IN VITRO BIOMECHANICAL COMPARISON OF DOUBLE VERSUS SINGLE PLATED TIBIAL PLATEAU LEVELING OSTEOTOMY CONSTRUCTS IN AXIAL LOADING

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ABSTRACT

The tibial plateau leveling osteotomy (TPLO) was introduced in 1993 as an innovative surgical procedure to treat stifle instability secondary to rupture of the cranial cruciate ligament (CCL) (Slocum 1993). The TPLO procedure eliminates cranial tibial thrust by converting this force into a compressive joint reaction force without restoring the passive constraint of the cranial cruciate ligament, resulting in a stable stifle during the stance phase of the gait cycle (Slocum 1993, Sahar 2006). The osteotomy created is stabilized by one of a variety of custom designed TPLO plates all of which vary in design characteristics, material and biomechanical properties.

Large and giant breed dogs have been shown to have a predisposition to develop rupture of the cranial cruciate ligament at a young age and biomechanical data regarding the performance of TPLO implants in these breeds is lacking (Duval 1999, Whitehair 1993). The objective of this study was to compare the axial stiffness of TPLO constructs stabilized with a single Synthes® broad TPLO plate (TPLO-broad) or a Slocum TPLO plate and a limited contact dynamic compression plate (LC-DCP) (TPLO-double).

Synthetic bone models were randomly assigned to one of two groups. All models underwent a standard TPLO and were stabilized with either a Synthes® Broad TPLO
plate or a Slocum TPLO plate and a LC-DCP. A gap was maintained at the osteotomy during plate application. Constructs were tested in axial compression and axial displacement and loads were recorded. Construct stiffness was calculated from load-displacement curves and comparisons of mean stiffness were performed.

The mean construct stiffness for the TPLO-broad constructs was not significantly different from that of the TPLO-double constructs. The minimum effective number of implants should be used clinically to achieve stability when performing a TPLO. Implant stiffness is a key contributor to the stability of an osteotomy and is integral to osteotomy healing.
Dedicated to

My Family and Friends
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CHAPTER 1

INTRODUCTION

1.1 Anatomy of the stifle

The stifle is a complex, condylar synovial joint (Evans 1993). The primary motion of the stifle joint is flexion and extension, however it permits motion in three planes (Carpenter 2000). Stifle stability is primarily controlled by four ligaments, including the medial and lateral collateral ligaments, as well as the cranial and caudal cruciate ligaments (Arnoczky 1977, Carpenter 2000).

The medial collateral ligament extends between the medial epicondyle of the femur and the medial border of the proximal tibia and has firm attachments to both the joint capsule and the medial meniscus (Evans 1993) (Figure 1.1). The medial collateral ligament has two functional components, a cranial and a caudal portion (Carpenter 2000). The caudal portion of the ligament is relaxed in flexion and taught in extension while the cranial portion is taught in both flexion and extension (Vasseur1981, Carpenter 2000).

The lateral collateral ligament is composed of a superficial and a deep portion and originates from the lateral femoropatellar ligament and lateral femoral
epicondyle (Carpenter 2000) (Figure 1.1). The lateral collateral ligament inserts on the head of the fibula and lateral condyle of the tibia (Evans 1993).

Figure 1.1: Schematic representation of the collateral ligaments of the canine stifle.

The cruciate ligaments are composed of twisted collagenous fascicles and fiber bundles containing >90% type I collagen and are each composed of 2 functional components (deRooster 2006, Amiel 1984). Collagen fibrils are characterized by a uniform crimp, parallel to the long axis of the fascicle and change length by straightening of the crimp (deRooster 2006, Hayashi 2003a, Muir 2005).
The CCL originates at the caudomedial aspect of the lateral condyle of the femur and inserts at the cranial intercondyloid region of the proximal tibia (Arnoczky 1977, Evans 1993) (Figure 1.2). The CCL is narrowest at its midsection and fans out proximally and distally (Evans 1993, deRooster 2006). The caudal cruciate ligament (CdCL) originates at the lateral surface of the medial femoral condyle and inserts on the edge of the popliteal notch of the tibia (Arnoczky 1977, Evans 1993) (Figure 1.2).

Figure 1.2: Schematic representation of the canine cruciate ligaments

Functionally, the CCL can be divided into two bundles, a craniomedial portion and a caudolateral portion (Arnoczky 1977). The caudolateral band is taught in extension
and relaxed in flexion, while the craniomedial band is taught in both extension and flexion (Arnoczky 1977). The major functions of the CCL include resisting cranial displacement of the tibia in relation to the femur (cranial tibial thrust), preventing hyperextension of the stifle, and limiting internal rotation of the stifle (Carpenter 2000). The CdCL functions to prevent caudal displacement of the tibia with respect to the femur (deRooster 2006).

In stifle extension, both collateral ligaments are taught and provide the primary restraint to internal rotation while the cruciate ligaments provide secondary restraint (Carpenter 2000). In flexion however, the lateral collateral ligament relaxes, permitting slight internal rotation. This is opposed by the twisting of the cruciate ligaments on one another, providing the primary restraint to internal rotation of the stifle in flexion (Arnoczky 1977, Vasseur 1981).

The cruciate ligaments are covered by folds of the synovial membrane and are supplied with blood from branches of the medial and lateral geniculate arteries, the popliteal artery and a direct branch of the descending genicular artery caudally (Arnoczky 1977). In addition to the vascular supply, the cruciate ligaments are nourished by passive permeation of the synovial fluid (deRooster 2006).

The major articular nerves to the stifle arise from the saphenous nerve, the tibial nerve and the common peroneal nerve (O’Connor 1982). Within the cruciate ligaments, most nerves course in the interfascicular areolar spaces (deRooster 2006). Various types of sensory nerve endings have been identified within the middle of the cruciate ligaments
and provide information about joint movement and position as well as noxious stimuli (Yahia 1992, Johansson 1991, Miyatsu 1993).

