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# Biomechanical Properties of Synovial Fluid in/Between Peripheral Zones of Articular Cartilage

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## 1. Introduction

The properties and behaviour of articular cartilage (AC) have been studied from numerous aspects. A number of biomechanical models of the properties and behaviour of AC are available today. The traditional model presents cartilage as homogeneous, isotropic and biphasic material (Armstrong et al., 1984). There also exist models of transversally isotropic biphasic cartilage material (Cohen et al., 1992; Cohen et al., 1993), non-linear poroelastic cartilage material (Li et al., 1999), models of poroviscoelastic (Wilson et al., 2005) and hyperelastic cartilage material (Garcia & Cortes, 2006), models of triphasic cartilage material (Lai et al., 1991; Ateshian et al., 2004), and other models (Wilson et al., 2004; Jurvelin et al., 1990). The published models differ, more or less, by the angle of their authors' view of the properties and behaviour of articular cartilage during its loading.

The authors base their theories on various assumptions concerning the mutual links between the structural components of the cartilage matrix and their interactions on the molecular level.

The system behaviour of AC very depend on nonlinear properties of synovial fluid (SF). Certain volumes of SF are moveable components during the mechanical loading in the peripheral zone of AC. Biomechanical properties of peripheral zone of AC are significantly influenced by change of SF viscosity due to mechanical loading.

The hydrodynamic lubrication systems and influences of residual strains on the initial presupplementation of articular plateaus by synovial fluid were not sufficiently analyzed up to now.

Our research has been focused on analyses of residual strains arising in AC at cyclic loading and on the viscous properties of SF. Residual strains in articular cartilage contribute the preaccumulation of articular surfaces by synovial fluid.

SF reacts very sensitively to the magnitude of shear stress and to the velocity of the rotation of the femoral and tibial part of the knee joint round their relative centre of rotation when the limb shifts from flexion to extension and vice versa. Shear stresses decrease aggregations of macromolecules of hyaluronic acid in SF.

Articular cartilage (AC) is a viscohyperlatic composite biomaterial whose biomechanical functions consist

1. in transferring physiological loads into the subchondral bone and further to the spongy bone,
2. in ensuring the lubrication of articular plateaus of joints and
3. in protecting the structural components of cartilage from higher physiological forces.

The macromolecular structure of AC in the peripheral zone (Fig. 1.) has two fundamental biomechanical safety functions, i.e. to regulate the lubrication of articular surfaces and to protect the chondrocytes and extracellular matrix from high loading.

The rheological properties of SF play the key role in the achievement of the optimum hyaluronan concentration.

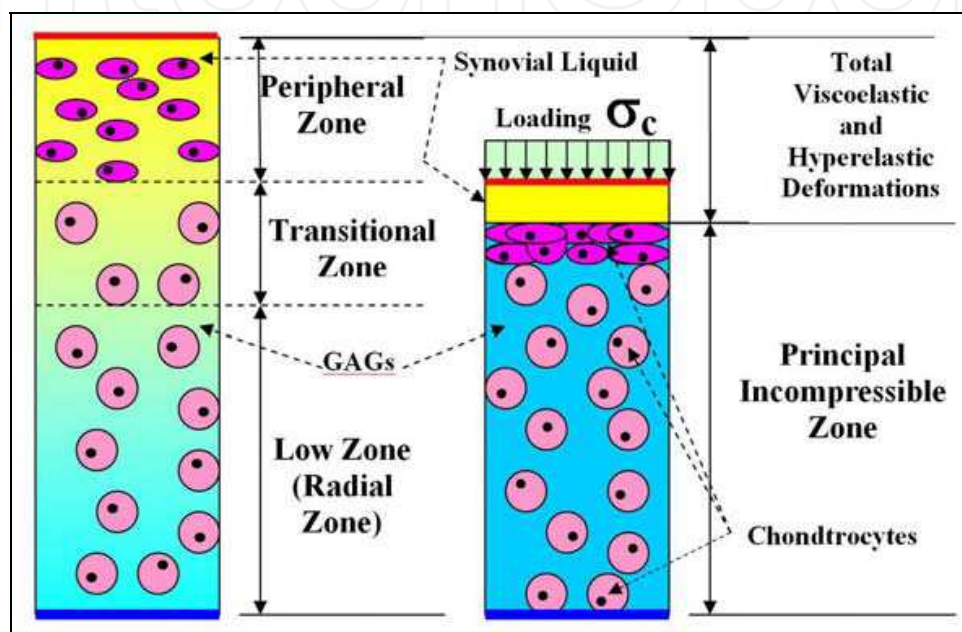


Fig. 1. Complex structural system of articular cartilage (collagen fibres of 2<sup>nd</sup> type are not drawn)

The properties of SF in the gap between the opposite surfaces of articulate cartilage are not homogeneous during loading. The properties of SF change not only during biomechanical loading, but also during each individual's life time. The viscous properties of this fluid undergo changes (in time) due to mechanical loading. As a consequence of its very specific rheological characteristics, SF very efficiently adapts to external biomechanical effects. Exact knowledge of the rheological properties of synovial fluid is a key tool for the preservation and treatment of AC. The significance of the specific role of SF viscosity and viscosity deviations from predetermined physiological values were first pointed out as early as the 1950s to 1990s (Johnson et al., 1955; Bloch et al., 1963; Ferguson et al., 1968; Anadere et al., 1979; Schurz & Ribitsch, 1987; Safari et al., 1990 etc.). The defects of concentrations of the dispersion rate components were noticed by Mori (Mori et al., 2002). In this respect, it cannot be overlooked that mechanical properties of SF very strongly depend on the molecular weight of the dispersion rate (Sundblad et al., 1953; Scott & Heatley, 1999; Yanaki et al., 1990; Lapcik et al., 1998) and also on changes in the aggregations of macromolecular complexes in SF during mechanical effects (Myers et al., 1966; Ferguson et al., 1968; Nuki & Ferguson, 1971; Anadere et al., 1979 and Schurz & Ribitsch, 1987).

Synovial fluid is a viscous liquid characterized by the apparent viscosity  $\eta$ . This viscosity depends on stress and the time during which the stress acts. SF is found in the pores of the

peripheral zone of AC and on its surface (in the gap between the opposite AC surfaces). The viscosity of synovial fluid is caused by the forces of attraction among its molecules being fully manifested during its flow. In other words, viscosity is a measure of its internal resistance during the SF flow. In the space between the opposite AC surfaces, its flow behaves like a non-Newtonian fluid.

As was pointed out above, biomechanical effects play a non-negligible and frequently a primary role in regulating rheological properties.

The principal components of synovial fluid are water, hyaluronic acid **HA**, roughly 3-4 mg/ml, D-glucuronic acid and D-N-acetylglucosamine (Saari et al., 1993 and others). By its structure, hyaluronic acid is a long polymer, which very substantially predetermines the viscous properties of synovial fluid. Its molecular structure is evident from Fig. 2. *Synovial fluid* also contains an essential growth hormone *prolactin* (PRL) and *glycoprotein lubricin*.

