MATHEMATICAL ANALYSIS OF STRESS RELAXATION
IN ARTICULAR CARTILAGE DURING COMPRESSION

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Introduction

Articular cartilage is the avascular bearing material covering the articulating ends of the mating bony segments of synovial joints. Functionally articular cartilage provides a near frictionless surface, whose coefficient of friction 0.002, which is tough and wear resistant. These biomechanical functions of the tissue are preserved so long as the tissue is maintained in a normal physiological state. The destruction and subsequent loss of articular cartilage as a result of degenerative joint diseases would lead to joint stiffness, pain and deformities. An abnormal state of mechanical stress exerted upon the tissue is an initiating factor or at least a precursor of chondrodegeneration [9]. The morphology of pre-clinical ultrastructural damage, resulting from biomechanical causes, of the surface collagen fibers from human femoral heads have been investigated and codified [8]. These alterations are believed to be mechanically induced and are related therefore to the inherent mechanical properties of the tissue.

The superb functional characteristics of articular cartilage are the outward manifestations of its unique deformational behavior during loading. The latter is in turn dependent on the composition of the substance and the ultrastructural architecture of the tissue. Relative to other tissues, articular cartilage has a very unusual composition. Sixty or eighty percent of its substance by weight is water. The remaining material, i.e., the solid phase, is fractionated approximately as follows: 72% collagen fibers and 28% proteoglycaus and other non-collagenous proteins [3]. It also dif-

fers from other tissue in that the substance of this solid phase is mostly extracellular. As such, the biomechanical behavior of articular cartilage, to the first approximation, may be considered as an inert hyperhydrated hydrogel.

As a result of the unique combination of tissue biology, substance and ultrastructure, cartilage may be viewed as a porous, permeable viscoelastic solid matrix filled with an incompressible fluid, i.e., water. A large fraction of the interstitial water moves freely about and may be exuded or imbibed upon loading and unloading [1]. It is the stiffest of all the soft tissues but ten times less stiff than the subchondral bone. It is able to maintain its original shape when subjected to the action of compressive stresses of short duration and creep considerably under compressive stresses on longer duration. At first glance, the whole tissue appears to behave as in a typical viscoelastic solid. However, it was soon discovered that total deformational recovery is not possible unless the tissue is bathed in a fluid. This suggests that the movement of the interstitial fluid is intrinsically coupled to the overall deformational behavior of the tissue.

The movements of the interstitial fluid across the articular surface are also vital in the understanding of the natural lubrication phenomenon occurring within synovial joints. Recently Mansour and Mow [5] and Torzilli and Mow [10] have calculated the exact manner of this fluid transport within the tissue and the flux of fluid across the surface of the tissue. It was shown that on the one hand, for material properties of normal articular cartilage (permeability, surface porosity, stiffness, etc.) the surface flux

distributions occurred in such a manner as to generate a thin film of fluid lubricant at the surface of that tissue. This form of animal lubrication in fact was suggested qualitatively by Radin and Paul [7]. On the other hand for material properties of very pathological tissues, the flow patterns and the surface flux distributions were substantially altered so that the existing natural lubrication process could be defeated. Finally, since the tissue is avascular, the transport of this interstitial fluid into the joint cavity, mixing of this exudate with the synovial fluid and its return into the tissue provides the necessary pathways for the transport of vital nutritive substances required by the chondrocytes and for the removal of their metabolic waste products. Thus it is important both biologically and biomechanically to be able to describe the fundamental biphasic rheological response of the tissue and predict its response during physiological function.