1.2 Pathophysiology of cruciate disease

The pathophysiology of cranial cruciate disease is not completely understood and is likely a multi-factorial process leasing to stifle instability and cranial cruciate rupture. Cruciate ligament disease has been theorized to be a progressive adaptive or degenerative change of the cruciate ligament. These changes are thought to be in response to micro-injury sustained from mechanical overload and the development of cellular hypoxia leading to gradual tearing of subunits of the cruciate ligament (Hayashi 2003a, Hayashi 2003b, Hayashi 2004). Cruciate disease may also result from dysregulation of the ligament matrix homeostasis resulting in generalized weakening of the ligament and ligament rupture (Gyger 2006). Alternatively a cascade of inflammatory mediators may lead to an intrinsic weakening of the ligament due to the actions of matrix degrading enzymes (Galloway 1995, Griffin 1992). However, no single factor accounts for all aspects of disease progression and the pathogenesis of cruciate disease may vary between different subgroups of patients.

Histologic changes seen in the core of cruciate ligaments from patients with naturally occurring cruciate disease include a decrease in the overall number of ligament fibroblasts due to a decrease in the normal fusiform and ovoid cells of the ligament (Hayashi 2003a). An increase in the number of fibroblasts exhibiting chondroid
transformation, or spheroid cells, is also present in core of ruptured CCLs (Hayashi 2003a). Additionally, disruption of the hierarchical architecture of the extracellular matrix collagen has been demonstrated in ruptured cruciate ligaments, indicated by a decreased birefringence and elongation of the collagen crimp (Hayashi 2003a). These findings support the theory that micro-injury to the ligament plays an important part of the signaling pathways that orchestrate remodeling of the CCL and lead to progressive CCL rupture (Hayashi 2003b).

An increase in the number of spheroid cells in the epiligamentous regions of the cranial cruciate has been shown in patients with cruciate disease, however there is no corresponding decrease in fusiform or ovoid cells in these regions as compared to the core regions of the ligament (Hayashi 2003a). Cellularity, vascularity, cell morphology and fibrous extracellular matrix in the epiligamentous regions of the ruptured CCL are similar in appearance to that of normal CCLs and an expansion of the epiligamentous region occurs, however this expansion fails to lead to the development of a bridging scar (Hayashi 2003a). In humans, a period of increased expression of alpha smooth muscle actin is seen post CCL rupture and may cause retraction of ruptured CCL fibers, thus preventing bridging scar formation. Whether this is true during the reparative phase after CCL rupture in the dog is unknown. None of the changes seen have been correlated with age, body weight, or duration of lameness (Hayashi 2003a).

It is unclear whether the cell loss noted in ligaments from patients with cruciate disease is due to necrosis or apoptosis of the cells. In one study, an absence of TUNEL+ cells (a cell marker for apoptosis) from the core of ruptured CCLs was suggested to
signify necrosis as a cause of cell death, however in this study there was an increase in the numbers of TUNEL+ cells in the epiligamentous regions of ruptured CCLs (Hayashi 2003b). Studies evaluating complete and partially torn cranial cruciate ligaments have demonstrated an increase in the number of apoptotic cells by the detection of active caspase within the intact and torn remnants of partially or completely torn ligaments suggesting that the intact as well as ruptured cruciate ligaments are affected by the same degree of degenerative change and an intrinsic factor may be responsible for ligamentous breakdown (Gyger 2006, Krayer 2008). No significant association between the degree of synovitis or osteophytosis has been found in correlation with the degree of apoptosis (Krayer 2008) and it was not able to be determined whether the apoptosis was a cause or result of the cruciate degeneration.

Cruciate ligament rupture is also associated with inflammation of the synovial lining of the stifle and the production of matrix-degrading enzymes may induce progressive rupture of the ligament (Muir 2005, Klocke 2005). Macrophages and cytokines TNF-alpha and IL-6 have been detected in the synovium of dogs with ruptured cruciate ligaments and have been associated with the degree of radiographically detected osteophytosis. However the importance of joint inflammation in the mechanism of CCL rupture is not completely understood (Klocke 2005, Muir 2005). Increased cellular localization of the collagenolytic agents cathepsin K and tartrate-resistant acid phosphatase have been demonstrated in the synovium and ruptured cruciate ligaments of dogs with cruciate disease and may induce progressive pathologic rupture of the CCL (Muir 2005).

1.3 Biomechanics of the TPLO

In addition to limiting internal rotation and hyperextension of the stifle, the CCL resists cranial translation of the tibia with respect to the femur (Reif 2002). Slocum and Devine introduced the concept of cranial tibial thrust force in 1983 (Slocum 1983). During weight bearing, cranial tibial thrust (CTT) is generated in the stifle as a result of tibial compression and the slope of the tibial plateau (Slocum 1983). The slope of the tibial plateau is approximately 10 degrees in humans, while the slope of the tibial plateau in dogs has been shown to be 25 +/- 2 degrees (Reif 2002). The compressive forces of weight bearing are divided into a force in the direction of the long axis of the tibia and a force cranial and perpendicular to it resulting in the generation of cranial tibial thrust force in the absence of a cranial cruciate ligament (Slocum 1983) (Figure 1.3).
Figure 1.3: Schematic representation of cranial tibial thrust indicated by the white arrow and the compressive forces of weight bearing indicated by the black arrow.

Transection of the cranial cruciate ligament resulted in a mean cranial tibial translation of 14 +/- 2.3mm in an in vitro cadaveric study (Reif 2002).

