Magnetic Resonance Imaging Analysis of
Kinematics in Osteoarthritic Knees

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Abstract: Kinematics in osteoarthritic knees may be impeding efforts to reproduce “normal” knee kinematics in the prosthetic knee. Fourteen subjects with unilateral symptomatic knee osteoarthritis performed a supine leg press from 0° to 90° flexion against a 150-N load. The tibiofemoral contact pattern was recorded for both knees using sagittal T1-weighted magnetic resonance imaging. Severity of osteoarthritis ranged from Kellgren Lawrence grade 2 to 4 in the symptomatic knees and from 0 to 3 in the contralateral knees. Contact in the lateral and medial compartments of osteoarthritic knees was more anterior on the tibial plateau than healthy knees, both in knee extension and to 90° flexion (P < .01). This anterior contact pattern was associated with severity of osteoarthritis (P < .01). Key words: total knee arthroplasty, knee osteoarthritis, knee kinematics, MRI.

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Osteoarthritis (OA) of the knee is a widespread problem, yet very little is known about kinematic changes due to OA. The incidence of total knee arthroplasty (TKA) is increasing, with the Swedish Arthroplasty Register reporting a 5-fold increase in TKA performed annually since 1976 [1], and in Australia, knee arthroplasty has overtaken hip arthroplasty for the first time [2]. The demand for TKA highlights makes it imperative to understand the kinematics of osteoarthritic knees and prosthetic knees.

The few published studies have shown that some characteristics of normal knee motion are lost in osteoarthritic knees, including the axial rotation that normally occurs at terminal extension [3,4]. In healthy knees, the axial rotation of the knee during flexion has been described in vitro [5-7] and in vivo [7-9]. But the loss of axial rotation may be associated with the severity of OA in the knee [4,10]. The loss of normal axial rotation during flexion in osteoarthritic knees could be due to either osteophytes, contracture, and thickening of capsular tissues constraining joint mobility or attrition of the cruciate ligaments (ACL), which have a role in controlling axial rotation [11].

Magnetic resonance imaging (MRI) has been used to analyze kinematics of healthy [8,9,12] and ACL-injured knees [13] by mapping the flexion axis through the femoral condyle. These studies have...
demonstrated axial rotation of the knee occurring during knee flexion from 0° to 120° with the rotation axis passing through the medial compartment of the knee [8]. Magnetic resonance imaging has also been used to analyze kinematics by mapping tibiofemoral contact patterns in healthy and ACL-injured knees [14,15]. Magnetic resonance imaging has not yet been used to describe the kinematic characteristics of osteoarthritic knees.

Osteoarthritis is assessable by MRI because changes to bone, soft tissue, and cartilage structures are all visible [16,17], and areas of damage to the articular cartilage can be recorded reliably [18-20]. Bone features that are visible on MRI include osteophytes, subchondral sclerosis, subchondral hematoma, and cysts [16]. In the present study, MRI was used to assess damage to the articular cartilage and menisci and ligament integrity in the osteoarthritic knees.

The aim of this study was to map the tibiofemoral contact pattern in osteoarthritic knees between 0° and 90° knee flexion and describe the differences between the contralateral asymptomatic knees and healthy knees. The associations between differences in the tibiofemoral contact pattern and severity of OA were also examined.

Method

Subjects

We recruited 14 subjects with symptomatic OA in one knee and no symptoms or history of OA in the contralateral knee (Table 1). Of the subjects, 3 were men, and 11 were women. Age of the subjects ranged from 54 to 81 years (mean ± SD, 65 ± 9.1 years). A total of 10 subjects had primary idiopathic arthritis, and 4 had OA secondary to injury. Four subjects had chronic anterior cruciate ligament injuries, including one subject who had an extra-articular knee reconstruction 12 years ago. One subject had OA secondary to patella fracture through the articular surface. There were 8 who had OA in the right knee and 6 in the left. Subjects were excluded if they had contraindications to MRI, including ferrous metal implants, pacemakers, and claustrophobia.

Twelve healthy subjects were used as controls for the comparison of knee kinematics. These subjects were described in a previous work [15]. These healthy subjects were aged 20 to 50 years; 7 were men, and 5 were women. None had any symptoms or history of injury in either knee. All subjects provided informed consent according to the approval of the University and Department of Health human research ethics committees.