The biphasic rheological model of articular cartilage proposed by Mow and Mansour [6] has been used to describe the nonlinear interaction between the creep-like behavior of cartilage in compression and the exudation of the interstitial fluid. Based upon previous experimental evidence in the literature, the tissue was regarded as a mixture of a porous permeable Kelvin-Voigt type viscoelastic solid matrix composed of chondrocytes, collagen fibers and proteoglycans and an incompressible interstitial fluid, water. The mechanical interactions giving rise to the flow of the fluid relative to the permeable solid matrix are described by Darcy's law and Biot's consolidation mechanism. The permeability (K) of the tissue has been measured as

a function of driving pressure and compression [4]. The function $K(e)$, where e is the compressive volume change per unit initial volume, was found to decrease monotonically with increasing loss of volume. The creep-like behavior of the cartilage in compression has been analytically treated [6]. In the present paper the stress relaxation behavior of articular cartilage during confined compression is to be described mathematically.

Stress relaxation of articular cartilage during confined compression was measured in an experimental apparatus, Fig. 1, especially designed for the purpose [2]. In this apparatus, circular cylindrical plugs of articular cartilage and its underlying bone were compressed at constant speeds against a porous platen attached to a load cell. Continuous records of the resulting axial load were monitored. Simultaneously, the change of the axial dimension of the tissue was by a measured displacement transducer and recorded. At selected times T or compressive strains, the motor drive was stopped and the stress relaxation, i.e., the decay of stress in time, was obtained from the load cell. For sufficiently long times a non-zero asymptotic stress was reached. A typical stress history curve at a compressive strain of 21.42% is shown in Fig. 2. In general qualitative terms it has been experimentally observed that the compressive stress at the articular surface under conditions of confined compression have the following properties: (i) it increased monotonically in time during the compressive phase of the experiment, (ii) it decayed monotonically in time during the relaxation phase of the experiment, (iii) it approached a non-zero steady state value as $t \rightarrow +\infty$, (iv) the relaxation time* depends only on the magnitude of the compressive strain at

* The relaxation time is mathematically defined by Eq. 6.

steady state [2]. Result (iv) is summarized in Fig.3 where the sixteen normal samples of articular cartilage plugs were compressed at various rates of compression. The abscissa of Fig.3 represents the equilibrium compressive strain. This curved semi-log plot of relaxation time, defined here by a single exponential decay function, shows that the stress relaxation of articular cartilage may not be described by a single exponential law. Thus the simple linear viscoelasticity theories does not accurately describe this basic deformational behavior of the tissue. These observed general stress history and stress relaxation characteristics can be attributed to the exudation of fluid from the tissue and the viscoelastic nature of the solid matrix. The former is controlled by the permeability and the stiffness of the tissue whereas the latter is determined by the inherent macromolecular conformation of the solid matrix. Thus another objective of this paper is to assess the relative importance of these two mechanisms of stress relaxation phenomenon in cartilage.

2. Mathematical Statement of Problem

Under compression the displacement of cartilage $u(x,t)$ is governed by a nonlinear partial differential equation [6] of the form

$$u_t = K(e)u_{xx} \quad 0 < t, \quad 0 \leq x < 1. \quad (1)$$

Here normalized and non-dimensional units are used to describe the dependent and independent variables. The function

$$e(x,t) = u_x(x,t) \quad (2)$$

represents the dilatation field and in this problem it should be strictly negative, i.e., compressive. The boundary conditions corresponding to the experimental configuration are

$$u(0,t) = U(t) \quad \text{and} \quad u(1,t) = 0, \quad 0 \leq t \quad (3)$$

where $U(t)$ is assumed to be a continuously differentiable, monotonically increasing function with $U'(t)$ nondecreasing for $t < T$ and

$$U(t) \equiv U(T), \quad t > T \quad (4)$$

where T is defined as the motor-shut off time.

The main concern of this analysis is the function

$$f(t) = -e(0,t) \quad (>0) \quad (5)$$

This function represents the non-dimensional magnitude of the compressive stress at the porous free draining platen boundary, Fig. 1. The general character of this function has been ascertained experimentally and described in the introduction. The function $K(e)$ is directly related to the permeability of the tissue.

Since Eq. (1) is nonlinear, a general closed form solution for $f(t)$ is not available. Further, since one does not have a precise a priori knowledge of the function $K(e)$, except that it is monotonically increasing and presumably continuously differentiable, it is then important to work in as general a framework as possible. From the mathematical analysis it is possible to deduce various functional properties of the stress history function $f(t)$.