The tibial plateau leveling osteotomy was introduced by Slocum in 1993 to reduce or eliminate cranial tibial thrust force thus stabilizing the cranial cruciate. It does this by converting cranial tibial thrust into a compressive joint reaction force without restoring the passive constraint of the CCL resulting in a stable stifle during the stance phase of the gait cycle (Slocum 1993, Sahar 2006). To achieve this, the tibial plateau angle (TPA), defined as the angle between the tibial plateau and a line perpendicular to the long axis of the tibia, is rotated to an angle more perpendicular to the functional long axis of the tibia (Slocum 1993) (Figure 1.4).
Figure 1.4: Schematic representation of the TPLO procedure. A) Creation of osteotomy, indicated by the curved line. B) Rotation of the proximal tibial segment by a predetermined degree to achieve a postoperative TPA of 5 degrees C) Application of custom designed TPLO bone plate across the osteotomy

Rotation of the tibial plateau to 0 degrees eliminated force in the CCL throughout the stance phase in one study and resulted in a mean CTT of -6.3mm indicating a caudally directed force (Warzee 2001). In a cadaveric study, rotation of the tibial plateau to 5 degrees resulted in a lesser, but persistent caudally directed force with a mean CTT of -2 +/-2.9mm (Reif 2002). The minimum rotation to achieve a stable stifle has been shown to be 6.5 +/- 0.9 degrees, however this still results in a caudally directed force and a mean CTT of -3.2 +/- 0.8mm accompanied by a 37.7% increase in CdCL strain.
The position of the osteotomy establishes accurate rotation of the proximal tibial segment to achieve joint stability. The osteotomy in the TPLO must be centered on the intercondylar tubercles in order to appropriately rotate the plateau and eliminate CTT force (Kowaleski 2005).

A distally placed osteotomy has been shown to result in a greater than expected TPA in one study where the tibial plateau achieved with a centered osteotomy was 5.5 +/- 0.32 degrees when attempting to achieve a TPA of 5 degrees, compared to a TPA of 7.92 +/- 0.49 degrees with a distally centered osteotomy (Kowaleski 2005). A distally centered osteotomy also results in a shift of the tibial long axis and results in an increase in the TPA equal to that of the tibial long axis shift (Kowaleski 2005). Consequentially, a distally placed osteotomy results in a greater degree of CTT postoperatively and a more unstable stifle than a centered osteotomy. Cranial tibial subluxation (CTS) was found to be 2.98 +/- 2.76mm in centered osteotomy models (similar to the theoretical zero that should be achieved) while the mean CTS in a distally placed osteotomy model was 14.78 +/- 1.96mm (Kowaleski 2005).

While the main effect of the TPLO is to eliminate CTT, it may also diminish internal rotation of the stifle. An in vitro study showed a mean internal rotation of the stifle of 23.3 +/- 6.8 degrees after transection of the CCL. Following TPL to an angle perpendicular with the long axis of the tibia, the amount of internal rotation decreased to 9.5 +/- 6.3 degrees (Warzee 2001). This may be due to the relative tightening of the collateral ligaments as the stifle flexes. During flexion, the lateral collateral ligament relaxes allowing caudal displacement of the lateral femoral condyle. Because the medial
collateral ligament remains taught throughout the range of motion, internal rotation of the tibia occurs. Although the femoro-tibial angle is unchanged post TPLO, tibial plateau leveling results in a relative increase in flexion of the femoro-tibial articulation resulting in a taught medial collateral ligament and internal tibial rotation (Warzee 2001).

1.4 TPLO complications

The reported complication rate post TPLO is 18.8-28% (Stauffer 2006, Pacchiana 2003, Priddy 2003) and may be grouped into infective, inflammatory and technical complications, including osteomyelitis, incisional infections, fractures of the tibia or fibula, broken drill bits, hemorrhage, intra-articular implant placement, intra-osteotomy screw placement, retained surgical sponges, broken holding pins or screws, septic arthritis, loose implants, draining tracts, ring sequestrum, incisional inflammation, dehiscence and swelling, edema and seroma formation, bruising, premature staple removal, patellar tendon swelling, and late meniscal injury.

In one study of 397 cases, the development of complications after surgery was not found to correlate with age or body weight of the dog, the pre-operative TPA or the experience level of the surgeon (Pacchiana 2003). Factors that have been significantly associated with complications include breed and the performance of an arthrotomy with the TPLO (Pacchiana 2003). Interestingly, another study also found a significant decrease in complications when a standard medial arthrotomy was no longer performed (Stauffer 2006). This may be related to the increased duration of surgery when
performing the arthrotomy. However, the length of surgery was not found to correlate to the rate of complications in a study of 696 TPLOs (Stauffer 2006).

A study of 193 cases found that dogs that underwent bilateral TPLOs during a single anesthetic episode had a higher complication rate than dogs that underwent unilateral TPLO or bilateral TPLOs during separate anesthetic episodes (Priddy 2003). This is similar to another study that did not find a significant difference in complication rates post staged surgeries in 118 dogs (Stauffer 2006).

Tibial tuberosity fractures were the most common long term complications reported in 28 of 696 (4%) TPLOs, followed by patellar tendon swelling (19/696, 2.3%) (Stauffer 2006). This is similar to a study of 397 cases with a 4% incidence of tibial tuberosity fractures (Pacchiana 2003) and another study with 4.3% of animals experiencing postoperative tibial tuberosity fracture (Carey 2005). Factors contributing to tibial tuberosity fracture have been reported to be a narrow mean absolute thickness of the tibial tuberosity and an increase in tibial plateau angle at follow-up versus immediately postoperatively (Bergh 2008).

Further studies evaluating the complication of patellar tendon thickening post TPLO have reported a higher incidence than that reported by Stauffer in 2006. A retrospective study of 94 cases found 54.3% of animals had moderate patellar tendon thickening while 25.5% had severe patellar tendon thickening post TPLO (Carey 2005). Risk factors for the development of patellar tendon thickening include a cranially placed osteotomy, a partially intact CCL in conjunction with a cranial osteotomy and post-operative tibial tuberosity fracture (Carey 2005).
1.5 Osteotomy healing

Bone healing may take place through primary (direct) bone healing or secondary (indirect) bone healing (Mann 1989). Primary bone healing is the direct formation of lamellar bone across a fracture line or osteotomy from the haversian systems of adjacent cortices (Mann 1989). For this type of boney healing to occur, absolute stability and anatomic reduction at the fracture site must be achieved (Mann 1989).

