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SYNOVIAL FLUID FILTRATION BY ARTICULAR CARTILAGE IN THE HUMAN SYNOVIAL JOINTS

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Summary: *The paper presents a critique of a squeeze-film lubrication model of the synovial joints with the fluid transport across the articular surface (J. S. Hou, V. C. Mow, W. M. Lai and M. H. Holmes, Journal of Biomechanics, 1992, 25, pp. 247-259). The asymptotic biphasic model of porous homogeneous articular cartilage in this lubrication model assumes a constant interstitial fluid pressure across the whole cartilage layer thickness and the continuity of the fluid pressure at the articular surface at the moment of a step-load application. The model does not allow for the fluid pressure jump that occurs there at that time. Thus, an intensive fluid flow across the interface and considerable filtration of the synovial fluid due to this jump is absent from the model.*

1. Introduction

Many studies have investigated the lubrication properties of articular cartilage (AC) in order to understand the normal and pathological behaviors and the reasons for degenerative processes in the human synovial joints. To explain the marvelous performance of normal joints, two conflicting conceptions have been proposed, with the fluid transport through the articular surface taken into account. "Weeping" lubrication (McCutchen, 1962) assumes that the squeezed synovial film is supplied by exudation of the interstitial fluid from the compressed cartilage, similarly to the exudation of a compressed sponge. By contrast, "boosted" lubrication (Walker et al., 1962; Maroudas, 1963) assumes that the solvent component of the synovial fluid (SF) flows into the pores of the compressed cartilage. During the squeeze-film action, the concentration of the macromolecular complex of hyaluronic acid and proteins present in the SF increases until a limit value is reached. A fixed synovial gel (SG) film is then formed between the surfaces, preventing intimate contact between them.

A successful biphasic mixture model for AC that takes into account the interstitial fluid flow has been developed by Mow, Lai and co-workers (1980). In this model, a porous elastic cartilage matrix is filled with an ideal (or viscous) interstitial fluid, both phases being

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intrinsically incompressible. The model reveals an important role of interstitial fluid pressurization due to loading proposed by McCutchen (1962; 1959). This fluid pressurization contributes to supporting a significant portion of the total applied load, particularly in the early time response of the material (Ateshian et al., 1994).

Hou et al. (1992) and Hou (1989) have presented a squeeze-film lubrication model of a rigid impermeable spherical indenter approaching a thin homogeneous permeable cartilage layer supported by an impermeable rigid subchondral bone, and step-loaded. A single-phase Newtonian fluid for SF and the above biphasic model (Mow et al., 1980) for AC (porous elastic, isotropic and homogeneous cartilage matrix filled with a viscous Newtonian interstitial fluid) are used. The calculations in the asymptotic model converge only for low loads. After accepting some simplifying assumptions, Jin et al. (1992) have succeeded in applying this model to the loads encountered in the human hip joint in standing. The model shows that cartilage porosity depletes the synovial film only slightly.

Hlaváček (1993a; 1993b; 2000) has applied the same biphasic model of AC (but with an ideal interstitial fluid) to an axially symmetric case of squeeze-film lubrication of the human hip joint in standing. In this model, SF is considered a mixture of two incompressible fluids: viscous (hyaluronic acid-protein macromolecular complex) and ideal (water and small solutes). Only the ideal phase passes through the interface, thus enabling an SG film description. According to this asymptotic model, the filtration by cartilage is intensive, the fluid film quickly becomes depleted, and an SG layer quickly develops over the greater part of the contact in the step-loaded joint. The gel serves as a boundary lubricant if a sliding motion follows before a fresh SF gets into the contact.

Both the above models corroborate the “boosted” lubrication conception for a step-loaded joint, but the filtration intensity that they predict differs. The fluid pressure is assumed to be continuous at the articular surface by Hou et al. (1992; 1989), while Hlaváček (1993b; 2000) takes into account a small jump in the fluid pressure at the articular surface at the moment of load application. Hou et al. seem to have omitted the existence of this boundary layer.