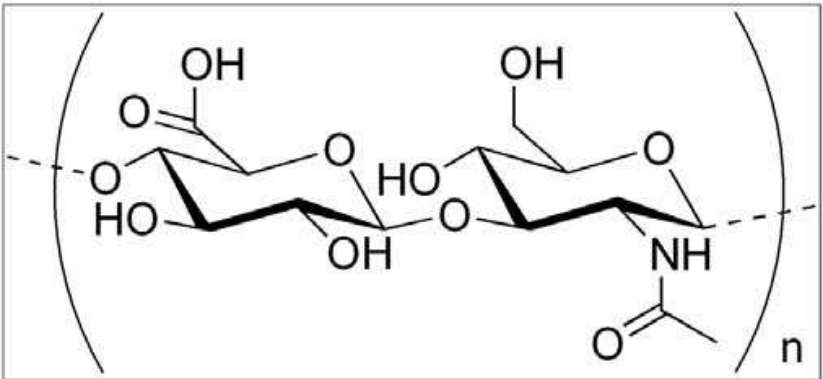


Fig. 2. Molecular complex of hyaluronic acid (HA)

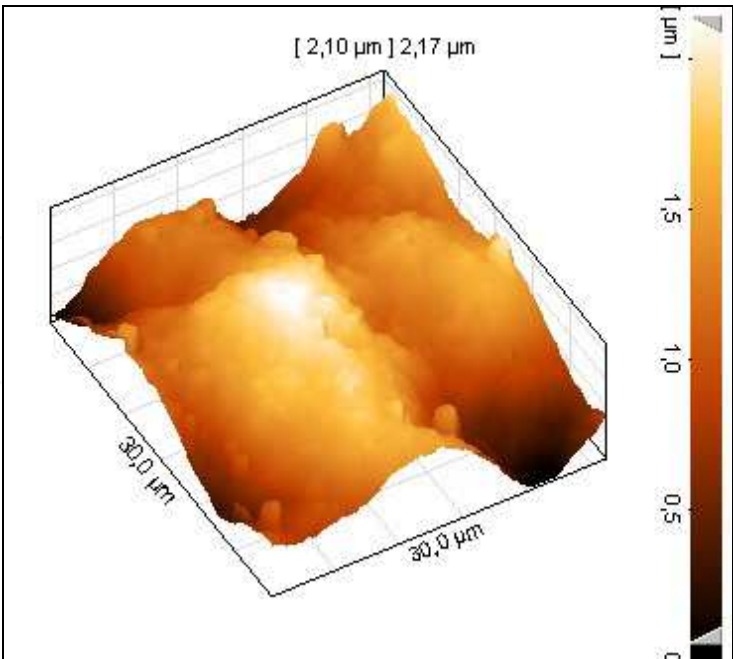


Fig. 3. Topography of the surface of articular cartilage verified by means of FAM (Force Atomic Microscope). The height differences of surface points range up to ca 200 nm - 2,4  $\mu\text{m}$ . In unloaded condition, they are flooded by synovial fluid

Prolactin induces the synthesis of proteoglycans and, in combination with glucocorticoids, it contributes to the configuration of chondrocytes inside AC and to the syntheses of type II collagen. The average molecular weight of human SF is 3 – 4 MDa.

Important components of SF are lubricin and some proteins from blood plasma ( $\gamma$ -globulin and albumin), which enhance the lubricating properties of SF (Oates, 2006). The importance of HA and proteins for the lubricating properties of SF was also described (Swann et al., 1985; Rinaudo et al., 2009).

In the gap between AC surfaces, synovial fluid forms a micro-layer with a thickness of ca 50  $\mu\text{m}$ . It fills up all surface micro-depressions (Fig. 3. and 4., Petrtýl et al., 2010) and in accessible places its molecules are in contact with the macromolecules of residual SF localized in the pores of the femoral and tibial peripheral zone of AC.

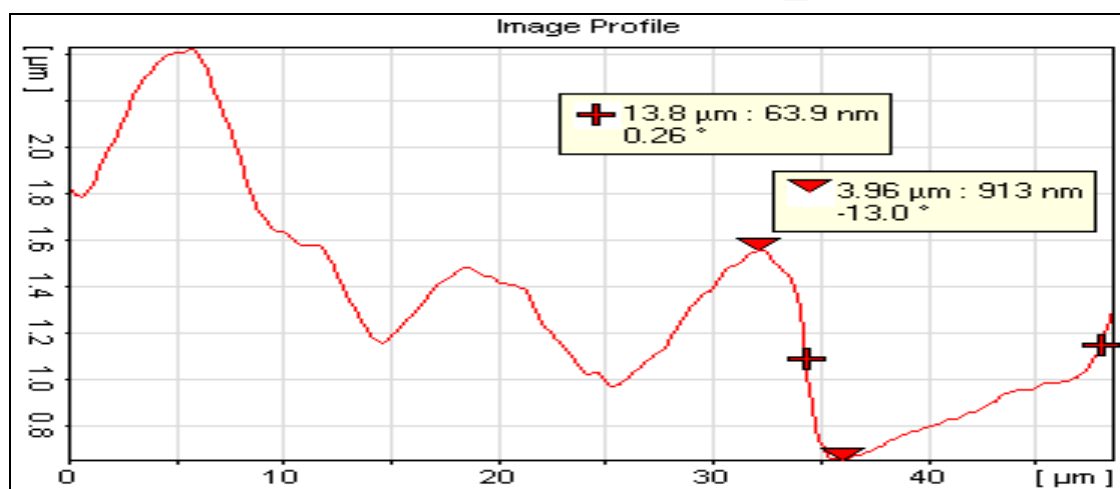


Fig. 4. Topography of the articular cartilage surface of a man (58 years of age). The AC surface oscillates to relative heights of 2.5  $\mu\text{m}$ . During fast shifts of the AC surface (due to the effect of dynamic shifting forces/dynamic bending moments or shear stresses), the AC surface is filled up with generated synovial gel (with less associated NaHA macromolecules) with *low viscosity*

SF is a rheological material whose properties change in time (Scott, 1999 and others). As a consequence of loading, associations of polymer chains of HA (and some proteins) arise and rheopexic properties of SF are manifested (Oates et al., 2006). Due to its specific rheological properties, SF ensures the lubrication of AC surfaces. The key component contributing to lubrication is HA/NaHA. In healthy young individuals, the endogenous production of hyaluronic acid (HA) reaches the peak values during adolescence. It declines with age. It also decreases during arthritis and rheumatic arthritis (Bloch et al., 1963; Anadere et al., 1979; Davies & Palfrey, 1968; Schurz & Ribitsch, 1987 and numerous other authors). Some AC diseases originate from the disturbance of SF lubrication mechanisms and from the defects of genetically predetermined SF properties. Therefore, the lubrication mechanisms of AC surfaces must be characterized with respect to the rheological properties of SF.

## 2. Contents

The objectives of our research has been aimed on the definition of the biomechanical properties of SF which contribute to the lubrication of the opposite surfaces of articular



cartilage, on the analysis of the effects of shear stresses on changes in SF viscosity and on the analysis of the residual strains arising in AC at cyclic loading.

## 2.1 Rheological properties of synovial fluid

With respect to the project objectives, the focus of interest was on the confirmation of the rheological properties of hyaluronic acid with sodium anions (sodium hyaluronan, NaHA) in an amount of  $3.5 \text{ mg ml}^{-1}$  in distilled water without any other additives. The use of only NaHA was based on the verification of the association of HA macromolecules and on the manifestation of highly specific rheological properties of SF, which regulate its lubrication function. The rheological properties were verified using the rotation viscometer Rheolab QC (Anton Paar, Austria). Viscosity values were measured continuously within 8 minutes.