Kinematic MRI

Kinematic MRI scans were performed as subjects performed a leg press in supine position. Sagittal images were taken of both knees at seven 15° intervals from full knee extension to 90° flexion. Subjects were scanned at each knee flexion angle twice, once while relaxed (unloaded) and again pressing down against a footplate weighted with a 150-N load (loaded). Eight slices were generated through each knee approximately 10 mm apart

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Side</th>
<th>OA Type</th>
<th>Kellgren Lawrence Grade</th>
<th>Ligament Status</th>
<th>Compartment Most Affected</th>
<th>Meniscal Damage</th>
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Details of joint damage were recorded from MRI and operation reports. Indicates normal appearance; d.tear, degenerative tear seen at surgery or abnormal signal intensity indicative of a tear; macerated, severe late stage degeneration with extensive tissue damage; F, female; M, male; R, right; L, left; PAM, previous arthroscopic meniscectomy; 1°, primary idiopathic OA; 2°, secondary OA as a result of injury, metabolic, or mechanical stress [21]; PCL, posterior cruciate ligament; MCL, medial collateral ligament.
using a T1-weighted spoiled gradient echo sequence (TR (repetition time), 160.0; TE (echo time), 2.3/1; TI (inversion time), 00:46) with a 256 × 256 matrix. The digital images were analyzed using Osiris software (Université de Genève, Switzerland).

The tibiofemoral contact point was measured as the distance from the posterior tibial cortex to the point at which the femoral condyle contacted the tibial plateau (Fig. 1). Because of the range of sizes of the knees studied, distances were scaled to a tibial plateau of mean proportions (Fig. 5). The reliability of this method was tested, including sources of error from choice of image slice, choice of MRI sequences, and accuracy of measurement, and was found to be good with an intraclass correlation of 0.94 (99% confidence interval, 0.91-0.97) [15].

Range of motion was measured in both knees using a goniometer in sitting. During the procedure for kinematic MRI, the limit of knee extension for each subject was used to measure the initial tibiofemoral contact point (Fig. 1).

**Details of OA and Damage in the Knee Joint**

The symptomatic and contralateral knees of subjects were graded for severity of OA as described by Kellgren and Lawrence [22] (Table 2).

Diagnostic MRI scans of the symptomatic osteoarthritic knee were used to record details of articular cartilage damage, meniscal damage, and ligament integrity in the knee joint. Magnetic resonance imaging sequences included proton density and STIR (short TI Inversion Recovery) sequences, T2 sagittal, coronal and axial sequences, and sagittal T1 sequences. A radiologist experienced in musculoskeletal MRI reported the images. Details of ligament, articular cartilage, and meniscal damage were also obtained from operating theatre records (Table 1).

**Statistical Analysis**

Differences between tibiofemoral contact patterns were compared for the medial and lateral compartments of the knee, for the loaded and unloaded conditions, and for differences between osteoarthritic and contralateral knees of both osteoarthritic subjects and healthy subjects using repeated measures analysis of variance.

Differences in tibiofemoral contact patterns associated with severity of OA were examined using repeated measures analysis of variance. Where differences were found, a Bonferroni post hoc analysis was conducted.

**Results**

**Severity of Disease in the Osteoarthritic Knees**

Range of motion of osteoarthritic knees was restricted compared with the contralateral asymptomatic knee (P = .009). In the osteoarthritic knees, flexion was 107° ± 9° (range, 83°-135°), and extension was 2° ± 3.8° (range, -5° to 10° extension), with 6 subjects unable to extend the knee to 0°. In the contralateral asymptomatic knee, flexion was 133° ± 13° and extension was −1° ± 2°.

The osteoarthritic knees of subjects showed one knee had Kellgren Lawrence grade 2, 5 knees had grade 3, and 8 knees had grade 4 OA. Magnetic resonance imaging reports described severe tricompartmental OA with full-thickness cartilage wear in...
Fig. 2. Gradient echo images of the medial and lateral compartments of the knee of a typical healthy subject, from 0° to 90° flexion. The medial femoral condyle was anterior on the tibial plateau in extension, then during flexion remained centered on the tibial plateau. The lateral femoral condyle continued to roll back through flexion.
Fig. 3. Gradient echo images of the medial and lateral compartments of the osteoarthritic knee of a typical subject, from 0° to 90°. The medial femoral condyle was fixed anteriorly on the tibial plateau. The lateral femoral condyle did not roll back through flexion but remained centered on the tibial plateau.
7 subjects. Six knees had full thickness cartilage wear in the medial compartment only, with less severe OA in the lateral and patellofemoral compartments. No knees had predominantly lateral compartment OA. One knee had patellofemoral OA, with minor changes in the lateral compartment. A total of 10 knees had medial meniscus damage, and 8 knees had lateral meniscus damage. Eight knees had ligament damage including 7 with ACL-deficiency confirmed at surgery. One ACL-deficient knee had concomitant PCL degeneration and degenerative MCL and LCL tears. Another ACL-deficient knee had a concomitant old MCL tear. One knee had an isolated LCL tear seen at surgery (Table 1).