Primary bone healing may be further subdivided into contact and gap healing (Mann 1989). Contact healing occurs when there is a defect of less than 0.01mm at the fracture or osteotomy site (Shapiro 1988). Cutting cones cross the fracture line and form resorption cavities. Perivascular osteoblastic precursors differentiate into osteoblasts and osteoid is produced (Mann 1989). This occurs parallel to the long axis of the bone and no external callus is formed in this process (Mann 1989). Gap healing occurs when there is a defect of less than 0.5mm at the fracture or osteotomy (Shapiro 1988). In this situation, lamellar bone is deposited perpendicular to the long axis of the bone and intracortical haversian remodeling leads to longitudinal re-orientation of the lamellar bone (Mann 1989, Newton 1985).

Secondary (indirect) bone healing occurs when the there is incomplete anatomic reduction or instability of a fracture or osteotomy and is characterized by callus formation (Blenman 1989). Secondary bone healing occurs in three phases: inflammatory, reparative and remodeling.
During the inflammatory phase, hemorrhage and vasodilation results in an influx of inflammatory cells and biochemical mediators of bone healing (Mann 1989). During the reparative phase, the initial hematoma is organized and a fibrocartilaginous callus is formed, later being converted to bone (Mann 1989). The remodeling phase is a balance between osteoclastic resorption and osteoblastic deposition and results in a change in bone shape to restore optimal strength and function (Mann 1989).

The progression of callus is dependent on the stability of the fracture site and its vascular supply (Mann 1989). Large interfragmentary movements have been shown to lead to the development of significantly more fibrocartilage and significantly less bone formation in osteotomy models (Claes 2002). Even well vascularized fractures will go on to non-union if mechanical stability is insufficient (Claes 2002).

In the early phases of bone healing, strain tolerance is of prime importance. Interfragmentary strain is defined as the relative displacement of the fracture gap ends divided by the initial fracture gap width and determines the subsequent differentiation of tissues in a fracture gap (Perren 1979). Interfragmentary strain is reduced by factors that increase the gap distance or decrease motion at the gap (Egol 2004, Perren 1979). Primary bone healing occurs at strains less than 2% while secondary bone healing occurs at strains between 2-10% (Egol 2004). Tissues that experience strain beyond their ultimate strain cannot form in a fracture gap (Perren 1979). Once tissues are formed, they stiffen the fracture gap and in turn lead to lower strains, allowing the formation of the next stiffer tissue, progressing to the formation of bone (Perren 1979, Claes 1997).

Large interfragmentary movements and strain have been shown to result in a
larger callus formation for small gaps (1-2mm) (Claes 1997). Callus formation appears to be greatest in groups with a mid-sized (2mm) gap compared to a small (1mm) or a large (6mm) fracture gap (Claes 1997). The smaller amount of callus in small fracture gaps may be explained by smaller interfragmentary movements while the deficiency in callus in the large fracture gaps indicates that the capacity of new bone to bridge large gaps must be limited despite the relatively low interfragmentary strain (Claes 1997). One reason for the relative lack of healing with large fracture gaps may be a limited capability of revascularization (Claes 2003). Large osteotomy gaps (5.7mm) have been found to have significantly more fibrocartilage and a lower number of newly formed blood vessels in the gap healing area than osteotomy gaps of a medium size (2.1mm) under similar conditions of interfragmentary movement and strain (Claes 1997).

Internal fixation of fractures has evolved over time to address issues of stability and blood supply to healing fractures. Initially, the goal of internal fixation was absolute stability to avoid micromovement at the fracture. The concept of biologic internal fixation to allow a more flexible fixation while minimizing surgical trauma and invasiveness is still developing (Perren 2002).

1.6 Biomechanics of bone plates

1.61 Conventional bone plates

Conventional bone plates rely on bone to plate contact for stability and convert
axial forces into shear stress at the plate-bone interface that is resisted by the frictional force between the plate and bone (Egol 2004). The frictional force equals the product of the coefficient of friction between the plate and bone and the force normal to the plate generated by screw torque (Egol 2004). If all screws do not have equal torque, the screw with the greatest torque contributes the most to the force normal to the plate and bears the greatest load (Egol 2004). Once the frictional force between the plate and bone is overcome, the strength of fixation becomes equal to the axial stiffness of the single screw farthest from the fracture in either the proximal or distal direction influenced by the screw position within the plate hole (Egol 2004). Plate screw construct resistance to bending is equal to the bending stiffness of the plate when fracture gaps are greater than zero (Egol 2004).

1.62 Locking bone plates

The goal of biologic fixation has lead to the modification and development of bone plates that limit contact between the bone and plate while still providing adequate stability. Locked plates do not rely on bone to plate contact for strength and function to control the axial orientation of the screw to the plate by allowing screws to be threaded into the plate, creating a fixed angle single-beam construct (Egol 2004). Locking plates convert shear stress to compressive stress at the screw-bone interface unlike conventional plates, which convert axial loads into shear stresses (Miller 2007). The strength of fixation is equal to the sum of all screw interfaces rather than of a single screw’s axial stiffness as seen in conventional bone plates (Egol 2004, Cordey 2003).
Locking head screws have been shown to provide superior resistance to displacement when only two screws per fracture segment were used in a mandibular reconstruction model when compared to conventional screws (Sikes 1998). However, no difference was found when more than 2 screws were used per fracture segment (Sikes 1998). Bicortical locking screws have also been shown to withstand significantly more loads to failure and experienced significantly less displacement after axial loading in a comminuted diaphyseal fracture model simulating osteoporotic bone (Fulkerson 2006). In a study evaluating the use of locking screws in a TPLO model using cadaveric canine tibiae, the screw type (conventional v. locking) had no significant effect on fixation stability under cyclic loading (Leitner 2008). Translation movement of the proximal tibial segment during screw insertion was found to be greater when using conventional screws and supports the use of locking screws to maintain reduction and alignment of the osteotomy during TPLO.