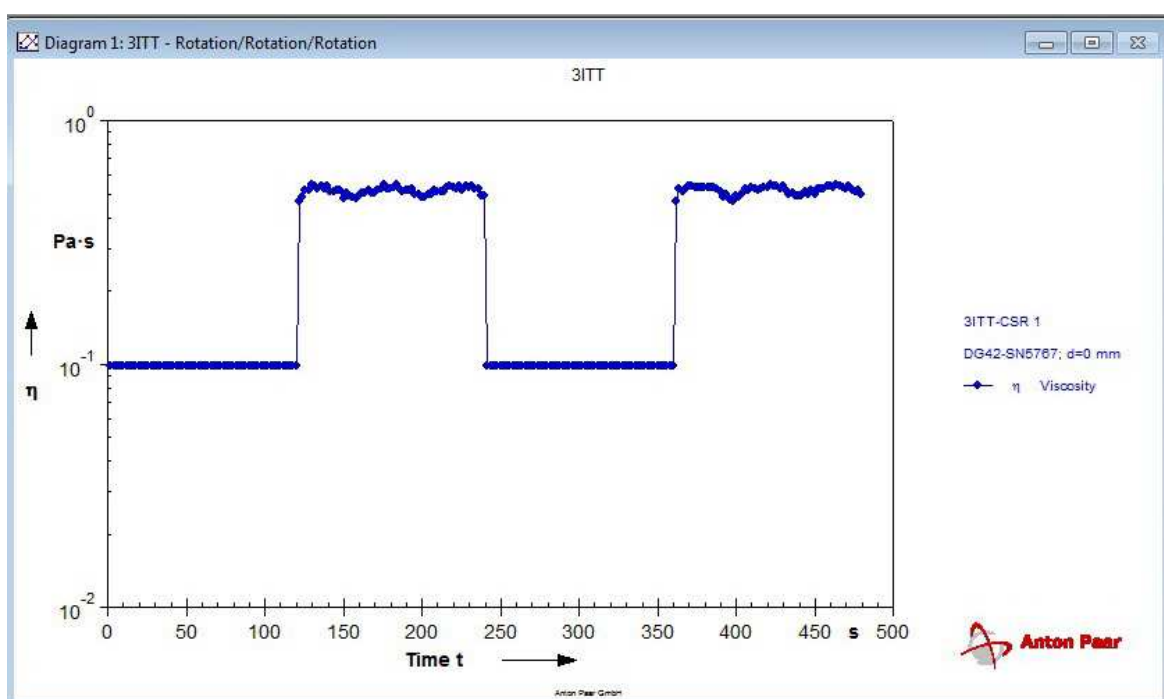


Fig. 5. SF apparent viscosity as related to time (velocity gradient  $100 \text{ s}^{-1}$ )

Samples were subjected to the effect of constant velocity gradient ( $100 \text{ s}^{-1}$  –  $500 \text{ s}^{-1}$  –  $1000 \text{ s}^{-1}$  –  $2000 \text{ s}^{-1}$ ) in time 0 – 120s and 240 – 360s. Samples were subjected in the tranquility state in time 120 – 240s and 360 – 480s. The measurements were performed at the temperature of human body ( $37^\circ\text{C}$ ).

Fig. 5. clearly shows that at the constant SF flow velocity gradient  $100 \text{ s}^{-1}$  there is a distinct time-related constant values in viscosity. The verified synthetic synovial fluid possesses *pseudoplastic properties*. It is evident that the *macromolecules of hyaluronic acid (NaHA/HA) in a water dispersion environment principally contribute to the pseudoplastic behaviour of the fluid. This property is of key importance for controlling the quality of the AC surface protection.*

Fig. 6. also shows that at the constant SF flow velocity gradient  $2000 \text{ s}^{-1}$  there is a distinct time-related constant values in viscosity. The viscosity of SF after unloading always returns to the same values (ca  $0.8 \text{ Pa s}$ ).

Fig. 7. shows that viscosity values of SF with increasing rate of flow velocity gradient 0 –  $2000 \text{ s}^{-1}$  (in time 0 – 60s) decrease. Viscosity values of SF are constant with constant rate of velocity gradient  $2000 \text{ s}^{-1}$  (in time 60 – 180s).

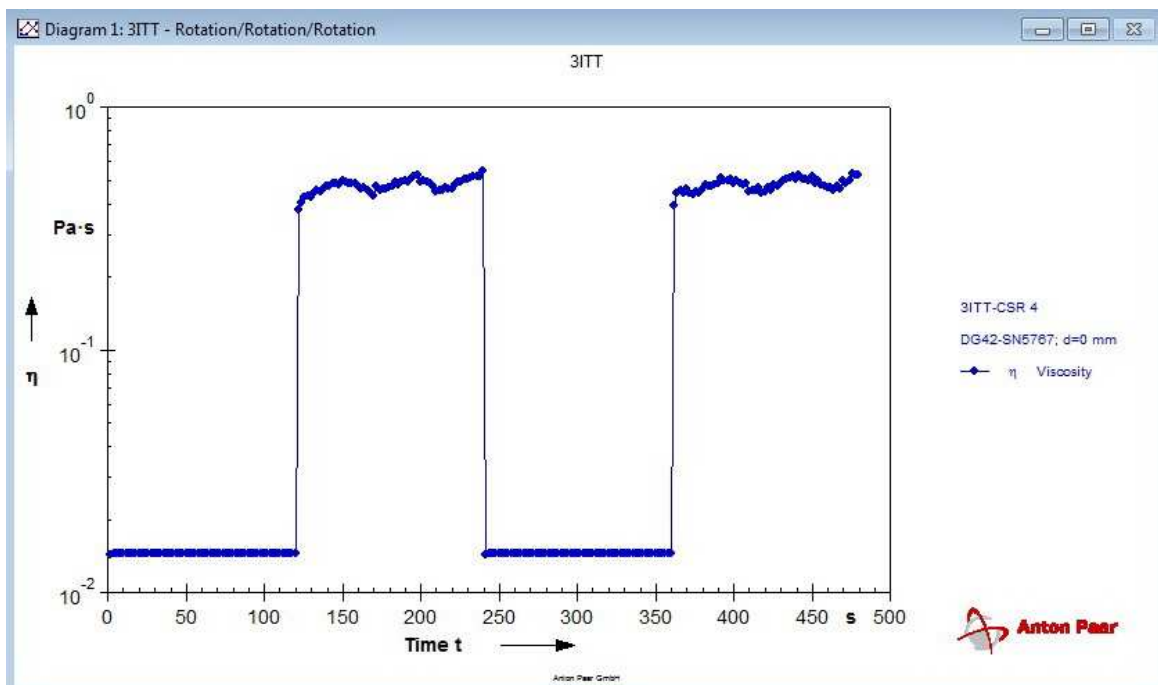


Fig. 6. SF apparent viscosity as related to time (velocity gradient  $2000\text{s}^{-1}$ )

Due to the fact that the lubrication abilities of SF strongly depend on the magnitude of viscosity, and SF viscosity depends on the SF flow velocities, the effects of the magnitudes and directions of shifting forces or shear stresses respectively on the distributions of the magnitudes and directions of SF flow velocity vectors in the space between the opposite AC surfaces had to be analyzed.

The kinematics of the limb motion (within one cycle) shows that during a step the leg continuously passes through the phases of flexion – extension – flexion (Fig. 8.). The effect of shifting forces (or shear stresses respectively) is predominantly manifested in the phases of flexion, while normal forces representing the effects of the gravity (weight) of each individual mostly apply in the phases of extension, Fig. 8. The distributions of the magnitudes of SF flow velocity vectors depend on the shifts of the tibial and femoral part of the knee joint, Fig. 9., reaching their peaks in places on the interface of SF with the upper and lower AC surface, Fig. 10. The velocities of SF flows very substantially affect the SF behavior contributing to the lubrication of AC surfaces and their protection.

At rest the bonds are created among the macromolecules of hyaluronic acid (HA) leading to the creation of associates. *By associating molecular chains of HA (at rest) into a continuous structure, a spatial macromolecular grid is created in SF which contributes to the growth in viscosity and also to the growth in elastic properties.*

The associations of HA molecules are the manifestation of cohesive forces among HA macromolecular chains. SF represents a dispersion system (White, 1963) in which the dispersion rate is dominantly formed by snakelike HA macromolecules. The dispersion environment is formed by water. Cohesive forces among NaHA polymer chains in SF are of physical nature. The density (number) of bonds among HA macromolecules is dominantly controlled by mechanical effects. Fig. 9. In relation to the magnitudes of velocity gradients, NaHA macromolecules are able to form “thick” synovial gel which possesses elastic properties characteristic of solid elastic materials, even though the dispersion environment of synovial gel is liquid.

