sensor (i.e. cranial-caudal tibial translation = 'X'). Corrections were made to 'X_M' to account for cranial-caudal displacement occurring due to internal-external rotation. A perpendicular was dropped from point C to line AB, making ABC a right angled triangle. Distance BC is equal to 'X_{CF}'. Key: EM sensor = electromagnetic sensor, Elevation = internal-external rotation, 'X_M' = measured cranial-caudal translation of sensor during testing, 'X_{CF}' = X correction factor, 'X' (cranial-caudal displacement) = 'X_M' $\pm$ 'X_{CF}'.

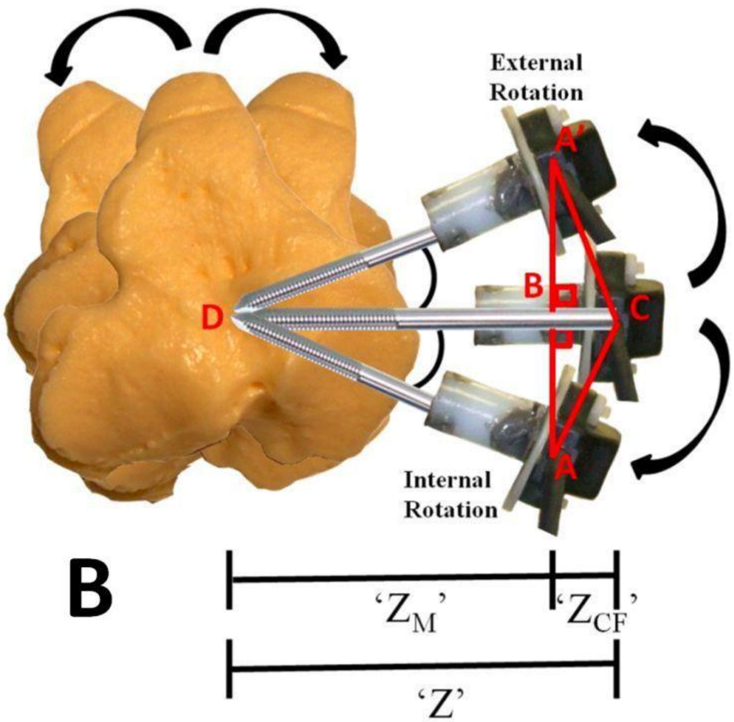

Figure 5B: Effect of tibial rotation on medial-lateral tibial translation.
Dorso-ventral view of tibia and sensors showing the effect of internal-external rotation (elevation) due to lever arm between the bone and the sensor on measurement of medial-lateral displacement of sensor (i.e. medial-lateral tibial translation = 'Z'). Corrections were made to 'Z_M' to account for medial-lateral displacement occurring due to internal-external rotation. Distance BC is equal to 'Z_{CF}'. Key: 'Z_M' = measured medial-lateral translation of sensor during testing, 'Z_{CF}' = Z correction factor; 'Z' (medial-lateral displacement) = 'Z_M' + 'Z_{CF}'.

