LAXITY IN HEALTHY AND OSTEOARTHRITIC KNEES

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Objective. Although it is a cause of osteoarthritis (OA) in animal models, laxity in human knee OA has been minimally evaluated. Ligaments become more compliant with age; whether this results in clinical laxity is not clear. In theory, laxity may predispose to OA and/or result from OA. Our goals were to examine the correlation of age and sex with knee laxity in control subjects without OA, compare laxity in uninvolved knees of OA patients with that in older control knees, and examine the relationship between specific features of OA and knee laxity.

Methods. We assessed varus–valgus and antero–posterior laxity in 25 young control subjects, 24 older control subjects without clinical OA, radiographic OA, or a history of knee injury, and 164 patients with knee OA as determined by the presence of definite osteophytes. A device was designed to assess varus–valgus laxity under a constant varus or valgus load while maintaining a fixed knee flexion angle and thigh and ankle immobilization. Radiographic evaluations utilized protocols addressing position, beam alignment, magnification, and landmark definition; the semiflexed position was used, with fluoroscopic confirmation.

Results. In the controls, women had greater varus–valgus laxity than did men (3.6° versus 2.7°; 95% confidence interval [95% CI] of difference 0.38, 1.56; \( P = 0.004 \)), and laxity correlated modestly with age (\( r = 0.29, P = 0.04 \)). Varus–valgus laxity was greater in the uninvolved knees of OA patients than in older control knees (4.9° versus 3.4°; 95% CI of difference 0.60, 2.24; \( P = 0.0006 \)). In OA patients, varus–valgus laxity increased as joint space decreased (slope \(-0.34; 95\%\) CI \(-0.48, -0.19; P < 0.0001\)) and was greater in knees with than in knees without bony attrition (5.3° versus 4.5°; 95% CI of difference 0.32, 1.27; \( P = 0.001 \)).

Conclusion. Greater varus–valgus laxity in the uninvolved knees of OA patients versus older control knees and an age-related increase in varus–valgus laxity support the concept that some portion of the increased laxity of OA may predate disease. Loss of cartilage/bone height is associated with greater varus–valgus laxity. These results raise the possibility that varus–valgus laxity may increase the risk of knee OA and cyclically contribute to progression.

A widely used paradigm depicts osteoarthritis (OA) in a specific joint as the result of local mechanical factors acting within a susceptibility-determining milieu (1). Variations in these factors affect the magnitude and distribution of the load transmitted to the articular milieu. Although mechanical factors are believed to be essential to the pathogenesis of knee OA (2,3), their contribution to disease development and progression in humans has been minimally examined.

Knee stability is an important component of the mechanical environment of the knee. Knee instability or laxity may be broadly defined as abnormal displacement or rotation of the tibia with respect to the femur (4). In the unloaded state, stability is provided by the ligaments, capsule, and other soft tissues, and in the loaded state by interactions between ligaments, other soft tissues, condylar geometry, and tibiofemoral contact forces generated by muscle activity and gravitational forces (4,5). Under dynamic conditions, normal knee mechanics and functional stability depend upon proprioceptive input and reflex and centrally driven muscle activity (6). As clinically assessed, joint laxity reflects an impairment of the passive restraint system for which muscle activity may or may not compensate. Laxity may adversely affect knee mechanics (7,8).

Different lines of investigation suggest that knee...
laxity may contribute to the development and progression of knee OA. First, OA develops in a canine model by inducing an unstable joint via complete anterior cruciate ligament (ACL) transection (9,10). Second, laxity is associated with abrupt motion with large displacements and suboptimal distribution of larger forces over the articular cartilage (7,8). Third, an association between generalized joint hypermobility syndrome and OA is suggested by clinic-based studies (11,12), although it is not clear that any predisposition is specific to the hypermobile sites. Fourth, with aging, both a decline in the material properties of knee ligaments (13–16) and an increase in the incidence of OA (17) occur.

While laxity may contribute to knee OA development and/or progression, it may also be a consequence of moderate-to-severe OA. In subjects without arthritis, laxity at the knee might represent primary capsuloligamentous laxity (possibly related to genetic factors or aging-related soft tissue changes) or the consequence of injury. Sex (18,19), ethnic factors (19–21), and genetic factors (19,22) appear to contribute to generalized hypermobility. In patients with knee OA, laxity, in theory, may relate to loss of cartilage and/or bone height (sometimes described in clinical settings as “pseudolaxity”), chronic capsuloligamentous stretch, or combinations of ligamentous, meniscal, muscular, and capsular pathology.

