FUNCTIONAL ANATOMY OF THE EQUINE MENISCUS: PATHOGENESIS AND PATHOPHYSIOLOGY OF INJURY TO THE CRANIAL HORN OF THE MEDIAL MENISCUS

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ABSTRACT

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The cranial horn of the medial meniscus is the most common site of soft tissue injury in the equine stifle. These injuries occur as tears that are commonly associated with secondary osteoarthritis and a poor prognosis for return to athletic performance. Experimental research evaluating equine meniscal tears is lacking. One goal of this thesis was to document the translocation and deformation of the equine menisci throughout the complete range of motion of the femorotibial joint in order to evaluate for potential mechanical factors in the pathogenesis of cranial horn injury. An additional goal was to assess the tensile forces and microanatomic structure of the cranial horn of the medial meniscus to further evaluate the pathogenesis of these lesions. In the third study, the pathophysiology of these lesions was evaluated by determining the effect of grade III cranial horn tears of the medial meniscus on the magnitude and distribution of contact forces on the medial tibial condyle. Results indicate that full extension of the femorotibial joint leads to cranial displacement and axial compression of the cranial horn of the medial meniscus, and an ensuing increase in tensile forces in the adjacent cranial horn attachment. Therefore, injury of the cranial horn of the medial meniscus may occur during hyperextension of the stifle joint. Histologic findings indicate that the characteristic pattern of these tears may be partly explained by the ultrastructure of the cranial meniscotibial ligament. Grade III meniscal tears resulted in alterations in the magnitude and distribution of contact forces on the medial tibial condyle that may contribute to the development of secondary osteoarthritis.

This work is dedicated to:

My parents:
Thank you for always believing in me and inspiring me to dream big.

Mark Fuller:
Thank you for you endless love, support, and understanding.

ACKNOWLEDGEMENTS

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LIST OF ABBREVIATIONS

AIFP	Arthroscopically Implantable Force Probes
ANOVA	Analysis of variance
Ca	Caudal
Cr	Cranial
CrMTL	Cranial meniscotibial ligament
CT	Computed tomography
L	Lateral
M	Medial
MFC	Medial femoral condyle
MICET	Medial intercondylar eminence of the tibia
MM	Medial meniscus
MRI	Magnetic resonance imaging

CHAPTER 1.

General Introduction

GENERAL INTRODUCTION

Recognition of meniscal tears in the horse is increasing because of recent access to large bore magnetic resonance imaging (1), improved ultrasonographic techniques (2), and increased application of new and old arthroscopic techniques.(3) However, basic research evaluating the pathogenesis and pathophysiology of these lesions is lacking for the equine species, particularly when compared to the extensive knowledge acquired from years of study on the human and canine menisci. There is a tendency to extrapolate scientific discoveries from one species to another, however, with regards to the menisci, there are multiple differences between species, such as quadruped vs. biped locomotion and the type of meniscal lesions that are commonly recognized. Therefore, extrapolation of experimental research from other species may be inaccurate with regards to the equine meniscus.

Meniscal tears have been recognized as the most common soft tissue lesion in the equine stifle, with cranial horn tears of the medial meniscus documented as the most common location of meniscal injury.(4, 5) A characteristic pattern of cranial horn meniscal injury has been recognized (6), and speculations have been made that hyperextension of the joint may be involved in the pathogenesis of these lesions.(5) The prognosis for return to previous athletic function with these lesions has been reported as guarded to poor, and the presence of concurrent osteoarthritis is common.(5) As recognition of this potentially career ending injury is increasing, it is evident that further understanding of these lesions is needed.

The objectives of this thesis were to evaluate the pathogenesis and pathophysiology of cranial horn tears of the equine medial meniscus. A comprehensive review of the equine meniscal literature and relevant research from other species was completed to assemble a broad understanding of the current knowledge on meniscal tears (chapter 2). In order to gain insight

into the pathogenesis of equine cranial horn meniscal tears, evaluation of meniscal translocation and deformation through the complete range of motion of the stifle joint was completed (chapter 3). Additionally, the tensile forces and the functional anatomic structure of the cranial meniscotibial ligament of the medial meniscus were evaluated for potential mechanical factors in the development of cranial horn tears (chapter 4). The final study evaluated the effect of grade III cranial horn tears of the medial meniscus on the magnitude and distribution of contact forces on the medial tibial condyle, in attempts to explain the common association between meniscal tears and secondary osteoarthritis (chapter 5).

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CHAPTER 2.

Review of the Literature

In order to gain a greater perspective on the pathogenesis and pathophysiology of meniscal tears, the equine literature and pertinent research from other species was reviewed. This chapter examines equine stifle anatomy, kinematics of the femorotibial joint, meniscal structure and function, meniscal translocation and deformation, pathogenesis of meniscal tears, sequelae of meniscal tears and clinical characteristics of meniscal tears in the horse. In closing, the purpose and hypothesis for each of the research studies in the thesis is presented.

Equine Stifle Anatomy

Anatomic descriptions of the equine stifle date back to 1884 (1), and from that time equine stifle anatomy has been extensively documented in veterinary anatomy textbooks.(2, 3) The stifle joint is the largest and most elaborate of all the equine articulations; it consists of three bones (the femur, tibia and patella) and three synovial compartments (the femoropatellar, and medial and lateral femorotibial).(2) The stifle has a complex arrangement of soft tissues structures including 2 menisci, 12 ligaments, and numerous musculotendinous support structures. During flexion and extension, the primary motion of the joint, the patella slides proximally and distally over the femoral trochlea and the medial and lateral femoral condyles rotate and glide over the corresponding tibial condyles.(2) The femoral condyles are slightly obliqued (craniomedial to caudolateral) and the articular surface of the lateral is more strongly curved than the medial.(2)

The medial and lateral menisci are crescent shaped fibrocartilaginous discs lying between the rounded femoral condyles and the flattened to slightly saddle shaped tibial condyles.(2) Menisci are wedge shaped, with a concave proximal surface and a flat distal surface, to provide congruency between the articular surfaces of the femur and tibia.(2) There are 4 meniscotibial

ligaments (Figure 2.1) which attach the menisci to the tibia axially (cranial and caudal

meniscotibial ligaments of the medial and lateral menisci), and one ligament which attaches the

caudal horn of the lateral meniscus to the caudal aspect of the intercondyloid fossa of the femur

(meniscofemoral ligament).(2) The joint capsule attaches to the majority of the peripheral border

of the menisci and meniscal ligaments (2), except where the the collateral ligaments and the

popliteal tendon pass through the joint. The joint capsule attachment to the peripheral menisci is

termed the coronary ligaments in other species and the term is intermittently used in horses.(1)
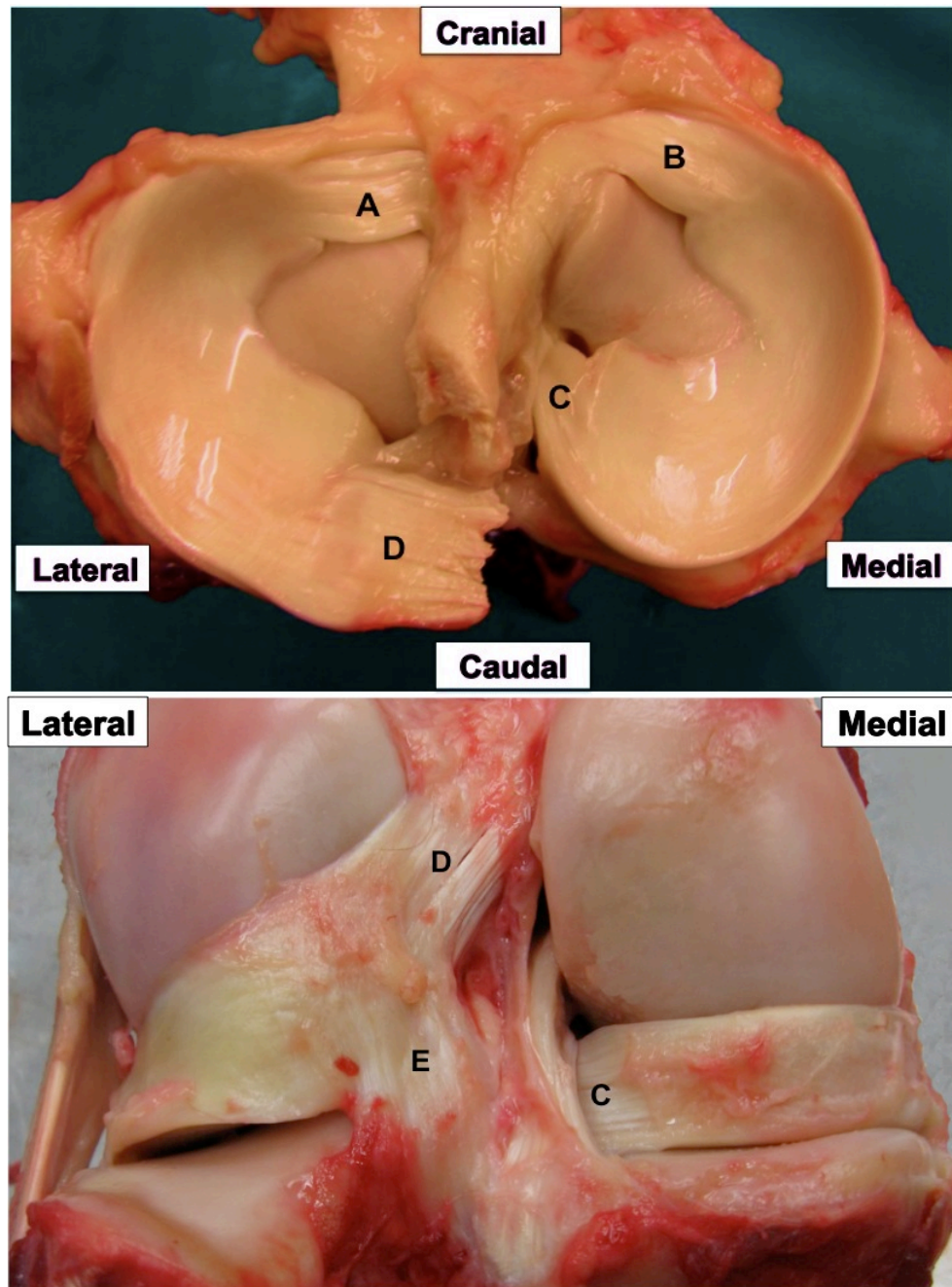
Figure 2.1 (Left) Proximal-distal view of the menisci from the left stifle of a cadaver. (Right) Caudal view of the left stifle of a cadaver. Cranial meniscotibial ligaments of the lateral (A) and medial (B) meniscus, and caudal meniscotibial ligaments of the lateral (E) and medial (C) meniscus. The meniscofemoral ligament of the lateral meniscus (D) is transected in the left image, but not the in the right. (For interpretation of the references to color in this and all other figures, the reader is referred to the electronic version of this thesis.)

The four ligaments responsible for stabilizing the femorotibial articulation are the cranial and caudal cruciate ligaments, which are located in the septum between the medial and lateral femorotibial joints, and the medial and lateral collateral ligaments, located on their respective aspects of the joint.(2) In humans, an intimate attachment of the medial collateral ligament to the medial meniscus is described (4), this has not been explicitly evaluated in the horse, however in some anatomy textbooks a small attachment is illustrated(2) and in other textbooks it is not.(5)

The main blood vessel supplying the stifle joint capsule and adjoining ligaments is the descending genicular artery, which arise from the femoral artery in the distal third of the thigh and passes craniodistal towards the patella and medial aspect of the femoropatellar joint between the sartorius and vastus medialis and the adductor muscles.(2) The popliteal artery, a direct continuation of the femoral artery beyond the origin of the caudal femoral artery, also provides blood supply to the stifle through collateral branches (genicular branches).(2) In humans and dogs, the meniscal blood supply originates from the joint capsule attachments to the periphery of the meniscus and from a vascular synovial covering of the meniscal ligaments.(6, 7) The meniscal blood supply has not been explicitly evaluated in the horse.

Kinematics of the Femorotibial Joint

The femorotibial joint is classified as a condylar joint (3) and functions primarily in uniaxial flexion and extension, with some slight internal and external rotation. Flexion of the equine stifle is limited by contact of the leg with the thigh and extension by the tension of the cruciate and collateral ligaments (2), though there are no published joint angles for maximal flexion or extension of the equine stifle. The standing caudal angle of the equine stifle joint is reported to be approximately 150°.(2) Kinematic studies have evaluated the equine stifle angle at

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a trot, and the overall impression is that the movement of the hindlimb is pendulum-like.(8-10) A kinematic study of the equine hindlimb during the gait cycle at a walk found that the caudal angle of the stifle varied between ~158° to ~142° during the weight bearing cycle.(8) One study used high speed cinematographic recording with reference markers on the limbs of Swedish Warmblood Riding horses to evaluate hindlimb joint angles at a trot on a treadmill, and found the stifle joint angle to vary from ~170-152° during weight bearing.(9) The angles of the equine stifle joint at a canter or a gallop have not been reported.

The motion of the femorotibial joint has been extensively evaluated in other species.(11-13) Complex models of the human knee have been created to evaluate its movement and how the soft tissue structures restrict such movement.(14, 15) For example, the human medial meniscus promotes internal rotation of the tibia and restrains femoral posterior translation, whereas the lateral meniscus and the medial aspect of the tibial eminence limit internal rotation of the tibia.(14) In the horse, the articular surface of the lateral femoral condyle is more strongly curved than the medial, and during flexion of cadaver stifles it was noted that the lateral femoral condyle and meniscus move further caudally on the tibial plateau than the medial.(2) Instant center of motion studies in humans and dogs have identified that the femoral condyle translocates caudally on the tibial plateau by a combination of sliding (gliding) and rolling.(12, 13) In humans, the femoral condyles exhibit predominantly rolling motion during early flexion, with the gliding component increasing during flexion to predominantly gliding motion at full flexion.(11) A 1:1.2 ratio of overall rolling:gliding was reported for the lateral femoral condyle, compared to a 1:3.8 ratio for the medial.(16) Sliding is the predominant mode by which the femur translocates in the canine stifle, and in normal stifles the direction of velocity at the joint contact surface is

tangential to the articular surface.(12) The instant center of motion in the equine femorotibial joint is not known.

Mild rotation occurs in the equine stifle joint during flexion and extension such that there is slight outward rotation of the tibia on extreme extension and slight inward rotation of the tibia on flexion.(2) This phenomenon is seen in many different species and has been described as the "screw home mechanism" of the stifle (17), and is attributed to the interactions of the cruciate and collateral ligaments and the disparate femoral condyles.(2, 11) Minor rotation within the equine stifle joint may occur at any flexion angle, although it is limited by the collateral and cruciate ligaments and is noted to be greatest in semi-flexion.(2)

Meniscal Structure and Function

Significant advancements have been made in understanding the function of the meniscus since 1897 when it was described as a functionless remains of a knee muscle.(18) The function of the menisci in the human knee was first described by King (19) in 1936, and in 1948 it was reported that removal of the meniscus lead to secondary deleterious changes in the femorotibial joint.(20) The multiple functions of the menisci have been documented to include providing congruency between the articular surfaces, equitable load transmission, shock absorption, joint stability, joint lubrication and proprioceptive information.(19, 21-24)

The exquisite functions of the menisci are directly related to the material component properties and ultrastructure of its extracellular matrix.(21) Because there are similarities in the basic composition and ultrastructure of the menisci between humans and other mammals, many different animal species (*ie.* canine, bovine and ovine) have been used as research models.(7, 21, 25, 26) Water makes up 74% of the meniscal contents, and collagen contributes to the majority

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(75%) of the dry weight of the meniscus, with non-collagneous proteins such as proteoglycans and elastin contributing only 8-13%.(27) The collagen fibers (predominantly type I collagen) are arranged in a parallel circumferential pattern, with the exception of superficial collagen fibers, that are parallel to the surface although randomly organized, and embedded radial tie fibers, that are oriented in a radial pattern (Figure 2.2).(27)
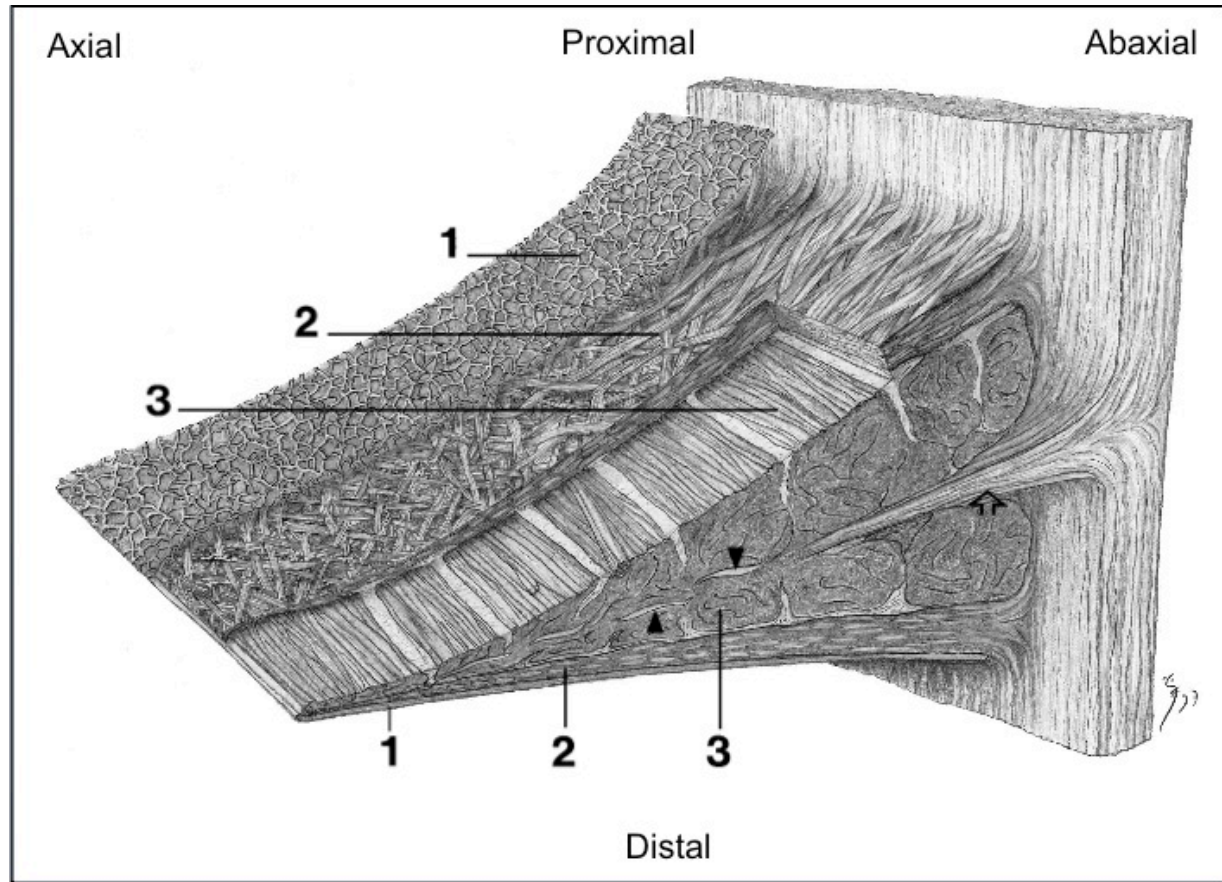
Figure 2.2 Schematic of a human meniscus demonstrating the collagen fiber orientation in the different regions. The superficial network [1] has a meshwork of thin fibrils, and the lamellar layer [2] consists of randomly orientated fibrils that are parallel to the surface of the meniscus. The central main layer [3] of the meniscus contains parallel circumferential collagen fibrils. Radial tie fibers are seen penetrating the central main layer (arrowheads). Image adapted from reference (28).