DEFINITION OF RELAXATION TIME. Consider an arbitrary function $f(t)$ which is defined in a neighborhood of $+\infty$. Suppose that (i) $f(t)$ tends to a limit, f_∞ , as $t \rightarrow +\infty$ and $f(t) - f_\infty > 0$ (for t sufficiently large) and (ii) $\lim_{t \rightarrow +\infty} t^{-1} \ln(f(t) - f_\infty)$ exists and is different from zero, then a characteristic time may be defined by the expression

$$\tau(f) = -[\lim_{t \rightarrow +\infty} t^{-1} \ln(f(t) - f_\infty)]^{-1} \quad (6)$$

To appreciate this definition consider the following two simple examples. In the case of a single decay function $g(t) = a_1 e^{-t/\tau_1}$, where a_1 and τ_1 are positive constants, Eq.(6) yields $\tau(g) = \tau_1$, the relaxation time of single decay function (2). In the case of a function of the form

$$h(t) = A + \sum_{n=1}^{\infty} a_n e^{-t/\tau_n} \quad (7)$$

where A, a_n, τ_n are constants such that $a > 0$ and $\tau_1 > \tau_2 > \dots > 0$, then Eq.(6) gives $\tau(h) = \tau_1$.

The stress relaxation function $f(t)$ $t > T$ is not a single decay function nor does it have the form presented in Eq.(7). It is however possible to deduce from the mathematical model, Eqs. (1) - (4), that $f(t)$ has the following properties

- 1°) $f(t)$ is non-decreasing for $t < T$
- 2°) $f(t)$ is non-increasing for $t > T$
- 3°) $f_\infty (\equiv \lim_{t \rightarrow +\infty} f(t)) = U(T)$
- 4°) $\tau(f) = (\pi^2 K(-U(T)))^{-1}$

The particular case which corresponds to the experimental configuration of Lipshitz et. al [2] is described by

$$U(t) = pt, \quad 0 < t < T \quad (8)$$

where p is positive constant. If in addition, $K(e)$ is assumed to be linearly dependent on e , i.e., $K(e) = 1 + \Gamma e$ then for this case $f_\infty = pT$ and 4°) becomes $\tau(f) = [\pi^2(1 - \Gamma pT)]^{-1}$.

The formula 4°) substantiates the experimental observations that the relaxation time appears to depend only on the magnitude of the imposed or equilibrium compressive strain $U(T)$, Fig. 3. Finally the consistency between experimental findings and the results of the present mathematical analysis is further demonstrated by comparing an experimental stress relaxation curve with a computer generated stress history curve, Fig. 4.

Before preceding to the mathematical derivation of the stress relaxation formula, Section 3, one might consider the following more intuitive argument. For long times the stress $-u_x(x, t)$ assumes a steady state value which is independent of x . Since $u(0, +\infty) = U(T)$ and $u(1, +\infty) = 0$, then the steady state value of $u(x, +\infty) = U(T)(1 - x)$ and $-u_x(x, +\infty) = U(T)$. Thus $u(x, t)$ is asymptotic to the solution of the linear partial differential equation problem

$$u_t^{(\infty)}(x, t) = K_\infty u_{xx}^{(\infty)}(x, t)$$

where

$$K_\infty = K(-U(T))$$

and $u^{(\infty)}(x, t)$ satisfies the same boundary conditions and initial conditions as $u(x, t)$. However, the linear problem has a closed form solution and one calculates this to be

$$\begin{aligned}
 f^{(\infty)}(t) &= -u_x^{(\infty)}(0, t) \\
 &= U(T) + \sum_1^{\infty} a_n^{(\infty)} \exp(-t/\tau_n^{(\infty)})
 \end{aligned}$$

for $t > T$, where

$$a_n^{(\infty)} = 2 \int_0^T U'(\tau) \exp(K_{\infty} n^2 \pi^2 \tau) d\tau$$

and

$$\tau_n^{(\infty)} = [K_{\infty} n^2 \pi^2]^{-1}$$

Now $f^{(\infty)}(t)$ has the form presented in Eq. (7) and so

$$\tau(f^{(\infty)}) = \tau_1^{(\infty)} = [K_{\infty} \pi^2]^{-1} = [\pi^2 K(-U(T))]^{-1}.$$

Since $f(t)$ is asymptotic to $f^{(\infty)}(t)$, the relaxation time $\tau(f)$ is given by this expression.

