Biomechanical Factors in Osteoarthritis: The Effects of Joint Instability

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Introduction

Osteoarthritis is now appropriately recognized as a joint disease (see the article by Robin Poole in this Supplement), and as such, the biomechanical factors that affect its etiology and progression can best be understood by considering the mechanical function of the whole joint.

Biomechanical Consequences of Joint Injury

A diarthrodial joint such as the knee or shoulder is intended to maintain an appropriate functional position throughout its range of motion [4]. Situations often arise, however, in which the ability of the joint to maintain a stable position is hampered by the loss of a beneficial constraint such as a ligament. Such occurrences impact the mechanical burden placed on the joint in important ways. The velocity with which the articular surfaces move relative to one another can increase dramatically in an unstable joint because rapid, large changes in position can occur. Examples include a torn anterior cruciate ligament (ACL), which can produce a threefold increase in anterior translation of the tibia relative to the femur [5], or a torn meniscus that not only alters the location of contact (again creating larger surface velocities) but also markedly increases the pressure distribution in the underlying cartilage [2].

The loads that are transferred across joints in the skeleton are high, even when performing normal activities. Direct measurements of joint loads have been performed in the knee and hip joints through the use of instrumented total joint

replacements. For the knee joint, in vivo loads exceed 2 to 2.5 times body weight for activities like walking, climbing stairs, or swinging a golf club [8]. Similar results were found for the hip for these same activities, though loads reached as high as 8.7 times body weight during an unanticipated stumble [3] (Fig. 1).

The altered loads and kinematics that accompany instability and trauma will impact all of the tissues of the joint (see, for example, the article by Harrie Weinans in this supplement for a discussion on periarticular bone). But if we focus on the cartilage that forms the bearing surfaces of the joint, we can begin to explain how altered biomechanics affects the damage to these surfaces that ultimately defines failure in an osteoarthritic joint. A great deal regarding the loads on these cartilage surfaces can be learned from the experience with joint replacements in which the cartilage surfaces are replaced most typically by a polyethylene-metal bearing couple.

Loading of Prosthetic Joint Surfaces

The surfaces of polyethylene components wear from at least two mechanisms: abrasive wear in which the harder surface cuts through the surface of the softer material, and fatigue wear, which occurs as result of the formation and growth of cracks [10]. Abrasive wear leads to direct damage at the surface, while fatigue wear is manifested as surface delamination and pitting created by subsurface initiation and propagation of cracks [1, 10]. Abrasive wear is a function of the load applied across the contacting surfaces times the distance that the surfaces slide under load. Fatigue wear is exacerbated in nonconforming joints where the local stresses are higher due to smaller contact areas compared to a more conforming joint [1].

If we apply these same concepts to understanding the damage that occurs to cartilage in osteoarthritis, then any situation that increases the combination of load times sliding distance should increase damage to the cartilage articular

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Fig. 1. Maximum compressive load across the knee joint (vertical axis) as a function of the joint position (knee flexion angle) for a number of activities of daily living as measured in an 81-year-old man 1.5 years after implantation of using a custom-designed instrumented knee prosthesis. Reprinted from Mündermann et al. [8] copyright 2008 with permission from John Wiley and Sons

surface. In a recent experimental study, Brown and colleagues set out to explore this hypothesis [9]. Using a rabbit model, they sought to alter joint stability (and, hence, load times sliding distance) by comparing animals with intact knee joints with knee joints in which the ACL had been partially transected and with knees in which the ACL had been completely transected. The comparison was based primarily

Fig. 2. Cartilage damage as reflected by a Mankin score (HHGS) was positively correlated with a measure of joint stability as reflected by the length of the neutral zone measured in the rabbit knees that had undergone partial transections of their ACLs. Reprinted from Tochigi et al. [9] copyright 2011 with permission from J Bone Joint Surg Am

on histological score as a marker for cartilage damage. They found that the severity of cartilage degeneration increased continuously with the degree of instability (Fig. 2). While the alteration in joint load could not be measured, the correlation suggests that tribological approaches can be applied to understanding surface damage in osteoarthritis.

By considering the interaction of joint stability and joint contact loads, the difficulty in interpreting the role of biomechanics in osteoarthritis animal models can be appreciated. Indeed, these models vary considerably in terms of their impact on joint biomechanics and, therefore, might yield markedly different results even when other factors (e.g., type of animal and animal genetic strain) are well controlled. For example, transection of the anterior cruciate ligament will no doubt lead to joint instability and larger sliding distances, and cause cartilage regions not normally loaded during routine activities by the animal to experience some level of contact loads. But presumably the animal is prone to co-contract muscles across the joint in an effort to add the stability in the same way that humans suffering an ACL tear co-contract their hamstrings and quadriceps [7]. Thus, not only will sliding distance (and velocity) increase in the ACL transaction model, but joint loads as well, but in an unpredictable manner that cannot be measured experimentally.

In contrast, other models, such as the surgical destabilization of the medial meniscus (DMM), may not create as much joint instability as ACL transaction, but will nonetheless have a large impact on the stresses in the cartilage as the cushioning role of the meniscus is lost in effect concentrating the load over smaller contact areas. Indeed, comparisons of the location and extent of osteoarthritic histological changes between the ACL transection and DMM model reflect differences consistent with these differences in altered biomechanics [6].

Conclusion

In an effort to better understand loading of normal and joints at risk of developing OA, the need exists for controlling or at least measuring the loads and motions that accompany osteoarthritis animal models. This will allow objective comparisons to be made of the effect of host alterations resulting from disease or injury as well as from treatments. Only through such understanding can the influence of biomechanical confounding factors be understood and controlled.

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