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Review

Measuring knee joint laxity: A review of applicable models and the need for new approaches to minimize variability

J.C. Küpper [a], B. Loitz-Ramage [b], D.T. Corr [b], D.A. Hart [c,*], J.L. Ronsky [a,b]

[a] Department of Mechanical and Manufacturing Engineering, University of Calgary, Calgary, AB, Canada
[b] McCaig Centre for Joint Injury and Arthritis Research, 3330 Hospital Dr. NW, Calgary, AB, Canada T2N 4NI
[c] Departments of Surgery, Microbiology and Infectious Diseases, and Medicine, University of Calgary, Calgary, AB, Canada

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Abstract

Knee joint laxity can result from soft tissue injury, such as a ligament tear, or from genetic factors such as joint hypermobility syndrome and various forms of Ehlers–Danlos Syndrome. The location of a subject's passive knee laxity along a continuous spectrum is dependent on the mechanical properties of the existing structures, and the increased motion that often follows joint injury. At a threshold along the spectrum, a patient will be at risk for joint instability and further injury to joint structures. Links between instability and laxity may be better understood if laxity can be reliably and accurately quantified. Current measures of laxity have not been compared to a 'gold standard' in all cases, and when they have, were found to overestimate the laxity values. This is attributed to soft tissue deformation. Consequently, a noninvasive measure of laxity with improved accuracy and repeatability would be useful clinically and in the research sector. In this review, current clinical measures of laxity are critiqued, criteria for a measure of laxity are identified, and three theoretical models of knee laxity are outlined. These include contact, lumped parameter, and finite element models, with emphasis on applicability, strengths, and limitations of each. The long term goal is to develop a model and method able to differentiate subjects along a spectrum of laxity, and understand the functional implications of altered joint integrity. This would allow careful scrutiny of clinical interventions aimed at improving joint health and provide a valuable research tool to study joint injury, healing, and degeneration.

Keywords: Stiffness; Ligament force; Magnetic resonance imaging; Contact; Lumped parameter; Finite element

1. Introduction

In biomechanical terms, passive laxity is a measure of joint movement within the constraints of ligaments, capsule, and cartilage (Cross, 1996) when an external force is applied to the joint during a state of muscular relaxation. Laxity depends on the shape of the involved bony surfaces, the mechanical behavior of the joint's soft tissue structures, such as the joint capsule and ligaments, and contributions from other supporting structures, such as menisci, that may improve the bony fit between relatively incongruent joint surfaces. Theoretically, laxity can be measured at any joint, although some joints are inherently very stable, such as the sacroiliac joints between the two halves of the pelvis and the centrally located sacrum. In these cases, laxity is not typically assessed by quantifying the motion of the joint, but by whether passive motion produces pain or other symptoms. The human shoulder is a good example of a lax joint that relies entirely upon ligaments for passive stability because the capsule is very loose and the bony anatomy provides minimal contributions to joint stability. In this case, the benefit of having a lax joint is a considerable increase in the total range of joint motion, allowing humans to reach overhead with relative ease. Therefore, a measure of laxity must be interpreted contextually, including the function of the joint (weightbearing support vs. functional reach or grasping).

* Corresponding author.
E-mail address: hartd@ucalgary.ca (D.A. Hart).

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Increased joint laxity can result from a local soft tissue injury such as a ligament tear or from genetic factors such as joint hypermobility syndrome and the various forms of Ehlers–Danlos Syndrome. Excessive joint laxity predisposes the joint to instability including recurrent dislocations and subluxations, and low grade inflammatory arthritis (Lewkonia, 1993). However, the link between instability and laxity is not fully understood (Maffulli, 1998; Patel et al., 2003). Knee joint laxity is of particular interest, and has been studied extensively, in part, due to the high incidence of knee injuries, knee joint pain, and degeneration that account for substantial morbidity, functional loss, and health care expenditures.