3. Summary of Mathematical Analysis

The details concerning the mathematical analysis used to obtain the properties 1°) thru 4°) are involved and therefore they will be presented elsewhere [12]. A brief summary of the mathematical argument is presented here.

The fundamental analytical tool used here is the well known maximum principle [11] which states, roughly, that the extreme values of a solution of a partial differential equation problem are taken on at the boundaries, i.e., the extreme values are determined by the initial conditions and the boundary conditions. For example one may apply the maximum principle to show that, if $u(0, t) \geq 0$, $u(1, t) \geq 0$ and $u(x, 0) \geq 0$, then

$$u(x, t) \geq 0, \quad 0 < x < 1, \quad t > 0 \quad (9)$$

Moreover, the partial derivative u_x and u_t are also solutions of a partial differential equation problem so that the maximum principle may be applied to deduce** that

$$u_t(x, t) \geq 0, \quad u_x(x, t) \leq 0, \quad 0 < x < 1, \quad t > 0. \quad (10)$$

Since $K(e) > 0$ and $u_{xx} = K^{-1}(e) u_t$, one has further

$$u_{xx}(x, t) \geq 0, \quad 0 < x < 1, \quad t > 0. \quad (11)$$

In the application of the maximum principle to u_t , if one takes into account the monotonicity of the data function $U(t)$, one finds that, if $t_0 \leq T$, $u_t(x, t)$ takes on its maximum value in the rectangle $0 \leq t \leq t_0$, $0 \leq x \leq 1$ at the point $x = 0$, $t = t_0$, i.e.,

$$0 \leq u_t(x, t) \leq U'(t_0), \quad 0 \leq t \leq t_0 \leq T, \quad 0 \leq x \leq 1 \quad (12)$$

Property 1°) follows from Eq. (12) and property 2°) may be deduced from Eq. (10) and the fact that $u(0, t) = U(t)$ is constant for $t > T$.

The derivations of properties 3°) and 4°) rely on comparisons of the solution of the nonlinear partial differential equation, $u(x, t)$, with solutions of linear problems which share the same boundary conditions and initial conditions. These comparisons are made also on the basis of the maximum principle. To do this let the bounds of the permeability function be given by K_0 and K_1 such that

** The argument makes use of the fact that e_t may not approach negative infinity in finite time.

$$K_0 \leq K(e) \leq K_1, \quad 0 \leq x \leq 1, \quad t \geq 0 \quad (13)$$

Since $K(e)$ is monotonically nondecreasing and since, from properties 1°) and 2°) and Eq. (10),

$$-f(T) \leq e(x, t) \leq 0, \quad 0 < x < 1, \quad t > 0 \quad (14)$$

then K_0 and K_1 are given by

$$K_0 = K(-f(T)), \quad K_1 = K(0) \quad (15)$$

Let $u^{(i)}$, $i = 0, 1$, be the solution of the linear problem

$$u_t^{(i)} = K_i u_{xxx}^{(i)}(x, t) \quad (16)$$

satisfying the same boundary conditions and initial conditions as $u(x, t)$. Then by the aid of the maximum principle and Eq. (10) one obtains the comparison

$$u^{(0)}(x, t) \leq u(x, t) \leq u^{(1)}(x, t) \quad (17)$$

It follows from the above relationship that

$$f^{(1)}(t) \leq f(t) \leq f^{(0)}(t), \quad t \geq 0 \quad (18)$$

where

$$f^{(i)}(t) = -u_x^{(i)}(0, t) > 0, \quad t \geq 0 \quad (19)$$

The $u^{(i)}(x, t)$'s, being solutions of a linear problem, have closed form solutions. Also $f^{(i)}(t)$ have closed form solutions and are given by