The natural history of laxity in knee OA is poorly understood. In the canine model, cartilage is preserved for not atypically 3 years after ACL transection. This lack of progression has been attributed to buttressing osteophytic growth and capsular thickening (23). As described by Buckwalter et al (8), based on clinical experience, in some patients, unstable knees become more stable with time without signs of progression, except perhaps osteophytes. Laxity in the frontal (varus–valgus) and sagittal (anteroposterior [AP]) planes has been examined in a small number of cross-sectional studies of patients with knee OA. These studies suggest that less AP translation can be achieved at advanced stages of OA than at earlier stages (24,25). In examining varus–valgus laxity by stage of OA, previous studies have exclusively used global measures of OA severity, and the results have been inconsistent. Specific aspects of OA pathology may have opposing effects, e.g., loss of cartilage and bone are likely to lead to increased varus–valgus laxity and osteophyte growth to decreased laxity (26). The paucity of information relates in part to the difficulty in obtaining reliable measurements.

At early stages of knee OA, laxity is unlikely to be a consequence of disease. Laxity that contributes to the development of knee OA may or may not be limited to the limb displaying the OA. If laxity were exclusively the local result of OA pathology that develops at later stages, then uninvolved knees of OA patients as well as knees with mild OA should not be more lax than the knees of older control subjects.

In this study, the following hypotheses were tested. To explore whether an increase in laxity might predate the disease, we tested whether (a) among subjects with healthy knees, varus–valgus and AP laxity increase with age, (b) the uninvolved knees of knee OA patients are more lax than the knees of older control subjects without OA, and (c) knees with mild OA are more lax than knees of older control subjects without OA. To better understand the impact of specific OA changes, we tested whether joint space narrowing and bony attrition are associated with greater varus–valgus laxity in patients with knee OA.

PATIENTS AND METHODS

Patients with knee OA. One hundred sixty-four patients with knee OA were enrolled in the baseline phase of a longitudinal study examining the contribution of specific mechanical, neural, and muscular factors to radiographic progression and functional decline in knee OA. Patients were recruited from the community through advertising in 67 neighborhood organizations and senior centers, local newspapers and magazines, press releases, letters to subjects in the Aging Research Registry of the Buehler Center on Aging of Northwestern University, and referrals from local physicians.

Inclusion and exclusion criteria were based on consensus recommendations (27,28). Inclusion criteria were as follows: definite osteophytes in the medial and/or lateral tibiofemoral compartment (i.e., Kellgren and Lawrence [K/L] radiographic grade ≥2) of one or both knees; at least “a little” difficulty with knee-requiring activities. Exclusion criteria were intraarticular corticosteroid injection into either knee within the previous 3 months, uncomplicated knee surgery within the previous 6 months, complicated knee surgery within the previous year, bilateral total knee replacement, and history of avascular necrosis, rheumatoid arthritis, or any other systemic inflammatory arthropathy, periarticular fracture, Paget’s disease, villonodular synovitis, joint infection, ochronosis, neuropathic arthropathy, acromegaly, hemochromatosis, Wilson’s disease, osteochondromatosis, gout or recurrent pseudogout, or osteopetrosis.

Control subjects. Older control subjects were recruited from the Aging Research Registry of the Buehler Center on Aging of Northwestern University. Exclusion criteria were (in either knee) any pain, swelling, stiffness, or other symptoms; any history of knee arthritis or injury; flexion ≤125°; effusion, or warmth; or any radiographic feature of OA (osteophytes, decreased joint space, sclerosis, cysts, or bone contour deformity). Twenty-four older control subjects were enrolled.

Twenty-five young control subjects were recruited from the students and staff working in the Rheumatology
Division at Northwestern University Medical School. Exclusion criteria were (in either knee) any pain, swelling, stiffness, or other symptoms and any history of knee arthritis or injury.