Macirowski et al. (1994) have measured in vitro the total surface stress and displacement on acetabular cartilage, when step-loaded by an instrumented femoral head prosthesis, together with the surface topography and constitutive properties of the intact cartilage. A simplified finite element model (with the shear and lateral strains in the cartilage neglected) is used to calculate the fluid and solid stress components together with the fluid velocity normal to the articular surface. The latter is obtained from the difference between the calculated and measured surface displacements. The results obtained by this combined method support the “weeping” lubrication concept.

Soltz et al. (2003) have presented an analysis of the contact of a rippled impermeable indenter against a biphasic cartilage layer. A numerical analysis demonstrates that, under contact creep, the trapped lubricant pool of an ideal fluid quickly depletes as a result of an intensive filtration by AC. A high time-decreasing fluid flow through the articular surface shortly after load application indicates the existence of a fluid pressure jump at the surface at the start of creeping, similarly to the model by Hlaváček (1993b; 2000).

The above papers present quite contradictory theoretical results that are still open for experimental verification.

The present contribution brings a critique on squeeze-film lubrication model presented by How, Mow, Lai and Holmes (1992; 1989) in order to explain the discrepancy with our model (Hlaváček, 1993a; 1993b; 2000).

2. Hou's model versus our model

The details of the Hou model (let us call it Model I) can be found in the Ph.D. theses of Dr. J. S. Hou (1989), while Hou et al. (1992) presents only the main results of the model for a step-load. Model I takes SF as a single-phase viscous (Newtonian) fluid and cartilage as a biphasic mixture of a porous elastic matrix and a viscous (Newtonian) fluid, with both phases intrinsically incompressible (Mow et al., 1980). The fluid can pass across the free cartilage surface s_c as a whole and its apparent viscosity is supposed to change during the passage from μ_f in SF to μ_a in cartilage and *vice versa*. The so called "pseudo-slip condition" (How, 1989) that guarantees the continuity of the surface weighted mixture velocity vector across s_c is applied in addition to all the jump and continuity conditions of the classical mixture theory. A rigid spherical indenter is loaded by a step steady load against a thin cartilage layer bonded to the rigid impervious substrate. In the asymptotic model two small parameters are introduced: $\varepsilon = H_0 / R_0$, $\delta^2 = \mu_a / K H_0^2$. Here the notation of Hou et al. (1992) is used: H_0, R_0 and K stand for the cartilage layer thickness, contact radius and diffusive drag coefficient, respectively. The model takes carefully into account boundary layers of a large change in the fluid velocity near s_c due to $\mu_a \neq 0$. However, the value of μ_a has never been measured. Hou (1989) solved the problems with $\mu_a \neq 0$ modeling the permeation and confined compression experiments (both stress relaxation and creep), but no values of $\mu_a \neq 0$ could be gained by comparing the measurement and calculation.

Denote our model (Hlaváček, 1993a; 1993b; 2000) by Model II. Model II considers cartilage a biphasic mixture as in Model I, but with the interstitial fluid inviscid, while SF is taken as a biphasic mixture of viscous (Newtonian) and ideal fluids. The ideal phase can pass across s_c, the macromolecules of the viscous phase (hyaluronic acid-protein complex) in the synovial gap being too large to get into small cartilage matrix pores. Only the classical boundary conditions are used on s_c, i. e. the jump conditions obtained from the balance laws and the kinematic condition for the non-inviscid phases. Two identical cartilage layers are considered, but the contact of Model I with one articular surface impervious and rigid can be also tackled. In the asymptotic formulation only the small parameter ε is used and all ε^2-order terms in the asymptotic expansions are considered. The cartilage immediate response to a step load is that of the single-phase elastic incompressible material. There are less boundary conditions for single-phase materials than for biphasic mixtures. Therefore, while the total stress vector remains continuous on s_c at the time of the load application $t = 0$, the two its parts, i. e. the fluid pressure p and the deviatoric solid part of stress, do not, the last part being different from zero, contrary to its zero value at the synovial side of s_c. (Strictly speaking, the viscous shear stress on the synovial side of s_c is small compared to the fluid pressure, or its jump across s_c, and is neglected.) In Model II the jump in the fluid pressure at $t = 0$ is to ε^2-order of the form $p_- - p_+ = H_0^2 V_+$, $V_+ = \partial(r \partial p_+ / \partial r) / \partial r / r$. Here $'-'$ and '+' indicate the AC and SF side of s_c and r stands for the radial coordinate, respectively. It has been found (Hlaváček, 1993a; 1993b; 2000) that these jumps on s_c form a boundary layer on s_c across which a large pressure gradient makes the interstitial fluid flow intensively across s_c shortly after the step load application, thus filtering intensively the synovial film. Another boundary layer is near the cartilage-subchondral bone interface, but it is unimportant for SF filtration. These boundary layers are present also if the total load changes continuously with time and their effect depends on the speed of this change. The ideal fluid imbibes in a large central part