$$f^{(i)}(t) = U(T) + \sum_{n=1}^{\infty} a_n^{(i)} \exp(-t/\tau_n^{(i)}), \quad t \geq T \quad (20)$$

where

$$a_n^{(i)} = 2 \int_0^T U'(\tau) e^{K_i n^2 \pi^2 \tau} d\tau \quad (21)$$

and

$$\tau_n^{(i)} = [K_i n^2 \pi^2]^{-1} \quad (22)$$

Since both $f^{(0)}(t)$ and $f^{(1)}(t)$ have a common limit as $t \rightarrow \infty$ Eq.(18) yields property 3°).

In order to obtain property 4°) a tighter comparisons must be obtained. One may show further, again with the aid of the maximum principle, that

$$-f(t_0) \leq e(x, t) \leq u_x(1, t_0), \quad 0 \leq x \leq 1, \quad t \geq t_0. \quad (23)$$

Let $t_0 > T$ be fixed and let J_0 and J_1 be constants defined by

$$J_0(t_0) = K(-f(t_0)), \quad J_1(t_0) = K(u_x(1, t_0)). \quad (24)$$

Since $k(e)$ is monotonically non-decreasing Eq. (23) yields

$$J_0(t_0) \leq K(e) \leq J_1(t_0), \quad 0 \leq x \leq 1, \quad t \geq t_0 \quad (25)$$

Now let $w^{(i)}(x, t; t_0)$ denote the solution of the linear problem

$$w_t^{(i)} = J_i w_{xx}^{(i)}, \quad i = 0, 1 \quad (26)$$

which satisfy the same boundary conditions and initial conditions as $u(x, t)$ in the region $0 \leq x \leq 1, \quad t \geq t_0$. One obtains, by the maximum principle and Eq. (25), the comparisons

$$w^{(0)}(x, t; t_0) \leq u(x, t) \leq w^{(1)}(x, t; t_0), \quad 0 \leq x \leq 1, \quad t \geq t_0. \quad (27)$$

It follows from this that

$$f^{(1)}(t; t_0) \leq f(t) \leq f^{(0)}(t; t_0), \quad t \geq t_0 \quad (28)$$

where

$$f^{(i)}(t; t_0) = -w_x^{(i)}(0, t; t_0), \quad t \geq t_0. \quad (29)$$

Since $w^{(i)}$ is a solution of a linear problem a representation of is available and one finds that

$$f^{(i)}(t; t_0) - U(T) = \sum_1^{\infty} C_n^{(i)} \exp[-n^2 \pi^2 J_i(t_0) t] > 0. \quad (30)$$

Observe that $f^{(i)}(t; t_0)$ has the form presented in Eq. (7) and so

$\tau(f^{(i)}(t; t_0)) = (\pi^2 J_i(t_0))^{-1}$. It then follows from Eq. (28) that

$$-\pi^2 J_1(t_0) \leq \liminf_{t \rightarrow +\infty} [t^{-1}(f(t) - U(T))] \leq \limsup_{t \rightarrow +\infty} [t^{-1}(f(t) - U(T))] \leq -\pi^2 J_0(t_0).$$

Recall that $f(t_0)$ tends to $U(T)$ as $t_0 \rightarrow +\infty$. One may also show in a similar fashion that $-u_x(1, t)$ is monotonically non-decreasing and

$$-u_x(1, t) \rightarrow U(T) \quad \text{as} \quad t \rightarrow +\infty \quad (31)$$

Thus, from Eq. (24), one obtains

$$\lim_{t_0 \rightarrow +\infty} J_0(t_0) = \lim_{t_0 \rightarrow +\infty} J_1(t_0) = K(-U(T)). \quad (32)$$

One concludes then, from the last chain of inequalities, that

$\lim_{t \rightarrow +\infty} [t^{-1}(f(t) - U(T))]$ exists and equals $-\pi^2 K(-U(T))$. Thus property 4° is established.