1.7 Effects of plates on bone

Internal fixation utilizing bone plates has been shown to have negative effects on the underlying bone, including porosis secondary to stress shielding and necrosis, potentially complicating and delaying fracture healing (Perren 2002).

Stress protection results in bone loss and thinning of the cortex following Wolff’s law (Claes 1989, Akeson 1976, Woo 1976). This bone loss results from a resorptive process characterized by endosteal porosis during early healing. Also the loss of porotic
endosteal bone results in an enlarged medullary cavity and cortical thinning late in the healing process (Uhthoff 1983). The degree of stress protection experienced by bone is related to the rigidity of the bone plate (Claes 1989). When comparing stainless steel plates of a greater modulus of elasticity and bending stiffness with a carbon fiber reinforced carbon plate, the stiffer stainless steel plates led to significantly higher bone loss (Claes 1989). Similarly, a significant amount of bone atrophy has been observed in canine femora after plating with two internal fixation plates with large difference in bending stiffness (Akeson 1976, Woo 1976).

Further negative effects of bone plates on the underlying bone include an interruption of blood supply. The necessary normal force between the conventional plate and the bone to prevent motion generates compressive forces, interrupting periosteal perfusion, compounding damage by the fracture mechanism and surgical intervention (Egol 2004). Areas of non-perfusion corresponding to the sector of cortex covered by a bone plate have been demonstrated experimentally (Uhthoff 1994).

Attempts to modify plates to result in a reduction in the area of bone-plate contact have resulted in plates with limited contact between the undersurface of the plate and bone. When the radius of the undercurvature of a bone plate is less than the radius of the bone surface, two-line contact between the bone and plate occurs (Perren 2002). When the radius of the plate is greater than that of the bone, a single line of contact may exist (Perren 2002). A bone plate with a radius that matches the radius of the underlying bone maximizes contact between the bone and plate with potential negative effects on boney perfusion (Perren 2002).
In an attempt to limit bone-plate contact area, the LC-DCP has been developed. The undersurface of the LC-DCP is scalloped and theoretically reduces bone-plate contact by 50% however the amount of bone-plate contact also depends on the radius of the bone. Despite this design feature, experimentally, no difference in cortical blood flow was found in a segmental fracture model of canine tibiae when plated with either a LC-DCP or a DCP (Jain 1999). Additionally, no differences in porosity of the bone was detected between tibiae plated with an LC-DCP versus a DCP (Jain 1999). This is theorized to be due to the extensive soft tissue and vascular damage induced by a segmental fracture rather than differences between the plating methods and indicates that simply modifying the plate may not result in improved postoperative blood supply in clinical cases of fractures and other factors should be taken into consideration (Jain 1999).

Locking plates do not rely on bone-plate contact and allow the local blood supply under the bone to be preserved, theoretically allowing for more rapid bone healing (Egol 2004). Despite the LCP’s independence of bone-plate contact for stability, it is recommended that the plate be placed at a distance less than or equal to 2 mm from the bone surface to maximize load to failure and minimize displacement (Ahmad 2007, Miller 2007, Stoffel 2003).
CHAPTER 2

IN VITRO BIOMECHANICAL COMPARISON OF DOUBLE VERSUS SINGLE PLATED TIBIAL PLATEAU LEVELING OSTEOTOMY CONSTRUCTS IN AXIAL LOADING

2.1 Introduction

Cranial cruciate ligament (CCL) disease is one of the most common orthopedic conditions affecting the canine stifle and may be attributed to a multi-factorial degenerative process of the cruciate ligaments under conditions of normal loading (Hayashi 2004, Duval 1999, Whitehair 1993, Rooster 2006, Moore 1996). The cranial cruciate ligament is a major stabilizer of the stifle, limiting internal rotation and hyperextension of the stifle as well as cranial translation of the tibia with respect to the femur (Moore 1996, Rooster 2006). Cruciate ligament disease predisposes to rupture of the cranial cruciate ligament and subsequent stifle instability, osteoarthritis and lameness (Hayashi 2004, Duval 1999, Moore 1996 part II).

The tibial plateau leveling osteotomy (TPLO) is a surgical technique that stabilizes the CCL-deficient stifle by reducing the tibial plateau slope in order to
neutralize cranial tibial thrust force (Slocum 1993). To reduce the tibial plateau slope, a radial osteotomy is made in the proximal metaphyseal region of the tibia and the fragment is rotated to five degrees (Slocum 1993, Warzee 2001). A custom designed TPLO plate is applied across the osteotomy for stabilization (Slocum 1993).

Stability of the osteotomy is vital for healing and determines the amount of strain at the site (Egol 2004). Fracture gap strain determines the type of healing that occurs and is reduced by factors that increase the gap distance or decrease motion at the gap (Egol 2004, Perren 1979, Perren 2002). Primary bone healing occurs at strains less than 2% while secondary bone healing occurs at strains between 2-10% (Egol 2004). Implant stiffness is an integral factor of construct stiffness and a major determinant of stability, an important consideration in large and giant breed dogs (Kloc 2009).

In a series of 394 cases, the most common complications involved fixation and included plate breakage, screw loosening, pin migration and wire breakage (Slocum 1993). In an attempt to address these complications, implant manufacturers have developed a number of different implant designs. Biomechanical data evaluating the relative performance of the various implants available for TPLO stabilization are lacking and further information is needed to draw comparisons between the different implants.

Specific recommendations for fixation of the osteotomy in giant breed dogs are unavailable. However clinically, surgeons may elect to utilize a more robust fixation for these patients. The purpose of this study was to compare the stiffness of a TPLO construct stabilized with standard 3.5mm Slocum TPLO plate and a 3.5mm seven-hole limited contact dynamic compression plate (LC-DCP) (TPLO-Double) to a TPLO
construct stabilized with a 3.5mm Synthes® broad TPLO plate (TPLO-Broad). Our hypothesis was that the TPLO-Broad plated constructs would not have a significantly different mean stiffness from the TPLO-Double plated constructs.