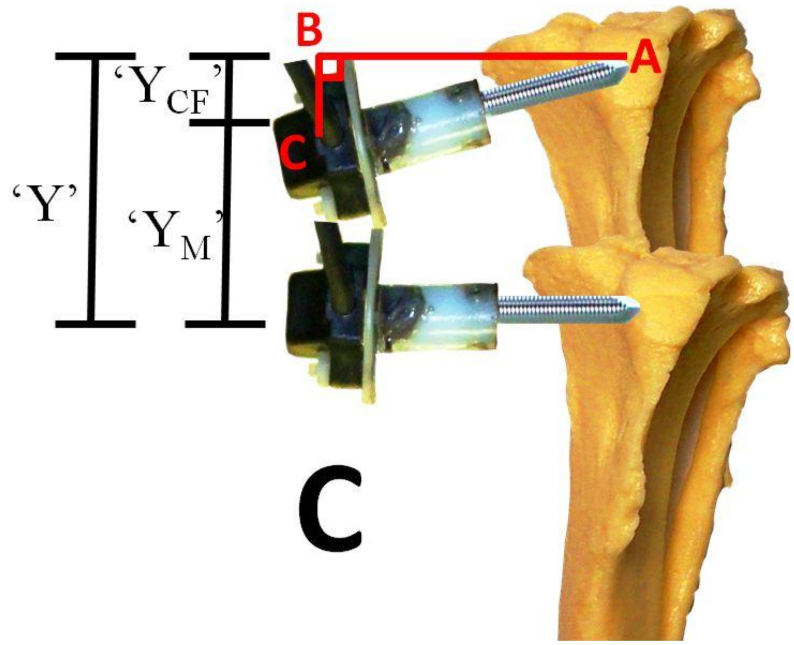

Figure 5C: Effect of tibial rotation on proximal-distal tibial translation.
Cranio-caudal view of tibia and sensors showing the effect of valgus-varus (roll) due to lever arm between the bone and the sensor on measurement of proximal-distal displacement of sensor (i.e. proximal-distal tibial translation = 'Y'). Corrections were made to 'Y_M' to account for proximal-distal displacement occurring due to valgus-varus rotation. Distance BC is equal to 'Y_{CF}'. Key: 'Y_M' = measured proximal-distal translation of sensor during testing, 'Y_{CF}' = Y correction factor; 'Y' (proximal-distal displacement) = 'Y_M' $\pm$ 'Y_{CF}'.

Note: the threaded pin was inserted through both cortices in actual specimens.

APPENDIX II: Tables

Table 1: Cranial-caudal, proximal-distal and medial-lateral tibial translations for all groups

Translations (mm)	**Stifle angle (degrees)**	**CrCL Intact [1]**	**CrCL-D$_{EG}$[2]**	**LSS$_{EG}$[3]**	**CrCL-D$_{TT}$[4]**	**LSS$_{TT}$[5]**
Cranial-caudal* (X; cm; mean ±SD)	125	0	1.71± 0.55 (p1-2 = **0.0000**)	0.30± 0.57 (p1-3 = 0.6842) (p2-3 = **<.0003**)	1.58±0.36 (p1-4 = **0.0000**) (p2-4 = 0.6471)	-0.02±0.56 (p1-5 = 1.000) (p4-5 = **<.0003**) (p3-5 = 0.0960)
	135	0	1.41± 0.25 (p1-2 = **0.0000**)	0.70±0.41 (p1-3 = **0.0001**) (p2-3 = **<.0003**)	1.45±0.46 (p1-4 = **0.0000**) (p2-4 = 1.0000)	0.33±0.51 (p1-5 = 0.2419) (p4-5 = **<.0003**) (p3-5 = **0.0432**)
	145	0	1.22±0.26 (p1-2 = **0.0000**)	0.51±0.42 (p1-3 = **0.0022**) (p2-3 = **<.0003**)	1.33±0.20 (p1-4 = **0.0000**) (p2-4 = 0.9237)	0.36± 0.44 (p1-5 = 0.0624) (p4-5 = **<.0003**) (p3-5 = 0.8346)
Proximal-distal* (Y; cm; mean ±SD)	125	0	0.20± 0.39 (p1-2 = 0.6737)	-0.14± 0.24 (p1-3 = 0.4637) (p2-3 =**<0.0001**)	0.11±0.27 (p1-4 =1.0000) (p2-4 = 0.8562)	-0.18±0.29 (p1-5 = 0.3255) (p4-5 = **<.0002**) (p3-5 = 0.6531)
	135	0	0.40±0.31 (p1-2 = **0.0014**)	-0.005± 0.25 (p1-3 = 1.0000) (p2-3 = **<.0003**)	0.31±0.25 (p1-4 =**0.0023**) (p2-4 = 0.9228)	-0.13±0.25 (p1-5 = 0.7217) (p4-5 = **<.0003**) (p3-5 = 0.4239)
	145	0	0.44±0.34 (p1-2 = **0.0015**)	0.14± 0.26 (p1-3 =0.6030) (p2-3 = **0.0033**)	0.49±0.21 (p1-4 = **0.0000**) (p2-4 =1.0000)	0.13±0.22 (p1-5 = 0.3754) (p4-5 = **<.0003**) (p3-5 = 1.0000)
Medial-lateral* (Z; cm; mean ±SD)	125	0	-0.86± 0.40 (p1-2 = **0.0000**)	0.30± 0.19 (p1-3 = **0.0002**) (p2-3 = **<.0003**)	-0.75±0.28 (p1-4 = **0.0000**) (p2-4 =0.4647)	0.39±0.21 (p1-5 = **0.0000**) (p4-5 =**<.0003**) (p3-5 =0.7167)
	135	0	-0.92± 0.25 (p1-2 = **0.0000**)	0.14± 0.26 (p1-3 = 0.6372) (p2-3 =**<.0003**)	-0.91±0.28 (p1-4 = **0.0000**) (p2-4 = 1.0000)	0.30±0.20 (p1-5 = **0.0003**) (p4-5 =**<.0003**) (p3-5 = 0.0978)
	145	0	-0.84± 0.24 (p1-2 = **0.0000**)	0.01± 0.25 (p1-3 = 1.0000) (p2-3 = <**.0003**)	-0.76±0.22 (p1-4 = **0.0000**) (p2-4 =0.9633)	0.19±0.20 (p1-5 = **0.0222**) (p4-5 =**<.0003**) (p3-5 = 0.0630)