The wedge shaped menisci provide congruency between the femoral and tibial articular surfaces. This allows for the weight-bearing load to be transferred to the peripheral aspects of the

tibial condyles, in addition to the central region and therefore allows for a more equitable load transmission to the tibial condyles.(21) Contact between the round femoral condyles and the relatively flat tibial condyles would result in a small area of joint contact (29) if it were not for the presence of the meniscus. The menisci must translocate over the tibial condyles and undergo deformational changes in order to maintain congruency between the tibia and femur during flexion and extension of the stifle joint.(30)

Shock absorption is one of the many important functions of the meniscus. During compression of the meniscus between the femoral and tibial condyles, the meniscus functions as a biphasic composite material consisting of a fluid phase and a porous-permeable solid phase.(21) Consequently, water is forced to flow through the matrix by a hydraulic pressure gradient and matrix compaction, which gives the meniscus its viscoelastic properties of creep and stress-relaxation.(21, 27) The predictable flow of interstitial fluid through the meniscal substance has allowed for the creation of research models such as the biphasic viscoelastic finite element model.(31) The low compressive stiffness and the highly deformable nature of the meniscus (25) makes it an ideal substance for shock absorption and equitable load distribution through the femorotibial joint.

The phenomenon of "hoop stress", where by compression of the meniscus leads to the development of circumferential tensile forces within the meniscal tissue, was first proposed by Fairbank in 1948.(20) The wedge shape and near frictionless surface of the meniscus results in the development of a radial extrusive force during compression from vertical loading.(21) The radial extrusion force is resisted by tethering of the menisci at their cranial and caudal poles by the meniscal ligaments, which leads the development of circumferential tensile forces ("hoop stress") (Figure 2.3).(20, 32) Collagen fibers are most resistant to tensile forces applied parallel

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to their long axis, and consequently are aligned accordingly in the bulk of the meniscus, to resist

the circumferential tensile forces that arise during limb loading.(21) Because of the predominant

parallel circumferential pattern of collagen fibers, meniscal tissue displays a differential response

to stresses applied in different directions, a phenomenon known as anisotropy.(33)
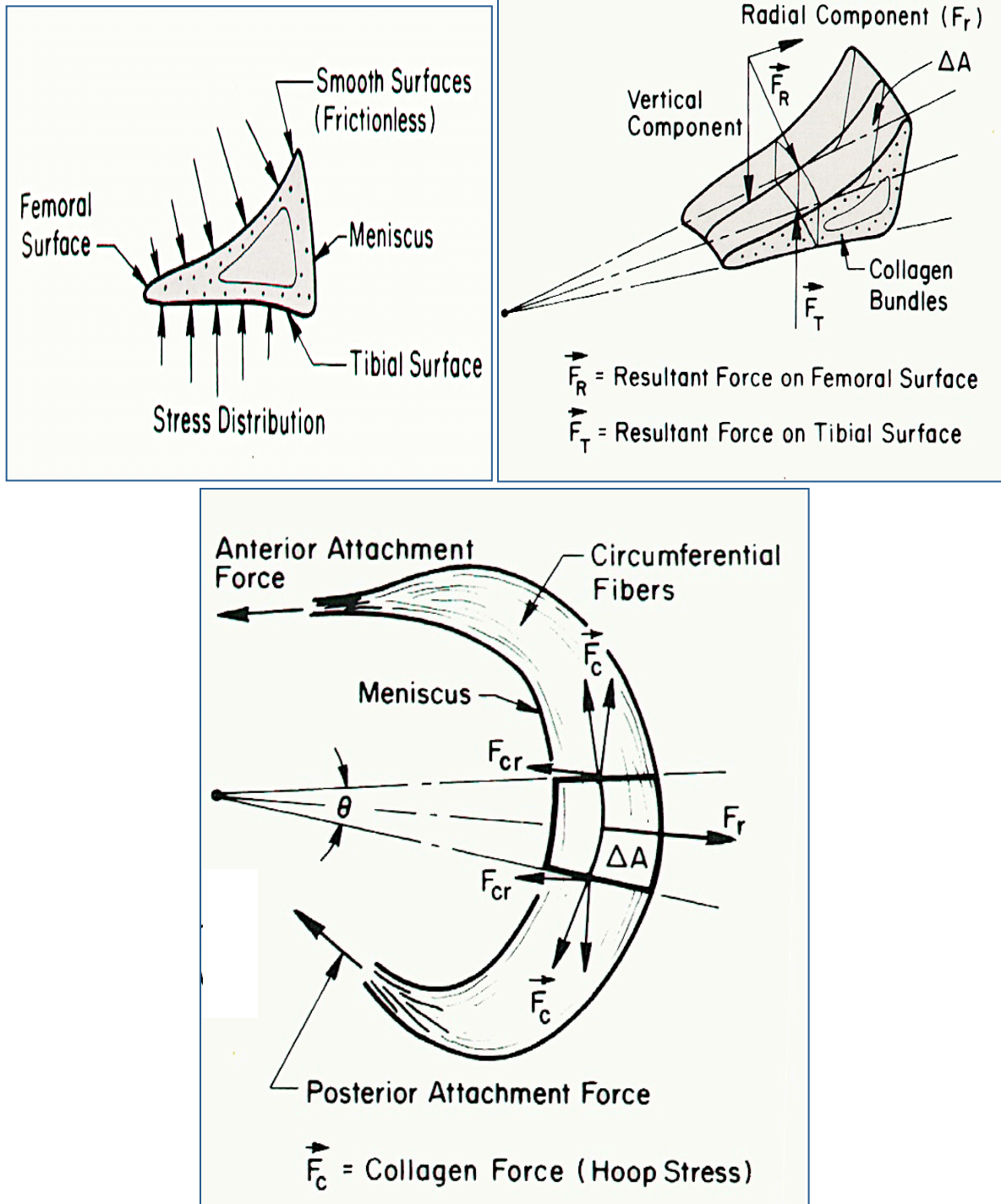
Figure 2.3 Free body diagrams demonstrating how compression of the meniscus results in circumferential oriented hoop stress. Image from reference (21).

Meniscal Translocation and Deformation

Meniscal translocation through the range of motion of the knee joint has been extensively evaluated in humans.(11, 30, 34, 35) However, to date there have been no experimental studies evaluating the motion of the equine meniscus. Alteration in the shape of the human menisci was noted during flexion, as the meniscotibial ligaments limit translocation of the cranial and caudal horns to a greater extent than the abaxial aspect of the meniscus, and the translocation distances of the anterior and posterior horns within a meniscus were not equal.(30) Meniscal deformation allows the menisci to maintain congruency with the femoral condyle despite alterations in the radius of curvature of its articular surface.(16) The human menisci were noted to translocate posteriorly over the tibial plateau during flexion of the stifle joint, with the lateral meniscus having a 2.3 times greater posterior excursion than the medial meniscus.(30) In Sisson and Grossman's 1975 anatomy textbook, it was noted that the lateral meniscus in the equine stifle, similarly, moved further caudally than the medial during joint flexion.(2) The posterior horn of the medial meniscus was reported to be the least mobile of the 4 horns in the human knee.(30) As the posterior horn of the human medial meniscus is the most common site of meniscal injury (36, 37), it was theorized that, because of its relative immobility, the posterior horn of the human medial meniscus may become trapped between the medial femoral and tibial condyles during hyperflexion of the knee and consequently be subjected to excessive forces that result in meniscal injury.(30) Accordingly, evaluation of meniscal translocation may provide insights into the pathogenesis of meniscal tears.

Pathogenesis of Meniscal Tears

A thorough understanding of the mechanisms involved in meniscal injury provides an explanation for the common locations and orientations of lesions, and allows for the identification of predisposing factors and therefore the opportunity for meniscal injury prevention. In order to provide a definitive explanation of the pathogenesis of meniscal tears, to fulfill Koch's postulates, the tear must be recreated under experimental conditions with the proposed risk factors. It is very difficult to experimentally create the complex conditions that may lead to *in vivo* meniscal tears. One cadaver study completed in rabbit stifles evaluated the morphology of failure with each of the ligaments and tendons, with the load applied along their anatomical axis until failure, which was compared to the morphology of clinical tears.(38) However, practically, understanding the pathogenesis of meniscal tears involves developing a thorough understanding of the forces acting on the meniscus (compression, shear, tension) and the ability of the meniscus to resist those forces, and then using this data to make predictions on how pathologic forces may lead to meniscal injury.(21, 25, 32, 33)

The collagen fibers of the meniscus are predominantly placed under tensile forces during limb loading due to the phenomenon of "hoop stress".(21) Tensile studies on meniscal tissue have shown a consistent pattern of failure of the circumferential collagen fibers, such that excessive tensile forces lead to shearing along the mechanically weaker planes between collagen bundles.(25, 31) As a result of this phenomenon, tears that are parallel to the collagen fibers (*ie.* longitudinal and horizontal tears), are more common in the human knee than tears that are perpendicular to the collagen fibers.(39) Understanding how the meniscus translocates and deforms has lead to predictions of how pathologic forces may develop and lead to meniscal injury.(11, 30) With the exception of one study which evaluated the shear properties of the

equine meniscus (33), there has been no documented experimental research evaluating the pathogenesis of meniscal tears in the equine stifle.

Sequelae of Meniscal Tears

In 1948 it was first reported that removal of the meniscus lead to secondary changes in the knee joint.(20) Since that time many studies have documented the development of secondary osteoarthritic changes with meniscal tears and loss/removal of meniscal tissue.(40-44) Complete or partial meniscectomy was the most common method used for treatment for meniscal tears in humans (20), until it was recognized that the technique was associated with the development of significant secondary articular cartilage injury.(42) Newer therapeutic techniques for meniscal tears, such as suture repair and stem cell therapy, provide improved restoration of meniscal geometry and as a consequence appear to result in a reduction in secondary cartilage injury compared to meniscectomy.(45-47)

Experimental studies in human and canine cadaver stifles have evaluated the effect of meniscal tears and meniscal resection on force transmission to the tibia.(29, 48-52) Similar results were seen in all of these studies, such that serial meniscectomy, meniscal release and complete meniscectomy resulted in alteration in the magnitude and distribution of pressure on the tibia.(29, 48-52) Loss of equitable load transmission with meniscal injury leads to the development of a focal central region of increased pressure on the tibial condyle with increasing severity of injury.(29, 48-52) Lesions that lead to a total loss of hoop stress, such as a complete tear to the posterior root, resulted in tibiofemoral contact mechanics similar to that seen with complete meniscectomy.(52) The focal central region of increased pressure on tibial condyle corresponds with the location that cartilage injury is seen in the central regions of the tibial and

femoral condyles in clinical cases of meniscal tears.(53, 54) Complete medial meniscectomy was evaluated in normal horses in one study, where focal erosion of the medial femoral condyle and tibial plateau was seen on post mortem examination of specimens euthanized 12 weeks after surgery.(55) Meniscal tears may also lead to a decrease in shock absorption by the meniscus, hence the articular cartilage and subchondral bone may be exposed to greater forces.(21) Loss of equitable load transmission and shock absorption as a result of meniscal injury may lead to altered mechanical loading and potentially injury of the articular cartilage and subchondral bone.

Provision of stability to the femorotibial joint is another meniscal function that may be altered by meniscal injury. For example, the posterior pole of the medial meniscus acts as a secondary stabilizer of cranial tibial translation, in addition to the primary stabilizer, the cranial cruciate ligament.(21, 56, 57) An experimental study documented significant increases in external rotation and lateral tibial translation with posterior root meniscal tears compared to intact menisci in a human cadaver stifle.(52) Interestingly, increased pressure (50) and cartilage lesions (53) were seen in the lateral aspect of the femorotibial joint in stifles with medial meniscal pathology, indicating that the biomechanics of the entire femorotibial joint may be altered by medial meniscal pathology. Alterations in joint stability and motion, in combination with alterations in joint contact forces (as described above), may alter the types of local stresses experienced by the articular cartilage (ie shear strain developing in a location previously under compression or vice versa).(58) Further research is indicated to evaluate the effects of equine meniscal tears on joint stability and secondary changes within the femorotibial joint.

Meniscal injury results in the release of tissue debris into the joint, which may lead to ongoing cartilage injury.(59) In one in vitro study, the presence of meniscal fragments resulted in an increased production of matrix metalloproteinases and prostaglandin E_2 in response to

interleukin-1.(60) Studies have shown that inflammatory and degradative mediators are produced when cyclic mechanical strain is applied to meniscal cells, which may play a role in the development and progression of osteoarthritis with meniscal injury.(61-63) Nitric oxide synthesis and a resultant inhibition of collagen and proteoglycan synthesis with meniscal injury may also lead to articular cartilage pathology.(60) In an experimental study of canine meniscal injury, several different configurations of medial meniscal injuries were created and insults that allowed a free-floating or entrapped segment resulted in the most severe degenerative changes in the joint.(64) The association between meniscal injury and osteoarthritis is complex, since osteoarthritis may conversely lead to degenerative changes in the meniscus.(65) The secondary osteoarthritic changes that are recognized with meniscal tears appear to have a multifactorial and complex pathophysiology. Further investigation of the molecular nature of the association between meniscal injury and osteoarthritis is needed.

Clinical Characteristics of Meniscal Tears in the Horse

Meniscal tears were first reported in the horse by Valdez and Adams in 1978.(55) Since that time, there has been a series of case studies published, primarily by Dr. John Walmsley, describing the clinical characteristics of these lesions.(66-68) An isolated lesion of the cranial horn of the medial meniscus and its associated cranial meniscotibial ligament is the most common arthroscopically identified site of meniscal lesions.(68, 69) A grading system (grade I-III) for cranial horn meniscal tears was established:(67)

Grade I- A tear in the cranial ligament extending into the meniscus but without significant separation of the tissues.

Grade II- A complete tear in the cranial pole of the meniscus and the cranial ligament, whose limits were visible arthroscopically.

Grade III- A severe tear of the meniscus and cranial ligament which extended beneath the femoral condyle and whose limits could not be seen. (Figure 2.4)
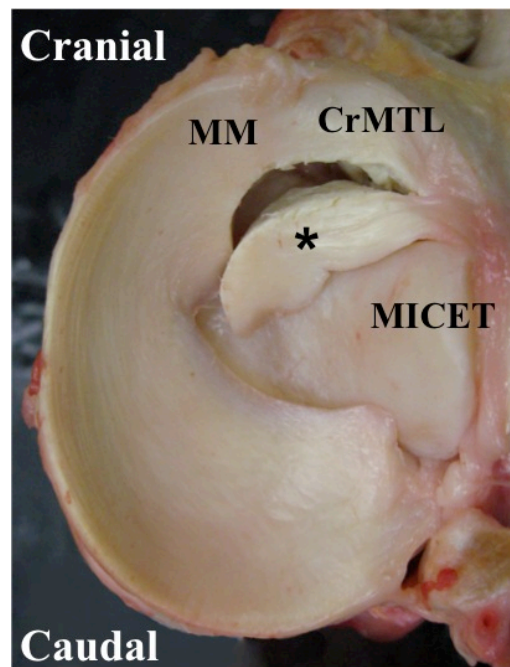
Figure 2.4 The medial meniscus of a right cadaveric stifle illustrating a simulated grade III meniscal tear (*). The tear extends longitudinally through the cranial meniscotibial ligament (CrMTL) into the cranial horn of the medial meniscus (MM). Medial intercondylar eminence of the tibia (MICET).

Unlike the dog or human, in which meniscal lesions are more commonly associated with concurrent cruciate (57) and/or collateral ligament injuries (70), only 14% of meniscal tears were associated with cranial cruciate ligament injury in one study.(68) As follows, meniscal tears are commonly recognized as a primary lesion, and primary source of lameness in the horse.(68, 69) In humans and dogs, meniscal tears in the posterior (caudal) horn of the medial meniscus are most common (36, 37, 57), in contrast to the horse where the cranial horn of the medial meniscus is the most common location for meniscal tears.(68)

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Clinical Presentation

Clinical signs of meniscal injury in the horse are generally non-specific. There is no documented pathognomonic history or clinical signs associated with meniscal injury; a traumatic incident or a fall may be part of the history, though generally the onset is insidious.(68) A sudden compressive force on the menisci with the joint in extension has been proposed as a potential mechanism of primary injury of the menisci.(68) Co-existing stifle pathology such as medial collateral or cruciate ligament injury may be recognized although more commonly, meniscal tears are seen as solitary or primary lesions.(68) A recent study documented the concurrent/sequential occurrence of subchondral cyst-like lesions in the medial femoral condyle with medial meniscal tears, though the pathogenesis of these lesions is not clear.(71)

In one case series, the average grade of lameness documented at presentation was 3/10 (with more severe lameness scores being reported at the time of injury) and the duration of injury at the time of presentation for veterinary evaluation was approximately 8 weeks.(67) Effusion of the femoropatellar and/or medial femorotibial joint was seen in only 39% of cases, and horses with effusion had a 9 fold increase in the relative risk of having a severe meniscal lesion.(67) Flexion tests were positive in 66% of cases and lameness was significantly improved with intra-articular anesthesia in 93% of cases.(67)

Diagnostics

Significant limitations exist in diagnostic imaging of equine meniscal injuries as a result of the size and anatomy of the equine stifle joint. Widely available diagnostic imaging techniques for the equine stifle joint include radiography, ultrasonography, arthroscopy and nuclear scintigraphy. Recent access to wide (or open) bore magnetic resonance imaging (MRI)

allows for ideal evaluation of the equine meniscus, articular cartilage and other soft tissue structures of the stifle.(72)

Radiographic changes that may be seen with meniscal injury include: enthesiophytosis, soft tissue mineralization, remodeling and osteoarthritic changes. Radiographic changes were seen in 38/80 (48%) cases with meniscal tears and the presence of radiographic changes worsened the chance of recovery by greater than 25%.(67) Another study reported radiographic changes in 83% of meniscal tears.(73) In one review of 80 cases, new bone was seen on the medial intercondylar eminence of the tibia (MICET) in 29% of cases, osteoarthritic changes in 23%, and dystrophic mineralization in 8%.(67) Interestingly new bone on the MICET is commonly documented as evidence of cranial cruciate ligament injury, however it was seen in 23% of stifles with meniscal injury and only 7% of stifles with cranial cruciate ligament injury.(68)

Ultrasonography is an important tool for diagnostic imaging of the equine meniscus.(74-76) Ultrasonographic signs of meniscal pathology include: loss of normal triangular shape on cross section, changes in echogenicity, extrusion from between the femoral and tibial condyles, fragmentation of the meniscus, separation from the collateral ligaments, and loss of femorotibial joint space.(68) One of the significant benefits of ultrasonography relative to arthroscopy, which is frequently considered the diagnostic gold standard in humans (77), is the ability to examine the abaxial region of the menisci that cannot be visualized at arthroscopy.(78) In a case series of 29 cases of meniscal tears, ultrasonographic evidence of meniscal injury was seen in 19 of the cases, and the remaining 10 cases were only identified at arthroscopy.(73) In contrast, three of the 29 cases had ultrasonographic evidence of tears that could not be visualized on arthroscopy.(73) A recent study evaluating soft tissue injuries in the equine stifle used arthroscopic exam as the gold

standard with which to verify ultrasonographic findings and found that ultrasound had a specificity and sensitivity of 56% and 79% respectively.(69) A potential explanation for the high number of false positives is the presence of meniscal tears in regions that are inaccessible with an arthroscope but can be seen with ultrasound. Owing to the complex shape and fiber orientation of the meniscal tissues, all lesions should be confirmed in two perpendicular planes with ultrasonography, and familiarization with the normal anatomy and comparison with the sound limb is necessary to avoid misdiagnoses.

