BIOMECHANICAL EVALUATION OF MEDIAL FEMORAL CONDYLAR SUBCHONDRAL CYSTIC LESIONS AND THE EFFECTS OF TREATMENT WITH INTERNAL FIXATION

Abstract

Purpose of the study: Subchondral cystic lesions (SCLs) in the medial femoral condyle are a common equine orthopedic problem occurring most commonly in the first two years of life. They are located on the weightbearing surface of the condyle and are frequently associated with lameness. While the pathophysiology of SCLs is poorly understood, they are believed to be the result of either osteochondrosis or trauma, or perhaps both. Similar to bone cysts in adolescent children, there are many treatments recommended for SCL, and like in children, recurrence and treatment failure rates are unacceptably high . Conservative management includes stall rest with or without intra-articular medication. Surgical management consists of intra-cystic injection with corticosteroids, cystic debridement, cancellous bone grafting, mosaic arthroplasty, and placement of chondrocytes or mesenchymal cells in fibrin glue. The goals of treatment are to maximize the amount and quality of subchondral and epiphyseal bone within the defect leading to a reduction in the inflammation caused by the contact of joint fluid with epiphyseal bone, and improve the attachment of the articular cartilage overlying the defect. There is very little information available about the biomechanics of the stifle joint of the horse either in health or disease, and no treatments of SCLs address the possible changes in biomechanics generated by what is often a large void in a critical weight-bearing area of the medial femoral condyle. The objectives of this study were to identify and measure the strain on the medial femoral condyle before and after the creation of a

defect similar to a SCL and evaluate if that strain is altered with the placement of a cortical bone screw placed in lag fashion across the defect. The first hypothesis was that there would be a decrease in the bone strain measured on the abaxial aspect of medial femoral condyle and proximal tibia, and a corresponding increase in strain on the medial meniscus, after the experimental creation of a medial femoral condylar subchondral bone defect. A second hypothesis was that internal fixation of the subchondral bone defect with a 4.5mm cortical bone screw placed in lag fashion would return bone strain to a level similar to the pre-defect measurements.

Research method: Stifle joints were collected from horses (<2 years old), from the proximal 1/3 of the tibia to the femoral head, that were euthanized for reasons unrelated to musculoskeletal disease. The limbs were dissected leaving the entire stifle joint with extra-and intra-articular ligaments intact. The function of the quadriceps muscle was recreated using a turnbuckle and wire, and the stifle was maintained in extension at approximately 160°. Uniaxial strain gages were placed peripherally on the abaxial aspect of the medial femoral condyle, medial meniscus, and proximal tibia. Axial compression was applied to the limb using a servohydraulic mechanical testing machine with a preload of 50 N (~11 lbs) and stepwise loading increments of 440 N (100 lbs) from 440-3960 N (100-900 lbs). Strains were recorded and averaged across each 100 lb increment. After testing the intact limb, the stifle was flexed and a small window was made into the craniomedial aspect of the femoro-tibial joint capsule to gain access to the medial femoral condyle. A subchondral bone defect was created on the weightbearing cartilage surface of the medial condyle approximately 20 mm X 15 mm in size. The limbs were then tested as previously, and strains were re-recorded. The final

trial was performed after a 4.5mm cortical bone screw was placed in lag fashion across the medial femoral condyle subchondral bone defect in a proximomedial to distolateral direction. Subchondral bone defect size and screw placement were radiographically assessed with a caudoproximal-craniodistal oblique projection. The data was tested for normal distribution and a 2-way repeated measure ANOVA used to determine significance ($p<0.05$).

Findings: Strain measured on the medial femoral condyle decreased following creation of the subchondral defect at all loads measured above 100lbs. Correspondingly, the strain measured on the adjacent medial meniscus increased following creation of this defect. Following insertion of the transcondylar screw, the strain on the medial femoral condyle increased to levels above which was measured prior to the creation of the defect, and the strain on the medial meniscus decreased back down to pre-defect measurements. The changes in strain on the tibia were minimal following creation of the defect and insertion of the transcondylar screw.

Implications: This study provides the first biomechanical assessment of the equine distal femur, meniscus, and proximal tibia, and offers experimental data for the clinical use of internal fixation for the treatment of SBC's. Furthermore, the data from this experiment can be used to validate the use of a previously created Finite Element model of the equine stifle, which can be the basis for future FE studies of this joint. The overall impact of FE analysis of the equine joint is profound, as the equine stifle has recently been suggested to be a model for joint disease involving the human knee.