Finally observe from Eq.'s (23), (31) and property 3°), that $e(x, t)$ converges uniformly to $-U(T)$ as $t \rightarrow +\infty$, i.e.,

$$e(x, +\infty) = -U(T) \quad (33)$$

DISCUSSIONS AND CONCLUSIONS: The biphasic rheological model proposed by Mow and Mansour [6] has been utilized to describe the stress relaxation phenomenon in articular cartilage during confined compression. The general characteristics of the stress history and the stress relaxation phenomenon, as determined experimentally at the porous platen, are: (i) it increased monotonically in time during the compressive phase of the experiment, (ii) it decreased monotonically during the relaxation phase of the experiment, (iii) it reached a non-zero steady state value as $t \rightarrow +\infty$ and (iv) the relaxation time τ depends on the magnitude of the average compressive strain. Experimental results (i), (ii), (iii) and (iv) correspond to the mathematical results 1°), 2°), 3°), and 4°) respectively.

Thus the general character of the mathematical results agree qualitatively, in all respects, with the experimental results. This provides confirmation as to the validity of the proposed biphasic rheological model of articular cartilage, i.e., its deformational behavior may be described by a nonlinearly permeable Kelvin-Voigt type viscoelastic solid matrix and an incompressible fluid. The two phase interactions are described by a Darcy type nonlinear permeability relationship and a consolidation mechanism. Further from the comparison result of Eq.'s (18) and (20) and 3°) an additional verification is obtained. Inherent in the rheological model is the assumption of linear elasticity for the spring portion of the Kelvin-Voigt type viscoelastic body. Equation (20) and 3°) provides the measure of an aggregate

linear elastic constant. A typical experimental result, Fig. 5 clearly shows linear relationship between the equilibrium stress and equilibrium strain of the solid matrix. The value of this aggregate elastic constant, the linear spring constant of the Kelvin-Voigt type viscoelastic body, for normal bovine cartilage is $0.873 \pm 0.35 \text{ MN/m}^2$ on sixteen samples.

A typical numerical result of the nonlinear stress relaxation phenomenon, corresponding to the example defined by Eq. (8) and $K(e) = 1 + \Gamma e$ is shown in Fig. 4. The solution $f(t)$ of the nonlinear problem is indeed bounded by the two linear approximations $f^{(0)}(t)$ and $f^{(1)}(t)$. The function $f^{(0)}(t)$ corresponds to the case of the constant low permeability K_0 and the function $f^{(1)}(t)$ corresponds to the case of the constant high permeability K_1 . These numerical results confirm the fact that a lower permeability producing a lower exudation rate would yield a higher stress and a slower stress relaxation phenomenon. The converse is also true. The transition of the nonlinear stress relaxation solution $f(t)$ from $f^{(0)}(t)$ during the initial period of stress relaxation to $f^{(1)}(t)$ as $t \rightarrow \infty$ shows the rate limiting effects of $K[-f(t)]$. By Eq. (13), $K_0 = K[-f(T)] \leq K[e(x,t)]$ and by Eq. (33) $K[e(x,t)] \rightarrow K[-U^{(m)}] \leq K_1$. Only if $K[f(t)]$ at the surface limits the rate of exudation can $f(t) \rightarrow f^{(0)}(t)$ during the initial period of stress relaxation. As stress relaxation progresses, $K[e(x,t)]$ increases and approaches the constant value $K[-U(T)]$. This results in $f(t)$ approaching $f^{(1)}(t)$ from above.

