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Computer simulation of damage on distal femoral articular cartilage after meniscectomies

E. Peña, B. Calvo, M.A. Martínez, M. Doblaré*

Department of Mechanical Engineering, Group of Structural Mechanics and Material Modelling, Aragon Institute of Engineering Research (I3A), University of Zaragoza, María de Luna, 3, E-50018 Zaragoza, Spain

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Abstract

It is commonly accepted that total or partial meniscectomies cause wear of articular cartilages that leads to severe damage in a period of few years. This also produces alteration of the biomechanical environment and increases articular instability, with a progressive and degenerative arthrosic pathology. Due to these negative consequences, total meniscectomy technique has been avoided, with a clear preference for partial meniscectomies. Despite the better results obtained with this latter technique, it has been demonstrated that the knee still suffers progressive long-term wear, which alters the properties of the surface of articular cartilage.

In this paper, a phenomenological isotropic damage model of articular cartilage is presented and implemented in a finite element code. We hypothesized that there is a relation between the increase of shear stress and cartilage degeneration. To confirm the hypothesis, the obtained results were compared to experimental ones. It is used to investigate the effect of meniscectomies on articular damage in the human knee joint. Two different situations were compared for the tibio-femoral joint: healthy and after meniscectomy. The distribution of damaged regions and the damage level distribution resulted qualitatively similar to experimental results, showing, for instance that, after meniscectomy, significant degeneration occurs in the lateral compartment. A noteworthy result was that patterns of damage in a total meniscectomy model give better agreement to clinical results when using relative increases in shear stress, rather than an absolute shear stress criterion. The predictions for partial meniscectomies indicated the relative severity of the procedures.

Keywords: Osteoarthritis; Damage; Articular cartilage; Shear stress; Finite element method; Meniscectomy

1. Introduction

Many authors agree that total meniscectomies lead to progressive articular cartilage damage after few years [1–3]. This is due to an alteration of the knee biomechanics that increases instability, resulting in to a progressive and degenerative arthrotic pathology [4–6]. Both, partial and total meniscectomies, alter the loading pattern [7], decrease the contact area and increase the magnitude and gradient of contact stresses [8]. The degree of damage appears to be directly related to the amount of meniscus removed [9].

Osteoarthritis is characterized by progressive changes in the ultrastructure and biochemical composition of articular cartilage and the surrounding tissues [10–12]. However, it is still unclear which are the main external factors and how they affect the normal structure, composition and mechanical properties of cartilage [13–16]. It is generally accepted, however, that osteoarthritis leads to major changes in cartilage function which adversely affect load bearing, stabilization and lubrication [17,18]. Macroscopically, cartilage degeneration has been described as fibrillation of the articular surface, the presence of cracks or fissures, and by partial or complete loss of the tissue [19]. In general, mechanical changes reported for degenerated human cartilage include a lower stiffness in compression, tension, and shear, as well as an increase in permeability [18]. Elliot et al. [17] found that the tensile modulus of cartilage significantly decreases following meniscectomy and indicated

* Corresponding author. Tel.: +34 976 76 19 12; fax: +34 976 76 25 78.
E-mail addresses: fany@unizar.es (E. Peña), bcalvo@unizar.es (B. Calvo), miguelam@unizar.es (M.A. Martínez), mdoblare@unizar.es (M. Doblaré).

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that this was consistent with changes in the collagen structure and proteoglycan–collagen interaction. Several researchers also noted a decrease in the shock-absorbing capability of articular cartilage after total meniscectomy [4,20–23]. Osteoarthritis has been shown to decrease the tensile modulus as much as 90%, reflecting significant damage of the cartilage solid network [10]. Guilak et al. [13] examined the changes in tensile mechanical properties and biochemical composition of the surface of articular cartilage in a canine osteoarthritis model. The tensile stiffness decreased 44%, the proteoglycan content 37% and the collagen content 36%. Other experimental studies in animal models confirm the association between altered loading of diarthrodial joints and arthritic changes.

Osteoarthritis can occur through many different pathways, but all pathways involve the interaction of mechanical and biological factors [24]. Fundamental mechanical perturbations that accelerate joint destruction include joint immobilization, impact, alterations in joint kinematics, and other mechanical, age and genetics-related alterations [24]. For example, Andriacchi et al. [25] found that the initiation of the degenerative changes was not directly because of the high contact pressures, but rather was associated with alterations in joint kinematics and Andriacchi et al. [26] included the hypothesis related to the magnitude of shear stress and cartilage degeneration in the context of ACL injury.