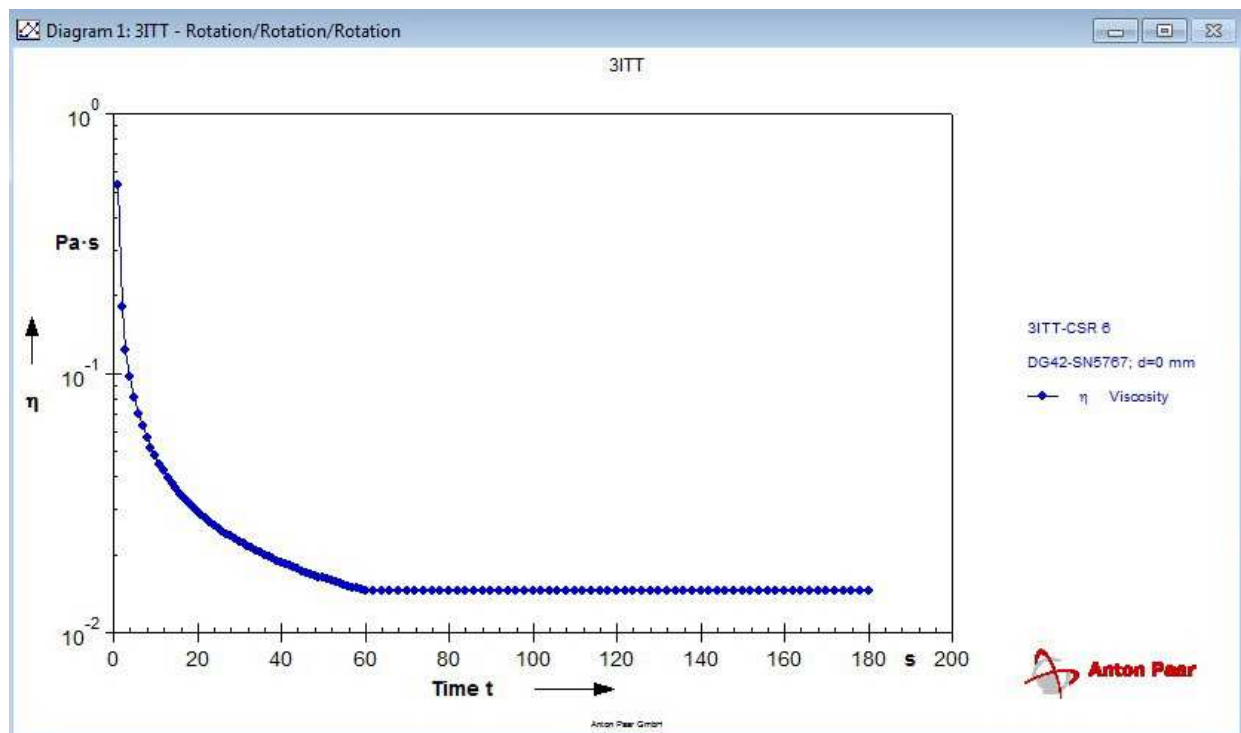


Fig. 7. Viscosity values of SF with increasing rate of flow velocity gradient  $0 - 2000 \text{ s}^{-1}$  (in time  $0 - 60 \text{ s}$ ) decrease. Viscosity values of SF are constant with constant rate of velocity gradient  $2000 \text{ s}^{-1}$  (in time  $60 - 180 \text{ s}$ )

SF represents a mobile dispersion system in which *synovial gel is generated due to non-Newtonian properties of SF*. Within this system, the macromolecules of hyaluronic acid can be intertwined into a three-dimensional grid, which continuously penetrates through the dispersion environment formed by water. The pseudoplastic properties of SF are manifested through mechanical effects (for example while walking or running), Fig. 8., Fig. 9. *Physical netting occurs, which is characterized by the interconnection of sections of polymer chains into knots or knot areas*. Generally speaking, the association of individual molecules of hyaluronic acid (HA/NaHA) occurs in cases of reduced affinity of its macromolecular chains to the solvent. In other words, the *macromolecules of hyaluronic acid (HA) form a spatial grid structure in a water solution* (Fig. 9.).

Mutually inverse shifts and inverse rotations of the opposite AC surfaces cause inverse flows of SF on its interface with the AC surface (Fig. 10.).

The greatest magnitudes of SF velocity vectors due to the effect of shear stresses  $\tau_{xy}$ , (or the effects of shifting forces respectively) are found near the upper and lower AC surface. They are, however, mutually inversely oriented. Fig. 10. displays the right-oriented velocity vector direction near the upper surface, and the left-oriented one near the lower AC surface. The magnitudes of velocity vectors decrease in the direction towards the central SF zone. In this thin neutral zone, the velocity vector is theoretically zero in value. A very thin layer (zone) of SF in the vicinity of the central zone, with very small to zero velocities, can be appointed **neutral SF zone**.

At very small velocities of SF flows, the *viscosity of the neutral central zone is higher than the viscosity in the vicinity of AC surfaces*. Under the conditions of very low viscosity, the SF material in the vicinity of AC surfaces is characterized by a low friction coefficient. Friction reaches values of ca  $0.024 - 0.047$  (Radin et al., 1971).