Introduction

Subchondral bone cysts (SBC), are a serious cause of lameness in the horse that can warrant a guarded prognosis for athletic potential.[1-3] They most commonly cause lameness in the first two years of life, and are associated with bone growth. They have been identified primarily in the medial femoral condyle of the femur and in the bones of the digit (metacarpus/tarsus and phalanges) but can occur in any location.[4] Many theories for etiology have been proposed, though only a few have been experimentally supported. Clinically significant bone cysts occur at areas of maximal/increased weight bearing, and one formation hypothesis proposes that trauma leads to subchondral bone damage. This hypothesis has been intentionally and coincidently reproduced experimentally, where a cartilage defect with subchondral bone damage was followed by secondary invasion of synovial fluid.[5-8] This hypothesis is regarded as the hydraulic theory because weightbearing leads to increased mechanical pressure on the subchondral bone due to synovial fluid being pumped into the defect.[5]

At initial diagnosis, young horses with uncomplicated SBC usually do not have radiographic or arthroscopic signs of degenerative joint disease. Lameness associated with SBC is believed to be the result of synovitis and increased intracystic or intraosseous pressures leading to subchondral bone pain.[5] In older or aging horses, the cystic lesions can be associated with osteoarthritis and a worsening prognosis.[9] All clinically significant SBC have a sclerotic periphery as a result of thickened trabecular bone. These thickened trabeculae indicate that strain is being transmitted around the defect, which will reduce bone remodeling.

Treatment recommendations for these lesions vary. Conservative management includes stall rest with or without intra-articular medication.[10] Surgical management consists of intra-cystic injection with corticosteroids, cystic debridement, cancellous bone grafting, mosaic arthroplasty, and placement of chondrocytes or mesenchymal stem cells in fibrin glue.[2,6,9-12] Collectively, the goals of these treatments are to maximize the amount and quality of subchondral bone within the defect leading to improvement or resolution of the cyst. Elimination of the subchondral bone defect should improve the congruity of the joint surface and contact of joint fluid with the cyst lining. A reestablished joint surface will reduce joint inflammation and pain, and may also reduce injuries to meniscus and cartilage that accompany large cysts and result in persistent lameness . We propose that introduction of a rigid and fixed transcondylar screw will return strain to the defect and promote subchondral bone and fibrocartilage formation in the articular communication of the cyst. To test this treatment an understanding of the forces and strains on the intact distal femur is essential.

However, to date, no experimental studies have evaluated the biomechanical forces on the equine stifle. Consequently, it is also largely unknown what the effect of a gap in the condyle, or cyst, would do to the strains of surrounding bone, cartilage, meniscus, and proximal tibia as they adapt to the altered loading. Once these crucial measurements are identified and understood, treatment regimens to normalize strain in the MFC with a SBC can be objectively assessed. The objectives of this study are to evaluate the strain measured on the medial femoral condyle before and after the creation of a “cyst-like lesion” and then to evaluate how those strains are altered with the placement of a cortical bone screw placed in lag fashion across the condylar defect.

Ultimately, this project intends to provide information regarding the biomechanics of the distal femur, specifically the medial femoral condyle. With this data we hope to establish and elaborate on a biomechanical method of critical evaluation about subchondral bone cysts that currently is not possible due to the lack of experimental evidence. We also hope to take this knowledge one step further by experimentally testing a method of fixation that may have an immediate effect in the clinical course and management of this disease. Currently the success rate of patients with a MFC SBC is <70% for return to full function. We hope that with the potential application of this treatment, this rate can reach into the 90th percentile assuming concurrent joint disease is not present; or that this treatment could slow down or eliminate further progression of concomitant disease related to the cystic lesion.

Materials and Methods

Sample collection and preparation:

Paired hindlimbs were collected from horses between the ages of 1 and 5 years of age that were euthanized for reasons unrelated to musculoskeletal problems and without evidence or history of stifle disease. These hindlimbs were disarticulated at the coxofemoral joint and dissected through the proximal 1/3 of the tibia, thus leaving the stifle intact. The soft tissues were removed down to, but not including, entrance into the femoropatellar, lateral femorotibial, or medial femorotibial joint pouches. To prepare the limbs for mechanical loading, a ½ inch transverse, medial-lateral drill hole was created in the proximal femur, approximately 1-2 cm distal to the femoral neck, through which a 15/32 inch pin was inserted (more on this in the **Load Application** subsection). Limbs

were wrapped in saline soaked gauze and stored in the freezer at -20 F until testing. Limbs were thawed under refrigeration for 12-16 hours prior to testing. The quadriceps apparatus was recreated by cranial placement of a turnbuckle. The turnbuckle was secured with 12 gauge wire fixation placed through 3.2 mm transverse holes made in the proximal patella and mid-diaphysis of the femur. A clinically relevant femorotibial angle (135°) was achieved by twisting the turnbuckle.