Measurement of AP translation. Knee laxity was assessed in the sagittal and frontal planes. Laxity in the sagittal plane (AP laxity) was measured using the KT1000 knee-testing system (MEDmetric, San Diego, CA). This system reproducibly quantitates the anterior and posterior displacement of the tibia that results from known forces (29–33) and is frequently used to assess anterior stability of the knee before and after ACL reconstruction. All measurements of AP translation were performed by a single examiner (LS) following a protocol. Intraseason reliability for this examiner testing subjects with knee OA of varying body habitus was 0.95–0.99.

Measurement of varus-valgus rotation. Devices to measure knee laxity in the frontal plane are not available. Varus-valgus rotation is most commonly assessed by physical examination. However, the reliability of physical examination estimates of varus-valgus laxity is poor in knee OA; within-observer agreement was found to be 0.55 (34). We designed a simple measurement system that addressed the major sources of variation during the physical examination test: inadequate immobilization of the distal thigh and ankle, incomplete muscle relaxation, variation of the knee flexion angle, variation of the load applied during the test, and imprecise methods of measuring varus-valgus rotation with load application (34–36).

The system consisted of a bench on which the subject sat and an attached arc-shaped, low-friction track. The track was 30 cm in radius, as measured from the center of the knee, and ran medially and laterally. The distal shank (30 cm from the knee) was firmly attached to a sled, which traveled within the track. A hand-held dynamometer was fitted into either side of the sled and was used to apply a fixed load. The subject was in a seated position, with the thigh and ankle immobilized, and the study knee was maintained at 20° flexion (4). To foster muscle relaxation, a comfortable bench and attached arc fully supported the study limb and a calming environment including music was provided; the examiner assessed muscle contraction visually and by palpation, and gently encouraged the subject to relax knee muscles.

Load was applied by the examiner and an auditory signal indicated when a load of 40N (12 Nm) was reached (25). Laxity was measured as the angular deviation at the sled after application of varus and valgus load. During load application, patients were asked if they were experiencing knee pain. Also, the examiner assessed the patients for nonverbal pain reaction criteria were (in either knee) any pain, swelling, stiffness, or other symptoms and any history of knee arthritis or injury.

Radiographic measurements. A detailed protocol (37) was followed, including knee position, criteria for beam alignment relative to the center of the knee, use of radiopaque markers to account for radiographic magnification, and definition of anatomic landmarks for measurement. The AP view was obtained in the weight-bearing, semiflexed position to superimpose the anterior and posterior joint margins. The heel was fixed and the foot rotated until the tibial spines were central relative to the femoral notch. Knee position was confirmed by fluoroscopy. A weight-bearing skyline view was also obtained (37).

To measure joint space width, the femoral boundary used was the distal convex margin of the condyles. The tibial boundary was the line extending from near the tibial spine to the outer margin, across the center of the articular fossa in the midcoronal plane of the joint, as defined by the superior margin of the bright radiodense band of the subchondral cortex (37,38). The interbone distance at the narrowest points of the medial and lateral compartments was measured as recommended (27,38), using electronic calipers. Measurements were corrected for magnification. To assess bony attrition, the 4-grade (0–3) scale with atlas representations from Altman et al (39) was used. Medial tibial, medial femoral, lateral tibial, and lateral femoral surfaces were each graded. The highest grade per knee was used for analysis. Because only a small number of patients had grade 3 bony attrition, those knees with bony attrition of grade 2 or 3 were considered in one group.

Global tibiofemoral radiographic severity was scored using the K/L grading system, in which 0 = normal, 1 = possible osteophytic lipping, 2 = definite osteophytes and possible joint space narrowing, 3 = moderate/multiple osteophytes, definite joint space narrowing, some sclerosis, and possible bony attrition, and 4 = large osteophytes, marked joint space narrowing, severe sclerosis, and definite bony attrition.

All radiographs were obtained in the same unit by 1 of 2 technicians trained in these protocols. One experienced reader (LS) performed radiographic measurements and assessments using an atlas (39). Reliability for the categorical scales, using kappa coefficients, was good (joint space narrowing 0.80–0.85; K/L grade 0.86). The reliability for minimum joint space measurement was calculated from ANOVAs with repeated measures and ICCs (model 3,1). The reliability was high for both medial and lateral compartments (r = 0.95–0.98).