Scintigraphy appears to have a very low sensitivity for diagnosing soft tissue lesions of the stifle; non-specific signs of bone injury or secondary osteoarthritis may be seen, and potentially though rarely insertional desmopathy.(79)

Computed tomography (CT) evaluation of the equine stifle has permitted detection and more extensive evaluation of lesions that would be inaccessible or incompletely identified with more conventional diagnostics.(80, 81) CT arthrography is available at select referral institutes and can be used to identify soft tissue lesions in the equine stifle, such as enthesis/subchondral sclerosis of the tibia at the cranial medial meniscotibial ligament attachment, that is not evident on radiographs.(80) Positive contrast stifle CT arthrograms in a canine cadaver model had a 90% sensitivity and 100% specificity for identifying simulated meniscal injuries.(82) Access to CT for evaluation of equine clinical cases has been relatively recent, therefore further evaluation of its benefits and limitations will be required.

Recent access to MRI's that have the capacity to accommodate an equine stifle has permitted excellent evaluation of the soft tissue structures in the stifle.(72) Horses must be placed under general anesthesia to perform the stifle MRI and generally imaging is limited to one stifle within an anesthetic period. The Siemens Magnetom Espree (Siemens Medical Solutions

USA, Inc. Malvern, PA, USA) is a high-field (1.5T) magnet with a wide (70cm) and ultra short (125cm) bore which can accommodate an equine stifle from horses with relatively long legs and narrow hips.(72) Open bore magnets such as the Vet-MR Grande unit (The Esaote Group, Genoa, Italy) with a low-field magnet (0.25 T) may also be used to image the equine stifle. The specificity and sensitivity of MRI in identifying meniscal tears has been reported to be 90% and 64% in dogs with a low field system (83), and 50-93% and 59-84% in humans with high and ultra-high field systems.(84, 85) Another study reported an accuracy of 85.9% and 73.8% in diagnosing medial and lateral meniscal injuries respectively in humans.(86) Evaluation of the accuracy of equine stifle MRI is yet to be determined.

Arthroscopic evaluation is considered the gold standard for evaluation of the meniscus in humans.(77) Arthroscopic examination of the equine meniscus is limited to the cranial and caudal horns as a result of superimposition of the femoral condyles and the inability to distract the equine stifle.(66) Arthroscopy allows direct visualization and assessment of the lesion, however horizontal tears and tears in a large portion of the abaxial menisci cannot be visualized. Intra-operative distraction of the canine stifle (87, 88) and human stifle (89) allows for complete evaluation of the menisci from a cranial approach. Distraction of the equine stifle is not possible unless there is disruption of a collateral ligament. Accordingly, evaluation of the caudal horns must be achieved with a caudal approach to the femorotibial joints (78, 90), although a novel cranial intercondylar approach has been described for the caudal horn of the medial meniscus.(91) Arthroscopy also allows for evaluation of co-existing cartilage or ligament injury. Evaluation of cartilage lesions on the femoral condyles can be completed with flexion of the femorotibial joint; however very little of the tibial condyles can be visualized arthroscopically.(78) Despite the limitations of arthroscopic evaluation of the equine meniscus, it

is frequently used as the gold standard from which the sensitivity and specificity of other diagnostic techniques is determined.(69) The most significant benefit of the use of arthroscopy as a diagnostic technique for meniscal injuries is that therapeutic procedures may be completed concurrently.

Treatment

Medical therapy for equine meniscal tears consists of rest and pain/anti-inflammatory medications (such as phenylbutazone and intra-articular steroids/hyaluronic acid).(90) However, if the lesion is accessible arthroscopically, the most common recommended treatment is arthroscopic resection of the damaged meniscal tissue (68, 69, 78, 90), as this treatment appears to lead to decreased joint inflammation and pain, and superior long-term results compared to medical treatment alone.(68) Cranial horn meniscal tears seen at arthroscopy commonly have severely fibrillated meniscal tissue, (68) and removal of this traumatized tissue in joints reduces the physical debris liberated into the joint which may lead to ongoing cartilage injury.(59) Evaluation and debridement of any additional cartilage or soft tissue injuries is also be performed during arthroscopy.

Suturing techniques for meniscal repair have been well documented in the human literature (45, 92, 93), and reports of successful suturing of equine meniscal tears exist.(78, 94) Suturing of meniscal tears is rarely completed, which may be as a result of limitations in case selection, expertise and/or access to specialized equipment. Local regenerative therapies, such as stem cells and fibrin clots, have been shown to improve meniscal healing in humans and dogs.(46, 47, 95, 96) Although there are positive anecdotal reports regarding stem cell therapy

for treatment of meniscal tears in horses, (94) there are no scientific studies documenting their efficacy in this species.

The overall goals for treatment of equine meniscal tears are removal of traumatized tissue, restoration of meniscal geometry and minimization of secondary osteoarthritic changes. In addition to the surgical and adjunctive therapies used, a sufficient rest period (often at least 6 months) is important for allowance of maximal healing prior to return to athletic function.(90)

Prognosis

The prognosis for return to previous athletic function with cranial horn meniscal injuries has been correlated with the severity of injury, and the reported prognosis for grade I, II and III tears is 36-63%, 29-56% and 0-6% respectively.(67, 69) In 71% of cases, articular cartilage injury was present at the time of meniscal tear diagnosis (68), and the outcome was significantly worse if there was articular cartilage injury or associated radiographic changes present at the time of diagnosis.(67) Overall, one clinic reported that 44% (39/110) of meniscal tears that have undergone diagnostic and therapeutic arthroscopy have returned to full use (68), compared to another study reporting only 21% (6/21) of cases returning to full work.(73) There are no published comparisons on the prognosis with medical versus surgical management; however anecdotal reports indicate that improvement in lameness is seen with arthroscopic debridement.(68, 69) A recent study reported a poor prognosis, with only 4/19 horses returning to previous athletic performance when meniscal lesions were seen concurrently or sequentially with medial femoral condylar subchondral cystic lesions.(71) As a result, the overall prognosis for recovery from meniscal tears is poor-fair, and secondary arthritic changes within the joint appear to be a significant factor affecting the prognosis.

Purpose and Hypothesis

Following a comprehensive review of the literature, it is evident that our understanding of the equine meniscus significantly lags behind that which is known about the human and canine meniscus. The characteristic cranial horn lesions seen in the equine stifle do not appear similar in appearance to those recognized in other species, therefore extrapolation of research data from these species may not be appropriate. The objective of this thesis was to gain a greater understanding of the pathogenesis of cranial horn meniscal tears in the horse, and the secondary pathologic changes that occur with these lesions. Three experimental studies were completed with these objectives in mind. The objectives and hypotheses of the studies were as follows:

Study 1 (chapter 3):

Objective: To quantitatively document meniscal translocation and deformation through the range of motion, in order to evaluate for potential mechanical factors in the pathogenesis of injury to the cranial horn of the medial meniscus.

Hypotheses: It was hypothesized that equine menisci would exhibit cranial-caudal translocation on the tibia during flexion and a reciprocal caudal-cranial translocation during extension. Additionally, it was hypothesized that the lateral meniscus would have a greater overall excursion, and that kinematic features of the cranial horn of the medial meniscus may place it at risk of injury during hyperextension.

Study 2 (chapter 4):

    Objective: To identify potential functional-anatomic characteristics of the cranial horn attachment of the medial meniscus (MM) that may explain the pathogenesis of the common tear patterns that have been reported.

    Hypotheses: It was hypothesized that, during full extension of the stifle, significantly higher tensile forces are generated within the abaxial (peripheral) portion of CrMTL when compared to the axial (inner) portion of the ligament. It was also hypothesized that the normal functional anatomic structure of the CrMTL predisposes this structure to injury at the reported site.

Study 3 (chapter 5):

    Objective: To evaluate the effect of grade III cranial horn tears of the equine medial meniscus (pre- and post- resection) on the pressure magnitude and distribution over the articular surface of the medial tibial condyle.

    Hypotheses: It was hypothesized that simulated grade III meniscal tears would lead to the development of an area of increased pressure in the central region of the medial tibial condyle throughout the weight bearing stifle angles, and that resection of the tear would further increase the magnitude of pressure in this region.

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CHAPTER 3.

Meniscal translocation and deformation throughout the range of motion of the equine stifle joint: An *in vitro* cadaveric study

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Abstract

The purpose of this study was to quantitatively document the translocation and deformation of the equine meniscus through the range of motion, to identify potential mechanical factors in the pathogenesis of injury to the cranial horn of the medial meniscus. Radiographic markers were embedded in the periphery of the menisci in 6 cadaver stifles. Proximal-distal radiographs were taken at 15° intervals ranging from full flexion (30°) to full extension (160°). Magnetic resonance imaging sequences of 3 additional cadaver stifles were obtained in axial and sagittal planes at the predetermined stifle angles. Data from the radiographic study showed that there was a significantly greater overall mean cranial-caudal translocation (1.6 times) of the lateral meniscus relative to the medial was seen from full extension to full flexion ($p = 0.002$). The cranial horn of the medial meniscus was the least mobile of the 4 horns, yet a significant cranial displacement relative to the cranial horn of the lateral meniscus was seen in the terminal 10° of extension. MRI images revealed a significantly greater axial compressive strain in the cranial horn of the medial meniscus relative to the cranial horn of the lateral meniscus in the terminal 10° of extension ($p = 0.017$). In conclusion, the equine menisci exhibit a cranial-caudal translocation over the tibia throughout the range of motion. While the cranial horn of the medial meniscus is the least mobile of the 4 horns, it undergoes significant cranial translocation and axial compression in the terminal 10° of extension. Thus, hyperextension of the stifle may place the cranial horn of the medial meniscus at risk of injury and thus explain the higher prevalence of meniscal tears at this location.

Introduction

Contrary to the human (1-5) and canine (6-8) literature, meniscal injuries in the horse have received relatively little attention. Meniscal tears in horses were first reported in 1978 (9) and, to date, the published literature on equine meniscal tears consists predominantly of retrospective cases studies.(10-12)

Clinically, an isolated lesion of the cranial horn of the medial meniscus and its associated meniscotibial ligament is the most commonly identified site of meniscal injury in horses.(12) It was noted that of 110 meniscal tears diagnosed at one referral institution, 79% involved the cranial horn of the medial meniscus.(12) Unlike the dog or man, in which meniscal tears are more commonly associated with concurrent cruciate and/or collateral ligament injuries, only 14% of meniscal tears were associated with cranial cruciate ligament injury in the horse.(12) Thus, extrapolation of the pathogenesis of meniscal injuries from other species to the horse may not be applicable.

Equine cranial meniscal tears tend to have a characteristic orientation with the lesion extending longitudinally through the cranial meniscotibial ligament and into the cranial horn a variable distance.(10) A classification system for cranial meniscal tears classifies lesions as *Grade I*, *II* or *III* based on the arthroscopic appearance of the degree of tissue separation, and the distance with which the tear extends towards the axial surface of the cranial horn underneath the femoral condyles.(11) Severe meniscal tears have been reported as career ending injuries with significant secondary osteoarthritic changes developing in the affected stifle joint.(12) Diagnostic tests to evaluate meniscal tears in horses have been limited based on the size and anatomy of the horse; however, with recent access to large bore magnetic resonance imaging (MRI) units (13), improved ultrasonographic techniques (14) and increased application of new

(15) and established arthroscopic approaches, recognition of meniscal tears is improving.

While medial meniscal injury has been associated with subchondral cystic lesions of the medial femoral condyle (16), it is not clear if these femoral lesions are a result or a cause of the observed meniscal injury. A recent report has suggested that compression of the cranial horn of the medial meniscus during hyperextension of the stifle may be the mechanism involved in the most commonly reported meniscal lesion, an isolated tear of the cranial horn of the medial meniscus.(12) However, none of these potential aetiologies has been rigorously investigated.

An elegant study examining meniscal translocation and deformation through the range of motion in man has led to a better understanding of potential mechanisms involved in meniscal pathology.(17) Therefore, the purpose of the current study was to document the *in vitro* translocation and deformation of the equine menisci through the full range of stifle motion. It was hypothesised that equine menisci would exhibit cranial-caudal translocation on the tibia during flexion and a reciprocal caudal-cranial translocation during extension and that kinematic features of the cranial horn of the medial meniscus may place it at risk of injury during hyperextension.

Materials and Methods

To determine the relative translocation and deformation of the equine menisci through the range of motion, cadaveric stifles were harvested from 10 mature light breed horses humanely subjected to euthanasia for reasons unrelated to this study. The specimens had no known history of lameness localized to the stifle joint and represented a range of ages, and both genders. Limbs were removed at the level of the mid femur and tibia within 8 h of euthanasia, stored at -20°C and thawed at room temperature for approximately 48 h prior to study.

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Part 1: Radiographic assessment of meniscal translocation and deformation

Six cadaveric equine stifles from 6 different horses were used for this portion of the study. The cadaver limbs were dissected on the day of experimentation and all soft tissues (including the joint capsule) were removed with the exception of the menisci and the collateral, cruciate and meniscal ligaments. The stifle specimens utilized in the study had no grossly visible joint pathology. Spherical radio-opaque markers (2 mm in diameter) were placed in the periphery of the menisci and cranial meniscotibial ligaments at 1 cm intervals starting 0.5 cm from the cranial meniscotibial ligaments attachment to the tibia. Access to the caudal medial meniscotibial ligaments was restricted due to caudal cruciate ligament superimposition and thus markers were not placed in the caudal meniscal ligaments. To place the radio-opaque markers, a 2.5 mm long, 2.5 mm deep incision with a No. 15 scalpel blade was made transversely (parallel to the circumferential collagen fibers) at the proximal to distal midpoint of the periphery of the menisci. Markers were embedded and a single cruciate suture of 2-0 monofilament was placed across the incision to prevent displacement of the markers. Two additional markers were placed in the most axial and proximal aspects of the cranial horns of the medial and lateral menisci.

Specimens were mounted in a jig constructed to stabilize the tibia while allowing passive, unconstrained flexion and extension of the femorotibial joint. The jig was secured to the digital radiography table, thus maintaining a constant tibia location and image angle, and proximal to distal radiographs were taken through the full range of motion. Digital radiographs (Eklin Medical Systems, Sound Eklin, Carlsbad, CA, USA) were taken at full extension (at or near 160° in all specimens), 150°, 135°, 120°, 105°, 90°, 75°, 60°, 45° and full flexion (at or near 30° in all specimens). Prior to each radiograph the menisci and femoral condyles were moistened with physiological saline and moved through the full range of motion to lubricate the cartilage and

meniscal surfaces.

Sequential digital images were superimposed using Adobe Photoshop software (Adobe Systems Incorporated, San Hose, CA, USA) and the distance each radio-opaque marker moved between the 9 intervals of stifle angle was recorded in mm (using a scale on the films) (Figure 3.1).
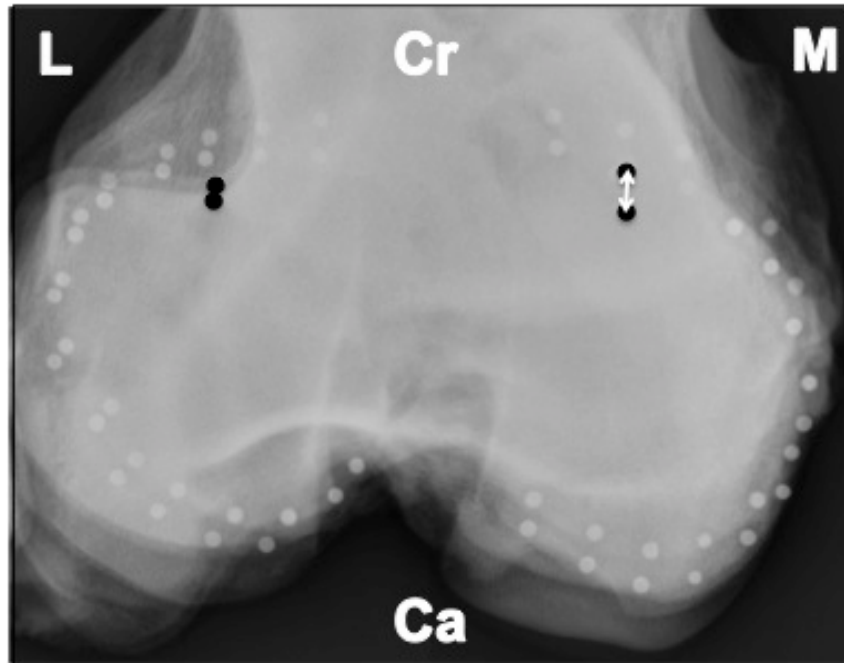
Figure 3.1 Superimposed proximal-distal digital radiographs taken at 160° and 150° stifle angle. Cr; cranial, Ca: caudal, L; lateral, M; medial. Radiopaque markers outline the periphery of the menisci and the cranial horns (highlighted in black). All markers translocated caudally over the tibial plateau between 160° and 150°. The distance each marker moved was measured (white arrow) between all the stifle angles.

