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Stride-to-stride variability of knee motion in patients with knee osteoarthritis

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Abstract

Purpose: Individuals with knee osteoarthritis (OA) experience pain, frontal plane joint laxity and instability. Co-contraction can control laxity and instability but may place constraints on the variability of the knee's motion during gait. Slight variation among gait cycles is normal, but reduced variability of joint motions could be detrimental. The purpose of this study was to quantify knee motion variability during gait and assess the influence of muscle activity, frontal plane laxity, and pain on knee movement variability in patients with medial knee OA.
Methods: Fifteen subjects with unilateral medial knee OA and 15 age and gender matched uninjured subjects underwent gait analysis, with electromyography to compute co-contraction. Stress radiographs were obtained for measuring frontal plane laxity. Knee motion variability was assessed from the phase angle (knee angle versus angular velocity) during early stance.
Results: Despite altered involved side knee kinematics and kinetics, individuals with knee OA showed involved side frontal plane variability which was not significantly different from the control group, but was significantly lower than the variability of the uninvolved knee's motion. Laxity and medial co-contraction influenced the amount of joint motion variability in the involved knee of the OA subjects. Pain did not influence variability.
Conclusion: Patients with medial knee OA displayed altered involved knee kinematics and kinetics, although stride-to-stride variability of knee motion was unchanged. Evidence of excessive joint motion variability on the uninvolved side, however, may provide insight into the development of OA in the contralateral cognate joint.

Keywords: Motor control; Gait; Muscle function; Instability; Varus

1. Introduction

Knee osteoarthritis (OA) is marked by the progressive erosion of articular cartilage, subchondral sclerosis, and osteophyte growth at the joint margins. Patients may also experience ligamentous laxity, muscle weakness, joint instability, and debilitating pain [1].

Joint pain in patients with knee OA can provoke a stereotypical knee-stiffening pattern during gait [2,3]. The measurement of joint angles at discrete time points provides valuable information, yet may represent an incomplete analysis because it ignores the inherent variability involved in the completion of a movement task. Uninjured subjects perform successive cycles (e.g. steps, strides) of rhythmic movements, such as gait, in a similar, but not identical manner with each repetition [4]. This is because the inherent redundancy of the motor components allows for multiple solutions to joint coordination in order to achieve the same control of the foot's path. The variability of joint motions may therefore reflect flexibility of movement patterns used to achieve control of important performance variables. We are particularly concerned with the variability of the knee joint's motion during gait, which we define as the stride-to-stride variability of the knee's angular position and velocity with each step.

Individuals with knee OA could potentially experience knee joint damage or pain due to the presence of either too

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much or too little knee motion variability. An increase in knee joint motion variability could indicate inadequate control of the joint. Conversely, a substantial reduction in knee motion variability could lead to an inability to adequately adjust to perturbations and attenuate impact shocks [5]. Additionally, joint surfaces would undergo similar stresses with each repetition, potentially leading to articular cartilage destruction. Therefore, the use of sufficient, but not excessively varied joint motions with each repetition may aid in the redistribution of stress across the joint [6].

Individuals with knee OA may reduce the variability of the knee joint's motion for several reasons. Pain could contribute to the overuse of similar, less painful knee joint motions [6] which may further damage a joint that has already begun to undergo structural and biochemical alterations. Radin and coworkers [7] speculated that progressive cartilage erosion was due to the continuous repetitive microtrauma that the joint undergoes on a daily basis. In vitro animal experiments have confirmed that cyclic loading of articular cartilage contributes to breakdown [8,9]. A failure to use more varied joint motions could therefore theoretically accelerate the degeneration of the articular surface. Perhaps a better explanation, however, is that the development of frontal plane laxity may lead to joint instability, requiring increased muscular control to stabilize the joint [10,11]. Individuals with medial knee OA attempt to stabilize the knee during gait with greater medial co-contraction, resulting in reduced joint excursions [12] and perhaps less varied knee joint motions.

The purpose of this study was to quantify the variability of knee motion in patients with medial knee OA. Our hypothesis was that patients with medial knee OA would demonstrate reduced variability of the knee's frontal and sagittal plane motions during gait compared to their uninvolved knees or the knees of a matched control group. In addition, we expected that pain, frontal plane joint laxity, and muscular co-contraction during gait would provide insight into the mechanism underlying alterations in knee motion variability.

2. Methods

2.1. Subjects

Fifteen patients (6 females, 9 males; age: 48.7 ± 7.4 years, height: 1.75 ± .09 m, weight: 91.9 ± 17.4 kg) with unilateral symptomatic, medial compartment knee osteoarthritis and genu varum (OA group) scheduled for high tibial osteotomy were tested. The diagnosis of OA was made from the clinical history, a physical examination, and radiographic changes observed during standing with the knees flexed to 30°. These radiographs showed definite joint space narrowing in the medial compartment (medial compartment: 1.6 ± 1.1 mm; lateral compartment: 6.2 ± 1.4 mm). All subjects were asymptomatic on the uninvolved side with adequate articular cartilage (medial: 4.6 ± 1.4 mm; lateral: 5.0 ± 1.6 mm). Assessment of skeletal alignment was made from a weight-bearing radiograph that contained the entire lower extremity, from the hip joints to the feet [13]. A ''weight-bearing line'' is drawn from the center of the femoral head to the center of the ankle mortise. The perpendicular distance from this weight-bearing line to the medial edge of the proximal tibia is divided by the width of the proximal tibia. A weight-bearing line of less than 50% therefore indicates varus alignment. The OA group had a weight-bearing line of 18.9 ± 12.7% on the involved side. Subjects who had torn knee ligaments, lateral compartment or patellofemoral osteoarthritis, other orthopedic problems or neurological damage in either lower extremity or a body mass index of ≥40 were excluded from the study.

A control group of 15 age- and gender-matched healthy subjects (6 females, 9 males; age: 48.4 ± 6.3 years, height: 1.71 ± .09 m, weight: 83.8 ± 17.3 kg) with no evidence of knee OA underwent identical testing to the OA group on a randomly chosen limb. The control group had a weight-bearing line of 45.1 ± 8.1% and had 5.0 ± 1.0 mm of joint space in the medial compartment and 6.3 ± 1.3 mm in the lateral compartment. All subjects were informed of the purpose of the study and signed informed consent forms approved by the IRB prior to testing.

2.2. Pain

Pain was assessed using the response to the following question: ''To what degree does pain affect your level of daily activity?'' taken from the Knee Outcome Survey-Activities of Daily Living Scale [14]. Responses are taken from a six-point scale where five represents no effect of pain on activities of daily living and zero represents an inability to perform activities of daily living because of pain. Reliability and responsiveness of the questionnaire for assessing knee function has been assessed and reported by others [14,15].

2.3. Frontal plane joint laxity

Measurements of frontal plane joint laxity has been described previously [12]. Briefly, frontal plane laxity was measured from stress radiographs obtained with subjects lying supine with the knee supported and flexed 20°. A TELOS stress device (Austin & Associates, Fallston, MD) was used to reliably apply a 15 da N (33 lb) force to generate varus and valgus forces [16]. Joint space was measured during both varus and valgus stresses. Medial joint laxity was calculated as the medial joint space during a valgus stress minus the medial joint space during a varus stress. Lateral laxity is the lateral joint space during a varus stress minus the lateral joint space during a valgus stress