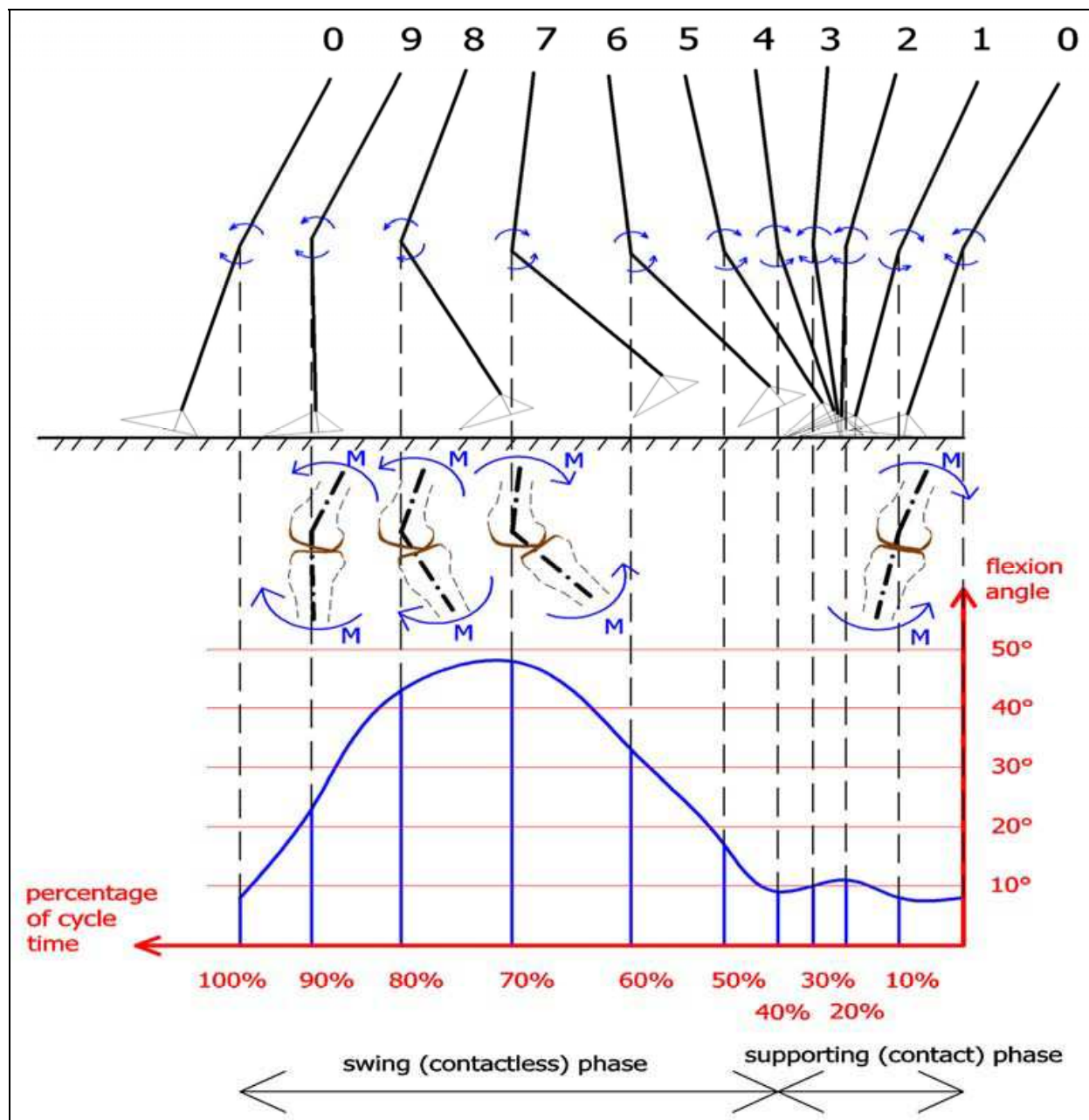


Fig. 8. Orientation diagram of the magnitudes of angles between the axes of the femoral and tibial diaphysis during the “flexion - extension - flexion” cycle of the lower limb in relation to the time percentage of the cycle

The total *thickness of the gap* between the opposite AC surfaces is only ca 50  $\mu\text{m}$ , including height roughness of the surfaces near both peripheral layers  $2 \times 2.5 \mu\text{m}$ , Fig. 4., Fig. 9. (Petrýl et al., 2010). In quiescent state, the AC surfaces are flooded with SF (synovial gel) while during the leg motion (from flexion to extension and vice versa) synovial sol with the relatively low viscosity is generated in SF in peripheral zones of AC. In other words, *due to the effect of shear stresses*  $\tau_{xy}$  the viscosity  $\eta$  of SF decreases and synovial sol is generated. Aggregations of macromolecules of hyaluronic acid decrease. *The most intense aggregations* are in places of the smallest SF velocities, i.e. in neutral (central) zone of SF between the AC surfaces.

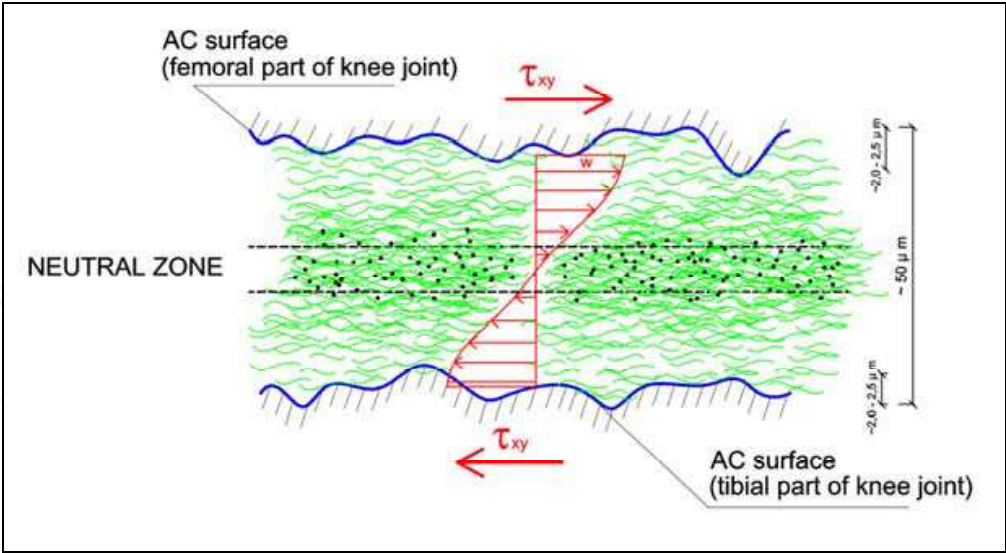


Fig. 9. Diagram of the distribution of magnitudes and directions of SF flow velocity vectors in the gap between AC surfaces. Associations of NaHA/HA macromolecules decline in places with the greatest SF flow velocity gradient, i.e. in zones adjoining each AC surface. The SF flow velocity gradient decreases in the direction towards the neutral zone

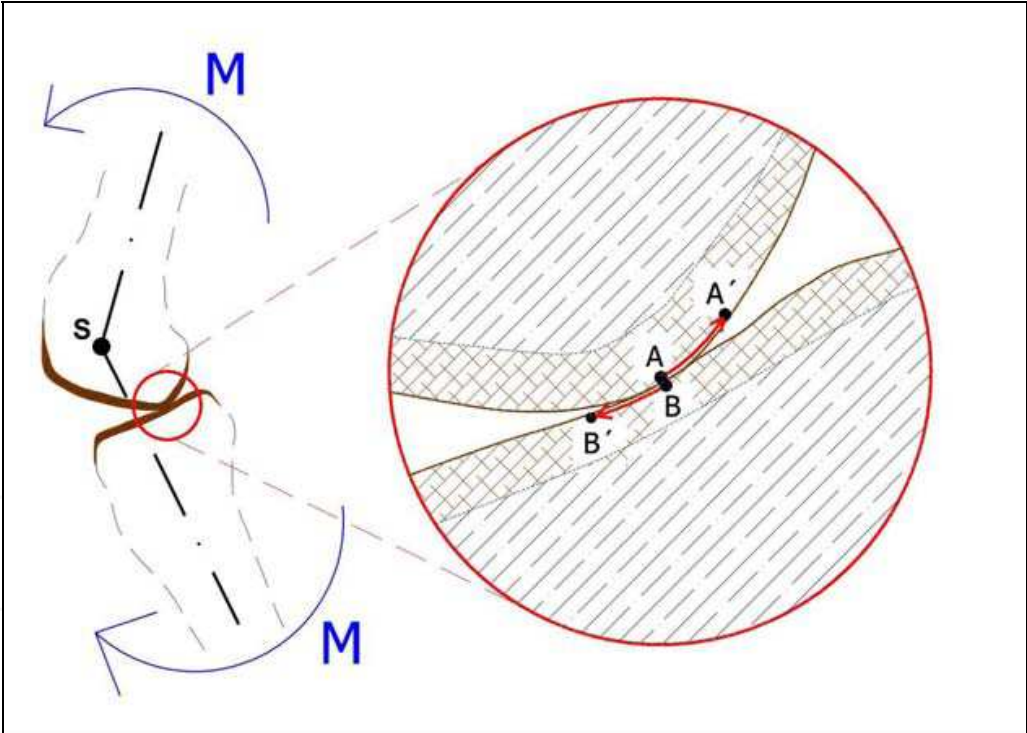


Fig. 10. Rotation of the tibial and femoral part of the knee joint during the transition from flexion to extension. During the rotation of the femoral part of the knee joint (due to the effect of the left-hand rotation moment  $M$ ) round the current (relative) centre of rotation (which is the intersection of longitudinal axes of the femur and tibia), point  $A$  moves to position  $A'$ . During a simultaneous rotation of the tibial part of the knee joint (due to the effect of the right-hand rotation moment  $M$ ) round the same current (relative) centre of rotation, point  $B$  moves to position  $B'$

Due to pseudoplastic properties of SF in the space between the opposite AC surfaces (Fig. 9.), non-physiological *abrasive wear* of the surfaces of AC peripheral zones is efficiently prevented.