Part 2: Magnetic resonance imaging assessment of meniscal translocation and deformation

Four additional cadaveric equine stifles from 4 different horses were used for this portion of the study. Minimal removal of soft tissues over the mid diaphysis of the tibia and femur was completed prior to mounting in a custom jig (Figure 3.2), which allowed passive, unconstrained flexion and extension of the stifle at set intervals. The remaining soft tissues in the stifle region

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were left intact.
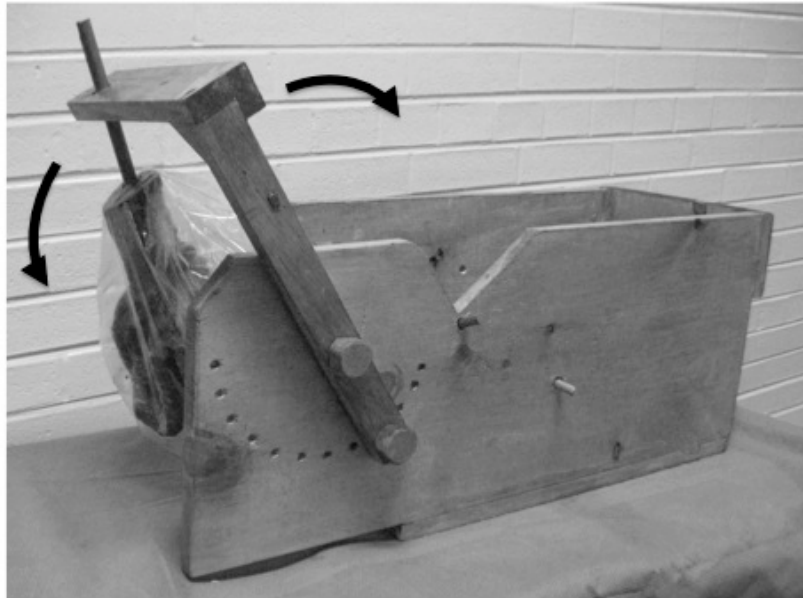
Figure 3.2 Equine stifle positioned in custom jig for MR imaging. The tibia is immobilized, while the cross bar assembly permits fixation of the joint at the standardized angles (160° to 30° at 15° increments) with minimal varus, valgus or rotational constraint.

The stifle/jig assembly was then secured in the MRI unit (Siemens Magnetom Espree, Siemens Medical Solutions, Malvern, PA, USA). Preliminary studies determined that optimal contrast between meniscal tissue and adjacent synovial fluid, cartilage and subchondral bone was achieved with T1-weighted volumetric interpolated breath hold examination (VIBE), T1-weighted Turbo-fast low-angle shot (FLASH) and T2*-weighted multiple-echo data image combination (MEDIC) with fat saturation sequences. An axial and sagittal series (of at least one of the above sequences) was acquired at each of the stifle angles used in Part 1 (160–30°). The height of the medial and lateral cranial horns were measured on parasagittal sections of the medial and lateral femoral condyles at a location corresponding to the minimal height of the cranial horn (maximal site of compression) in the 160° stifle angle image. The height of the cranial horns at the same location was measured at 150° stifle angle. The compressive strain on

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the medial and lateral cranial horns in the terminal 10° of extension (between 150° and 160°) was then calculated using the following formula:

$$\varepsilon = (\text{height at } 160° - \text{height at } 150°) \ \text{height at } 150°$$

Statistical Analysis

Student's paired *t* tests were used for all statistical analyses; allowing for comparison of medial and lateral meniscal translocation and height while avoiding confounding from variation in cadaver size. Significance was set at $p<0.05$.

Results

Part 1: Radiographic assessment of meniscal translocation and deformation

Evaluation of the superimposed images revealed that the menisci translocate caudally over the tibial plateau from full extension to full flexion. The peripheral meniscal outline, portrayed by the radiographic markers (Figure 3.3), changed from a C-shape in extension to an L (and inverted L) shape in full flexion.
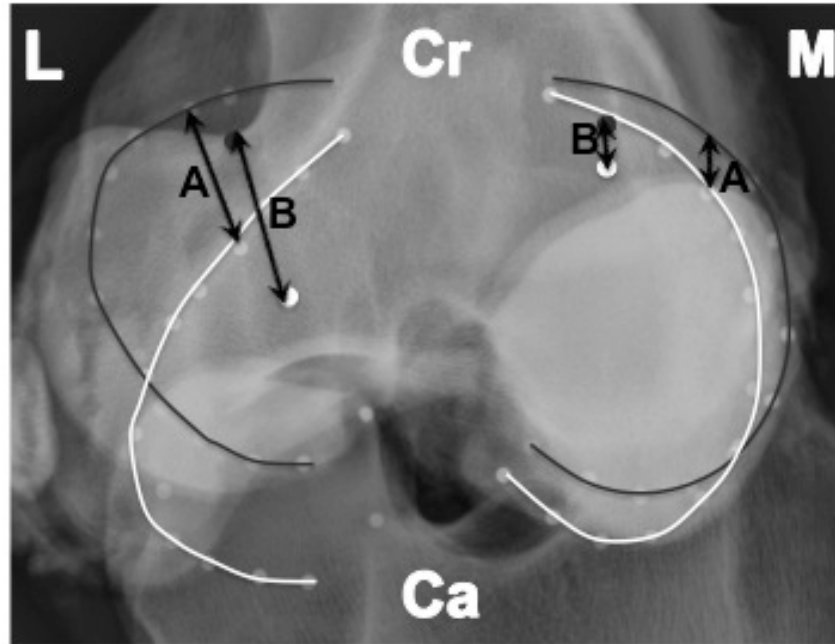
Figure 3.3 Superimposed proximal-distal digital radiographs of 160° (full extension) and 30° (full flexion) stifle angle for Cadaver 2, Cr; cranial, Ca: caudal, L; lateral, M; medial. Radiopaque markers outline peripheral meniscal location at 160° (grey line) and 30° (white line). Distance A represents the distance moved by each marker that was measured and averaged for overall meniscal translocation distance. Distance B indicates the distance moved by the cranial horn markers.

Quantitative assessment of meniscal translocation revealed that the mean ± s.d. medial and lateral meniscal cranial-caudal translocation was 1.69 ± 0.41 cm and 2.53 ± 0.31 cm, respectively. This resulted in a significantly ($p = 0.002$) greater cranial-caudal translocation of the lateral meniscus relative to the medial through the full range of motion. The mean ratio of lateral/ medial meniscal translocation was 1.6 ± 0.47.

When focusing solely on the movement of the markers at the periphery of the cranial and caudal horns of the medial and lateral menisci, the cranial horn of the medial meniscus had the lowest average cranial-caudal translocation through the full range of motion of the 4 horns, while the caudal horn of the lateral meniscus had the greatest translocation (Table 2.1).

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	Medial Meniscus		Lateral Meniscus	
Horn	Cranial	Caudal	Cranial	Caudal
Specimen #1	1.155	1.527	1.433	2.749
Specimen #2	0.76	0.919	2.05	2.386
Specimen #3	1.693	1.997	2.307	3.184
Specimen #4	1.87	2.006	2.061	3.151
Specimen #5	1.813	2.191	2.006	3.064
Specimen #6	1.520	1.749	1.950	2.401
Average ± SD	1.47 ± 0.43*	1.73 ± 0.46	1.97 ± 0.29	2.82 ± 0.37

* Significantly shorter translocation than all other horns ($p < 0.05$)

Table 3.1 Average cranial-caudal translocation distances (cm) of the cranial and caudal horns of the medial and lateral menisci through the full range of motion (160°-30° stifle angle).

The cranial horn of the medial meniscus translocated a shorter distance than the cranial horn of the lateral meniscus for all the stifle angle intervals except the terminal 10° of extension (150–160°). Statistical analysis revealed that the cranial horn of the medial meniscus had a significantly ($p = 0.004$) greater cranial translocation between 150 and 160° stifle angles. On average the ratio of translocation of the cranial horn of the medial meniscus compared to the cranial horn of the lateral meniscus in the terminal 10° of extension was 3.8 ± 3.2 times. Figure 3.4 illustrates the average cranial-caudal translocation distance of the cranial horns of the medial and lateral menisci between each interval of stifle flexion. The cranial-caudal translocation of the cranial horns of the medial and lateral menisci at each stifle flexion interval is expressed as a percentage of the total cranial-caudal translocation of each cranial horn in Figure 3.5. The cranial horn of the medial meniscus undergoes 29.5 ± 7.5% of its total cranial-caudal translocation in the first 10° of flexion, relative to the cranial horn of the lateral meniscus which only translocates 4.4 ± 1.5% of its total excursion during this interval (Figure 3.5). Thus, the cranial horn of the

medial meniscus appears to be forced cranially to a greater degree than the cranial horn of the

lateral meniscus in the terminal 10° of extension.
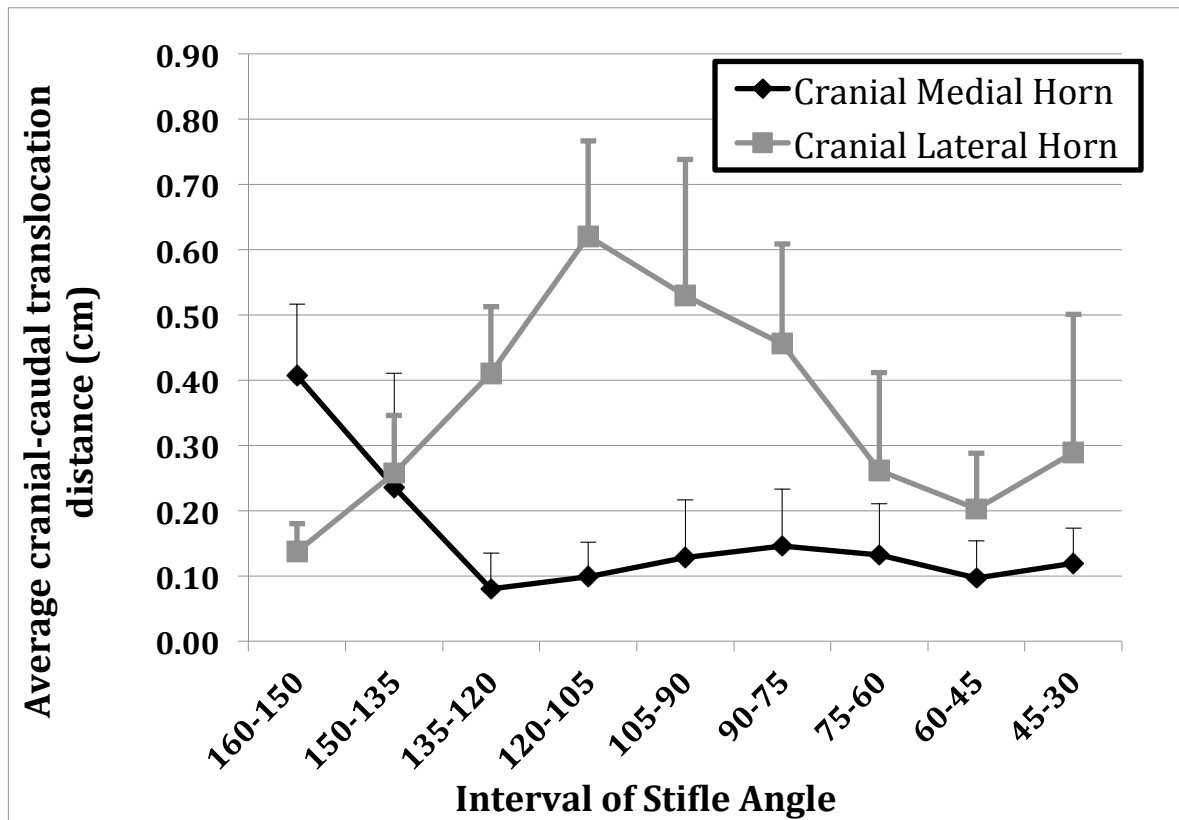
Figure 3.4 Mean ± s.d. cranial-caudal translocation distance of the cranial horns of the medial and lateral menisci at each interval of stifle flexion.
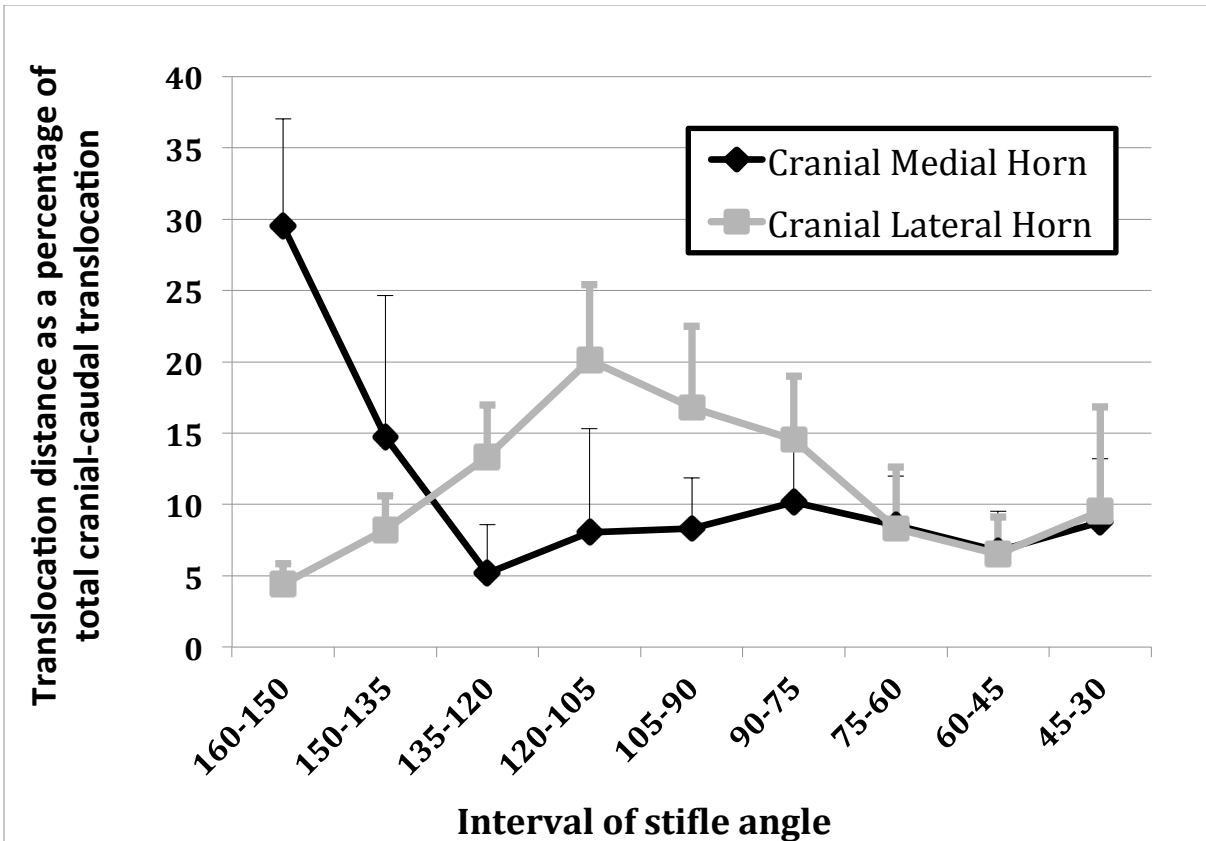
Figure 3.5 Mean ± s.d. cranial-caudal translocation of the cranial horns of the medial and lateral menisci at each interval of stifle flexion as a percentage of total distance moved by each horn.

Part 2: MRI assessment of meniscal translocation and deformation

Qualitative assessment of MR images confirmed meniscal translocation and deformation similar to that quantified in Part 1 (Figure 3.6).
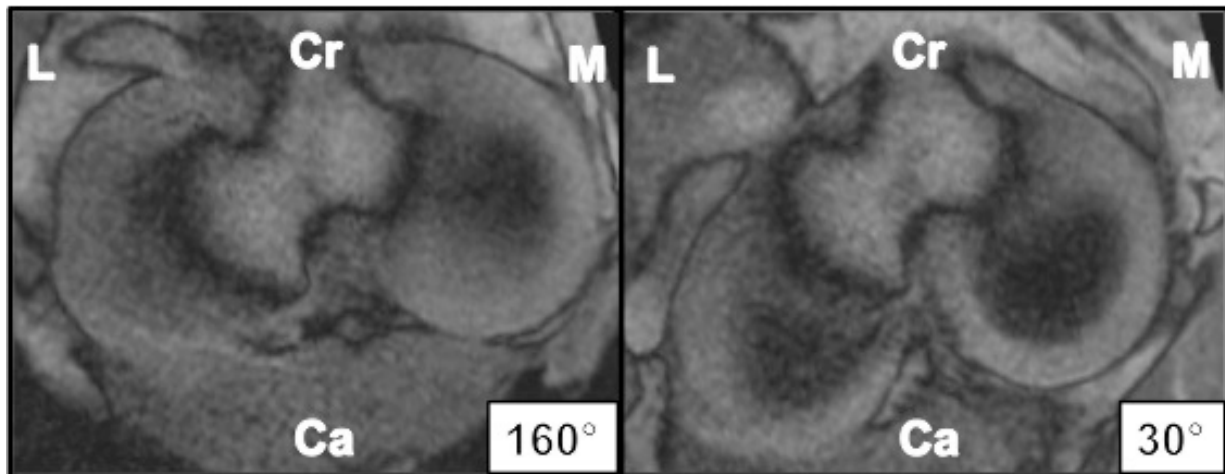
Figure 3.6 Transverse MR images (T1-weighted turbo-FLASH) through the proximal to distal midpoint of the meniscus at 160° (full extension) and 30° (full flexion). Cr; cranial, Ca; caudal, L; lateral, M; medial. Menisci change from a c-shape to a curved L (and inverted L) shape and a greater overall cranial-caudal translocation of the lateral meniscus relative to the medial is seen. Note similarity to figure 3.3.