of the step-loaded contact and flows out in a narrow circumferential part (for a positive effective curvature radius of the contact). For physiologic parameters, within about a fraction of second the SF film is depleted in the central part and turned into a stable synovial gel, acting probably as a boundary lubricant if sliding motion follows. The model is valid for $0 \le t \ll H_0^2 / k H_A$, when the boundary layer thickness is small compared to H_0. Here k, $H_A = \lambda + 2\mu$ and t stand for the cartilage matrix permeability, its Lame's constants and time after the load application, respectively. Only later, the fluid flow in the bulk cartilage and in the radial direction becomes apparent. Contrary to Model I, Model II describes also the gel forming process. The power law, binding μ_f and the hyaluronic-acid macromolecular concentration in SF and used in Model II, has not been verified in the whole concentration range. Fortunately, the filtration process depends only slightly on the form of this law, showing that the effect of the increased viscosity on the squeezed film due to filtration is relatively small.

Another simple example of a pressure jump on the surface is the case of a free (unconfined) elastic single-phase isotropic and incompressible rectangular element under uni-directional compression. It follows from the Hooke law that the hydrostatic pressure is even as low as one third of the outer compressive traction in this case. On the contrary, for confined compression of the above element the stress tensor is spherical and the hydrostatic pressure equals the outer traction.

Written in the notation of How et al. (1992), the surface weighted mixture velocity v_n and the fluid flux q_z (the component perpendicular to s_c) are related by $v_n = \varphi^f v_z^f + \varphi^s v_z^s = \varphi^f (v_z^f - v_z^s) + v_z^s = q_z + v_z^s$. Here ϕ^f, ϕ^s denote the volume fractions of the fluid and solid phases. Thus, v_n equals q_z plus the matrix velocity v_z^s. According to Model II, in a boundary layer of s_c the fluid transport is one-dimensional (perpendicular to s_c) and the continuity equation of the mixture yields $v_{n-} = 0$, i. e. $q_{z-} = -v_{z-}^s$. On the other hand, eq. (27) in How et al. (1992) gives $v_{n-} = k H_0 V_+$. The last expression is also the flux into a rigid porous cartilage (Hlaváček, 1993b). After the immediate compression at $t = 0$, the vertical displacement of a point on s_c, u_{z-}^s, increases as $t^{1/2}$ according to Model II. At $r = 0$ cartilage swells, while in Model I u_{z-}^s quickly decreases shortly after $t = 0$, as shown in Fig. 6 of How et al. (1992). Measuring there $\partial u_{z-}^s / \partial t = v_{z-}^s$ at s_c for $r = 0$ at time A (at the film thickness of 10μm) yields $v_{z-}^s = -8.1 \times 10^{-3}\,\mathrm{ms}^{-1}$. The SF pressure distribution p_+ at later times *B, C* (see Fig. 5, How et al. (1992)) can be approximated by a thin-layer dry contact pressure distribution with zero pressure gradient at the contact edge, applied to the case of one cartilage layer. For the parameters of How et al. (1992) this yields the contact radius 6.4 mm, the maximum pressure 1.2 MPa and $V_+ = -2.3 \times 10^{11}\,\mathrm{Nm}^{-4}$. This gives for Model I (and $H_0 = 1\mathrm{mm}$, $k = 1 \times 10^{-15}\,\mathrm{m}^4 / \mathrm{Ns}$): $v_{n-} = k H_0 V_+ = -2.3 \times 10^{-7}\,\mathrm{ms}^{-1}$. But this is just the value shown in Fig. 8, How et al. (1992) at $r = 0$ for the same k. Thus, Fig. 8 shows the distribution of the mixture velocity v_{n-} and not the fluid flux q_{z-} at time *B*. Thus, in How et al. (1992) the surface weighted mixture velocity v_n is erroneously taken for the interstitial fluid flux q_z. Fig. 5 indicates that V_+ at $r = 0$ at time *A* must be lower (in the absolute value) than at times *B* or *C*. As $|v_{n-}| \ll |v_{z-}^s|$ at time *A*, we get $q_{z-} = -v_{z-}^s = 8.1 \times 10^{-3}\,\mathrm{ms}^{-1} > 0$, i. e. the interstitial fluid flows intensively out of the cartilage at $r = 0$ and time *A*, which means the McCutchen "weeping" lubrication (McCutchen, 1962). Only later (at times *B, C*), already with very small v_{z-}^s, the fluid imbibes into the cartilage at $r = 0$. This alternating flow behavior is due to a