The SF solution process in the gap between the AC surfaces is not an isolated phenomenon. It is interconnected (during walking, running etc.) with residual SF in the pores of the intercellular matrix in peripheral zones of the tibial and femoral part of AC. Under high loads, an *integrated unit* is generated which, after the *formation of an incompressible “cushion”*, is able to transfer extreme loads thus protecting the peripheral and internal AC structures from their destructions.

2.2 Residual strains during the cyclic loading in the articular cartilage

In agreement with our analyses, the properties and behaviors of articular cartilage in the biomechanical perspective may be described by means of a complex viscohyperelastic model (Fig. 11.). The biomechanical compartment is composed of the Kelvin Voigt viscoelastic model (in the peripheral and partially in the transitional zone of AC) and of the hyperelastic model (in the middle transitional zone and the low zone of AC). The peripheral zone is histologically limited by oval (disk shaped) chondrocytes. The viscohyperelastic properties of AC are predetermined by the specific molecular structures.

The mechanical/biomechanical properties of articular cartilage are topographically non homogeneous. The material variability and non homogeneity depends on the type and the size of physiological loading effects (Akizuki et al., 1986; Petrtyl et al., 2008).

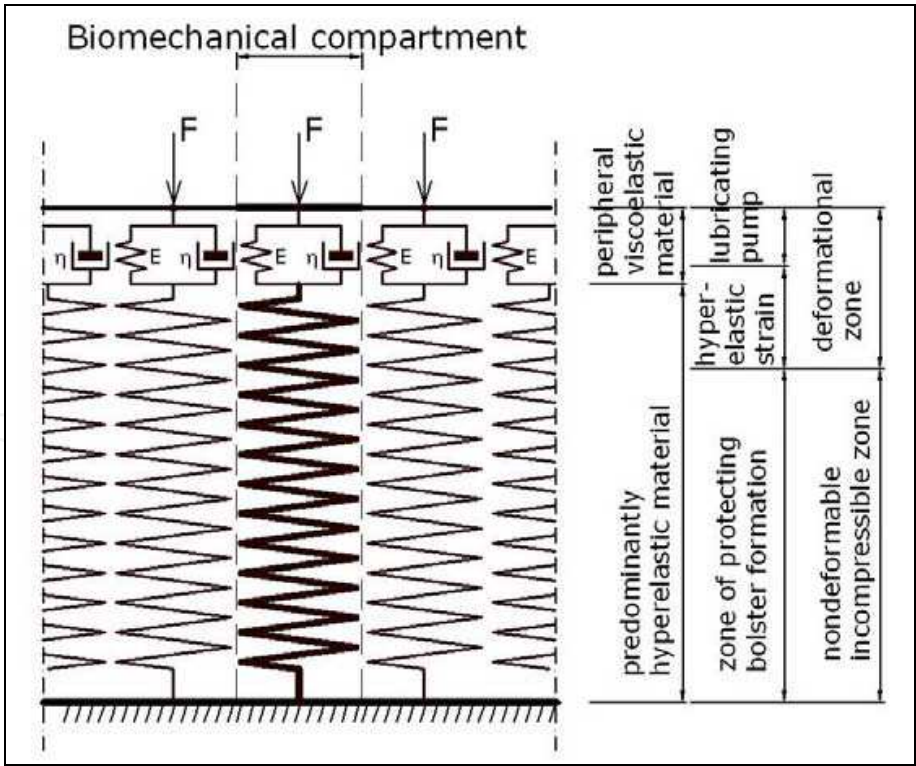


Fig. 11. Mechanical diagram of the complex viscohyperelastic model of articular cartilage. The mechanical compartment is composed of the Kelvin Voigt viscoelastic model (in the peripheral and transitional zone of AC) and of the hyperelastic model (in the middle transitional zone and the low zone of AC)



AC is composed of cells (chondrocytes), of extracellular composite material representing a reinforcing component – collagen 2<sup>nd</sup> type (Benninghoff, 1925) and of a non reinforcing, molecularly complex matrix (Bjelle, 1975). A matrix is dominantly composed of glycoprotein molecules and firmly bonded water. In the peripheral zone, there is synovial fluid unbound by ions.

The principal construction components of the matrix are glycoproteins. They possess a saccharide component (80-90 %) and a protein component (ca 20 - 10 %). Polysaccharides are composed of molecules of chondroitin-4-sulphate, chondroitin-6-sulphate and keratansulphate. They are bonded onto the bearing protein, which is further bonded onto the hyaluronic acid macromolecule by means of two binding proteins. Keratansulphates and chondroitinsulphates are proteoglycans which, through bearing and binding proteins and together with the supporting macromolecule of hyaluronic acid, constitute the proteoglycan (or glycosaminoglycan) aggregate. As the saccharide part contains spatial polyanion fields, the presence of a large number of sulphate, carboxyl and hydroxyl groups results in the creation of *extensive fields of ionic bonds with water molecules*.

The proteoglycan aggregate, together with *bonded water*, creates an amorphous extracellular material (matrix) of cartilage, which is bonded onto the reinforcing component – collagen 2<sup>nd</sup> type. Glycosaminoglycans are connected onto the supporting fibres of collagen type II by means of electrostatic bonds. In articular cartilage, nature took special efforts in safeguarding the biomechanical protection of chondrocytes in the peripheral zone. In the biomechanical perspective, chondrocytes are protected by glycocalix (i.e. a spherical saccharide envelope with firmly bonded water). Glycocalix is composed of a saccharide envelope bonded onto chondrocytes via transmembrane proteoglycans, transmembrane glycoproteins and adsorbed glycoproteins. *The glycocalix envelopes create gradually the incompressible continuous layer during the loading in peripheral zone of AC* (Fig. 12.).

Our research has been focussed on analyses of viscoelastic strains of the upper peripheral cartilage zone, on the residual strains arising at cyclic loading, on the analyses of strain rate and on the creation of a peripheral incompressible cartilage cushion.

The peripheral cartilage zone consists of chondrocytes packaged in proteoglycans (GAGs) with firmly bonded molecules of water. In the intercellular space, there is unbound synovial fluid which contains water, hyaluronic acid, lubricin, proteinases and collagenases. Synovial fluid exhibits non-Newtonian flow characteristics. As was pointed out above, under a load the synovial fluid is relocated on the surfaces of AC.

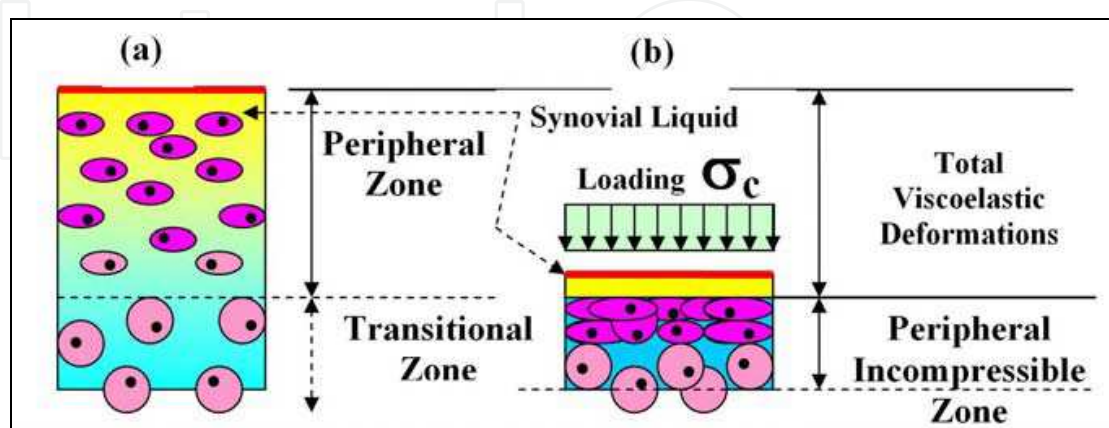


Fig. 12. Peripheral zone of articular cartilage without (a)/with (b) loads. The peripheral incompressible zone is integrated with the incompressible zone in the middle (transitional) zone and low (radial) zone

During loading, the chondrocytes with GAGs encapsulation (in the peripheral zone) create a continuous incompressible mezzo layer with protected chondrocytes. Simultaneously, an incompressible peripheral zone arises in the middle of the transitional zone and in the low (radial) zone of AC. There are dominantly hyperelastic properties in the transitional and the low radial zone (Fig. 11.). Stress states can be simulated by the modified Cauchy stress tensor for incompressible hyperelastic material.