However detailed examinations of the experimental results on the stress history, e.g., Fig. 2, and numerical results, Fig. 4, reveals a number of fine discrepancies. During the compression phase, i.e., $t < T$, the experimental stress history curve is slightly concave whereas the numerical

results are slightly convex. Figure 4 also shows that the experimental stress decay curve tends to stay up near the low permeability linear bound $f^{(0)}$ during the initial period of relaxation and transit gradually to the high permeability linear bound $f^{(1)}$ as $t \rightarrow \infty$. Further at $t = 40$ seconds, the numerical solution has already reached its asymptotic limit whereas the experimental data shows a continuous downward trend. It is believed that these discrepancies between the experimental data and the nonlinear solution are due to the assumed functional dependence of the permeability on the dilatational field; $K = 1 + \Gamma e$. This form, suggested by Mow and Mansour [6], is the simplest model to the system. However this simple linear dependence can quickly lead to the non-physical situations of negative permeability, i.e., $K < 0$. Finding the appropriate $K(e)$ remains a problem. A more realistic functionality would be the choice $K = \alpha \exp(\Gamma e)$ where α and Γ are positive constants. Qualitative arguments show that this form could rectify the above discrepancies. Low compressive strains would produce smaller contributions toward decreasing the permeability and thereby produce lower stresses in the initial portion of the compressive phase and asymptotic portions of the relaxation phase of the experiment. By the same token, high compressive strains would produce substantial decreases in tissue permeability. Concomitant with this decrease of tissue permeability is an increase of the peak stress. Stress relaxation would progress slower and would tend toward the low permeability

bound during the initial phase of relaxation. This could result in a concave stress history curve during the compression phase. The asymptotic value $t \rightarrow +\infty$ would not be affected by this choice since $\alpha^{-1}K(e) = \exp(\Gamma e) \approx 1 + \Gamma U(T)$ for small strains. The overall effect would be to lower the asymptotic stress relative to the peak stress; as observed experimentally.

In conclusion this mathematical analysis of experimental findings provides an assurance as to the general validity of the proposed biphasic rheological model of Mow and Mansour [6] for articular cartilage. The general agreement is sufficiently strong to warrant numerical determination of the fundamental physical constants utilizing this proposed biphasic rheological model for cartilage. Concomittant with these investigations are parallel investigations on biochemically and biologically altered tissues [3]. The comparison of normal cartilage properties, as defined by the present proposed model, with abnormal properties ultimately will provide insights into the biomechanical pathogenesis of degenerative joint disease.

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CAPTIONS

- Fig. 1 Schematic view of the experimental apparatus, Lipshitz et. al. [2], Proc. VIIth Intern. Cong. on Rheology, Gothenburg, Sweden, 1976.
- Fig. 2 Typical stress history at the articular surface of a plug of normal cartilage specimen, no. NS-45-N during confined compression showing a rapid rise of stress during the compressive phase of the experiment and a gradual decay of compressive stress during the relaxation phase of the experiment.
- Fig. 3 Mean relaxation time, defined here by a single exponential decay function, as a function of the imposed compressive strain. Strain rates in each cluster of points ranged from 0.05S^{-1} to 0.20S^{-1} . Data also shows strain dependence and strain rate independence of this definition of relaxation time.
- Fig. 4 Typical computer generated stress history curves, solid lines, as compared with a typical experimental relaxation curve, dash lines. The numerical case corresponds to the prescribe data:
 $K = 1 + 0.3e$, $p = 5.0$, $U(T) = 0.2$. The computed non-dimensional time $\tau = 0.1078$, from Eq. 6, corresponds to a physical time of 5.39 seconds. The measured value, using a single term exponential law, is 5.07 seconds. The functions $f^{(0)}$ and $f^{(1)}$ are the upper and lower bounds corresponding to Eq. 18 and to $K_0 \leq K \leq K_1$.
- Fig. 5 Stress vs strain on a plug of normal cartilage specimen, no. NS-60-N, during confine compression. Equilibrium stress is linearly related to equilibrium strain. On sixteen such samples, the mean value of this proportionality factor, E_∞ , is $0.873 \pm 0.353 \text{ MN/m}^2$

FIGURE 1

FIGURE 2

FIGURE 3

FIGURE 4

FIGURE 5