2.2. Materials and methods

2.2.1 Synthetic bone models

A tibial bone model was designed based on a CT scan (Siemens/Sensation 16, Siemens Corp, NY, NY, 0.7mm slice thickness, 15.8cm field of view, 120kVp, 100mAs, 512x512 resolution, 0.309mm pixel size) of the left tibia of a female spayed Great Dane with naturally occurring cranial cruciate disease, weighing 52kg. The initial bone model was fabricated in a laser-cured photopolymer using stereolithography (Stereolithography Apparatus (SLA 190), 3D Systems, Rock Hill, SC). The bone model was truncated at the distal end of the tibia in a block shape to allow it to be secured into a custom designed grip attached to servo-hydraulic testing frame (Bionix 585, MTS Systems Corp., Eden Prairie, MN). The proximal aspect of the tibia featured a dome shaped depression centered over the intercondylar tubercles. This feature was designed to allow axial load to be applied in the testing frame. The cranial aspect of the platform was blunted so as to not interfere with the saw when performing the proximal tibial osteotomy. A 4 cm stainless steel sphere attached to the actuator of the testing frame was used to apply axial load to the dome shaped depression during testing (Figure 2.1).
Figure 2.1: Caudocranial and proximodistal view of the 3D CT reconstruction of the tibia used for bone models demonstrating proximal hemispherical platform for mounting in testing apparatus and the blunted distal portion of the tibia.

The initial SLA bone model was test fitted to the testing apparatus. Once satisfactory alignment and positioning was achieved, this model was used to make 22 identical replicas using a blend of epoxy, bone powder and shredded fiberglass in a room temperature vulcanization (RTV) silicon mold. The tibial models were numbered and randomly assigned to one of two groups.
2.22 Construct preparation

Medio-lateral radiographs (Siemens Optitop 150/40/90 HC radiographic machine, Siemens Corp., NY, NY, 81 kVp and 2.8 mAs) of the left hind limb of the Great Dane were taken to assess the tibial plateau angle (TPA) to aid in planning the appropriate rotation for tibial plateau leveling osteotomy to achieve a postoperative TPA of 5 degrees. The stifle and tarsal joints were positioned in approximately 90 degrees of flexion. A line was drawn along the functional axis of the tibia by centering at the intercondylar tubercles and extending through the center of the talocrural joint. Another line was drawn parallel to the tibial plateau. The TPA was determined as the angle formed between a line perpendicular to the functional axis of the tibia and the line parallel to the tibial plateau as reported by Slocum (Slocum 1993).

The osteotomy was completed by utilizing a 60 mm diameter hole saw mounted in a drill press (Delta 15-017, Rockwell Manufacturing Co, Pittsburgh, PA) to create a repeatable 30mm osteotomy. To minimize variability between models, the tibial models were mounted in a vice and custom created jig, which was visually aligned with the axis of the saw blade such that the osteotomy was perpendicular to the sagittal plane of the tibial models. The fibula was removed from the bone models due to interference with rotation due to the firm attachment between the fibula and the tibia in the model. After performing the osteotomy, a Slocum TPLO jig (Slocum Enterprises Inc., Eugene, OR) was applied and the proximal tibial segment was rotated 13.5mm to convert the TPA from its original pre-operative value of 31 degrees to a postoperative TPA of 5 degrees.
After rotation of the proximal segment, two pieces of a 3.2mm diameter Steinmann pin (Securos Inc., Charlton, MA) were placed within the cranial and caudal aspects of the osteotomy to achieve a gap at the osteotomy. The tibial models were then randomly assigned to one of two groups. The first group received a single 3.5mm Synthes® broad TPLO plate (Synthes Vet, Paoli, PA) and the second group received a single 3.5mm Slocum TPLO plate (Slocum Enterprises Inc.) and a caudally placed 7 hole 3.5mm LC-DCP (Synthes Vet). The plates were applied so that the distal portion of the TPLO plate and the LC-DCP were visually aligned and parallel. After plate application, the Steinmann pins were removed, resulting in a uniform 3.2mm gap at the osteotomy.

2.23 Plate application

All implants were cleaned with an ultrasonic cleaner (Blue Wave Ultrasonics, Andover, MA, Spectrasonic Ultrasonic Cleaning Solution, Spectrum, Stow, OH) and sterilized by vacuum steam sterilization (Vacuum Steam Sterilizer M/C 4133, Rochester, NY) prior to use to replicate the treatment of implants used in clinical applications.

Group one (TPLO-broad) consisted of 11 TPLO constructs utilizing the 3.5mm Synthes® broad TPLO plate. The proximal aspect of the Synthes® broad TPLO plate was minimally contoured to the medial aspect of the proximal tibia by slightly unbending the plate. The plate was applied so that the narrowest portion of the plate distal to the expanded proximal portion was positioned over the osteotomy gap. The plate was applied by placing a standard 3.5mm non-self tapping stainless steel cortical bone screw in the
neutral position in the first combi-hole of the distal portion of the plate using standard AO/ASIF guidelines. The proximal plate holes were filled with 3.5mm self-tapping stainless steel locking cortical bone screws in standard AO/ASIF fashion utilizing a 2.8mm LCP threaded drill guide (Synthes Vet), and 2.8mm drill bit to prepare the screw holes. A 1.5N/m torque-limiting device was utilized to place these screws with a uniform torque. The remaining distal plate holes were filled with 3.5mm non self-tapping stainless steel cortical bone screws (Synthes Vet) in neutral fashion using standard AO/ASIF guidelines to maintain the 3.2mm osteotomy gap (Figure 2.2). After plate application, the TPLO jig was removed.

