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Articular cartilage MR imaging and thickness mapping of a loaded knee joint before and after meniscectomy[1]

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Summary

Objective: We describe a technique to axially compress a sheep knee joint in an MRI scanner and measure articular cartilage deformation. As an initial application, tibial articular cartilage deformation patterns after 2 h of static loading before and after medial meniscectomy are compared.

Methods: Precision was established for repeated scans and repeated segmentations. Accuracy was established by comparing to micro-CT measurements. Four sheep knees were then imaged unloaded, and while statically loaded for 2 h at 1.5 times body weight before and after medial meniscectomy. Images were obtained using a 3D gradient echo sequence in a 4.7 T MRI. Corresponding 3D cartilage thickness models were created. Nominal strain patterns for the intact and meniscectomized conditions were compared.

Results: Coefficients of variation were all 2% or less. Root mean squared errors of MR cartilage thickness measurements averaged less than 0.09 mm. Meniscectomy resulted in a 60% decrease in the contact area ($P = 0.001$) and a 13% increase in maximum cartilage deformation ($P = 0.01$). Following meniscectomy, there were greater areas of articular cartilage experiencing abnormally high and low nominal strains. Areas of moderate nominal strain were reduced.

Conclusions: Medial meniscectomy resulted in increased medial tibial cartilage nominal strains centrally and decreased strains peripherally. Areas of abnormally high nominal strain following meniscectomy correlated with areas that are known to develop fibrillation and softening 16 weeks after medial meniscectomy. Areas of abnormally low nominal strain correlated with areas of osteophyte formation. Studies of articular cartilage deformation may prove useful in elucidating the mechanical etiology of osteoarthritis.

Key words: Articular cartilage, Meniscectomy, Cartilage thickness, Contact area, Osteoarthritis, MR imaging, Joint loading.

Introduction

Osteoarthritis (OA) is among the most common diseases in orthopedics and a leading cause of physical disability and health care expense throughout the world. Approximately 11% of people aged over 65 and 6% of entire U.S. adult population suffer from symptomatic OA[1]. Various factors, including mechanical insults such as meniscectomy, intra-articular fracture, mechanical malalignment, and ligamentous instability, alter the mechanical environment on the articular surface and can initiate OA[2].

Meniscectomy is a common and successful surgical procedure for treating pain and mechanical locking of the knee after a meniscus tear. However, several studies have shown that a large number of meniscectomized patients subsequently develop OA[3,4]. Post-meniscectomy OA of the knee is generally believed to be a consequence of biomechanical alterations at the knee due to the absence of a meniscus to distribute the joint reaction force, absorb impact force, and maintain joint stability.

Experimental and computational models have demonstrated increases in articular cartilage contact stress following meniscectomy[5,6], and in animal models articular cartilage degradation and subchondral bone changes have been reported[7–11]. More recently, Appleyard *et al.* and Oakley *et al.* mapped region-specific cartilage morphology and material properties following meniscectomy in a sheep model of OA[12,13]. Sixteen weeks after medial meniscectomy, the medial tibial articular cartilage developed fibrillation and softened centrally, while osteophytes developed peripherally[13].

Various mechanical factors are believed to initiate and promote the progression of OA[14]. Among these factors, joint unloading has been postulated to result in decreased articular cartilage fluid pressure and a progression of the subchondral growth front[14]. Osteophytes are commonly seen in such areas[15]. Conversely, articular cartilage has been noted to be fibrillated in areas of pathologic overloading[16].

Though contact stress has been previously studied in the laboratory, the region-specific deformation in the articular cartilage tissue itself has not been previously mapped in a knee before and after meniscectomy. To understand how known alterations of contact stress translate to known patterns of articular cartilage degeneration, it is necessary to characterize the deformation of the articular cartilage tissue. It is likely that the local deformation of the tissue is

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involved in transmitting the mechanical signal from the surface to the chondrocyte, the active element embedded in the tissue that is responsible for matrix synthesis and degradation. Further, it is likely that areas of increased cartilage deformation are more susceptible to mechanical damage and fibrillation.

Various methods have been described to determine articular cartilage thickness or deformation in an intact joint. Among those methods, computed tomography (CT) and scanning electron microscopy (SEM) have been shown to be capable of providing high resolution cross sectional images of cartilage[17,18]. CT scans, however, do not delineate the interface between soft tissues, such as cartilage–cartilage or cartilage–meniscus boundaries, well when entire intact joint is scanned and these surfaces are pressed together. SEM is incapable of revealing the thickness of the articular cartilage over its entire surface and through its depth, and necessitates destroying the specimen, making repeated measurements of the same specimen impossible.