Para-sagittal sections through the medial femoral condyle revealed an axial compression (decrease in height) and an increase in cranial translocation of the cranial horn of the medial meniscus relative to the cranial horn of the lateral meniscus between 150° and full extension (Figure 3.7). The resulting average compressive strain on the cranial meniscal horns between 150° and full extension (160°) was 0.27 ± 0.03 for the medial and 0.06 ± 0.06 for the lateral meniscus. Thus, the cranial horn of the medial meniscus experiences a significantly greater compressive strain relative to the cranial horn of the lateral meniscus ($p = 0.017$). The increase in cranial translocation and axial compression experienced by the cranial horn of the medial meniscus at full extension appears to occur in response to a change in the contour of the articulating surface of the distal femur (Figure 3.7). At full extension it appears that the femoral contact point with the cranial horn of the medial meniscus changes to a region with a greater radius of curvature, which corresponds to the anatomic location of the transition from the medial femoral condyle to the medial trochlear ridge. This change in contact surface geometry of the

femoral condyle resulted in an axial compression and cranial displacement of the cranial horn of the medial meniscus.
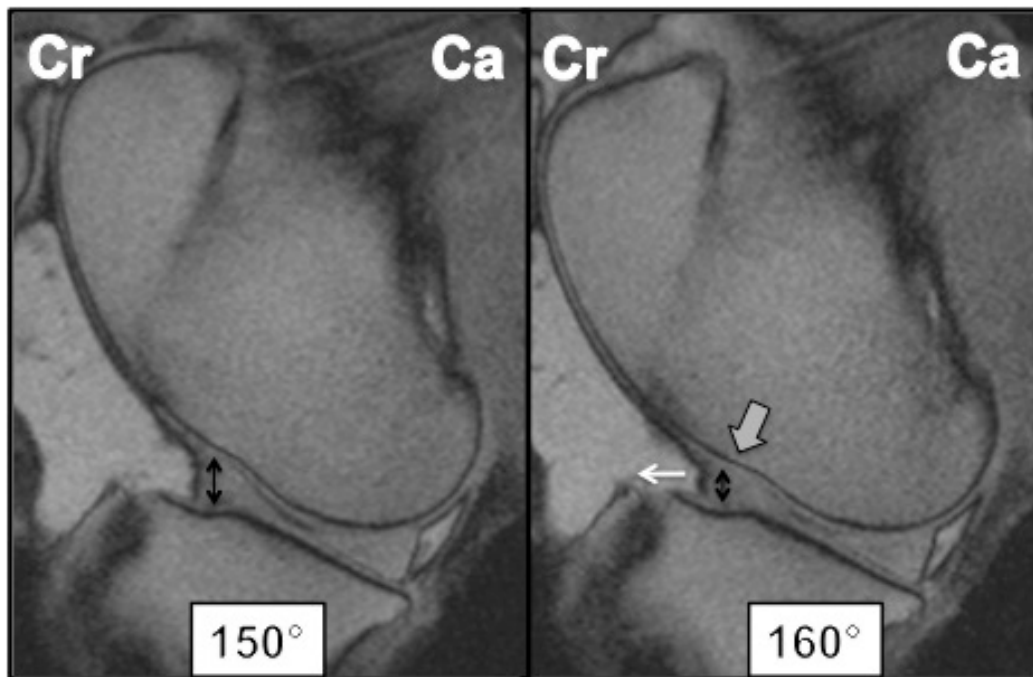
Figure 3.7 Parasagittal MRI images (T1-weighted turbo-FLASH) through the medial femoral condyle at 150 and 160° stifle angles. Cr; cranial, Ca; caudal. Proximal-distal compression (black arrows) and cranial displacement of the cranial horn (white arrow) seen at full extension (160°). At full extension the cranial horn of the medial meniscus articulates with the transition (grey arrow) from the medial femoral condyle to the femoral trochlear ridge where a visible change in radius of curvature occurs.

Discussion

The results of the current study demonstrate that the cranial-caudal translocation of the equine menisci during passive flexion and extension of the stifle joint is similar to that reported in man (17), with the lateral meniscus having a significantly greater overall cranial-caudal translocation relative to the medial. The cranial-caudal translational movements of the menisci were also associated with subtle changes in the overall shape of both the medial and lateral menisci. The ability of the menisci to change position and shape through the range of motion allows them to maintain congruency and equitable load transfer between the femoral and tibial

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articular surfaces.(18) The change in shape of the equine menisci is, in part, due to the fixed axial ligamentous attachments of the cranial and caudal horns, which appear to limit their translocation to a greater extent than the peripheral, abaxial attachments of the menisci.

In the human knee, the posterior horn of the medial meniscus was reported to be the least mobile of the 4 horns.(17) As the posterior horn of the human medial meniscus is the most common site of meniscal injury (19, 20), it has been theorized that, because of its relative immobility, the posterior horn of the human medial meniscus may become trapped between the medial femoral and tibial condyles during hyperflexion of the knee and consequently be subjected to excessive forces that could damage the meniscus.(17)

In the current study, the cranial horn of the medial meniscus of the horse was found to be the least mobile horn, and significant axial compression and cranial translocation of this horn relative to the cranial horn of the lateral meniscus was seen on full extension of the stifle. Thus, it is possible that the cranial horn of the medial meniscus may become trapped between the femoral and tibial condyles during hyperextension, leading to injury of this region. At full extension (160°) it was noted that the cranial horn of the medial meniscus comes into contact with the distal aspect of the medial femoral trochlear ridge. The transition from the smaller radius of curvature of the femoral condyle to the larger radius of curvature of the trochlear ridge resulted in compression and cranial translocation of the cranial horn of the medial meniscus during full stifle extension. While the medial femoral condyle is confluent proximally with the medial femoral trochlear ridge, there is a narrow ridge connecting the lateral condyle and trochlear ridge.(21) Thus, the contour of the lateral aspect of the distal femur does not appear to place the same constraints on the cranial horn of the lateral meniscus and therefore this area is not exposed to the same degree of axial compression or cranial displacement that was observed in the medial

meniscus.

Hyperextension of the stifle joint may lead to the development of pathological forces and injury in the cranial horn of the medial meniscus, similar to that observed in the human posterior medial horn upon hyperflexion.(3) The most commonly described meniscal injury in horses is an isolated longitudinal tear of the cranial medial meniscotibial ligament that extends into the cranial horn of the medial meniscus a variable distance.(10) Currently, it is unclear which forces play a role in the creation of this lesion. There are multiple forces that have the potential to be involved in injuring this region of the medial meniscus during hyperextension: compression (22) due to the contour of the medial distal femur, shearing (23) from cranial translocation of the meniscus, and tension (24) through the adjacent meniscotibial attachment when the horn is cranially displaced. However, the contribution of these forces, either singularly or in combination, to the creation of meniscal injuries in the horse has yet to be determined. As with other viscoelastic soft tissues, the degree, direction, rate and magnitude of the forces applied influence the occurrence and degree of clinical meniscal injury.(22, 25) Thus, a comprehensive understanding of the pathogenesis of isolated injury to the cranial horn of the medial meniscus in horses will require evaluation of the local forces experienced by the menisci during hyperextension.

In the current study, meniscal translocation data were obtained from passive, nonweightbearing range of motion in cadaver specimens with the musculotendinous supports of the stifle joint transected. As such, mechanical factors associated with weightbearing in the *in vivo* state such as hoop stresses and compression (22, 26, 28) were not taken into account. While the current *in vitro* test system represents a simplification of the *in vivo* state, the bony constraints of the tibial plateau and femoral condyles, as well as the static ligamentous stabilizers

of the stifle remain unchanged in the *in vitro* state. Therefore, the menisci must translocate cranially and caudally to adapt to the variation in the contour and the gliding/rolling motion of the femoral condyles. Similar findings have been reported in the human and canine knee. (17, 28)

In summary, the equine lateral meniscus was shown to have a greater overall cranial-caudal translocation over the tibia than the medial meniscus. The cranial horn of the medial meniscus was found to be the least mobile horn in the horse, although this horn undergoes approximately one-third of its total caudal-cranial translocation in the final 10° of extension. The imposed cranial translocation and axial compression of the cranial horn of the medial meniscus during this interval may be exacerbated during hyperextension. Thus, hyperextension of the stifle may lead to injury of the cranial horn of the medial meniscus and its attachment and thus explain the considerably higher prevalence of cranial medial vs. cranial lateral meniscal tears in horses.

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CHAPTER 4.

Stifle extension produces a significant difference in the tensile forces experienced by the abaxial and axial components of the cranial meniscotibial ligament of the equine medial meniscus: A mechanistic explanation for the reported injury patterns

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Abstract

The purpose of this study was to identify potential functional-anatomic characteristics of the cranial horn attachment of the medial meniscus (MM) that may help explain the pathogenesis of the common tear patterns that have been reported. The effect of femorotibial angle (160°, 150°, 140° and 130°) on tensile forces in the axial and abaxial components of the CrMTL was examined in six adult cadaver stifles using an implantable force probe. Additional specimens (n=3) were used to examine the histologic structure of the CrMTL and its connection to the cranial horn of the MM. Full extension of the stifle (160°) resulted in a significantly greater tensile force in the abaxial component of the CrMTL when compared to the axial component (p=0.001). The tensile force in the abaxial component of the CrMTL increased significantly between 150° and 160° of stifle extension (p=0.011). The CrMTL appears to be comprised of two functional components, which become more visually distinct as the stifle is extended. Histologically, these components are separated by a cleft of highly vascularized, less organized connective tissue, which becomes less prominent at the junction of the ligament and the cranial horn of the MM. In conclusion, a significant difference in tensile forces experienced by the two functional components of the CrMTL during hyperextension may generate shear forces, which, during weight bearing, may be sufficient to result in damage at this site. The functional anatomy of the CrMTL may place this region at greater risk of injury during hyperextension of the stifle and, therefore, may provide a mechanistic rationale for the commonly reported meniscal tear patterns in the horse.

Introduction

The most common arthroscopically diagnosed meniscal tears in horses involve the cranial horn of the medial meniscus (MM) and its associated cranial meniscotibial ligament (CrMTL).(1-3) Tears in the cranial horn of the MM are approximately 4 times more commonly diagnosed than similar tears in the cranial horn of the lateral meniscus.(2) Cranial horn meniscal tears are recognized in combination with multiple soft tissue injuries of the stifle, although they are commonly recognized as an isolated lesion, and therefore are suspected to be the primary cause of lameness in such cases.(2)

A characteristic orientation of equine cranial horn meniscal tears has been recognized and a grading system developed.(1, 2, 4) The grading system (grade 1-3) describes tears extending longitudinally down the CrMTL into the cranial horn of the meniscus, with the higher grade tears involving greater separation of tissues and further extension into the cranial horn(4). However, the anatomic origin of these tears and the mechanism(s) by which they occur is unknown. As tears have been documented to be present solely within the CrMTL, and may extend variable distances into the cranial horn meniscal tissue, the meniscotibial ligament, itself, may be a potential site for the initiation of the tear.

Evaluation of meniscal translocation through the range of motion has lead to a greater understanding of the pathogenesis of medial meniscal tears in humans.(5) A recent study evaluating meniscal translocation through the range of motion of the equine stifle joint, found that the cranial horn of the MM is the least mobile of the four meniscal horns.(6) However, during the terminal 10° of extension, it was noted that the cranial horn of the MM undergoes cranial displacement and axial compression to a greater extent than cranial horn of the lateral meniscus.(6)

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Evaluation of the forces that develop within the meniscal tissues during limb loading has been completed in other species.(7-10) An experimental analysis of forces occurring within a finite element model of a human meniscus under compressive load revealed a marked difference in the circumferential stain between the axial and abaxial components of the meniscus.(10) It was suggested that this difference in strain magnitude between the inner and peripheral portions of the meniscus during high compressive loads could be a mechanism for meniscal injury.(10) It is possible that, during hyperextension of the stifle, significant differences in tensile forces may develop within the CrMTL of the equine MM leading to the characteristic tear patterns that have been reported.

Therefore, the objective of this study was to evaluate the tensile force patterns that exist within the CrMTL of the MM through the range of motion in an effort to provide a possible mechanistic explanation for the pathogenesis of commonly described tear patterns. In addition, the gross and microscopic structure of the CrMTL was examined to determine if there is a functional anatomic predisposition for these tear locations. It was hypothesized that, during full extension of the stifle, significantly higher tensile forces are generated within the abaxial (peripheral) component of CrMTL when compared to the axial (inner) component of the ligament. It was also hypothesized that the normal functional anatomic structure of the CrMTL predisposes this structure to injury at the reported site.

Materials and Methods

In a previous study, which evaluated meniscal translocation through the range of motion of the equine stifle (6), a consistent observation of the CrMTL of the MM was made. A shallow longitudinal cleft in the proximal surface of the medial CrMTL became apparent as the joint was

moved towards full extension (from 120-160 degrees), with depth of the cleft increasing with increasing stifle angle. The cleft appeared to longitudinally divide the ligament into approximately equally sized abaxial and axial components (figure 4.1). Assessment of this visibly apparent longitudinal cleft was completed in the present study through evaluation of the tensile forces and anatomic characteristics of the CrMTL of the MM.
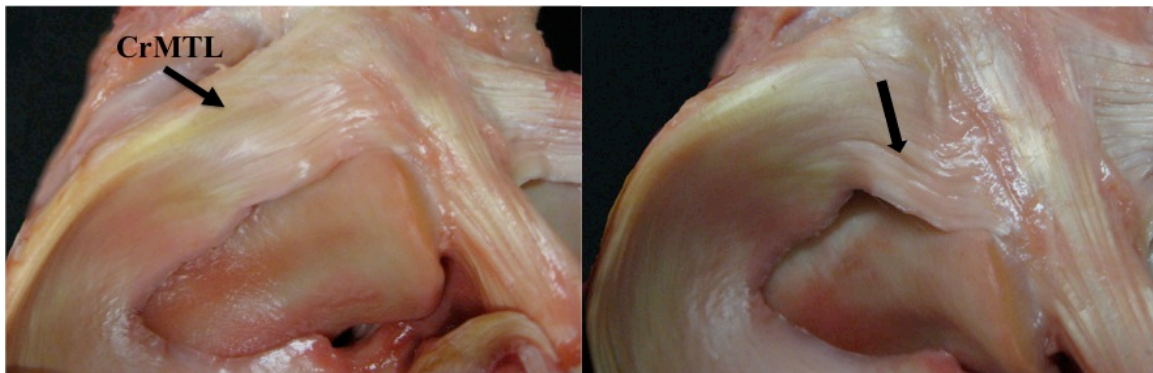
Figure 4.1 (Left) Proximal to distal view of the MM and it's CrMTL, simulating the position of the meniscal tissues at a stifle joint angle of ~100°-110°. (Right) Longitudinal cleft (arrow) appears in the CrMTL of the MM when the MM is moved cranially simulating the position of the meniscal tissues at a stifle angle of ~160°.

Cadaveric equine stifles utilized in all aspects of the study were harvested from horses euthanized for reasons unrelated to the study. All specimens were free of visible stifle joint pathology upon dissection and had no known history of lameness localized to the stifle.

Evaluation of tensile forces in the abaxial and axial components of the CrMTL of the MM

Cadaveric equine stifles (4 left and 2 right limbs) were harvested from six mature light breed horses (3 Quarter horses, 2 Thoroughbreds and 1 Warmblood). The horses represented a range of ages (2-16 years; mean 9.5 years), and both genders (2 geldings and 4 mares). Limbs were removed at the level of the proximal femur and distal tibia within 8 hours of euthanasia. All

soft tissues were removed with the exception of the menisci, and the collateral, cruciate and meniscal ligaments. The cadaver stifles were stored at – 20°C and thawed at room temperature for approximately 48 hours prior to study.

The femurs and tibias were potted in epoxy resin to provide stability upon placement in the cylinders of a custom aluminum testing fixture. The jig was designed to secure the specimens at a femorotibial angle of 160°, 150°, 140° and 130°. These angles were chosen to evaluate contact forces through the weight bearing angles. Previous literature documented the equine standing caudal joint angle to be approximately 150° (11) and to vary between approximately 160-140° during the weight-bearing segment of the gait cycle at a walk and trot.(12, 13) Measurements from a slow motion video of a racehorse at a gallop indicated foot strike to occur at a stifle angle of approximately 130° and limb protraction at approximately 145° (unpublished results).

Tensile forces within the abaxial and axial components of the CrMTL of the MM were measured with Arthroscopically Implantable Force Probes (AIFP6) (MicroStrain Inc., Williston, VT, USA). The probes (~1.4mm x 1.8mm x 5mm) use a half bridge semiconductor strain gauge (460 ohm) bonded to a steel probe to convert tensile forces into an electrical current. Implanted into the ligament, the walls of the probe are subjected to a compressive force when the ligament fiber bundles are subjected to tensile forces. A linear relationship was established between voltage output (volts) and force (newtons), with calibration up to 30N force. Arthroscopically implantable force probes were fully embedded vertically into the approximate middle of both the abaxial and axial components of the CrMTL of the MM using a knife insertion tool (figure 4.2). The probes were connected to a compensated signal conditioning board (DEMOD-AIFP, MicroStrain Inc., Williston, VT, USA), and a data acquisition system (LABTECH, Omega

Engineering Inc, Stamford, CT, USA) was used for collection of voltage data at 50Hz for 30

seconds/trial and converted to force data (Newtons) using a calibration curve.
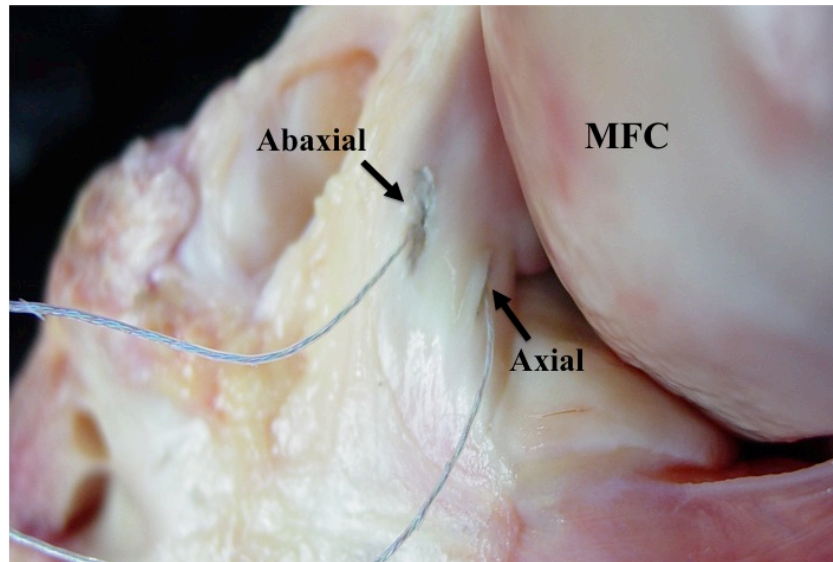
Figure 4.2 Image of the CrMTL of the MM with the AIFP's inserted into the abaxial and axial
components. MFC; medial femoral condyle, Cr; cranial aspect of the joint

Force data was collected simultaneously from the abaxial and axial components of the

CrMTL of the MM while a standard nominal load (6.8kg) was applied. This was the maximum

load that could be applied while staying within the calibration limits (30N) of the AIFP in trial

specimens. Testing was completed at the four stifle angles (160°, 150°, 140° and 130°), with the

progression of testing from 160° to 130° or 130° to 160° assigned randomly. The mean tensile

force within the ligament components was calculated over a continuous span of ten seconds of

data collection with minimal noise. Testing was completed in triplicate for each angle and

averaged. The experimental protocol was repeated with all specimens (n=6).

Histologic evaluation of the CrMTL and adjacent cranial horn of the MM

Stifle joints (n=3) were collected from fresh cadavers within 30 minutes of euthanasia. Cadaveric stifles (2 left and 1 right) were harvested from light breed horses, representing a range of ages (5-28 years; mean 13) and both genders (2 geldings and 1 mare). Immediate dissection involved removal of the femur and securing of the MM to the tibial plateau with a loop of suture material (to prevent contraction of the tissues). After at minimum of 72 hours formalin (10%) fixation the MM and associated CrMTL were removed and transected in longitudinal (transverse) section and cross (sagittal/frontal) sections. The histological specimens were embedded in paraffin and 5-μm thick sections were cut, and stained with hematoxylin and eosin (H&E) or Safranin-O stains. Slides were examined under light and polarized light microscopy for evaluation of tissue microstructure.

Statistical Analysis

A repeated measure ANOVA with Bonferroni Post-Hoc tests was used to evaluate for significant differences between tensile forces in the abaxial and axial components at the four stifle angles, and to evaluate for significant differences between the abaxial and axial components within each stifle angle.