Key: * Positive values indicate cranial, proximal and lateral tibial translation and negative values indicate caudal, distal and medial tibial translation relative to the femur. Values for Normal (CrCL intact) group were normalized to zero. CrCL-D$_{EG}$ = Cranial cruciate ligament deficient (transected CrCL) group that received LSS$_{EG}$ later. CrCL-D$_{TT}$ = Cranial cruciate ligament deficient group that received LSS$_{TT}$ later. The numbers associated with the p means that statistical comparisons are being made between respective groups. For example p1-2 means statistical comparison is being made between group #1 and group #2.

Table 2: Internal-external and varus-valgus rotations for all groups.

Rotations (^{0})	Stifle angle (degrees)	CrCL Intact [1]	CrCL-D_{EG}[2]	LSS_{EG}[3]	CrCL-D_{TT}[4]	LSS_{TT}[5]
Internal-external* (Elevation; mean ± SD)	125	0	-12.57±4.86 (p1-2 = **0.0000**)	5.95±2.51 (p1-3 = **0.0000**) (p2-3 = **<.0003**)	-14.02±3.43 (p1-4 = **0.0000**) (p2-4 = 0.3930)	6.85±1.80 (p1-5 = **0.0000**) (p4-5 = **<.0003**) (p3-5 = 1.0000)
	135	0	-11.4±4.31 (p1-2 = **0.0000**)	4.48±3.58 (p1-3 = **0.0019**) (p2-3 = <**.0003**)	-12.31±3.52 (p1-4 = **0.0000**) (p2-4 = 1.0000)	8.16±3.44 (p1-5 = **0.0000**) (p4-5 = **<.0003**) (p3-5 = **0.0009**)
	145	0	-11.20±4.82 (p1-2 = **0.0000**)	0.89±4.18 (p1-3 = 1.0000) (p2-3 = **<.0003**)	-11.53±3.31 (p1-4 = **0.0000**) (p2-4 = 1.0000)	5.31±3.35 (p1-5 = **0.0002**) (p4-5 = **<.0003**) (p3-5 = **<.0003**)
Varus-valgus* (Roll; mean ± SD)	125	0	6.10±4.3 (p1-2 = **0.0005**)	-6.61±2.9 (p1-3 =**0.0000**) (p2-3 = <**.0003**)	6.69±3.1 (p1-4 =**0.0000**) (p2-4 =1.0000)	-6.15±3.02 (p1-5 =**0.0000**) (p4-5 =<**.0003**) (p3-5 =1.0000)
	135	0	3.6±2.90 (p1-2 =**0.0021**)	-5.20±2.94 (p1-3 =**0.0000**) (p2-3 =<**.0003**)	4.64±3.17 (p1-4 =**0.0004**) (p2-4 = 1.0000)	-4.92±1.62 (p1-5 = **0.0000**) (p4-5 =<**.0003**) (p3-5 = 1.0000)
	145	0	-0.19±3.40 (p1-2 = 1.0000)	-4.5±2.8 (p1-3 =**0.0001**) (p2-3 =<**.0003**)	0.75±2.98 (p1-4 =1.0000) (p2-4 =1.0000)	-4.33±1.82 (p1-5 =**0.0000**) (p4-5 =<**.0003**) (p3-5 = 1.0000)

Key:* Positive values indicate external tibial rotation and valgus of the stifle and negative values indicate internal tibial rotation and varus of the stifle.
See Table1 for rest of the key.

APPENDIX III: ABBREVIATIONS

CrCL	–	cranial cruciate ligament
LSS_{TT}	–	lateral suture stabilization with distal anchor through tibial tuberosity
LSS_{EG}	–	lateral suture stabilization with distal anchor through cranial eminence of extensor groove
CTT	–	cranial tibial translation
NLL	–	nylon leader line