MRI is a promising technique for articular cartilage imaging because it reveals the superficial surface and the rim to subchondral bone over the entire cartilage surface. It also has the advantage of being non-invasive, allowing maintenance of periarticular supporting structures, and allowing repeated measurements of the same joint under various loading conditions. Several studies have evaluated the deformation of articular cartilage in tissue plugs and exposed articular surfaces with mechanical loading[19–23], and another has evaluated the *in vivo* and *in vitro* deformation of articular cartilage from the patella-femoral joint during mechanical loading[24,25]. At the tibio-femoral joint, MRI has been used *in vivo* to evaluate the deformation of articular cartilage after various activities such as running[26]. There have been, however, no MRI studies done on the tibio-femoral joint to evaluate articular cartilage deformation while load is applied, and no MRI studies have evaluated articular cartilage deformation following OA-inducing changes to the joint such as meniscectomy. Information about articular cartilage deformation in an intact knee joint with and without a meniscus could shed light on the pathogenesis of OA after meniscectomy.

The purpose of this study was to develop a device that can accurately and precisely measure cartilage deformation in a sheep knee joint under axial compressive loading at a physiologic magnitude. As an initial application of this device, we compared tibial articular cartilage deformation before and after medial meniscectomy under static loading and steady state conditions. We postulated that the pattern of tibial plateau articular cartilage deformation in a meniscectomized knee is different than in an intact knee. Specifically, we hypothesized that in a statically loaded knee joint following meniscectomy, the contact area will decrease, the maximum cartilage deformation will increase, and the pattern of articular cartilage deformation will be significantly changed. We further hypothesized that changes in articular cartilage deformation with meniscectomy will relate to articular cartilage degeneration patterns that are seen *in vivo*.

Methods

LOADING SYSTEM

The entire loading system consists of a cylindrically shaped MR compatible pneumatic loading apparatus and an electric air flow control device. The loading apparatus (Fig. 1) holds a joint specimen and applies uniaxial compressive load across the tibio-femoral joint inside a 4.7 T MRI scanner. A flow control device (Fig. 2) controls the air flow to generate both static and cyclic compressions. For imaging of cyclically loaded specimens, it also provides a gate signal to the MRI scanner synchronized with the loading cycle.

The loading apparatus is sized to test a sheep knee. It consists of an outer cylinder, a supporting shell, a loading shell, and an air bladder. The outer cylinder is a container holding the specimen and fluid, and is a frame for the loading mechanism. The outside diameter of the cylinder fits into the 6 cm radio-frequency coil of the 4.7 T MRI scanner. The cylinder is made of high strength cast acrylic to maintain its original shape under high pressure, to resist fatigue failure after numerous loading cycles, and to allow X-ray transmission should micro-CT applications be needed. The supporting shell is also made of high strength cast acrylic and is fixed on the bottom part of the outer cylinder. An acrylic pin on the supporting shell anchors the outer part of the tibia when the specimen is in place, preventing anterior/posterior rotation and medial/lateral movement of the tibial portion of the specimen. PVC tubing is used for the loading shell because of its flexibility. The loading shell is driven by an inflatable air bladder in the gap between the loading shell and the outer cylinder, and is guided by two guide pins on the top of the outer cylinder. The bladder has two air inlets to

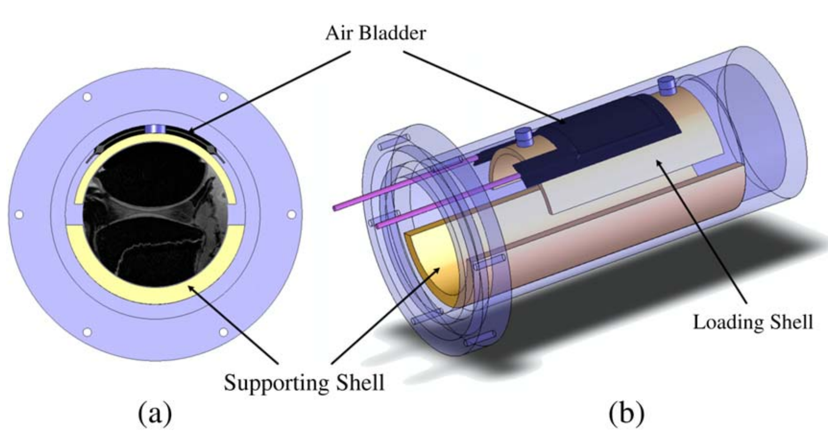

Fig. 1. (a) A front view and (b) perspective drawing of the pneumatic loading apparatus are shown.