**Severity of Disease in the Asymptomatic Knee**

In the asymptomatic knees of 7 of the 14 subjects, OA appeared on x-ray. Of the asymptomatic knees,
5 had grade 0 (normal) and 3 had grade 1 (doubtful) radiographs on the Kellgren Lawrence scale. However, 4 asymptomatic knees had grade 2 (minimal), and 2 subjects had grade 3 (moderate) OA on x-ray. These 7 asymptomatic knees with evidence of OA on x-ray included 6 with primary idiopathic OA and 1 subject with OA secondary to ACL injury in the contralateral knee.

Tibiofemoral Contact Patterns

The tibiofemoral contact pattern of osteoarthritic subjects did not translate as far posterior on the tibial plateau during knee flexion as the healthy control subjects \( (F_{(1,1884)} = 19.3; P < .001) \). Post hoc analysis showed the contact pattern was limited in posterior translation in both symptomatic \( (P < .001) \) and asymptomatic knees \( (P < .001; \text{Fig. 4}) \). The symptomatic and contralateral asymptomatic knees of osteoarthritic subjects were not significantly different \( (F_{(1,104)} = 1.475; P = .227) \).

In healthy subjects, the medial compartment tibiofemoral contact pattern began in knee extension anteriorly on the tibial plateau (mean distance from the posterior tibial cortex, 34 ± 4 mm). The femur then rolled back on the tibial plateau to 30° knee flexion (to be 21 ± 3 mm from the posterior tibial cortex). For the remainder of knee flexion to 90°, the healthy knee moved little, moving back to 19 ± 2 mm from the posterior tibial cortex at 90° knee flexion (Fig. 4). In this respect, the healthy medial condyle could be described as predominantly rolling between 0° and 30° and gliding between 45° and 90°. In osteoarthritic knees, the medial compartment had similar contact in knee extension, but the femur did not move back on the tibial plateau as far, remaining 26 ± 5 mm from the posterior tibial cortex, that is, a difference of 5 mm more anteriorly than the healthy knee contact position.

In the lateral compartment of the symptomatic osteoarthritic knees, the tibiofemoral contact did not move posteriorly during flexion as far as healthy control subjects (Figs. 2 and 3). In the healthy knees, the femur moved from an anterior position on the tibial plateau in knee extension (27 ± 4 mm from the posterior tibial cortex reference point) to a posterior position at 90° knee flexion (11 ± 2 mm from the posterior tibial cortex) fairly steadily. The femoral contact pattern of the osteoarthritic knees did not move back as far on the tibial plateau: in knee extension, the contact point was 29 ± 5 mm from the posterior tibial cortex, and at 90° knee flexion, it had only rolled back to 19 ± 4 mm from the posterior tibial cortex. Thus, there was less rollback in both the medial and lateral compartments of the symptomatic osteoarthritic knees (Fig. 3).

Loading or unloading the knees did not change the tibiofemoral contact pattern of the osteoarthritic knees \( (F_{(1,104)} = 0.095; P < .759) \) or the asymptomatic knees of osteoarthritic subjects \( (F_{(1,104)} = 1.184; P < .909) \) (Figs. 4 and 5).

Knees with more severe OA, as recorded by the Kellgren Lawrence grades, showed incrementally less posterior translation of tibiofemoral contact patterns during knee flexion \( (F_{(1,4)} = 8.6; P < .001) \) (Fig. 6). Post hoc analysis explained that although there was no difference between contact patterns of knees with Kellgren Lawrence grades 0 and 1 (minimal and doubtful OA), there was significantly less posterior translation of contact points between grade 0 and grade 3 \( (P = .001) \) and grade 4 knees \( (P = .014) \). Knees with Kellgren Lawrence grade 2 had a different tibiofemoral contact pattern from those with grade 3 \( (P = .012) \). There was no significant difference detected between the contact pattern of knees with Kellgren Lawrence grades 3 and 4 \( (P = .689; \text{Fig. 6}) \).
An interaction was seen between the tibiofemoral contact pattern and severity of OA and the medial or lateral compartments ($F_{(1,2)} = 3.2; P = .014$), indicating that disease severity affected the contact pattern in lateral compartment more than in the medial compartment. In the lateral compartment, when the knees were flexed at $90^\circ$, the tibiofemoral contact point of the healthy knee was $11.7 \pm 1.6$ mm from the posterior tibial cortex. In grade 2 OA, the contact point was $15.9 \pm 1.5$ mm, and in grade 4 OA, the tibiofemoral contact was $19.9 \pm 2.3$ mm from the posterior tibial cortex. However, the power of the statistical analysis to detect changes is reduced by the small sample sizes of the groups for each Kellgren Lawrence grade. For statistical analysis, the changes in the lateral compartment were detected with power of at least 80%; however, in the medial compartment, the power was insufficient to detect significant changes between the 4 Kellgren Lawrence grades.

**ACL integrity and kinematics**: The 7 subjects with ACL-deficient knees did not have a significantly
different contact pattern to the ACL-intact knees ($F_{(1,1)} = 1.062; P = .304$). There was also no significant difference when the contact pattern of the ACL-deficient knees was considered in the medial and lateral compartments separately ($F_{(1,1)} = 2.89; P = .092$) (Fig. 6).