The knee joint exhibits a wide spectrum of laxity, from inherently stable joints at one end, to excessively lax joints at the other. The causes of abnormal laxity are numerous and complex. Individuals with high joint laxity, such as those with Anterior Cruciate Ligament (ACL) tears, are more likely to incur subsequent knee injuries. Interestingly, even the normal, uninjured population displays a wide range of knee laxity. For example, young, fit military recruits, who are otherwise healthy, have exhibited laxity at the high end of the spectrum, without any prior injury or existing pathology (Uhorchak et al., 2003). This normal range of laxity is further complicated in sexually mature females where, at least in a subpopulation of them, changes in joint laxity have been reported to occur during the menstrual cycle (Deie et al., 2002; Shultz et al., 2004, 2006; Wojtys et al., 2002). However, this point is still controversial (Belanger et al., 2004), and it is not clear if there are biologically different populations, or subtle differences in methodology that are confounding the findings.

In the ACL deficient knee (ACLD), laxity values lie at the far end of the spectrum. ACLD subjects are often subdivided into copers, who functionally adjust to the injury, and noncopers, who experience increased instability, including recurrent subluxations (Eastlack et al., 1999). Noncopers are often candidates for ACL reconstruction (ACLR), where the torn ligament is commonly replaced by either the central third of the patellar tendon or the gracilis/semitendinosis tendon (Herrington et al., 2005). After reconstruction, laxity is reduced but the joint does not return to normal function (Almekinders et al., 2004; Ejerhed et al., 2003).

Another factor that affects joint laxity is an individual's genetic predisposition for pathologies such as Marfan's syndrome, Ehlers–Danlos syndrome, and joint hypermobility syndrome. This latter disorder appears to affect connective tissue matrix proteins, thereby altering the mechanical properties of the soft tissues and creating an inherent joint laxity (Hakim and Grahame, 2003). The majority of individuals with joint hypermobility syndrome are female (Acasuso-Diaz et al., 1993; Baum and Larsson, 2000; Bridges et al., 1992), and the incidence has been reported to vary from 5% of the Caucasian population to ~30% of females of Middle Eastern descent (Al-Rawi et al., 1985; Bridges et al., 1992; Fitzcharles, 2000). These subjects are more lax than normal, and are unique from an injured population because the musculoskeletal laxity is something they have matured with rather than having to adjust to a sudden change in joint laxity following an acute injury. Those with joint hypermobility syndrome are also unique because the laxity may not be restricted to a particular joint. Some patients with joint hypermobility syndrome demonstrate laxity throughout all joints, while others may experience laxity in only upper extremity or only lower extremity joints.

It is important to distinguish *passive* laxity, which is measured during a state of muscle relaxation, from *functional* or *active* laxity, which describes the joint motion that occurs during functional activities. In the latter, the forces applied through the joint arise from muscle contraction or external loads related to movement, such as inertial or ground reaction forces. This distinction is clinically important because some patients with passive laxity do not demonstrate functional laxity (Snyder-Mackler et al., 1997). Muscle contraction or co-contraction (Aalbersberg et al., 2005a) that is well-timed and of an appropriate magnitude may play a role in controlling dynamic joint function by preventing excessive joint laxity from limiting function or increasing joint injury risk. Regardless of the underlying cause or number of affected joints, at a certain threshold along the spectrum a patient will be at risk for joint injury because of instability. The link between instability and laxity may be further understood if laxity can be reliably and accurately quantified.

The objective of this review is to critique the current clinical measures of laxity, identify the criteria for a measure of laxity, and outline three potential theoretical models of knee laxity. The long term goal is to develop a model and method that can be used to differentiate subjects along the laxity spectrum, and understand the functional implications of altered joint integrity.

2. Current measures of joint laxity

Traditionally, passive tests have been used to assess knee laxity in patients. These measures include the Lachman test, the anterior/posterior drawer test, the pivot shift test, the quadriceps active test, and the varus/valgus stress test (Malanga et al., 2003). The primary structures being tested are the ACL, posterior cruciate ligament (PCL), and medial and lateral collateral ligaments (MCL, LCL). These clinical measures can be effective for an experienced physician, and have been useful for determining treatment protocol. However, they do not allow for quantitative comparison between subjects or testers since the results are qualitative and primarily used for diagnosis (Malanga et al., 2003). These clinical measures are not sufficient for understanding the impact of the injuries or genetic pathologies.

In response to this need, instrumented devices such as the KT-2000 arthrometer (http://www.medmetric.com, MedMetric, San Diego, CA, USA), the Genucom Knee