Other measurements. Pain was recorded as average amount of pain in the previous week, on separate 0–100 mm visual analog scales for each knee (0 = no pain, 100 = most severe pain). Functional status was assessed using the Western Ontario and McMaster Universities Osteoarthritis Index Physical Function scale (WOMAC-PF) (40,41). Likert version (higher score worse; range 0–68). Body mass index was measured as the weight (in kg)/[height (in meters)]². To ascertain sessions. Reliability was calculated using analysis of variance (ANOVA) with repeated measurements and intraclass correlation coefficients (ICCs). For within-session reliability, the ICC (model 3,4) was used because the mean of the sessions was to be used in future analyses. Between-sessions reliability was determined using the mean of the 4 measurements each day and the ICC (model 3,1). The within-session coefficients ranged from 0.85 to 0.96. The between-sessions coefficients ranged from 0.84 to 0.90.
any history of knee injury, patients with knee OA were asked in separate questions for the right and left knees: “Have you ever injured your knee badly enough to limit your ability to walk for at least a week?”

**Subgroup definitions.** Definitions were based upon the widely utilized K/L grade 2 definition of knee OA, reflecting the presence of definite osteophytes. In patients with knee OA, **uninvolved knees** were defined as knees graded K/L 0 or 1. Of the 164 patients with knee OA, 39 had unilateral involvement. Therefore, there were 39 knees graded K/L 0 or 1 among the 328 patient knees. **Knees with mild OA** were defined as those knees assigned K/L grade 2. Of the 328 OA patient knees, 154 knees were graded K/L 2.

**Statistical analysis.** To compare knee laxity (averaged for both knees) in women and men, as well as to compare all patients with knee OA with the control subjects, a *t*-test was used. The relationship between age and laxity was evaluated using a Pearson correlation coefficient.

Next, laxity in older control knees was compared with that in 2 subgroups of OA patient knees: uninvolved knees and knees with mild OA. In this analysis, laxity was analyzed as a knee-specific (as opposed to person-specific) measure. Generalized estimating equations (GEE) were used to account for correlated measures (i.e., 2 laxity measurements) from the same subject (42).

The relationship between knee-specific measures of laxity and joint space width was evaluated in OA patients by use of GEE to account for the correlated measures in the same subject. Laxity was regressed on joint space width to determine the slope between the 2 measures. Comparisons of laxity across bony attrition grade and K/L grade categories used GEE to calculate 95% confidence intervals (95% CIs) for the pairwise mean differences in laxity between the categories.

The sample size in some analyses may have been too small to satisfy the asymptotic assumptions of GEE; when right and left knees were examined in separate analyses, the results were not affected.

**RESULTS**

Characteristics of the older control subjects and of the patients with knee OA are provided in Table 1. Of the 164 patients, in 76 the highest K/L grade was 2, in 53 the highest grade was 3, and in 35 the highest grade was 4. The mean pain scores in patients with OA were 36.8 mm (±26.7 SD, range 0–95) for right knees and 35.5 mm (±30.4 SD, range 0–100) for left knees. Of the 164 patients, 145 indicated that they had had pain in at least 1 knee for most of the days of the previous month.

**Laxity in the knees of control subjects.** Among the young and older control subjects, women had greater varus–valgus laxity than did men (average of both knees 3.6 ± 1.1° [SD] versus 2.7 ± 0.8°, 95% CI of difference 0.38, 1.56). AP laxity did not differ between men and women (average of both knees 6.0 mm ± 3.0 versus 6.2 mm ± 1.8, respectively). In young controls (age range 20–40 years), mean varus–valgus laxity was 2.9 ± 1.0°, and in older controls (age range 54–85 years), mean varus–valgus laxity was 3.4 ± 1.1°. A modest correlation between varus–valgus laxity and age was detected (r = 0.29, 95% CI 0.01, 0.53). A correlation between AP laxity and age was not detected (r = −0.08).

**Laxity in elderly control subject knees versus uninvolved OA patient knees and patient knees with mild OA.** Patients with knee OA (all stages) had greater varus–valgus laxity than did those of the older controls (average of both knees 4.8 ± 1.7° [SD] versus 3.4 ± 1.1°, 95% CI of difference 0.84, 1.93).

As shown in Figure 1, varus–valgus laxity was greater in the uninvolved knees of the OA patients (K/L grade 0 or 1) than in the older control knees (95% CI of difference 0.60, 2.24). Varus–valgus laxity was also greater in the knees with mild OA (K/L grade 2) than in the older control knees (95% CI of difference 0.43, 1.52). AP laxity did not differ between patients with knee OA (either all stages or only those knees with mild disease) and older control subjects.