Strain Gage Application:

The sites for strain gage attachment were based on data obtained from preliminary studies. Specifically, the first site was approximately 1-2 cm cranial to midpoint of the medial collateral ligament of the medial femorotibial joint on the distal medial femoral condyle. The second site was along the medial aspect of the medial meniscus 1-2 cm cranial to the medial collateral ligament of the medial femorotibial joint. The final site was approximately 3-4 cm distal to the femorotibial joint on the proximal tibial epiphysis 1-2 cm cranial to the medial collateral ligament of the medial femorotibial joint. Access to these points was obtained by dissecting the soft tissue and joint capsule covering down to the cartilaginous surface with a #10 Bard-Parker scalpel blade. Then an approximately 3 cm x 3 cm square of cartilage was removed with a #15 Bard-Parker scalpel blade exposing the subchondral bone. The subchondral bone was degreased with acetone and isopropyl alcohol, and left to dry by room air. Vishay general purpose unidirectional strain gages (Model # C2A-06-250LW-350, Vishay Intertechnology, Inc., Malvern, PA) pre-fitted with leads were secured to each of the aforementioned subchondral bone and meniscal sites with an adhesive (cyanoacrylate). At each site the gage was oriented in a vertical (proximal to distal) position.

Strain Measurement:

The leads of the unidirectional strain gages were soldered to the connections of a strain gage amplifier (Model # 2150, Vishay Intertechnology, Inc., Malvern, PA), which amplified the microstrain signal measured by the gages at each load. The amplifier was connected to an oscilloscope (Model # 54622A, Agilent Technologies, Inc., Santa Clara, CA), which was used to observe and record the amplified strain. The oscilloscope recorded a measurement every 0.25 seconds. Once obtained, the recorded strain measurements were saved as a .CSV file. This file was opened in the Microsoft Excel program and saved as an .XLS file for further analysis.

Load Application:

Testing was carried out using a servohydraulic mechanical testing machine [Bionix 858, MTS Corp., Eden Prairie, MN]. The femur was secured in the machine with an aforementioned 15/32 inch pin placed through the previously drilled ½ inch medial to lateral transverse hole and the axial loading arm of the MTS frame. Once in the machine, the testing commenced with a preload of 50 N and then stepwise loading in increments of 100 lbs (444.8 N) from 0-900 lbs (0 – 4003.2 N). This load was chosen for two reasons. Most importantly it encompasses physiologic loads applied to an average equine stifle during various phases of the stride. Secondly, through our preliminary data, we have learned that bone fatigue and wire fatigue to failure occur at loads greater than 1000 lbs. The load was applied to both ends of the pin by the axial loading arm, thus evenly applying axial compression to the femur, stifle, and proximal tibia. A custom circular plate was fitted with side walls to secure the distal aspect of the

construct and prevent it from moving during loading. Load applied at each 100 lb increment was maintained for a 20 second before a 5 second transitional loading period to the next 100 lb increment. The result of this testing period was 80 saved strain measurements per each 100 lb loading increment (one strain measurement per 0.25 seconds x 20 seconds).

Control Testing:

Once the specimen was prepared, fitted with gages, and secured in the testing apparatus, the testing began as described above. Each limb was loaded to 900 lbs and strain recorded. The first testing cycle represented the controls. Immediately following the completion of the control specimen, a defect was created into the medial femoral condyle.

Medial Femoral Condyle Defect Preparation:

A subchondral bone defect was created through the weight bearing cartilage surface of the medial condyle using a 15mm drill bit in a distal to proximal direction approximately 20mm in depth. The site chosen for defect creation was determined through examinations of cadaveric limbs of clinical cases of MFC SBC that presented to the Goss Pathology Laboratory, as well as through arthroscopic identification and mapping in clinical cases that presented to the hospital for elective surgery performed by the investigators. To access this location, the turnbuckle was loosened and the stifle flexed to expose the weight bearing surface of the medial femoral condyle cranially. Following the creation of the defect, the turnbuckle was tightened to recreate the 160° femorotibial angle. The limbs were tested in axial compression and the strains were re-

recorded as per the controls. Following testing of the stifle with the subchondral defect in the medial femoral condyle, the defect was internally fixed with a cortical bone screw placed in lag fashion from proximomedial to dorsolateral.