**Discussion**

This study has demonstrated how the kinematic characteristics of osteoarthritic knees differ from healthy knees. The tibiofemoral contact pattern remained relatively anteriorly on the tibial plateau compared with healthy controls. Loss of the normal posterior movement of femoral contact demonstrated the change to the normal roll/glide characteristics of knee motion. In the healthy knee, the femur predominantly rolls across the tibial plateau between $0^\circ$ and $30^\circ$, then from $30^\circ$ onward, the femur combines roll with glide on the surface of the tibial plateau; otherwise, it would reach the posterior tibial plateau rim before flexion was completed [23]. In addition, insufficient rollback would cause the shaft of the femur to impinge on the posterior tibial rim, also limiting knee flexion. Control of the roll/glide behavior was attributed to the cruciate ligaments [23]. In the osteoarthritic knee, the amount of rollback was reduced, and tibiofemoral contact occurred anteriorly on the tibial plateau. This loss of normal rollback may be a contributor to reduced flexion range of motion.

Reduced range of knee flexion often accompanies OA [24]. In several studies of deep flexion of healthy knees, the medial femoral condyle is described sitting upon the posterior horn of the meniscus and the lateral femoral condyle rolled back to the posterior rim of the tibial plateau, also taking the posterior horn of the meniscus back [25]. In TKA, loss of normal femoral rollback has been considered a limiting factor in achieving knee flexion, and cruciate retaining prostheses have been designed to facilitate rollback [26]. It remains to be seen whether limited rollback in osteoarthritic knees is related to similar kinematics post arthroplasty.

The loss of the normal rollback in the osteoarthritic knees was more apparent in the lateral than the medial compartment. In the healthy knees, it is the rollback of the lateral femoral condyle while the medial femoral condyle remains centered on the tibial plateau that indicates the axial rotation of the knee during flexion. The loss of rollback in the lateral compartment, in particular, demonstrated the loss of axial rotation in the osteoarthritic knees [12]. Loss of axial rotation in osteoarthritic knees has previously been described in standing subjects in the last $30^\circ$ knee extension [3]. Further studies are needed to explore whether loss of axial rotation is implicated in disease progression. Kinematic changes were apparent in early stages of OA and in asymptomatic knees with radiographic signs of OA. Furthermore, knees with grade 2 OA already had a different tibiofemoral contact pattern to healthy knees.

Medial compartment contact patterns may be related to wear in the medial tibial plateau. The tibiofemoral contact pattern was more anterior on the tibial plateau throughout knee flexion in knees with grade 3 and 4 OA than in healthy knees. At TKA, anteromedial wear areas have been described on the tibial plateau [27]. These wear areas increase in size as the severity of knee OA increases [28,29]. It is possible that the area of wear in knees with grade 3 OA tends to hold the femur in the wear cupola, anteriorly on the tibial plateau, and restrains the rollback of the femoral condyle.

The severity of OA of the knees of subjects in this study ranged from minimal to severe in the symptomatic knees and from normal to moderate in the asymptomatic knee. The high incidence of asymptomatic radiographic OA in the contralateral knees was probably because many of these subjects had primary idiopathic OA, which is most commonly bilateral [30]. One subject had an ACL injury in the symptomatic knee but may have had concurrent primary idiopathic OA. Therefore, it is not surprising that 7 of 14 subjects had asymptomatic OA in the contralateral knee.

The incidence of ACL deficiency in the present study was similar to that previously reported [31,32]. However, conclusions about the attrition of the ACL in knee OA cannot be drawn from this small sample of convenience. Intraoperatively, Wada et al [31] found 50% of knees had a completely absent ACL, 25% partly torn, and only 25% had an intact ACL [31]. The PCL, however, was intact in all the knees. Allain et al [32] found similar incidence of ACL damage in osteoarthritic knees, but in 75% of cases where the ACL was torn, the PCL also had histological evidence of degeneration. He suggested that the absence of the ACL indicated the quality of the PCL as a viable restraint structure was probably also compromised. The menisci also are frequently found to be macerated or absent in advanced knee OA [33]. In our sample of 14 subjects, only 1 had no meniscal damage. Thus, the evidence of joint damage in this sample of osteoarthritic subjects is consistent with that reported by others.
Although both medial and lateral compartments exhibited a loss of the normal posterior translation of contact points during knee flexion, the loss of posterior translation was greatest in the lateral compartment of osteoarthritic knees. In the healthy knees, the lateral compartment contact pattern demonstrated that the femur moved posteriorly during knee flexion from 30° to 90°, more than in the medial compartment, which indicated the axial rotation of the knee during flexion. The changes to knee kinematics were related to the severity of OA.

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References