Results

Evaluation of tensile forces in the abaxial and axial components of the CrMTL of the MM

The magnitude of tensile force measured in the CrMTL of the MM was found to change with stifle angle (figure 4.3). The greatest tensile force was recorded in the abaxial component of CrMTL at 160° (full extension); this value was significantly greater than the tensile force in the

abaxial component at 150° (p=0.011) and 140° (p=0.037). There were no significant differences in the tensile force in the axial components between the 4 stifle angles measured.
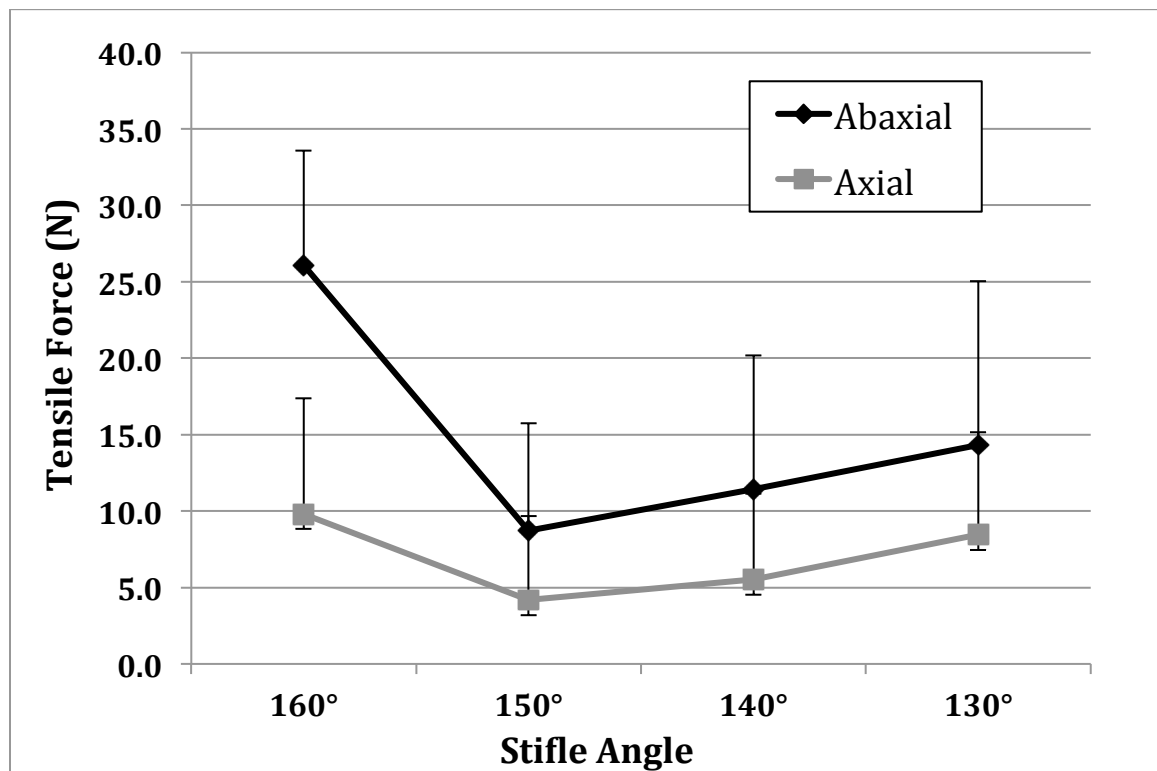
Figure 4.3 Average (n=6) tensile force (newtons) recorded in the abaxial and axial components of the CrMT of the MM at the four weight-bearing stifle angles during limb loading.

The abaxial component of the CrMT ligament of the MM experienced a greater tensile force than the axial component at all the stifle angles (figure 4.3). The abaxial component of the CrMTL of the MM had a significantly greater (p=0.001) tensile force than the axial component upon full extension of the stifle (160°). There was, on average, a 4.1 fold greater tensile force in the abaxial component compared to the axial component at 160°. Although the abaxial component of the CrMT ligament of the MM had on average a greater mean tensile force of 4.5, 5.9 and 5.9 N compared to the axial component at 150°, 140° and 130° respectively (figure 4.3), the differences were not statistically significant (p>0.05). Thus, the difference in tensile forces

between the abaxial and axial components was greatest at 160° and was significantly greater than all other angles ($p < 0.05$) (figure 4.4).
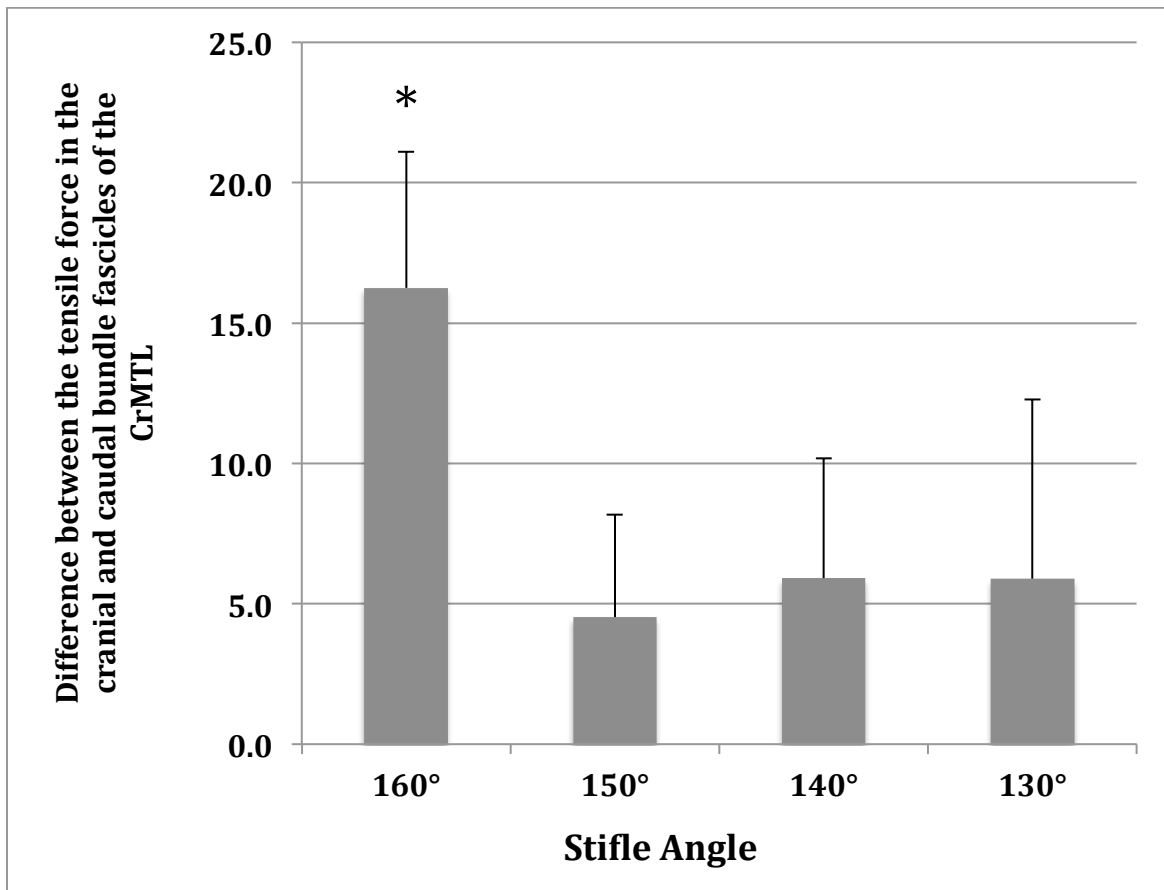
Figure 4.4 Average (n=6) difference in tensile force (newtons) between the abaxial and axial components of the CrMT of the MM at the four stifle weight-bearing stifle angles during limb loading. Significant difference from all other angles indicated by (*).

The lowest magnitude of average tensile force, and the smallest average difference between the abaxial and axial components in the CrMT ligament of the MM occurred at 150°, which corresponds to the reported standing angle of the equine stifle joint.

Histologic evaluation of the CrMTL and adjacent cranial horn of the MM

Transverse and coronal sections of the cranial horn and CrMTL of the MM reveal that the collagen bundles are predominantly oriented in a parallel circumferential pattern, similar to that seen in other species. A longitudinal, central region of highly vascularized, less organized connective tissue was observed to separate the collagen fibers in the CrMTL of the MM into abaxial and axial components (figure 4.5b-d). This longitudinal region of less organized connective tissue was at the approximate cranial-caudal midpoint of the ligament and was wide enough to be evident macroscopically (figure 4.5a). The prominent region of highly vascularized, less organized connective tissue appears to correspond with the location and orientation of the grossly visible cleft seen in the proximal surface of the intact ligament (figure 4.1). Microscopically, this longitudinal region of less organized connective tissue in the meniscotibial ligament extends down the length of the meniscotibial ligament and becomes less prominent at the junction of the ligament and the cranial horn of the MM.
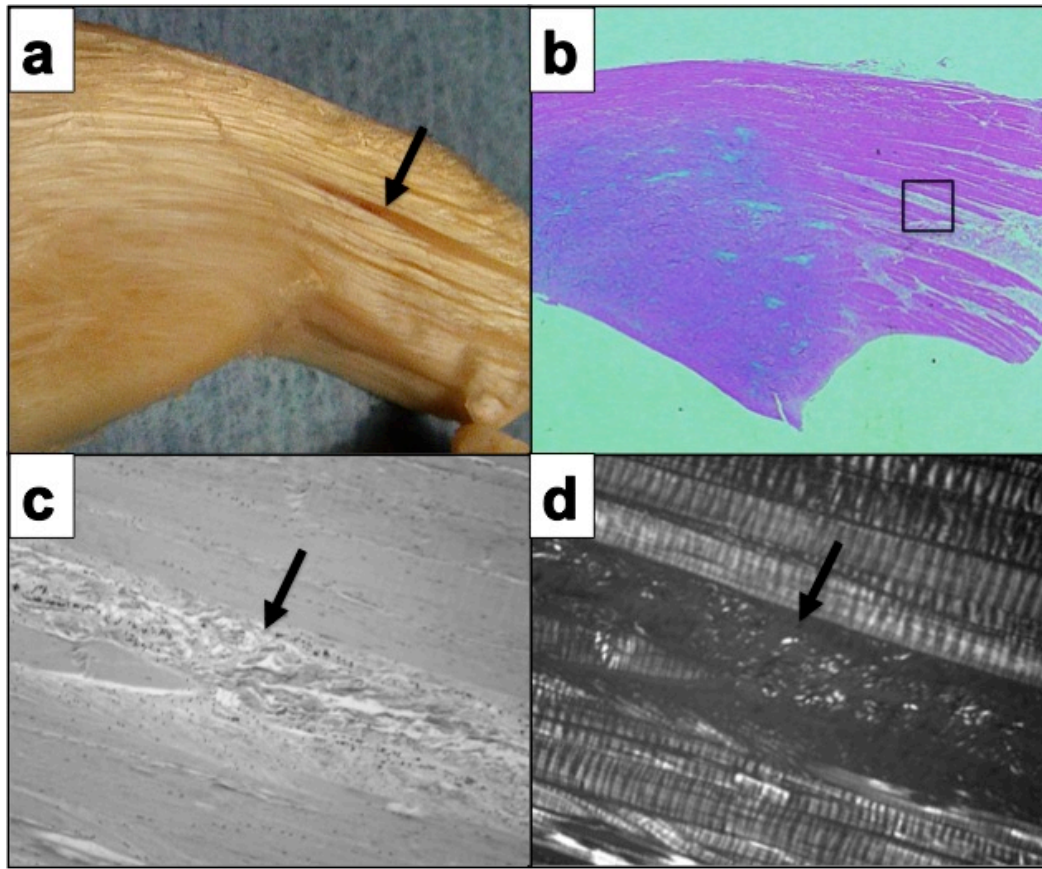
Figure 4.5 (a) CrMTL and cranial horn of the MM from a 6 year old QH gelding, cut in transverse section following formalin fixation, showing the grossly visible longitudinal region of connective tissue dividing the CrMTL into abaxial and axial components. H&E stained sections of the same region at 1X magnification (b) and 50X magnification (c) showing the longitudinal region to be composed of highly vascularized, less organized connective tissue, between parallel collagen fiber bundles. (d) Polarized light microscopy highlighting the typical banding pattern of the collagen fibrils and the collagen fiber void longitudinal region of less organized connective tissue.

Discussion

Compression of the menisci during load bearing leads to the development of circumferential tensile forces within the meniscal tissue, a phenomenon known as "hoop stress".(7,14) The wedge shape and near frictionless surface of the meniscus results in the development of a radial extrusion force during vertical loading, which is resisted by tethering of the cranial and caudal horns of the meniscus to the tibia by the meniscotibial ligaments.(7) A

recent study documented that the cranial horn of the equine MM experiences significant axial compression and cranial displacement upon full extension of the stifle joint.(6) The geometry of the medial aspect of the distal femur appears to lead to the cranial extrusion and compression of the cranial horn of the MM at full extension, and the abaxial component of the cranial horn and it's adjacent ligamentous attachment appeared to be more significantly compressed than the axial component.(6) This likely leads to an increase in circumferential tensile forces in the cranial horn of the MM in this abaxial component. Cranial displacement of the cranial horn of the MM is counteracted by the CrMTL, which limits the translation of the cranial horn of the MM relative to the tibia. Alteration in meniscal position during flexion and extension of the femorotibial joint has been shown to alter the tensile forces within the meniscotibial ligaments.(15, 16) Thus, an increase in the tensile forces in the CrMTL was anticipated with hyperextension of the equine stifle joint based on the compression and cranial displacement of the cranial horn of the MM seen in the previous study.(6)

The results of the current study quantitatively demonstrate that full extension of the stifle (160°) leads to a significant increase in the tensile forces in the abaxial component of the CrMTL of the MM compared to 150°. The lowest overall tensile forces in the CrMTL (and the smallest difference between the abaxial and axial components) was seen at the reported standing stifle angle (150°); indicating that CrMTL experiences minimal tensile force while standing at rest. The development of excessive tensile forces within the CrMTL during hyperextension may play a role in cranial horn injury in horses. Tensile studies on meniscal tissue have shown a consistent pattern of failure of the circumferential collagen bundles, such that excessive circumferential tensile forces lead to shearing along the mechanically weak planes between collagen bundles.(8)

Full extension of the equine stifle resulted in the development of a differential in tensile forces within the CrMTL in addition to the highest overall magnitude of tensile force. The appearance of a shallow cleft between the abaxial and axial components of the CrMTL of the MM during extension, alluded to the potential for a differential in tensile forces to be developing within the ligament. At a stifle angle of 160°, a 4.1 fold greater tensile force was measured in the abaxial component compared to the axial on average, whereas a significant difference between the abaxial and axial components was not seen at any other angle evaluated. Differential tensile forces within the CrMTL during stifle hyperextension may increase the magnitude of shear forces that develop between the abaxial and axial components when the CrMTL is placed under tensile forces. Differential ligamentous strain has been recognized in the canine cruciate ligaments, as each cruciate ligament is composed of two parts that undergo reciprocal loosening and tightening dependent on joint positioning.(17) Understanding of the functional mechanics of the cruciate ligaments, through evaluation of the associations between joint angle and tensile forces in the components of the cruciate ligaments, has allowed for a greater understanding of the mechanisms of cruciate ligament injuries.(18) Similarly, experimental evaluation of the human scapholunate interosseous ligament revealed a relationship between differential regional strain within the ligament and joint position during loading; thus leading to greater understanding of the pathogenesis of ligament injury.(19) The development of a significant differential in tensile forces and the highest overall magnitude of tensile force in the CrMTL of the MM at 160°, indicates the potential for shearing forces to develop resulting in injury to the cranial horn attachment with hyperextension of the femorotibial joint in the horse.

Histologic evaluation of the CrMTL and the cranial horn of the MM in the current study revealed a potential microanatomic predisposition for the characteristic cranial horn meniscal

tears in the horse. The CrMTL of the MM, composed primarily of parallel circumferentially aligned collagen fibers, was found to be anatomically divided into abaxial and axial components by a longitudinal region of highly vascularized less organized connective tissue in the cadaver specimens. This longitudinal region of connective tissue corresponded in location and orientation to the shallow cleft that developed in the proximal surface of the CrMTL of the MM during the final 40° of extension. Research has shown that tears in collagenous based tissues generally initiate in weaker areas of the extracellular matrix (less organized connective tissue), and that the collagen fibers act to deflect the direction of the propagating tear between collagen fibers.(20) As a result of this phenomenon, tears that are parallel to the collagen fibers (*ie.* longitudinal and horizontal tears), are more common in the human knee than tears that are perpendicular to the collagen fibers.(21) Equine cranial horn meniscal tears are documented to run longitudinally down the middle of the CrMTL in a vertical plane (1), which appears to be similar to the location and orientation of the prominent longitudinal region of less organized connective tissue seen on histology. Therefore, the longitudinal region of connective tissue in the CrMTL may result in a structural weakness, relative to the tensile resistant collagen fibers of the abaxial and axial components, and may represent a location from which tears originate or extend through. Histologic descriptions of human and canine meniscal tibial ligaments do not document a longitudinal region of vascularized, less organized connective tissue such as that seen in the horse.(22, 23) Further evaluation of this vascularized region is indicated to determine if it is present congenitally or if it develops overtime, potentially as a result of variable tensile forces acting on the two regions or progressive invasion and development of the vascular supply to the CrMTL's and cranial horn of the MM.

Interestingly, a previous study evaluated the viscoelastic shear properties of the equine meniscus and found that the shear modulus of the meniscal tissue was lower in the cranial horn compared to the caudal horn, indicating that the a cranial horn may be less resistant to shear forces.(24) Thus, further evidence exists that the cranial horn of the medial meniscus may be predisposed to injury in the horse.

Arthroscopically Implantable Force Probes (MicroStrain Inc., Williston, VT, USA) allow for quantitative evaluation of intra-ligamentous tensile forces with minimal tissue disruption, and thus can even be used for in vivo experiments.(25) Several studies have documented the susceptibility of AIFP to variable outputs over time with cyclic loading, and with removal and replacement of the probe.(26, 27) To minimize error in the present study, the order of completion from 160-130 degrees was chosen randomly, abaxial and axial component readings were made simultaneously, three readings were averaged for each value and probes were not removed and replaced within a specimens testing period.

In the current study, the force applied to the cadaver specimens was limited by the maximum voltage output of the AIFP. As a result, a physiologic load equivalent to that experienced during weight bearing could not be used. However, the results of this study focused on the relative, as opposed to absolute, force within the ligament. A previous study found that the compressive component of the loading force is balanced by the tensile stresses that develop in the circumferentially oriented fiber bundles (28), and thus an increase in vertical load (compressive force) would be expected to result in a proportional increase in tensile forces.

In conclusion, the results of the present study indicate that there is a potential anatomic and functional rationale for the described cranial horn tears of the equine MM. Multiple factors may play a role in the development of injury to this region, including structural inhomogeneity

and variation in the forces acting on the region with joint angle. The potential for shear forces to develop along a mechanically weaker longitudinal region of less organized connective tissue between the abaxial and axial components of the CrMTL of the MM provides rationale for the location, orientation and incidence of cranial horn tears of the MM.