Viscous properties in the peripheral zone of articular cartilage result from the interaction between the molecules of the extracellular matrix and the molecules of free (unbound) synovial fluid. The transport of SF molecules through the extracellular space and the lack of bonding of these molecules onto glycosaminoglycans create the basic condition for the viscous behaviors of cartilage. High dynamic forces are dominantly undertaken by the AC matrix with firmly bonded water in its low and middle zone with a simultaneous creation of an incompressible tissue, a cushion (Fig. 1.).

The articular cartilage matrix with viscoelastic properties functions dominantly as a *protective pump* and a regulator of the amount of SF permanently maintained (during cyclic loading) between articular plateaus. The importance of the *protective pump* is evident from the function of retention of AC strains during cyclic loading. Due to slow down viscoelastic strain, part of accumulated (i.e. previously discharged) SF from the preceding loading cycle is *retained in articular cartilage* (Fig. 13.).

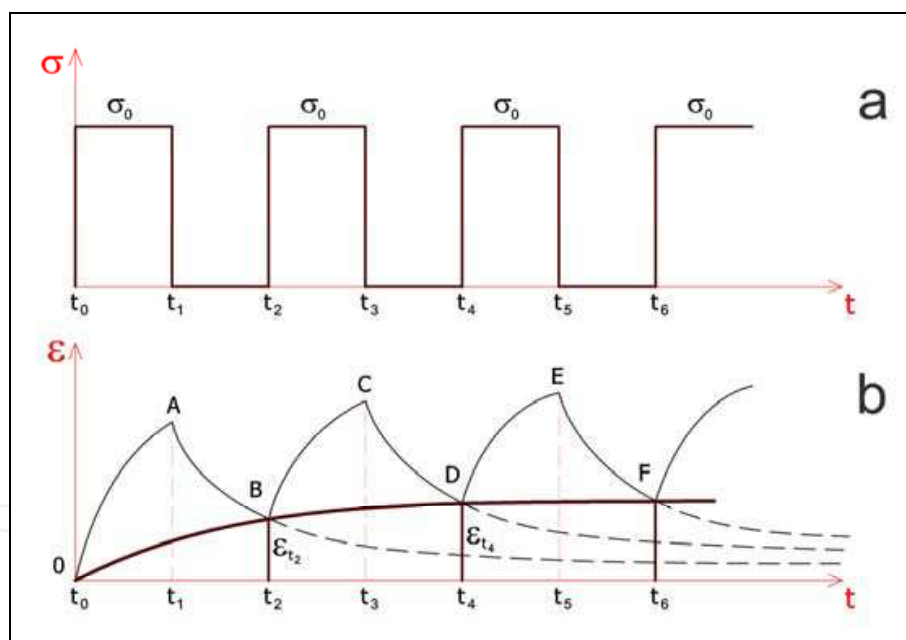


Fig. 13. Application of Kelvin Voigt viscoelastic model for the expression of step by step increments of strains  $\varepsilon_{ti}$  in the peripheral zone of AC during cyclic loading (e.g. while walking or running)

Fig. 13. in its upper part (a) shows the loading cycles e.g. during walking, while in the lower part (b) strains during the strain time growth and during strain relaxation are visible. The strain time growth occurs during the first loading (see the first concave curve OA of the strain growth). At the time  $t_1$  after unloading strain relaxation occurs (see the convex shape of the second curve AB). At the time  $t_2$  the successive (second) loading cycle starts. The strain time growth during the successive loading cycle, however, does not start at a zero



value (as was the case during the initial, first loading cycle), but at point B, or at the value of the residual strain  $\varepsilon_{t2}$ . *The first residual strain provides the initial presupplementation of articular plateaus with synovial fluid.* Fig. 13. manifests that the envelope curve OBDF slightly grows during cyclic loading to stabilize after a certain time at a steady value characterizing long-term strain (during the time of cyclic loading) and long-term presupplementation of articular space with synovial fluid. After cyclic loading stops (i.e. after AC unloading) during the last loading cycle, as seen in Fig. 1., the strain relaxation follows the convex curve, and strains asymptotically approach to the time axis  $t$  (or zero). After the termination of the last loading cycle, SF (in the form of synovial sol) is sucked back into the peripheral layer of AC. The mechanism of viscous strain time growth and viscous strain relaxation creates a highly efficient *protective pump* functioning not only to discharge and suck back synovial fluid, but also to pump (accumulate) it into the articular space. Stresses in the peripheral zone may be expressed for the Kelvin Voigt model by the constitutive equation:

$$\sigma(t) = \eta \frac{d\varepsilon(t)}{dt} + E\varepsilon(t) \quad (1)$$

where  $\eta$  is the coefficient of viscosity,  $E$  is the modulus of elasticity,  $\varepsilon(t)$  is the strain of AC and  $\frac{d\varepsilon(t)}{dt}$  is the strain rate of cartilage tissue in the peripheral zone.

Equation (1) is a first order linear differential equation for an unknown function  $\varepsilon(t)$ . The solution to the non-homogeneous equation (1) under the given initial conditions determines the time related strain of articular cartilage. In our case, it is in the form:

$$\varepsilon(t) = e^{\frac{-1}{\eta}Et} \left[ \frac{1}{\eta} \int_{t_0}^t \sigma(\tau) e^{\frac{1}{\eta}E\tau} d\tau \right] \quad (2)$$

Let us further consider the case where articular cartilage is loaded by a constant load  $\sigma(\tau) = \sigma_c = \text{const}$  (Fig. 13.) :

$$\varepsilon(t) = \frac{\sigma_c}{E} \left[ 1 - e^{\frac{-1}{\eta}E(t-t_0)} \right] \quad (3)$$

Equation (3) implies that the strain of AC is a function of time depending on the magnitude of the constant stress  $\sigma_c$  also (for example by shifting an individual's weight onto one foot).

The presence of residual strain (marked by a thick line in Fig. 13.) ensures the accumulation of synovial fluid between articular plateaus. It means that during each step (during cyclic loading) articular plateaus are presupplemented with the lubrication medium – synovial fluid. The magnitudes of residual strains of AC play a key role in the presupplementation of AC surface plateaus with synovial fluid. The magnitudes of residual strains may be determined from the functions expressing strain during the strain time growth and from the functions expressing strain during the strain relaxation of AC, this may be performed separately for each loading cycle of cartilage (Fig. 13.).

For the 1<sup>st</sup> phase of the first loading cycle, for  $t \in < t_0; t_1 >$ , (Fig. 13.) the concave curve is defined by function (3) for the articular cartilage strain.