**Relationship between specific aspects of disease and laxity in patients with knee OA.** In patients with knee OA, with greater joint space narrowing, varus–valgus laxity increased (Figure 2). The estimate of the slope between laxity and joint space width was −0.34 (95% CI −0.48, −0.19). The negative slope reflects an average decrease of 0.34 mm of joint space width for every 1° increase in laxity. Varus–valgus laxity was

### Table 1. Characteristics of older control subjects and patients with knee osteoarthritis (OA)*

<table>
<thead>
<tr>
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<th>Older control subjects, mean ± SD (range)</th>
<th>Patients with knee OA, mean ± SD (range)</th>
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</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>71.4 ± 8.3 (53.9–85.4)</td>
<td>62.6 ± 11.5 (33–91)</td>
</tr>
<tr>
<td>BMI</td>
<td>26.3 ± 4.1 (20.9–37.7)</td>
<td>31.9 ± 7.2 (19.3–61.9)</td>
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<tr>
<td>WOMAC-PF</td>
<td>0.8 ± 1.8 (0–8)</td>
<td>23.5 ± 13.8 (0–58)</td>
</tr>
<tr>
<td>Sex, no. female/no. male</td>
<td>14/10</td>
<td>118/46</td>
</tr>
</tbody>
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* Body mass index (BMI) was calculated as the weight (in kg)/[height (in meters)]². WOMAC-PF is the Western Ontario and McMaster Universities Osteoarthritis Index Physical Functioning scale (range 0–68; 0 represents a perfect score, i.e., no knee- or hip-related limitation in physical function).
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Significantly greater in knees with than in those without bony attrition (Figure 3); the difference in laxity between grade 1 and grade 0 was 0.79° (95% CI of difference 0.32, 1.27).

With increasing K/L grade, an initial stabilization or trend toward a decrease in varus–valgus laxity was followed by an increase in varus–valgus laxity (Figure 4). Varus–valgus laxity rose significantly between K/L grades 2 and 3, by 0.63° (95% CI of the difference 0.12, 1.14), and between K/L grades 2 and 4, by 1.24° (95% CI of the difference 0.57, 1.91).

No relationship was detected between joint space width or bony attrition and AP laxity. AP translation tended to decline with increasing K/L grade, but the differences between grades were not significant.

No correlation was detected between laxity and body mass index (e.g., for varus–valgus laxity and body mass index \( r = -0.09 \)). One hundred ten patients with knee OA had injured one or both knees in the past.

Laxity (varus–valgus or AP) did not differ between previously injured and uninjured knees.

DISCUSSION

Varus–valgus laxity was greater in the uninvolved or the mildly involved knees of OA patients than in the knees of older control subjects. Among young and older control subjects without OA, varus–valgus laxity was greater in women, and correlated modestly with age. In patients with OA, narrower joint space width or the presence of bony attrition was associated with greater varus–valgus laxity. Increasing K/L grade was associated with stabilization or a trend toward a decrease followed by an increase in varus–valgus laxity. Collectively, these results suggest that, while varus–valgus laxity is worse with joint space loss and bony attrition, a portion of the increased varus–valgus laxity found in patients with knee OA may precede disease development.

The uninvolved and mildly involved knees of patients with OA had greater varus–valgus laxity than the healthy knees of the older control subjects. This would not be the case if varus–valgus laxity in primary OA was exclusively a consequence of OA pathology that develops at later stages. Subgroup definitions were

Figure 1. Varus–valgus laxity in older control subject knees, uninvolved osteoarthritis (OA) patient knees, and patient knees without OA. Mean (±SEM) varus–valgus laxity was greater in the uninvolved knees of OA patients compared with the knees of elderly control subjects (\( P = 0.0006 \)). Mean varus–valgus laxity was greater in the knees with mild OA compared with the knees of elderly control subjects (\( P = 0.0005 \)). K/L = Kellgren/Lawrence.

Figure 2. Varus–valgus laxity in the knees of osteoarthritis (OA) patients, by minimal joint space width. In the knees of OA patients, varus–valgus laxity increased as joint space width decreased (\( P \) for slope < 0.0001).
based upon the K/L grade 2 definition of knee OA. The small number of knees graded K/L 0 precluded analysis of these knees as a subgroup. Mean varus–valgus laxity did not differ between K/L grade 0 knees and K/L grade 0–1 knees (4.8° versus 4.9°).