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CHAPTER 5.

Grade III cranial horn tears of the equine medial meniscus alter the contact forces on the articular surface of the medial tibial condyle

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Abstract

The purpose of this study was to evaluate the magnitude and distribution of contact forces on the medial tibial condyle after the creation and resection of grade III cranial horn tears of the equine medial meniscus. Peak pressure and contact area on the tibial plateau was recorded following application of a vertical load (1800N) to cadaveric stifles (n=6) at 130°, 140°, 150° and 160°. Testing was repeated following surgical creation of a grade III cranial horn tear of the medial meniscus, and after resection of the simulated tear. In the intact specimens, a significantly smaller tibial contact area was observed at a stifle angle of 160° compared to 150°, 140°, and 130° (p=0.005, 0.010 and 0.036 respectively). Creation of a grade III cranial horn tear in the medial meniscus did not significantly alter the pressure or contact area measurements at any stifle angle compared to the intact specimens (p>0.05). Resection of the simulated tear resulted in a significant increase in peak pressure in the central region of the medial tibial condyle at a stifle angle of 160° relative to the intact (p=0.026) and torn specimens (p=0.012). In conclusion, resection of grade III cranial horn tears in the medial meniscus resulted in a central focal region of increased pressure in medial tibial condyle at 160° stifle angle. Altered loading of the tibial condyle as a result of grade three meniscal tears may contribute to secondary osteoarthritis in the horse, and restoration of normal meniscal geometry may limit these degenerative changes.

Introduction

The menisci are c-shaped discs of fibrocartilage interposed between the condyles of the femur and tibia (1). Their functions have been described as providing equitable load transmission, shock absorption, joint stability, joint lubrication, and proprioception (2-5), and numerous studies in man and animals have documented the degenerative changes in the femorotibial joint which occur following damage or removal of meniscal tissue.(6-8)

Loss of meniscal function in load distribution secondary to a meniscal tear has been shown to lead to an increase in focal pressure in the central region of the tibial plateau in humans and dogs.(9-13) Cartilage injury subsequent to meniscal release or meniscectomy in these species has been identified in the central regions of the tibial and femoral condyles, and appears to correspond to the central region of increased pressure seen experimentally.(6,14) Partial or complete meniscectomy has been used as an experimental model to predictably create osteoarthritis (7,15), and has been shown to lead to degenerative joint disease in human clinical patients.(8)

Meniscal tears have been reported as the most common soft tissue injury in the equine stifle.(16,17) The most common location of meniscal injury in horses, as identified by arthroscopy, is a lesion extending longitudinally through the cranial meniscotibial ligament and into the cranial horn of the meniscus; and cranial tears of the medial meniscus have been reported to occur 4 times more commonly than cranial horn tears of the lateral meniscus.(17-19) A grading system was developed for cranial horn meniscal lesions: grade I lesions have no significant separation of tissues, grade II have a greater separation of the injured tissue, and grade III lesions defined as severe tears that extend beneath the femoral condyle and whose limit can not be seen arthroscopically.(19)

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The prognosis for return to athletic function in horses with cranial horn tears precipitously declines with the grade of lesion, with reports of 63%, 56% and 6% with grade I, II, and III tears respectively.(19) Articular cartilage injury of the medial femoral condyle was noted on arthroscopy in 71% of cases of meniscal tears at the time of diagnosis, and the outcome was significantly worse if there was concurrent articular cartilage disease.(17) Thus, limiting the development of osteoarthritis secondary to meniscal tears is important for maximizing a horses athletic potential.

Currently, there are no published studies evaluating the effect of meniscal tears on force transmission through the femorotibial joint in horses. Additionally the effect of partial meniscal resection on force transmission is unknown, although this is the predominant technique used for treatment of equine meniscal tears presently.(17, 20) The purpose of the current study was to evaluate the effect of grade III cranial horn tears of the equine medial meniscus (pre- and post-resection) on the pressure magnitude and distribution over the articular surface of the medial tibial condyle. We hypothesized that simulated grade III meniscal tears would lead to the development of an area of increased pressure in the central region of the medial tibial condyle throughout the weight bearing stifle angles, and that resection of the tear would further increase the magnitude of pressure in this region.

Materials and Methods

Cadaveric equine stifles (3 left and 3 right limbs) were harvested from six mature light breed horses (4 Quarter horses and 2 Thoroughbreds) humanely euthanized for reasons unrelated to this study. The specimens had no known history of lameness localized to the stifle joint, and represented a range of ages (4-24 years; mean 12.5 years), and both genders (3 geldings and 3

mares). Limbs were removed at the level of the proximal femur and distal tibia within 8 hours of euthanasia. All soft tissues were removed with the exception of the menisci, and the collateral, cruciate and meniscal ligaments. The cadaver stifles utilized in the study had no grossly visible joint pathology. Limbs were stored at – 20°C and thawed at room temperature for approximately 48 hours prior to study.

The femurs and tibias were potted in an epoxy resin to provide stability upon placement in the cylinders of a custom aluminum testing fixture (Figure 5.1). The fixture was designed to secure the specimens at a stifle angle of 160°, 150°, 140° and 130°, thus allowing for a vertical force to be applied at each angle. These angles were chosen to evaluate contact forces through the weight bearing angles. Previous literature documented the equine standing caudal joint angle to be approximately 150° (1) and to vary between approximately 160-140° during the weight-bearing segment of the gait cycle at a walk and trot.(21, 22) Measurements from a slow motion video of a racehorse at a gallop indicated foot strike to occur at approximately 130° stifle angle and limb protraction at approximately 145° (unpublished results).

Figure 5.1 Cadaver stifle loaded in the custom aluminum testing fixture at 150° stifle angle.

Digital electronic pressure sensors (I-Scan Sensor Model 5051,Tekscan Inc, South Boston, MA, USA) were equilibrated using Tekscan-provided equipment. A 3-point equilibration at 80 psi, 90 psi and 100 psi was performed using an air-pressure equilibration device and subsequently conditioned and calibrated using the calibration software. The calibration method utilized the 2-point power method, which has been shown to have low error.(23) The calibration sequence involved loading the sensor between two flat polycarbonate plates linearly to 200N and unloading linearly to 10N three consecutive times.

The testing fixture and specimen were secured in the 858 Mini Bionix® II testing system (MTS Systems Corporation, Eden Prairie, MN, USA). The I-Scan sensor was then inserted between the medial meniscus and the medial tibial condyle following transection of the medial collateral ligament. A vertical load of 1800N, corresponding to the peak ground reaction force on the hindlimb of an average light breed horse (~450 kg) during a walking gait was applied. Limbs were loaded at 200N/s until 1800N was reached, held constant at this force for 5 seconds, prior to unloading at 200N/s. Using the Tekscan software (Tekscan Inc, South Boston, MA, USA), pressure data maps were collected through the loading cycle for quantitative evaluation at the time point in which the maximum peak pressure was reached.

Pressure data maps were collected at stifle angles of 160°, 150°, 140° and 130° for the intact specimen, with the order of progression from 160°-130° or 130°-160° assigned randomly. A simulated Grade III tear of the cranial horn of the medial meniscus (Figure 5.2) was then created with a #15 scalpel blade. A longitudinal incision extending down the middle of the cranial meniscotibial ligament was extended as far as possible into the cranial horn (~0.75-1cm, because of restricted access under the femoral condyle) and exited out its axial aspect. Pressure data collection was repeated for the four stifle angles. The simulated tear was then resected by

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transecting the portion of the meniscotibial ligament involved in the simulated tear at its attachment to the tibia and removing the "torn" ligament and cranial horn tissue before testing was repeated.
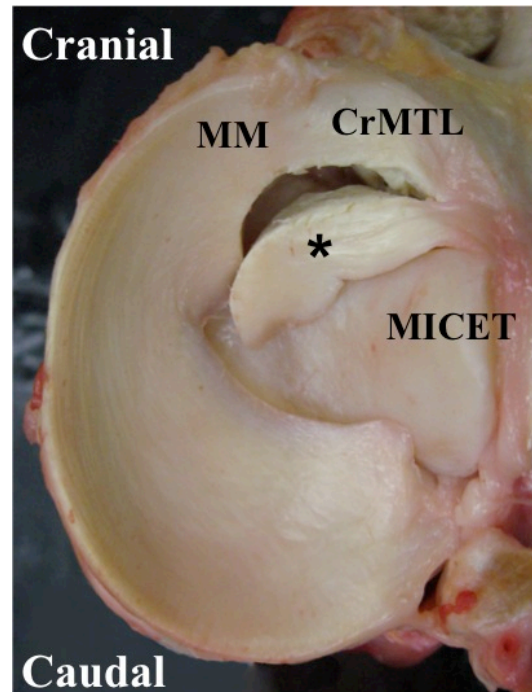
Figure 5.2 The medial meniscus of a right cadaveric stifle illustrating a simulated grade III meniscal tear (*). The tear extends longitudinally through the cranial meniscotibial ligament (CrMTL) into the cranial horn of the medial meniscus (MM). Medial intercondylar eminence of the tibia (MICET).

Pressure data collected included peak pressure (PSI), and contact area (mm^2). Contact points of less than 30 PSI were not included in the contact area calculations to remove the effects of mild background noise and regions of very minimal contact. Post processing of the data was completed to define the central region of the tibial condyle that is not covered by the meniscus, using an overlay of a digital photograph. A standardized sized region of fixed dimensions was used for evaluation of peak pressure in this central region of the tibial condyle.

A repeated measure ANOVA with Bonferroni Post-Hoc tests was used to evaluate for significant differences between the intact, torn and resected treatment groups for peak pressure and contact area for the entire joint contact area, and for peak pressure within the central area of the joint not covered by the meniscus at the four stifle angles. Significance was set at $p<0.05$.

Results

Evaluation of the pressure maps from the intact cadaver specimens revealed a relatively diffuse even distribution of pressure over the medial tibial condyle, with minimal pressure in the central region of the condyle (Figure 5.3). There were no significant differences in the peak pressures measured between flexion angles in the intact specimens ($p>0.05$). A trend was noted in all specimens such that the regions of higher magnitude force appeared to migrate from a more caudal location on the tibial condyle at 130° stifle angle to a more cranial location at 160° (Figure 5.3).
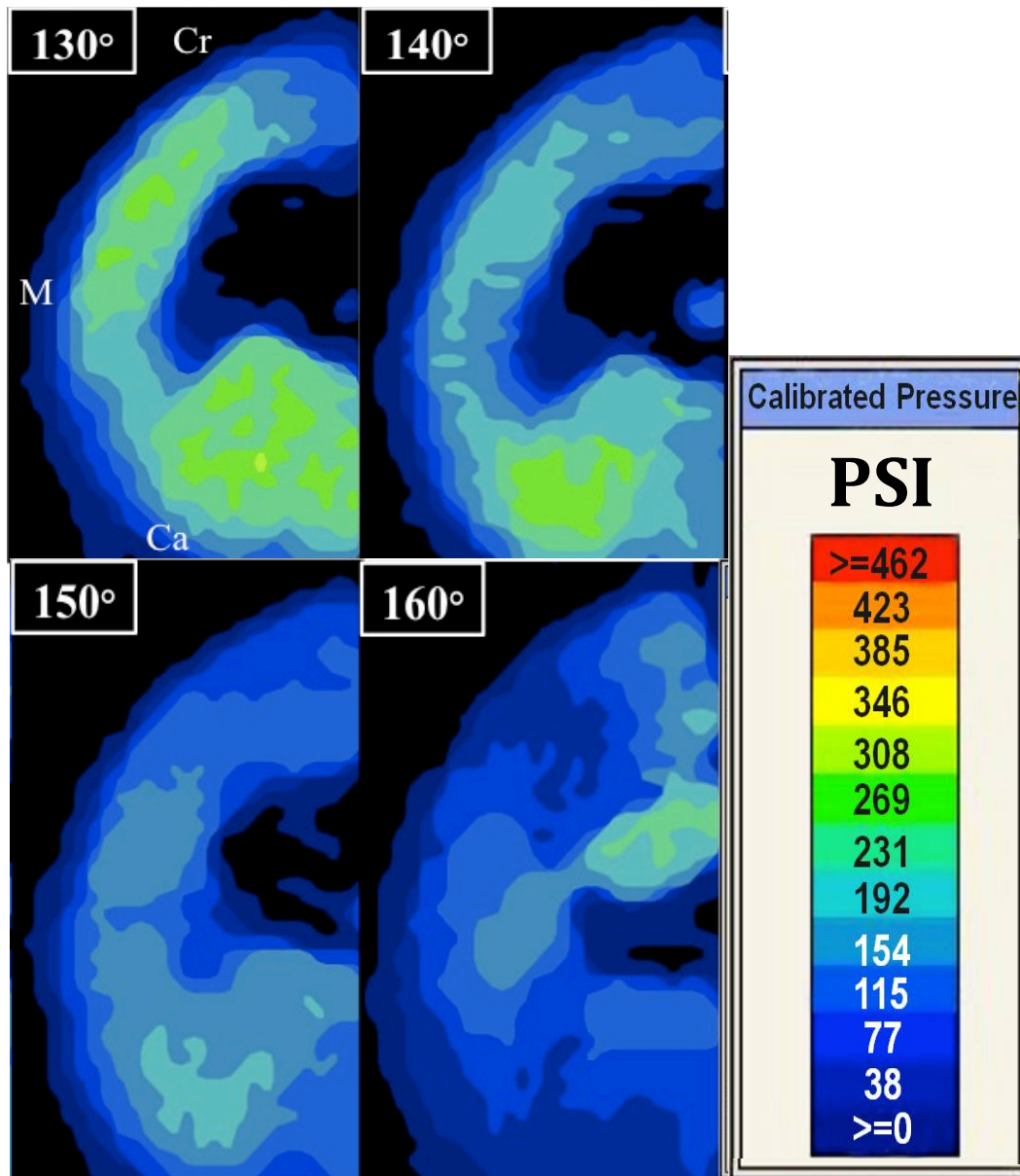
Figure 5.3 Contact maps obtained with Tekscan software of a cadaveric stifle with an intact medial meniscus. The pressure distribution and magnitude (see scale) on the medial tibial condyle are illustrated at stifle angles of 130°-160°. Cranial (Cr), medial (M), and caudal (Ca).

The contact area in the intact specimens (Figure 5.4) was significantly lower at 160° compared to 150°, 140°, and 130° (p= 0.005, 0.010 and 0.036 respectively). In 2/6 specimens, an extreme reduction in contact area was seen at 160° stifle angle, with force tending to localize centrally and cranially.
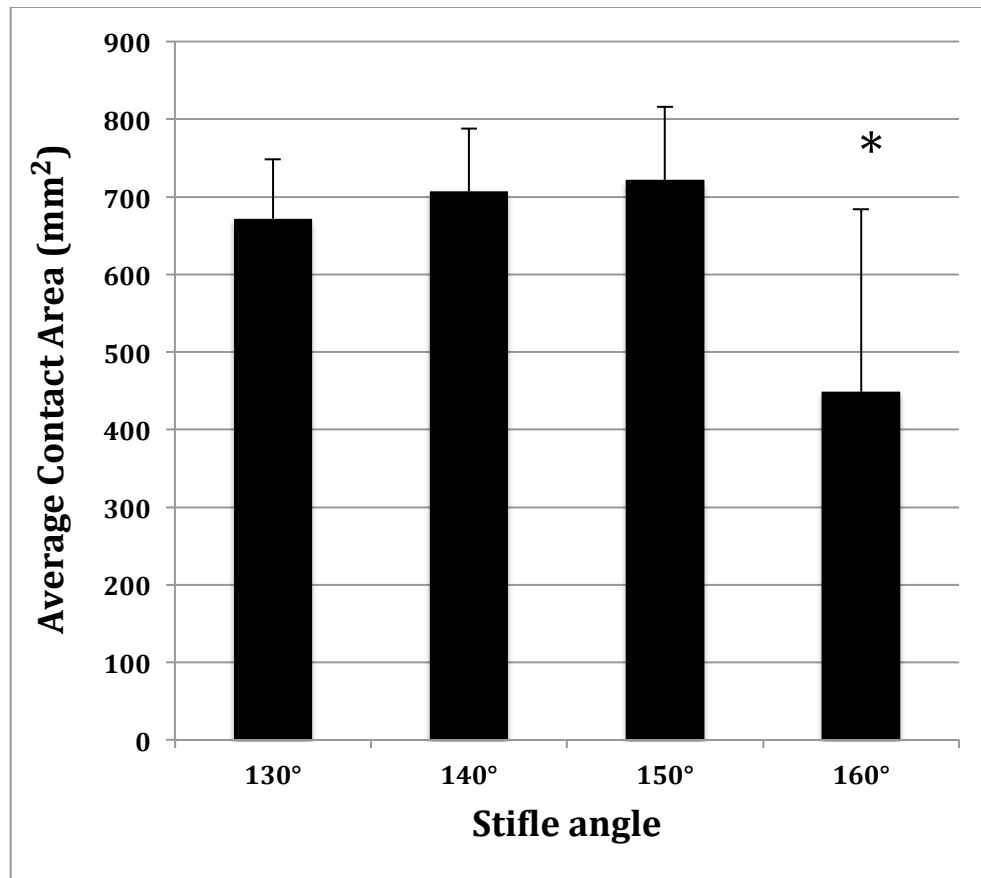
Figure 5.4 Average (n=6) contact area (mm^2) on the medial tibial condyle at the four stifle angles (160°, 150°, 140° and 130°) for the the intact specimens. Significant difference from all other values noted (*).

Pressure data collected following the creation of a simulated grade III cranial horn medial meniscus tear, revealed that there was no significant difference in peak pressure compared to the intact specimens at all four stifle angles. Therefore, there were no significant differences in peak pressures when evaluating the entire tibial condyle or the central region of the tibial condyle. Additionally there were no significant differences in contact area. During loading the simulated torn tissue appeared to remain in place and did not show obvious displacement.