Internal Fixation of Defect:

The position of screw placement was determined and standardized by placing the screw approximately 1cm caudal to the midpoint of the medial collateral ligament of the medial femorotibial joint. To place the screw, a 4.5mm hole was first drilled in a proximomedial to distolateral direction into the cis cortex until the defect was encountered. This hole represented the glide hole. A drill guide with a 4.5 mm outer diameter and 3.2 mm inner diameter was placed into the glide hole. A hole was then drilled in the trans cortex with a 3.2 mm drill bit placed through the drill guide until the bit exited through the condyle. This hole represented the thread hole. The screw hole was then countersunk 2-3 mm and the entire hole was measured for screw length. The thread hole was then tapped with a 4.5 mm tap, and a 4.5 mm fully threaded cortical bone screw placed in lag fashion across the medial femoral condyle subchondral bone defect in a proximomedial to distolateral direction. The limbs were then loaded in axial compression for a third time and strain measurements were re-recorded as previously described.

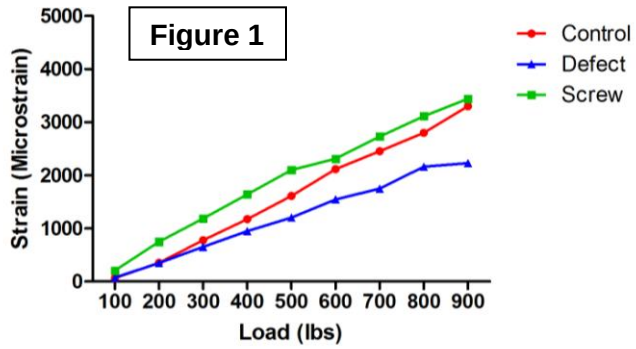
Statistical Analysis:

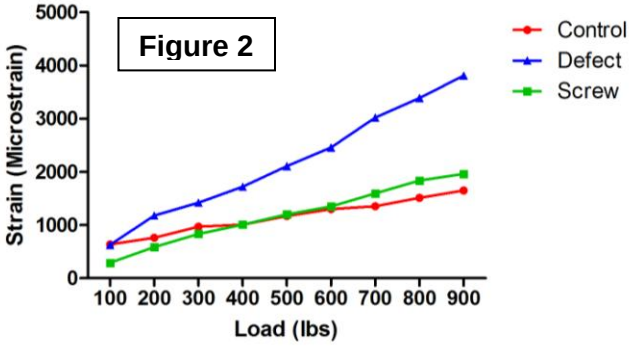
As mentioned above, strain recordings were made every 0.25 seconds for 20 seconds of testing at each 100 lb load; thus creating 80 strain measurements for each load applied. These 80 strain recordings were averaged together to create one strain

value for each 100 lb load. Therefore, each testing specimen had 9 values (one for each 100 lb load from 100 to 900 lbs) for each of the 3 testing runs (control, condyle with defect, and condylar defect with internal fixation). Each of the values was then entered into GraphPad Prism under the appropriate load measured (i.e. 100 lb, 200 lb, etc) for each of the controls, condylar defects, and condylar defects with internal fixation. Once entered, each load had strain measurements for each of the 3 testing runs. This strain data at all loads was evaluated and standard error of the means was calculated for the data. The data was then tested for normal distribution with a D'Agostino and Pearson omnibus normality test, and the controls, condylar defect group, and condylar defect with internal fixation group were statistically compared with a paired t-test at all recorded loads. Significance was determined at $p<0.05$.

Results

Figure 1 displays the effects of the creation of a subchondral bone defect in the medial femoral condyle and on bone strain measured from just cranial to the medial collateral ligament in a proximal to distal orientation compared to strain measured before the creation of the defect. It also demonstrates the effects of placing a transcortical bone screw within that defect under stepwise loading. Evaluation of **figure 1** shows that as load is increased compressive strain is increased at this point. When the defect is created the amount of strain is decreased; however when a transcortical screw is placed across this defect the strain increases to above the





level measured before the defect was created. **Figure 2** demonstrates that under identical testing conditions, strain on the meniscus is increased with the creation of a subchondral bone cyst. This increase in strain is reduced back to the levels of the pre-defect control with the placement of the transcondylar screw. These results would indicate that changes to femoral bone strain induced by a subchondral bone defect can translate into altered load on the meniscus. Furthermore, placement of a transcortical screw in this defect appears to "protect" the meniscus from the increased load and strain translated to it following creation of the bone defect.