Theoretically, in the absence of injury, such laxity preceding disease may be influenced by genetic factors, sex, or variably acquired as a consequence of healthy aging. Neither body mass index nor past injury was associated with laxity, making it unlikely that these were confounding factors. Confounding by past physical activity cannot be excluded based on this study. Nevertheless, these results offer support that some varus–valgus laxity precedes OA; in theory, varus–valgus laxity may represent one mechanism by which certain use patterns contribute to knee OA.

Wada et al (24) did not find a difference in varus–valgus laxity between the knees of older control subjects and those of OA patients with K/L grade 1. Their inability to detect a difference might be linked to methodologic issues: the criteria by which patients with OA were defined as having OA are not provided; subclinical, radiographic OA was not excluded in the control subjects. Brage et al (25) found no difference in either varus–valgus laxity or AP laxity between controls or knees with mild-to-moderate OA. However, although Brage et al did not statistically compare the knees with mild OA with the knees of older control subjects, mildly diseased knees appear to be more lax in both the sagittal and frontal planes than do the knees of healthy controls.

In the present study, among the controls, varus–valgus laxity was greater in women than in men, suggesting that attributes of the mechanical environment of the knee may be influenced by sex-related factors. Both incident and progressive OA have been shown to be more common in older women than men (43,44). A modest correlation between varus–valgus laxity and age was detected. The incidence of symptomatic knee OA appears to rise with age (17), as does the prevalence of symptomatic knee OA and radiographic knee OA (45,46). The process of healthy aging includes variable alterations in the neural, mechanical, and muscular environment of the knee, which might contribute to this increase in knee OA with age. In animal models, femur–
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medial collateral ligament–tibia complex stiffness and strength have been shown to decline and ligament substance to become more compliant with age (13,14). Based on studies of cadaver knees, the material properties of the ACL tissue appear to decline with age (15,16). Such age-related changes may be intensified by anatomic factors, by patterns of use, and by comorbid conditions (47). Although soft tissue changes with age may predispose toward ligament insufficiency, little is known about the relationship between age and clinical knee instability.

Specific aspects of OA pathology are likely to have opposing effects on varus–valgus laxity. The relationship between specific radiographic features and laxity in patients with knee OA has not previously been examined. In the present study, varus–valgus laxity increased as joint space decreased, and was greater in knees with evidence of bony attrition. These findings are likely to relate to the points of ligamentous attachment to the femur and tibia moving closer together as a result of loss of bone and cartilage height. It is likely that osteophytes prevent even greater instability, as clearly demonstrated by Pottenger et al (26), who measured varus–valgus laxity before and after intraoperative osteophyte removal in patients with advanced OA. Given their findings, one possible explanation for the collective results of the present study is that, at earlier stages of OA, osteophytes make an effective contribution to varus–valgus stability. With progressive disease, loss of cartilage and bone height appear to override this stabilizing effect. At advanced stages, although osteophytes continue to have some stabilizing activity, as shown by Pottenger et al (26), they cannot prevent further increases in varus–valgus laxity.

Previous studies have utilized global assessments of disease severity and have had mixed results. Using a Genucom knee system, Wada et al (24) examined 68 knees of 34 patients with medial compartment OA and found that varus–valgus laxity did not differ between knees graded K/L 1 and 2, was greater in knees graded K/L 3, and slightly greater at grade 4. Brage et al (25) examined 22 patients with knee OA and found that varus–valgus laxity was greatest at mild stages of OA, and was equivalent at moderate and severe stages. The present study excluded OA in the older controls by explicit clinical and radiographic criteria, used a widely accepted definition of knee OA and consensus inclusion/exclusion criteria, utilized advances in optimizing the quality of radiographic methods, examined the relationship of age and sex to laxity in subjects without OA, compared laxity in the uninvolved knees of patients with knee OA with the knees of older controls, examined the relationship between laxity and specific aspects of OA pathology as well as a global score, and accounted analytically for the within-subject correlation between knees.