Following resection of the simulated grade III tears, a significant increase in peak pressure was seen at 160° in the central region of the medial tibial condyle compared to the intact

(p=0.026) and torn specimens (p = 0.012). (Figure 5.5 and Figure 5.6). The peak pressure in the

resected specimens increased by 1.3 fold on average (range 1.05-1.6) relative to the torn

specimens. There was no significant differences seen in the peak pressures measured in the

central region of the medial tibial condyle at 150°, 140° and 130°. When evaluating the peak

pressure and contact area for the entire tibial condyle there was no significant differences seen

between the three experimental treatments (normal, resected, torn) at any angles (p = >0.05).
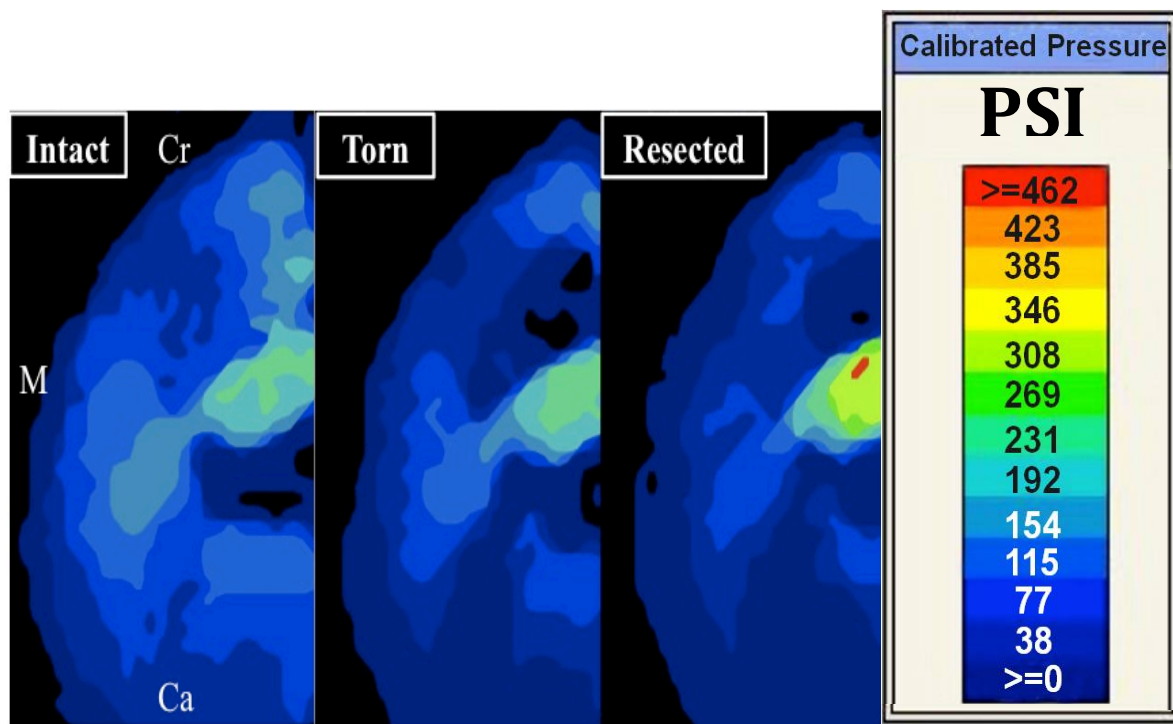
Figure 5.5 Representative contact maps of a cadaveric stifle illustrating the pressure distribution and magnitude on the medial tibial condyle at 160° with an intact medial meniscus meniscus (left), simulated grade III cranial horn tear of the medial meniscus (center) and with resection of the tear (right). Cranial (Cr), medial (M), and caudal (Ca).
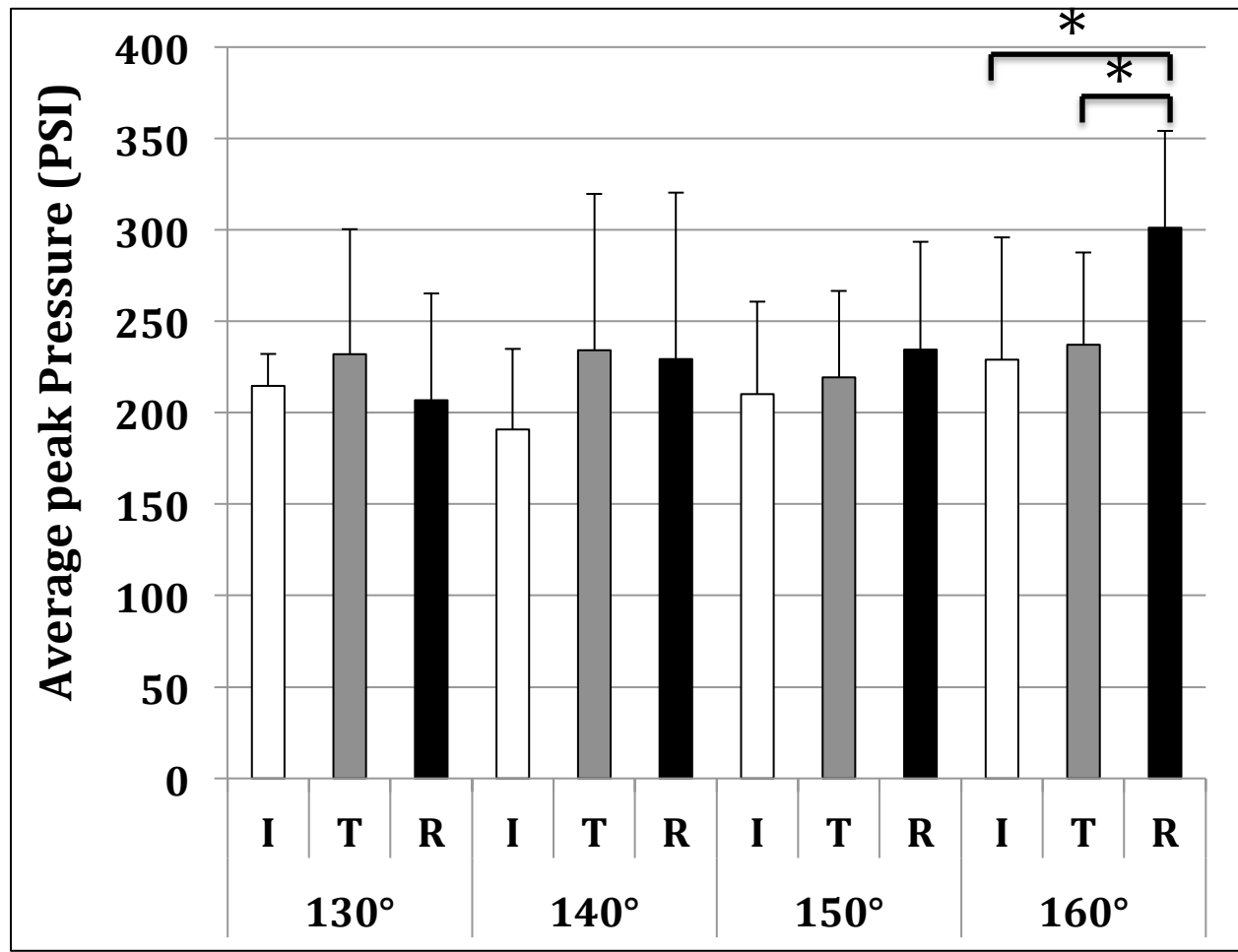
Figure 5.6 Average (n=6) peak pressures (PSI) in the central region of the medial tibial condyle at the four stifle angles (160°, 150°, 140° and 130°) for the three treatment groups; intact (I), torn (T), resected (R). Significant differences noted (*).

Discussion

The results of the current study demonstrate that resection of a grade III meniscal tear of the cranial horn of the medial meniscus leads to alteration in the magnitude and distribution of contact forces on the articular surface of the medial tibial condyle. A 1.3-fold increase in the magnitude of pressure in the central region of the medial tibial condyle was seen at a stifle angle of 160° in the resected specimens relative to the torn specimens. Therefore, resection of the simulated grade III meniscal tears shifted the pressure distribution on the medial tibial condyle to

a more centralized location, a region that had minimal contact forces in the intact and torn specimens. This central region of the medial tibial condyle corresponds to the area in which there is no meniscal coverage, therefore resection of grade III tears led to increased pressure in the region of direct joint contact. This alteration in force transmission may be associated with an increased risk of mechanical cartilage injury in the central region of the medial tibial condyle.

At 160° stifle angle, full extension, the contact area was significantly smaller than any of the other angles in the intact specimens. Having a smaller area of contact appeared to result in more focused pressure in the region of the cranial horn of the medial meniscus, near the attachment of the cranial meniscotibial ligament. A previous *in vitro* study indicated that axial compression and cranial displacement of the cranial horn of the medial meniscus occurs at 160° stifle angle based on 3-dimensional MRI images.(24) Accordingly, the localization of pressure in this region, seen with the contact maps at 160° stifle angle, helps to support the data from this previous *in vitro* study. Human research indicates that excessive compression of meniscal tissue may lead to the formation of a circumferential crack that propagates parallel to the collagen fibers.(25, 26) The development of a focal central region of increased pressure with grade III meniscal tear resection occurred only at 160° stifle angle. In this way, hyperextension may also be involved in the pathogenesis of cartilage injury secondary to cranial meniscal tears, or the concurrent development of these pathologies.

No significant differences were seen in the peak pressures measured in the central region of the tibial condyle between the intact and torn specimens at any of the stifle joint angles. These results indicate that even with a tear in the cranial horn and cranial horn attachment, if the tissue remained in place, load transmission was similar to that of intact menisci under the experimental conditions. Additionally, the results indicate that the portion of the cranial meniscotibial ligament

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that remained intact (the cranial bundle), was able to provide adequate hoop stress to allow for equivalent pressure distributions to those seen in the intact specimens. There are limitations in extrapolating these findings to clinical cases, such that in clinical cases the torn segment of tissue is often severely fibrillated and displaced and hence provides minimal mechanical support. In this study, the simulated grade III tear was noted to remain in place as the load was applied, and the load applied was only equivalent to that experienced at a walking gait. Accordingly, if forces were applied to simulate those generated in a horse during athletic activities, the results could certainly have been different, especially if the torn meniscus would become displaced. In fact, it is likely that the peak pressures generated may very well have been similar to or exceed those that were found after resection.

Results from this study parallel experimental work completed in humans and dogs;(9-12) wherein transection and/or removal of meniscal tissue is associated with a loss of meniscal function in equitable load distribution and the development of a focal central region of increased pressure on the tibial condyle. The nature of the femorotibial joint, which consists of a round femoral condyle articulating with a flat tibial condyle, leads to a focal centralization of the weight bearing forces when meniscal function is lost. Articular cartilage is adapted to distribute weight-bearing loads, and to withstand physiologic forces; however, as the loads applied to articular cartilage increase beyond critical thresholds, irreversible matrix destruction may occur.(27, 28) Therefore, alteration in the joint contact forces secondary to meniscal tears may result in cartilage injury and secondary arthritis. Experimentally, caudal medial meniscal release via meniscotibial ligament transection in normal dogs was associated with significant articular cartilage loss, further meniscal pathology, degenerative joint disease and lameness within 12 weeks.(6) The focal articular cartilage pathology seen on the medial tibial plateau and medial

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femoral condyle(6) correlated with the region of focal concentration and increased magnitude of pressure seen with experimental meniscal release in canine cadaveric stifles.(10) Complete medial meniscectomy has been evaluated experimentally in normal horses and focal cartilage erosion off the medial femoral condyle and tibial plateau was seen on post mortem examination of specimens euthanized 12 weeks after surgery.(29)

In clinical cases, arthroscopic resection of damaged meniscal tissue is recommended (17, 30, 31), as this treatment appears to lead to decreased joint inflammation and pain, and superior long-term results compared to medical treatment. The theory of debriding traumatized tissue in joints is based on evidence that physical debris liberated into joints either due to the original insult or the healing process may lead to ongoing cartilage injury.(32) As a consequence, removal of fibrillated tissue and debris from meniscal tears is a logical treatment based on general principles of joint therapy and can not be refuted based on the results of this study. However, the data from the current study indicates that physical support from tissue in the region of the meniscal tear may improve pressure distribution across the joint. For that reason, the potential benefits of techniques that restore the meniscal geometry should be considered. These techniques include: suturing techniques to salvage the torn tissue (33-35), regenerative therapy that may improve healing (36-39), and the allowance of a sufficient rest period for maximal healing prior to return to athletic function.

The risk of developing osteoarthritis secondary to meniscal tears is likely to be multifactorial. In addition to the magnitude of pressure experienced by the articular surface, alterations in the shock absorption and joint stability functions of the meniscus may increase the risk of osteoarthritis with meniscal tears.(2-4) In the current study the distribution of contact forces on the medial tibial condyle were altered secondary to resection of the meniscal tear.

Changes in the distribution of joint contact forces may alter the type of local stresses experienced by the articular cartilage (ie shear strain developing in a location previously under compression or vice versa).(40) Additionally, mild increases in the magnitude of contact forces may still lead to irreversible cartilage injury with the cummulative effects of cyclic loading.(41)

Arthroscopic exam of the tibial condyles is limited in clinical cases of equine meniscal tears; however, the weight bearing surface of the femoral condyle is readily visualized, and cartilage erosion in the central region of the femoral condyle is commonly seen.(17) Additionally, sequential or concurrent development of medial femoral condylar subchondral bone cysts with medial meniscal tears has been documented.(16) It is possible that the central region of increased pressure seen on the medial tibial condyle with grade III meniscal tears may be mirrored on the medial femoral condyle. Further evaluation of the effect of grade III meniscal tears on the pressure distribution over the femoral condyles is indicated.

Limitations exist when using an *in vitro* model to simulate an *in vivo* condition. In the current study, femorotibial joint pressure data was obtained from cadaver specimens with the medial collateral ligament and musculotendinous supports of the stifle joint transected. As the force was applied in a uniplanar vertical manner to a stable fixture-specimen assembly, no grossly visible displacement of the stifle in any other plane was noted, but could have occurred. Another limitation of this *in vitro* simulation is that upon natural creation of a grade three meniscal tear *in vivo*, there may be injury and stretching of the remaining intact segment of the meniscotibial ligament. This may lead to a further reduction in hoop stress that was not simulated in this model.

In conclusion, grade III meniscal tears of the cranial horn of the medical meniscus lead to mechanical overloading of the articular cartilage and subchondral bone in the central region of

the medial tibial condyle and as a result predispose the medial femorotibial compartment to the development of secondary osteoarthritis. Early recognition, treatment and enforcement of an adequate rest and rehabilitation program are important in the management of equine meniscal tears in order to decrease pathologic loading of the articular cartilage. Additionally, restoration of meniscal tissue within the tear defect should be the goal for treatment of equine cranial horn meniscal tears.

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CHAPTER 6.

Concluding Discussion

Concluding Discussion

The primary goals of this project were to evaluate the pathogenesis (study 1 and 2) and pathophysiology (study 3) of injury to the cranial horn of the MM in the horse. Extensive research has been completed evaluating the pathogenesis and pathophysiology of meniscal tears in other species, however, variation in the anatomy and pathology of the equine meniscus limits extrapolation of this data to the horse. The basic principles of meniscal injury and the development of secondary osteoarthritis were assessed through evaluation of the functional anatomy of the equine meniscus.

Meniscal translocation and deformation was documented in study 1, presented in chapter 3 of the thesis. The lateral meniscus was shown to have a greater overall cranial-caudal translocation than the medial meniscus through the range of motion, similar to that seen in the human knee. The cranial horn of the medial meniscus was found to be the least mobile of the 4 meniscal horns. However, during the terminal 10° of stifle extension, the cranial horn of the MM is forced to undergo significantly greater cranial translocation and axial compression than the cranial horn of the lateral meniscus, as a result of the geometry of the distal femur. In consequence, it was hypothesized that hyperextension of the equine stifle joint may result in the development of pathologic forces and injury to the cranial horn of the MM and it's adjacent meniscotibial ligament, which was further evaluated in study 2.

Evaluation of the tensile forces in the CrMTL of the MM was completed in study 2, presented in chapter 4 of this thesis. The results of this study demonstrated that full extension of the stifle (160°) results in a significant difference in the tensile forces in the abaxial and axial components of the CrMTL of the MM. The abaxial component of the CrMTL of the MM develops a significant increase in tensile forces at full extension of the stifle. Histologic

evaluation reveals division of the CrMTL into abaxial and axial components by a longitudinal region of highly vascularized, less organized connective tissue, which corresponds to the location and orientation of the reported meniscal tear patterns in this region. Differences in tensile forces may lead to shear forces developing along this region of less organized connective tissue, between the abaxial and axial components of the CrMTL of the MM, during hyperextension of the stifle. Thus, the results of study 1 and 2 indicate that cranial displacement and axial compression of the cranial horn of the medial meniscus during full extension of the stifle, places forces on the cranial horn and it's adjacent meniscotibial attachment that may lead to injury of this region with hyperextension of the stifle. Additionally, histologic findings indicate that the ultrastructure of the CrMTL may help explain the characteristic cranial horn meniscal tear patterns reported in the horse.

Cartilage injury and secondary osteoarthritis are reported to occur commonly with cranial horn tears in the medial meniscus of the horse. In order to evaluate the pathophysiology of these lesions, the magnitude and distribution of contact forces on the medial tibial condyle after the creation and resection of grade III cranial horn tears in the medial meniscus was completed in study 3, presented in chapter 5 of this thesis. Resection of the simulated grade III cranial horn tear resulted in a significant increase in peak pressure in the central region of the medial tibial condyle at a stifle angle of 160° relative to the intact and torn specimens. Thus altered loading of the tibial condyle as a result of displacement or resection of grade three meniscal tears may contribute to secondary osteoarthritis in the horse, and restoration of normal meniscal geometry may limit these degenerative changes.

In conclusion, the results of these experimental studies resulted in significant advancements in our understanding of the functional anatomy of the equine meniscus. However,

as this research essentially represents the first experimental studies specifically evaluating the pathogenesis and pathophysiology of cranial horn injury in the horse, it is apparent that considerable additional investigation is warranted.

Future Directions

Numerous unanswered questions arose throughout this project, and further research into these areas would help to establish a comprehensive understanding of the pathogenesis and pathophysiology of cranial horn injury in the horse. Potential areas of future study include:

- Comprehensive histologic evaluation of the equine meniscus, including elucidation of the meniscal vascular supply.

- Evaluation of the instant center of motion of the equine femorotibial joint, and documentation of the rolling and sliding components of femoral translation over the tibia.

- Determination of the effect of joint rotation on meniscal translocation, meniscal deformation and tensile forces within the CrMTL of the MM.

- Evaluation of the athletic endeavors and potential risk factors that lead to hyperextension of the equine stifle joint.

- Documentation of the incidence of medial collateral ligament attachment to the medial meniscus, and evaluation of the effect of this attachment on meniscal translocation and meniscal injury.

- Mapping of the magnitude and distribution of pressure over the medial femoral condyle with grade III cranial horn tears of the MM.

- Evaluation of the effect of suture repair of meniscal tears on the magnitude and distribution of femorotibial joint contact pressure.

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- Evaluation of the association between subchondral cyst-like lesions and meniscal tears may include: mapping of the location on the femoral condyle that the cysts occur with comparison to pressure data, and sequential MRI evaluation of test subjects in which arthroscopic meniscal tears were created.
- Evaluation of the ability of cranial horn tears in the medial meniscus to heal, including correlation with blood supply, and evaluation of therapeutic treatments (stem cell and growth factor therapies).

Recognition of meniscal tears in the equine stifle is increasing as a result of improved access to advanced diagnostic imaging techniques such as MRI, CT and ultrasonography. Elucidation of the mechanisms involved in the creation of these lesions and determination of the factors that lead to secondary cartilage injury in the stifle joint, are fundamental steps in understanding these lesions. On account of the relatively poor prognosis for return to athletic function with these lesions, establishment a greater understanding of these lesions is imperative for further development of preventive and therapeutic strategies.