Besides extensive statistical analyses and experimental tests, numerical modelling is progressively becoming a significant tool in quantitative and qualitative assessment of the overall biomechanical behavior and evolution of many different organs [27–29]. Despite the increasing possibilities of numerical methods, and especially finite element techniques, information about the constitutive behavior of some of the most common biological tissues, such as ligament, tendon and cartilage, is still insufficient. Even with this limitation, finite element simulations are able to provide a better understanding of the joint biomechanics, helping in the prevention of injuries and pathological degeneration [30–35]. Three-dimensional finite element models can also be used to estimate the consequences of surgical treatments such as total or partial meniscectomies [6,36–39].

In this paper, a mechanical damage model of articular cartilage is presented. Several authors suggested that damage of cartilage can be related to maximal shear stresses [33,40,41]. We hypothesize, however, that there is a relation between the increase of shear stresses and cartilage damage. A three-dimensional finite element model of the human tibio-femoral joint including the femur, tibia, cartilage layers, menisci and main ligaments has been used to estimate the osteoarthritis damage in femoral cartilage surfaces after meniscectomy. The obtained results could help to explain cartilage degeneration after total or partial meniscectomies [18,42].

2. Material and methods

2.1. Damage model of articular cartilage

An osteoarthrosis index (OA) is now frequently used in clinics and usually normalized between $\mathrm{OA_{min}}=0$ and $\mathrm{OA_{max}}=4$ [43]. $\mathrm{OA_{min}}$ corresponds to healthy cartilage and $\mathrm{OA_{max}}$ to completely disrupted cartilage with only a residual elastic stiffness that corresponds to of fibrillated cartilage.

Although a consideration of fluid flow and matrix consolidation is important in understanding joint lubrication, and matrix damage leads to changes in permeability, following Carter et al. [24], we shall only consider the damage of the solid phase (matrix)

$$E_{\mathrm{damaged}} = (1-\zeta)E_{\mathrm{healthy}}, \tag{1}$$

where ζ is a scalar internal variable $\zeta \in [0,1]$, referred to as isotropic damage. The factor $(1-\zeta)$ plays the role of a reduction factor and was first proposed by Kachanov [44].

Armstrong et al. [45] reported that the elastic modulus of human articular cartilage decreases almost linearly with the degree of structural disorganization. We have considered therefore, a linear relation between the OA index and the decrease in the elastic modulus of articular cartilage, such as

$$E_{\mathrm{damaged}} = E_{\mathrm{healthy}} - (\mathrm{OA}-\mathrm{OA_{min}})\frac{E_{\mathrm{healthy}}-E_{\mathrm{res}}}{\mathrm{OA_{max}}-\mathrm{OA_{min}}}, \tag{2}$$

where E_{damaged} is the elastic modulus of osteoarthritic cartilage, E_{healthy} the elastic modulus of healthy cartilage and E_{res} the residual elastic modulus corresponding to $\mathrm{OA_{max}}$. Comparing (2) with (1) it is clear that damage ζ can be expressed in terms of the more clinical variable OA as

$$\zeta = \frac{\mathrm{OA}-\mathrm{OA_{min}}}{\mathrm{OA_{max}}-\mathrm{OA_{min}}}\left(1-\frac{E_{\mathrm{res}}}{E_{\mathrm{healthy}}}\right). \tag{3}$$

Several authors suggested that cartilage damage is related to a maximal shear stress criterion [26,40,41,46]. This type of criteria, however, can only explain articular cartilage damage in high weight-bearing areas, but not the experimental results presented by other authors [10,47,48]. They found damage both, in low and high weight-bearing areas in femoral and tibial cartilage. In contrast, a shear stress increase criterion is able to explain these experimental results. Therefore, we have considered the increase of shear stress $\Delta\tau$ as the main damage-producing variable. Maximal shear stress τ is calculated as $\tau=(\sigma_1-\sigma_3)/2$, where σ_i are the eigenvalues of $\boldsymbol{\sigma}$, see appendix.

With this, the appearance of cartilage degeneration in low weight-bearing areas is possible [10]. Within the closed time interval $[0,t]$ and at each point, we define α as the relative difference between the maximum value of $\Delta\tau$ obtained in that interval at that point and the physiological value of the shear stress (we identify this physiological value with the value of the shear stress that appears at that point in the intact cartilage for the same load). Therefore:

$$\alpha(t) = \frac{\max_{s\in[0,t]}\Delta\tau(s)}{\tau_{\mathrm{phys}}}, \tag{4}$$

where the dependence on the point has been dropped for clearness.