high compression of surface s_c shortly after $t = 0$ according to Model I. Contrary to this, Model II gives q_{z-} at $r = 0$ decreasing in the absolute value monotonically with time as $t^{-1/2}$.

The parameters (the total load $F = 50\,\mathrm{N}$, $\mu_f = 1\,\mathrm{Pas}$, the effective curvature radius $R = 5\,\mathrm{cm}$) as used in How et al. (1992) do not refer to a real hip joint. Jin et al. (1992) used Model I for the parameters corresponding to the human hip joint loaded by the body weight ($R = 0.5\,\mathrm{m}$, $H_0 = 2.5\,\mathrm{mm}$, $H_A = 2\,\mathrm{MPa}$, the Poisson ratio $\nu = 0.2$, $\mu_f = 0.001\,\mathrm{Pas}$, $k = 1.6 \times 10^{-15}\,\mathrm{m}^4/\mathrm{Ns}$ and $F = 2.5$ kN). Fig. 1 shows the variance of the central fluid film thickness with time for both models, both with and without filtration. According to Model I, the effect of porous cartilage is small. Both curves, with and without filtration, taken from Fig. 4 in Jin et al. (1992), practically merge. Model II, however, shows a quick SF film depletion at $r = 0$ due to filtration by cartilage. In order to compare both models, the curve for Model II is calculated assuming μ_f independent of the HA macromolecular concentration in the SF. When this concentration reaches the twenty fold of the original concentration, SF is assumed to change into a stable gel (Maroudas, 1969), the fluid transport across s_c stops and the film thickness already remains practically constant. The small difference between the curves of both models with no filtration ($k = 0$) is probably due to the fact that the time independent dry contact pressure distribution is used in Model II for $p_+(r)$, while in Model I $p_+(r,t)$ is obtained in the course of a more complicated solution and slightly varies with the early time.

