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Hypothesis of regulation of hip joint cartilage activity by mechanical loading

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Summary Hypothesis of regulation of proteosynthetic activity of chondrocytes is suggested. A deformation of the cartilage caused by contact hip joint stress and consequent deformation of the chondrocytes are considered as main factors that could influence the metabolism of the cartilage.

INTRODUCTION

Articular cartilage is a complex structure, which consists of cells (chondrocytes) and intercellular matrix (fluid and structural macromolecules). Although progress has been made in understanding and predicting development of the hip by biomechanical parameters, there is no decisive answer regarding the effect of the load yet. To understand the effect of the load on the human cartilage, regulation mechanisms acting on the cellular and molecular levels should be clarified. It is the aim of this article to suggest such a theory.

STRESS AND DEFORMATION OF THE CARTILAGE

Recently, a noninvasive method was developed for determination of contact stress distribution on the hip joint weight bearing area (1–3). This method is based on the mathematical models of the resultant hip joint force in the one-legged stance (4) and of the corresponding contact stress distribution (5). The resultant hip joint force and the contact stress distribution can be computed for each patient individually according to geometrical parameters of the hip and pelvis obtained from a standard antero-posterior rentgenograph (1). By using this method it was shown (6) that in dysplastic hips the peak stress on the weight bearing area is considerably and statistically significantly higher than in normal hips. According to the Hooke's law higher stress in the dysplastic hip is proportional to larger deformation of the cartilage. It was found that the deformation of the cartilage causes the deformation of the chondrocytes (7). The changes in structure of the cartilage were found in dysplastic hips with respect to normal hips (8), (e.g., changes of the content and type of structural macromolecules – collagen and proteoglycans that are produced by the chondrocytes) indicating that the proteosynthetic activity of the chondrocytes is altered in dysplastic hips.

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HYPOTHESIS

According to the above we suggest a hypothesis of the regulation of the proteosynthetic activity of the chondrocytes by the change of the shape of the chondrocytes induced by the load upon the cartilage.

In normal hips, the contact stress is on the average relatively low and also the peak stress is relatively low (6) so that under normal conditions the cartilage is not extensively deformed. The chondrocytes are only slightly

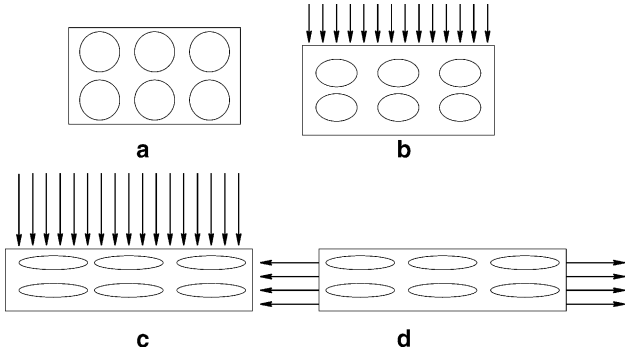

Fig. 1 Schematic representation of the deformation of the cartilage and chondrocytes when unloaded (a), in small compressive loading (b), in large compressive loading (c) and in stretching (d).

deformed from the spherical shape (Fig. 1b). Hypothetically such loaded chondrocytes product structural molecules that serve as a barrier against compression – proteglycans.

In dysplastic hips the compressive stress is on the average higher and the peak stress is higher (*6*). The cartilage, therefore, deforms to a greater extent and the deformation of the cartilage is transduced to chondrocytes (Fig. 1c). The change of the shape of the chondrocyte under high compression is similar to the change of the shape of the chondrocyte when stretched, i.e., the chondrocytes are elongated (*7*) (Fig. 1d). Hypothetically such deformed chondrocytes behave like mesenchymal cells subject to stretching (fibroblasts) and produce structural molecules which serve as a barrier against stretching – collagens. As the collagen cannot resist compressive stress (*9*), the cartilage is deformed more and more and the vicious circle starts.

DISCUSSION

Evidence exists that supports our hypothesis. The hypothesis explains the structure of the cartilage. The superficial layer of the cartilage, which undergoes highest stretching (*9*), contains mostly collagen (10). Deeper layers that are subject mostly to compressive load contain more proteoglycans (*9*).

Further, in dysplastic hips the collagen type I was found (8) which is characteristic for tendons and is normally not present in the cartilage.

It should be noted that the dysplastic hips exhibit higher gradient of stress on the lateral edge of the acetabulum (11). Although articular cartilage is a porous viscoelastic material, and the flow of an interstitial fluid is different than the flow of an ideal liquid (12), it can be assumed in the first approximation that the velocity of the efflux of the interstitial fluid is proportional to the gradient of contact stress in the pores of the cartilage. Efflux of the interstitial fluid changes mechanical properties of the cartilage and allows higher deformations of the cartilage (12). This would imply that the gradient of stress is important too as was suggested on the basis of clinical investigations (13).

To describe the effect of the load on the cartilage regulation more exactly, further studies based on the cellular level are needed. Also other mechanisms should be considered such as: reduction of the fluid film lubrication between the articular surfaces, loosening of the collagen network, disruption of the collagen fibers and loss of the proteoglycans.

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