Figure 2.2: Schematic of bone model with a 3.5mm Synthes® Broad TPLO plate applied
Group two (TPLO-double) consisted of 11 tibial constructs prepared with a single 3.5mm Slocum TPLO plate and a single 7 hole, 3.5mm LC-DCP applied caudal to the Slocum TPLO plate. The Slocum plate was contoured to the medial aspect of the proximal tibia and standard 3.5mm non self-tapping stainless steel cortical bone screws were used in neutral fashion in all holes to maintain the osteotomy gap. The plate was applied so that the narrowest portion of the plate, just distal to the triangular shaped head, was positioned over the osteotomy gap. The LC-DCP was contoured to the medial aspect of the proximal tibia immediately caudal to the Slocum TPLO plate. The plate was placed so that 2 holes were over the proximal tibial segment and the remaining 5 holes were over the distal tibial segment. The plates were applied so that the cranial aspect of the LC-DCP was in direct contact with the caudal aspect of the Slocum TPLO plate over the osteotomy gap. All plate holes were filled with standard 3.5mm stainless steel non self-tapping cortical bone screws placed in neutral fashion to maintain the osteotomy gap in standard AO/ASIF fashion (Figure 2.3). After plate application, the TPLO jig was removed.
2.24 Mechanical testing

The TPLO constructs were mounted in a servo-hydraulic testing frame (Bionix 858, MTS Systems Corporation, Eden Prairie, MN). The truncated distal aspect of the tibia was mounted within a square fixture secured to the testing machine (Figure 2.4).
Load was applied utilizing a 4 cm stainless steel sphere attached to the actuator of the testing frame. The sphere articulated with the depression feature on the tibial plateau to allow axial compression to be applied without construct slippage.

Models were tested by applying a monotonically ramped load from 10N to 3000N (or a point at which catastrophic failure occurred) at a rate of 10N/S with load and actuator measurements taken at a rate of 5 Hz. Catastrophic failure was defined as fracture of the tibia, screw pullout, plate or screw breakage or slippage of the construct from the actuator.

Load displacement curves were generated for each construct and a linear regression line was calculated for the linear portion of the load-displacement curve.
The slope of the linear regression line was calculated to represent the stiffness of the construct (Figure 2.5). An $r^2$ value of $\geq 0.85$ was considered acceptable.

Figure 2.5: Sample Load-Displacement curve. Load in Newtons on the y-axis and displacement in mm on the x-axis.
2.3 Results

Evaluation of load-displacement curves revealed a linear portion between the loads of 250 and 1000N for all tests. All linear regions evaluated had an $r^2$ value $\geq 0.85$. (double plated constructs range 0.960-0.999, Synthes® constructs range 0.990-0.999) (Figure).

The mean stiffness for the TPLO-broad constructs was 411.44 +/- 70.54 N/mm (95% CI 364.06 – 458.83N/mm) and the mean stiffness of the TPLO-double constructs was 432.19 +/- 63.55 N/mm (95% CI 389.49 – 474.89 N/mm). (Graph 2.1).
There was not enough evidence to suggest a significant difference between mean construct stiffness for the two groups (P = 0.48). No construct underwent catastrophic failure and the most common modes of failure included plastic and elastic deformation of the bone plates exhibited by bending of the plate and collapse of the osteotomy with a resultant valgus malformation of the proximal tibia. Failure loads were not reported because no distinct yield point or failure point was observed for many of the specimens.
2.4 Discussion

Six of nine dog breeds identified as being predisposed CCL rupture at a young age may be considered giant breed dogs (including the Rottweiler, Newfoundland, Mastiff and Neapolitan Mastiff, Akita, and the Saint Bernard) (Duval 1999, Whitehair 1993). It would be reasonable to assume these breeds may require a more robust fixation after TPLO than a dog of average size. The AO/ASIF recommends using a 3.5mm broad or 4.5mm DCP for fixation of tibial fractures in dogs greater than 25kg (Johnson 2005), however TPLO plates were previously not available in these sizes. Due to concerns of achieving adequate stabilization of the osteotomy, surgeons may apply an additional bone plate across the osteotomy as well as the standard TPLO plate in larger dogs (Kowaleski 2005). Broad TPLO plates (both conventional and locking) have recently been introduced by multiple implant manufacturers to address stabilization of the tibial osteotomy in larger and giant breed dogs.

A gap model based on a previous report (Kloc 2009) was used to evaluate the contribution of TPLO implants to construct stiffness in this study. A gap model was utilized to isolate the contribution of the plate or plates to construct stiffness to compare the stiffness between construct groups. This information can aid surgeons in deciding what type of fixation to apply to stabilize the TPLO in giant breed dogs. This model underestimates the actual stiffness of a clinical TPLO without an osteotomy gap, however during a clinical TPLO, poor osteotomy reduction or limb alignment corrections may result in a significant cortical gap (Kloc 2009, Wheeler 2003). The standard for
biomechanical evaluation of bone plate constructs is 4 point bending (ASTMST 1994), however constructs were tested in axial compression in our study due to the complex geometry of the plates and the desire to have a more accurate representation of how the implants perform when loaded in a fashion similar to the clinical situation.

Bone models instead of cadaveric bones were used due to relative unavailability of cadaveric tibiae from giant breed dogs. Bone models have an additional advantage over actual bone, since they are very uniform in size, shape and stiffness, which mitigates variability between constructs. The relative concentrations of the bone model components creates a composite that closely resembles cortical bone (Hildreth 2006). These bone models have a comparable flexural modulus of elasticity to the natural canine tibia (Hildreth 2006). The bone model played a limited role in our testing, since a construct’s resistance to bending loads equals the stiffness of the plate when an osteotomy gap exists and does not rely on the plate-screw-bone interaction (Egol 2004).