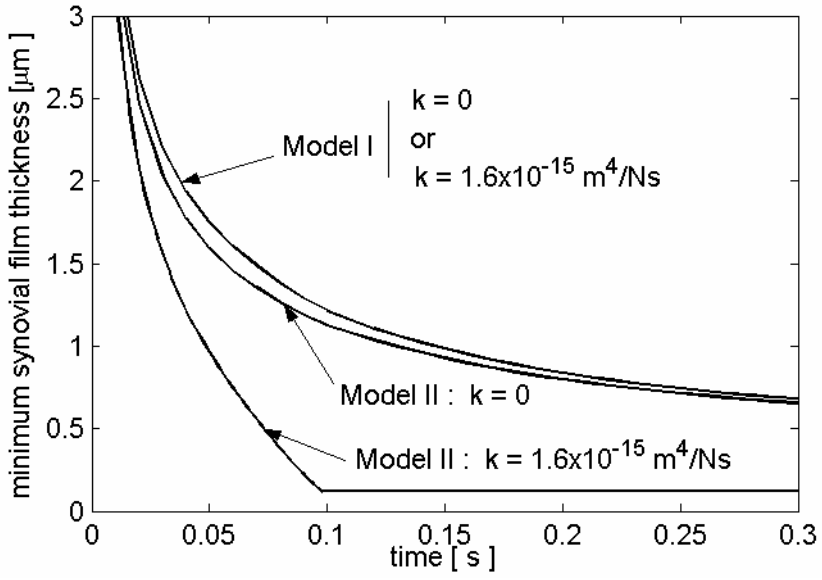

Fig. 1 The variance of the central synovial film thickness with time for Models I and II with and without SF filtration ($k \neq 0$ and $k = 0$) by articular cartilage

The introduction of viscosity μ_a of the interstitial fluid in the cartilage by Dr. Hou and his colleagues in their Model I seems unfortunate. The assumption $\mu_a \neq 0$ makes the analysis much complicated, while the value of μ_a remains unknown. Moreover, the fact that the

resulting equations (32), (23-24), (34) and (19) in How et al. (1992) do not contain μ_a at all is surprising and looks suspicious. In Model I for the fluid pressure in the cartilage only the ε^0-order terms in the asymptotic expansions were considered (eqs. (4.99)-(4.101) in How (1989)), which makes p to ε^0-order independent of the z-coordinate. Then, setting this p equal the SF pressure p_+ excludes any jump of the fluid pressure on s_c at the load application. It is here where one should see the reason for the discrepancy of both models. On summing equations (4.105)-(4.106) from How (1989), the derivative $\partial p_1 / \partial z$ is obtained as a function of the ε^0-order terms of u_r^s, u_z^s. It is this p_1, a term of ε^2-order, that causes to order ε^2 the above mentioned jump in p in Model II. In Model I this term is absent and the fluid transport across s_c due to this jump is not taken into account. But it is just this fluid pressure jump that, according to Model II, makes filtration of the synovial film by cartilage and its depletion much intensive. Assuming μ_a of order 10^0 Pa s (even 3 orders higher than μ_f), it follows that $\delta^2 \sim 10^{-8}$, while $\varepsilon^2 \sim 10^{-2}$. As $\varepsilon^2 \gg \delta^2$, it is not right to consider some δ^2-order terms in a model and at the same time to leave out some ε^2-order terms.

Let us note in closing one important point. Both Model I and Model II (as described in the above references) consider articular cartilage homogeneous and isotropic. However, the intact superficial zone of the normal articular cartilage (about 100 to 200 micrometers thin only) has quite different microstructure and mechanical properties as compared to the bulk cartilage. Due to the tangential orientation of the collagen fibres in this superficial zone of the normal cartilage the extendibility of this intact zone is very low, which should change the articular surface fluid transport considerably. From this point of view, the models discussed above describe, in fact, a pathological case of the articular cartilage with the surface zone already worn out or disrupted, which seems to correspond to the early stage of primary osteoarthritis. Model II has been recently also applied to the human ankle joint and generalized to non-homogeneous transversely isotropic cartilage matrix (the case of normal AC) and used for periodic loading encountered in walking (Hlaváček, 2002; 2005).

3. Results

The asymptotic biphasic model of porous homogeneous articular cartilage in the lubrication model by How et al. (1992) assumes a constant interstitial fluid pressure across the whole cartilage layer thickness and the continuity of the fluid pressure at the articular surface at the moment of load application. The model does not allow for the fluid pressure jump, present in the model by Hlaváček (1993b; 2000), that occurs on the articular surface at that time. Thus, an intensive fluid flow across the interface and considerable filtration of the synovial fluid due to this jump is absent from the How model. The existence of an intensive fluid flow across the articular surface is also corroborated by Soltz et al. (2003).

4. Acknowledgements

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