The paucity of information on varus–valgus laxity in knee OA relates in part to the absence of reliable measurement systems. The computerized system utilized in previous studies (Genucom) is no longer available. Cushnaghan et al (34) demonstrated the poor reliability of physical examination assessments of laxity within the same examiner. It was therefore necessary to design a measurement system for static laxity. Methods to measure knee laxity under dynamic conditions were not available. The relationship between laxity as measured here and joint instability under dynamic conditions is not known. In the system described here, we sought to address major sources of variation in the usual physical examination maneuver and were able to substantially improve the reliability of measurement of varus–valgus rotation.

Pain and muscle contraction during the laxity measurement were assessed by protocol. Pain or muscle guarding would be more likely in patients with knee OA than in control subjects, and most likely in those with advanced OA. If unreported pain or undetected muscle activity were present, it is likely that the true differences would be in the same direction and even larger than those reported here.

Unlike varus–valgus laxity, AP laxity did not appear to correlate with age or to differ between subjects with knee OA and controls. Age-related changes involving the ACL substances have been demonstrated (15,16). However, such changes may not result in functional instability detectable using a KT1000 arthrometer. Among the patients with OA, AP laxity did not correlate with joint space width and was not affected by the presence of bony attrition, but tended to decline with increasing K/L grade, although the difference between grades was not significant. The direction of this trend is consistent with the findings of Wada et al (24) and of Brage et al (25), who used a Genucom system and demonstrated that AP laxity steadily declined with K/L grade in the former study, and from mild to moderate to severe disease in the latter study. In the study by Wada et al (24), a decline in AP translation was noted with increasing severity of disease, in spite of the fact that among those with severe OA, ACLs were absent in 20, partially torn in 10, and intact in only 11 subjects. ACL type did not predict AP translation. These previous studies suggest that joint stiffness due to capsular changes or osteophytic growth overrides the cruciate...
ligament insufficiency that may occur in progressive knee OA.

While hypermobility as a generalized syndrome has received attention, few studies (48) have dealt with the significance of increased mobility at specific joint sites. A relationship between hypermobility and OA may reflect the mechanical consequences of hypermobility and/or the same basic abnormality leading to both hypermobility and to OA (22). Studies of varus–valgus or AP laxity cannot be directly compared with studies of joint hypermobility, which in general, equate knee hypermobility with knee hyperextension and utilize criteria systems (Beighton and Carter and Wilkinson [49]) that evaluate mobility at the knee in only 1 plane of motion. Knee hyperextension is a common feature of hypermobility syndrome (49); whether patients with hypermobility syndrome have increased mobility in other planes is not clear. It is possible that excessive mobility in different planes of the same joint will have different mechanical consequences and, at the knee, may more greatly stress either the tibiofemoral or patellofemoral compartments.

This study is limited by its cross-sectional design and is hypothesis generating; these questions should be examined in longitudinal studies. This study is not population based. These data were obtained during the baseline evaluation phase of a longitudinal study involving a cohort of diseased patients. Subjects were recruited from several sources in the community to maximize the generalizability of the results, efficiently target a sufficient number of patients with knee OA, and ensure a reasonable distribution of disease severity.

The results reported here suggest the possibility that varus–valgus laxity may precede the development of knee OA and become worse with progression of the disease. In knee OA, the magnitude of varus–valgus laxity is variable, and, even as a consequence of disease, from a mechanical perspective, greater laxity may contribute to subsequent progression. Structural change has mechanical consequences which may lead to further structural changes; such cycling is likely to contribute to joint deterioration in knee OA. On the basis of its effects on the magnitude and distribution of load, laxity may represent one mechanism by which knee OA progresses.
Figure 5 is an illustration of the potential relationships between varus–valgus laxity and knee OA. Whether greater laxity is associated with accelerated progression should be examined in longitudinal studies. If this is demonstrated, intervention directed toward correcting laxity may have a disease-modifying effect.

In summary, varus–valgus laxity was greater in OA patient knees that were uninvolved or had mild OA than in older control knees, suggesting that laxity in knee OA is not exclusively a consequence of pathologic changes that develop at later stages of disease. Varus–valgus laxity might increase variably with healthy aging, predisposing some people to develop knee OA. With progressive OA, loss of cartilage/bone height appeared to override a potentially stabilizing effect of osteophytes. These results raise the possibility that varus–valgus laxity may increase risk of knee OA and may cyclically contribute to disease progression.

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