A significant difference in stiffness was not found between the TPLO-Broad constructs and the TPLO-Double constructs in our study (P = 0.48). The bending stiffness of a bone plate is proportional to the area moment of inertia (AMI). For a plate with a rectangular cross section, AMI = BH^3/12, where B is the base of the rectangle and H is the height, multiplied by the modulus of elasticity for that material. (Skinner 2006). Calculating this for our models, one would expect a similar stiffness between our two construct types given the dimensions of the implants. This assumes that the AMI can be estimated by adding the widths of two separate bone plates together for calculation purposes and does not take into account any gap between the plates that may alter the
observed AMI of the construct. Efforts were taken to ensure contact between the Slocum TPLO plate and the LC-DCP without an appreciable gap in this study, however this was difficult due to the irregular surface of the medial tibia and contouring of the plates to the bone surface but is a problem that is also encountered in a clinical TPLO. Because we evaluated TPLO constructs, the observed stiffness should closely replicate those experienced in vivo.

The comparison of stiffness calculations also assumes that all implants are made of similar materials with a similar modulus of elasticity and limited variability within the implant itself. This may not be true in our study as the Synthes® broad TPLO plate and the LC-DCP are both manufactured from cold worked 316L stainless steel, while the Slocum TPLO plate is manufactured from a cast stainless steel that may lead to inconsistencies within the implant (Boudrieau 2006). Variations in chemical composition among plates and among regions within the same plate as well as inclusions and cavities have been identified (Boudrieau 2006). In a previous study, variability in the stiffness of the Slocum TPLO constructs was attributed to the cast nature of the plate that could result in inconsistencies within the implant (Kloc 2009). This was not observed in our study as both construct types had similar variability (similar standard deviations). This may be due to the addition of the LC-DCP manufactured from cold worked stainless steel to the Slocum plate, compensating for variations in the stiffness of the Slocum plate.

Load at failure was not reported as we were unable to determine this value for many of the load-displacement curves. The observed mode of failure was plastic deformation of the plate with axial collapse and a resultant valgus deformity of the
proximal tibia. Reported failure modes of bone plates and screws include screw loosening or breakage by shearing at the screw head-shaft junction, plate and screw bending and screw cut-out (Egol, 2004, Leitner 2008). This typically results in a “rock back” of the proximal tibial segment and a resulting increase in tibial plateau angle and valgus deformity of the tibia (Kloc 2009, Bergh 2008, Leitner 2008).

The use of a single rather than multiple implants may be preferable when the stability afforded by either choice is comparable. The application of multiple implants increases anesthetic and operating times as well as surgical trauma to the soft tissues and bone, with potential negative effects on vascularization and healing.

The Slocum and LC-DCP plates require extensive contouring prior to application compared to the pre-contoured Synthes plate. The Synthes® plates were minimally contoured in this study to maintain a small (less than 2mm) gap between the plate and bone as currently recommended (Ahmad 2007). While the time for contouring and application was not specifically measured in this study, one may reasonably assume that the application of multiple implants and contouring required of the Slocum plate and LC-DCP would increase surgical and anesthetic time.

Conventional plates and screws rely on friction between the plate and bone surface to resist axial forces (Egol 2004). Bone-plate contact interrupts vascular perfusion and leads to weakening of the bone due to stress shielding or necrosis (Perren 1987, Uhthoff 2006, Akeson 1976). In previous studies, it has been shown that a large zone of disturbed blood supply persisting for weeks after surgery can be seen under bone plates (Gunst 1979). Delayed bridging of the osteotomy may be seen due to interruption
of cortical perfusion caused by compression between the plate and the bone surface (Perren 1987, Uhthoff 2006). The deleterious effects of bone plates may be assumed to be magnified when double plating the tibial osteotomy as the area of bone covered by the implant is greater than when using a single implant. The clinical effects of this are unknown, but should be considered when planning fixation in clinical cases. Additionally, application of the more caudally located LC-DCP when double plating may cause compromise of the medial collateral ligament.

In addition to limiting the number of implants, the amount of contact between the bone and plate and thus interruption of vascular perfusion may be theoretically decreased by design in the LC-DCP and potentially eliminated with the LCP. However, differences in the contact area between the bone and plate were not found however in a study comparing the DCP to the LC-DCP in human femora and equine third metacarpi (Field 1997). This effect may be due in part to the geometry of the particular bone and plate (Field 1997).

Some studies have demonstrated a beneficial effect on blood flow when utilizing the LC-DCP (Jacobs 1981) while other studies have not shown an effect on the overall cortical bone porosity or cortical blood flow when comparing the two plates (Jain 1999). This may again be due to the plate-bone contact and is dependant in part on the design of the plate as well as the geometry of the underlying bone (Field 1997). The Slocum TPLO plate resembles a DCP. The proximal aspect of the Synthes® TPLO plate employs locking technology, which does not rely on friction between the bone and plate for stability, while the distal aspect of the plate resembles an LC-DCP and relies on contact
between the plate and bone for stability. Examination of clinical cases of double- versus single-plated TPLOs will be necessary to evaluate the potential impact of the implants on clinical boney union.

Limitations of our study include the limited sample size, which account for the inability to detect a significant difference in construct stiffness if one existed, or type II error. Additionally, by design, the gap model did not allow us to evaluate the contribution of bone contact to construct stiffness. However it did allow us to evaluate the contribution of the bone plates themselves and eliminated possible variations due to locking plate technology in the Synthes® TPLO constructs. Lastly, bending and torsional stiffness were not evaluated by our study, however axial load as applied in this model creates a combination of axial collapse and bending typical of the failure modes experienced in vivo (Kloc 2009).
Our study did not find a significant difference in construct stiffness between a single Synthes® broad TPLO construct and a double plated construct consisting of a Slocum TPLO plate and an LC-DCP. Further evaluation of the stiffness of other broad TPLO plates would add to available knowledge and comparison of TPLO constructs using conventional cortical screws instead of locking screws proximally would assist in decision making in clinical cases. Based on this in vivo determination of gap stiffness, the clinical performance of the Synthes® Broad TPLO plate should be comparable to the double plated Slocum + LC-DCP construct. The clinical performance of the Synthes® broad TPLO plate and double plated constructs should be evaluated to determine if there is a difference in the rate of boney healing, complications or failure.
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