Conclusion

These findings indicate that the experimental creation of a subchondral "cyst-like" defect in the medial condyle of the femur decreases strain on the periphery of the femur, while increasing strain on the periphery of the meniscus. We believe that a similar scenario could exist in naturally occurring medial femoral condylar bone cysts. Due to Wolff's Law, bone strain is essential for the initiation of bone production. Therefore, we believe that the decreased strain observed in the medial femoral condyle could explain why subchondral bone cysts in this area frequently do not heal. Furthermore, in patients with chronic cysts, we believe that the increased strain observed on the meniscus could lead to increased soft tissue damage.

The findings of this study also demonstrate that placing a 4.5mm cortical bone screw in lag fashion across a subchondral defect increases the strain measured in the

periphery of the femur beyond that seen in the control group. It is believed that this increase in strain could be the inciting factor needed to establish healing of the subchondral defect with bone. Furthermore, it was observed that placement of the transcondylar screw also decreased strain on the periphery of the meniscus to levels similar to that observed in the control group. This study suggests that treatment of medial femoral condylar cysts with internal fixation may promote healing of the defect, while minimizing the soft tissue damage associated with this pathologic condition. This study also presents experimental data that could be used for the establishment of clinical testing of this novel treatment for subchondral bone cysts.

References

1. McIlwraith CW. Subchondral cystic lesions (osteochondrosis) in the horse. *Comp cont Educ pract Vet* 1982; 4:S282-S291.

2. Hogan PM, McIlwraith CW, Honnas CM, et al. Surgical treatment of subchondral cystic lesions of the third metacarpal bone: results in 15 horses (1986-1994). *Equine Vet J* 1997; 29(6):477-482.

3. Bramlage LR. Osteochondrosis-related bone cysts. *Proc Am Ass equine Practnrs* 1993;39:83-85

4. von Rechenberg B, McIlwraith CW, and Auer JA. Cystic bone lesions in horses and humans: A comparative review. *Vet Comp Orthop Traumatol* 1998;11:8-18

5. Kold SE, Hickman J, and Melsen F. An experimental study of the healing process of equine chondral and osteochondral defects. *Equine Vet J* 1986;18:18-24.

6. Ray CS, Baxter GS, McIlwraith CW, et al. Development of subchondral cystic lesions after articular cartilage and subchondral bone damage in young horses. *Equine Vet J* 1996;28:225-232.

7. von Rechenberg B, Akens MK, Nadler D, et al. Changes in subchondral bone in cartilage resurfacing: An experimental study in sheep using different types of osteochondral grafts. *Osteoarthritis Cartilage* 2003;11:265-277.

8. von Rechenberg B, Akens MK, Nadler D, et al. The use of photooxidized, mushroom structured osteochondral grafts for cartilage resurfacing: A comparison to photooxidized cylindrical grafts in an experimental study in sheep. *Osteoarthritis Cartilage* 2004;12:201-216.

9. Howard RD, McIlwraith CW, and Trotter GW. Arthroscopic surgery for subchondral cystic lesions of the medial femoral condyle in horses: 41 cases (1988-1991). *J Am Vet Med Assoc* 1995;206:842-850.

10. Wallis TW, Goodrich LR, McIlwraith CW, et al. Arthroscopic injection of corticosteroids into the fibrous tissue of subchondral cystic lesions of the medial femoral condyle in horses: a retrospective study of 52 cases (2001-2006). *Equine Vet J* 2008;40(5):461-7.

11. Bodo G, Hangody L, Modis L, and Hurtig M. Autologous osteochondral grafting (mosaic arthroplasty) for treatment of subchondral cystic lesions in the equine stifle and fetlock joints. *Vet Surg*. 2004;33(6):588-96.

12. Jackson WA, Stick JA, Arnoczky SP, and Nickels FA. The effect of compacted cancellous bone grafting on the healing of subchondral bone defects of the medial femoral condyle in horses. *Vet Surg.* 2000;29(1):8-16.