Discrete strain at the time  $t_0$  is  $\varepsilon_{t_0} = 0$ , at the time  $t_1$  discrete strain is:

$$\varepsilon_{t_1} = \frac{\sigma_c}{E} \left[ 1 - e^{\frac{-1}{\eta} E(t_1 - t_0)} \right] \quad (4)$$

For the 2<sup>nd</sup> phase of the first loading cycle (for  $t \in \langle t_1; t_2 \rangle$ ) (Fig. 13.), the convex curve AB is defined by the function for articular cartilage strain:

$$\varepsilon(t) = \varepsilon_{t_1} e^{\frac{-1}{\eta} E(t - t_1)} \quad (5)$$

Discrete strain at the time  $t_0$  is  $\varepsilon_{t_0} = 0$ , at the time  $t_1$  discrete strain is:

$$\varepsilon_{t_2} = \varepsilon_{t_1} e^{\frac{-1}{\eta} E(t_2 - t_1)} \quad (6)$$

The magnitudes of strains during cyclic loading at the starting points of loading and unloading of articular cartilage may be expressed by recurrent relations. For the time  $t_i$  with an odd index, the strain at the respective nodal points is:

$$\varepsilon_{t_{(2k+1)}} = \frac{\sigma_c}{E} \left[ 1 - e^{\frac{-E}{\eta} (k+1)l} \right] \quad ]_{k=0,1,2,\dots} \quad (7)$$

where  $l$  is the length of the time interval  $\langle t_i; t_{i+1} \rangle$ . For the time  $t_i$  with an even index, the strain is :

$$\varepsilon_{t_{2k}} = \frac{\sigma_c}{E} \left[ e^{\frac{-E}{\eta} l} - e^{\frac{-E}{\eta} (k+1)l} \right] \quad ]_{k=0,1,2,\dots} \quad (8)$$

where  $l$  is the length of the time interval  $\langle t_i; t_{i+1} \rangle$ ,  $i = 0, 1, 2, \dots$ . During long-term cyclic loading and unloading, for  $k \rightarrow \infty$  the strain  $\varepsilon_{t(2k+1)}$  asymptotically approaches the steady state  $\sigma_c/E$ ; for  $k \rightarrow \infty$  the strain  $\varepsilon_{t2k}$  asymptotically approaches the steady state  $\frac{\sigma_c}{E} e^{\frac{-E}{\eta} l}$ . It is evident that for  $k \rightarrow \infty$  it holds true that:

$$\varepsilon_{t_{(2k+1)}} = \frac{\sigma_c}{E} > \varepsilon_{t_{2k}} = \frac{\sigma_c}{E} e^{\frac{-E}{\eta} l} \quad (9)$$

### 2.2.1 The strain rate of articular cartilage in peripheral zone during the strain time growth

Strain  $\varepsilon(t)$  of AC during the strain time growth in the interval of  $t \in \langle t_0; t_1 \rangle$  is given by equation (3). Because  $\frac{d\varepsilon(t)}{dt} = \dot{\varepsilon}(t) > 0$  (in the indicated interval) the function  $\varepsilon(t)$  is increasing. The strain rate of AC during the strain-time growth in interval of  $t \in \langle t_0; t_1 \rangle$  is given by equation (10):

$$\frac{d\varepsilon(t)}{dt} = \frac{\sigma_c}{\eta} e^{\frac{-1}{\eta} E(t-t_0)} \quad (10)$$

From equation (10) is evident, that the strain rate of articular cartilage in interval of  $t \in \langle t_0; t_1 \rangle$  decelerates. The strain rate shortly after the load is the highest.

### 2.2.2 The strain rate of articular cartilage in peripheral zone during the strain relaxation

The strain of peripheral zone in time  $t_1$  during unloading is given by equation (11):

$$\varepsilon(t) = \varepsilon_{t_1} e^{\frac{-1}{\eta} E(t-t_1)} \quad (11)$$

The strain rate  $\frac{d\varepsilon(t)}{dt} = \dot{\varepsilon}(t) < 0$ . It means that the strain function  $\varepsilon(t)$  in interval of  $t \in \langle t_1; t_2 \rangle$  is decreasing. The strain rate in the same interval of  $\langle t_1; t_2 \rangle$  is decreasing also. Strain rate during the strain relaxation in interval of  $\langle t_1; t_2 \rangle$  is given by equation (12):

$$\frac{d\varepsilon(t)}{dt} = \varepsilon_{t_1} e^{\frac{-1}{\eta} E(t-t_1)} \left[ -\frac{E}{\eta} \right] \quad (12)$$

The strain rate of articular cartilage shortly after the unloading (during the strain relaxation) is distinctly higher than to the end of interval of  $\langle t_1; t_2 \rangle$ . Strain rate  $\left| \frac{d\varepsilon(t)}{dt} \right|$  with increasing time in interval of  $t \in \langle t_1; t_2 \rangle$  is decreasing.

## 3. Conclusions

The above described analyses lead to the formulation of the following key conclusions: Synovial fluid is a viscous pseudoplastic non-Newtonian fluid. Apparent viscosity of SF decreases with increasing rate of flow velocity gradient. SF does not display a decrease in viscosity *over time at a constant flow velocity gradient* (as it is typical for thixotropic material). The rheological properties of synovial fluid essentially affect the biomechanical behaviour of SF between the opposite AC surfaces and in the peripheral AC zone also. During the shifts of the femoral and tibial part of AC in opposite directions the velocities of SF flows decrease in the direction towards the neutral central zone of the gap between the AC surfaces. **Non-linear abatement in viscosity** in the direction from the neutral ("quiescent") layer of SF towards the opposite AC surfaces contributes to the lubrication quality and very efficiently protect the uneven micro-surfaces of AC.

The viscoelastic properties of the peripheral zone of AC and its molecular structure ensure the regulation of the transport and accumulation of SF between articular plateaus. The hydrodynamic lubrication biomechanism adapts with high sensitivity to biomechanical stresses. The viscoelastic properties of AC in the peripheral zone ensure that during cyclic loading some amount of SF is always retained accumulated between articular plateaus, which were presupplemented with it in the previous loading cycle. During long-term harmonic cyclic loading and unloading, the strains stabilize at limit values.

The limit strain value of AC during loading is always greater than its limit strain value after unloading. Shortly after loading, the strain rate is always greater than before unloading. In

this way, the hydrodynamic biomechanism quickly presupplements the surface localities with lubrication material. Shortly after unloading, the strain rate is high. During strain relaxation, it slows down. This is the way how the articular cartilage tissue attempts to retain the lubrication material between the articular plateaus of synovial joints as long as possible during cyclic loading.

Analogically to the low and the middle zone of AC where an incompressible zone arises under high loads whose dominant function is to bear high loads and protect chondrocytes with the intercellular matrix from destruction, in the peripheral zone as well a partial incompressible zone arises whose function is to bear high loads and protect the peripheral tissue from mechanical failure. The appearance of the incompressible tissue in all zones is synchronized aiming at the creation of a single (integrated) *incompressible cushion*. The existence of an incompressible zone secures the protection of chondrocytes and extracellular material from potential destruction.

### 3.1 Significance of results for clinical practice

Metabolic processes during the HA synthesis are very dynamic. The chondrocytes in AC actively synthesize and catabolise HA so that its optimal “usability” is achieved (in a relatively short time). The HA synthesis is usually in equilibrium with catabolic processes. These processes result in the achievement of the optimum HA concentration. The studies of metabolic processes (Schurz et al., 1987) implied that the half-life of the functional existence of HA molecules are mere 2-3 weeks. The solved project makes it evident that the “short lifecycle” of HA is dominantly caused by dynamic (biomechanical) effects. During leg movements, long snakelike NaHA/HA macromolecules are exposed to fast changes in shape accompanied by permanently arising and vanishing physical (non-covalent) bonds. To avoid the shortage of HA/NaHA, old polymer chains are replaced with new chains. The disturbance of HA new formation processes may lead to initiations of pathological processes. Mechanical effects during movements continuously initiate new groupings of HA macromolecules and newly arising (and vanishing) bonds among them. Frequent variations of kinetic energy transfers into HA molecular structures contribute to HA fragmentations in the biophysical perspective. These fragmentations may also be biochemically accelerated by hyaluronisades (Saari et al., 1993). HA fragments may initiate the formation of macrophages and extensive inflammations of AC.

The above examples of the interrelated nature of the causes of some AC defects show the key role of the rheological properties of non-Newtonian synovial fluid.

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