An Anatomical Model of Stress Induced Fluid Flow in Osteons

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Abstract: The objective of the present study is to present a new model for intraosteonal stress induced fluid flow, based on the principle that fluid exchange occurs between the lacunar-canalicular system and the matrix microporosity as well as the lacunar-canalicular system and the Haversian Canal. The model represents an extension of the earliest models of Pollack et al., (1984) and Kufahl and Saha (1990) and is an alternative to the Weinbaum et al., (1994) model.

INTRODUCTION

An osteonal model consisting of canaliculi and lacunae bounded in series, as on Figure 1, was used by of Piekarski and Munro [1] to illustrate stress induced fluid flow in osteons. Pollack et al., [2] developed an anatomical model for a system of independent lacunae each connected to a Haversian canal with a set of canaliculi. The fluid balance for each lacuna in the case of step loading is represented by the initial problem:

\begin{equation}
\frac{dP}{dt} = -P \quad t \in (0, t_0] , \quad P(0) = -\frac{K_w}{3K_1} \frac{4\mu_1 + 3K_1}{4\mu_1 + 3K_w} S , \quad \tilde{\tau} = \frac{4\eta L V_0}{n \pi R^4} \frac{K_w - K_{\text{eff}}}{K_w K_{\text{eff}}} ,
\end{equation}

where $P, L, R, V_0, n, \eta, K_w, K_{\text{eff}}, K_1, \mu_1, S$ are lacunar pressure, canalicular length (lacuna-lacuna distance), canalicular radius, lacunar volume, number of canaliculi connecting the lacuna and Haversian Canal (or two lacunae in the next model), fluid viscosity, bulk fluid modulus, effective lacunar bulk modulus, bulk bone matrix modulus, shear stress modulus of bone matrix and uniaxial stress respectively. Excellent agreement was found with the experimental data measured by Pienkowski [3] for samples composed of many osteons. The model was extended later by Kufahl and Saha, [4] for a series of lacunae and canaliculi as in the Piekarski and Munro schematic osteonal model (Figure 1).
Figure 1: Osteonal model of Piekarski and Munro

The governing equation in Kufahl and Saha theory of the case of step loading is:

\[
\frac{d}{dt}\{P\} + \frac{1}{\tau}\{S\}\{P\} = 0 , \quad t \in (0, t_0] ,
\]

where \( \{P\} \) is a vector composed of lacunar fluid pressures and \([S]\) is the quadratic matrix

\[
[S] = \begin{pmatrix}
2 & -1 & 0 & 0 & \ldots & 0 & 0 & 0 & 0 \\
-1 & 2 & -1 & 0 & \ldots & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \ldots & 0 & -1 & 2 & -1 \\
0 & 0 & 0 & 0 & \ldots & 0 & 0 & -1 & 1 \\
\end{pmatrix}
\]

with dimension equal to the number of the lacunae in series. A problem however appears with the lacunar effective bulk modulus, a model constant, which in the Kufahl and Saha theory is considered as an experimentally determined parameter. In fact, however, there is no experimental method currently available for its evaluation. For the particular case of step loading its value was theoretically evaluated by Pollack et al. [2] for a single lacuna connected with a Haversian canal. Recently Petrov [5] proved, that the same value, namely

\[
K_{eff} = \frac{4\mu_1 K_w}{8\mu_i + 3K_w}
\]

applies for an arbitrary network composed of lacunae and canaliculi.

A model of stress induced fluid flow based on a hypothetical matrix structure within the canaliculus was proposed by Weinbaum et al. [6] and Zeng et al. [7]. The presence of the fiber network, occupying the inner one half of the radius of the canaliculus with the osteocytic process results in sufficient increased drag to enable this model to predict the experimentally observed order of the relaxation time constant of 1.0 sec. There are, however some principle problems connected with the Weinbaum et al. model. In the paper of Cowin et al., [8] data from Scott and Korostoff [9], Otter et al., [10] and Pienkowski and Pollack [3], [11] were used, and it is remarkable that streaming potential data from each of these experiments is fit by a decay constant of about one second independently of the fact that specimens have been subjected to completely different tissue preparation. Otter used fairly fresh specimens, Scott and Korostoff fixed them in formalin, and Pollack and Pienkowski kept the specimens for a considerable time in an unfixed state such that it would be expected that the proposed osteocyte’s glycocalyx structure would be degraded. Because of the fact that, in Weinbaum et al. theory, the detailed organization of the fiber network plays a principle role in determining the drag increase, it is a serious problem that the characteristic decay constants are the same irrespective of the method of preparation of the specimens. It is possible that the drag increase is caused by some more stable structure of the canalicular-lacunar system. The other aspect of the Weinbaum model with which this work takes issue is the assumption that the total water within the microporosity compartment is in a frozen state and is not free to flow. They base their argument on Neuman and Neuman’s work [12] in which they centrifuged a single synthetic specimen of L-Apatite and found that enough water was retained to account for a 100 Angstrom film that accounted for all the bound water. This work, however, was not done on bone. In addition, there is the implicit assumption in Neuman’s interpretation that in the bone microporosity there are no dead ended pores. The existence of dead ended pores would change tremendously the interpretation of the Neuman
experiment. From Newman’s model of the porous space in L-apatite and from gas adsorption studies, Weinbaum et al. concluded that the water in bone was bound water with thickness of 100 Angstroms. This value is however in absolute conflict with the current conception that for a wide set of materials the shear layer (or slip plane) thickness at the water-solid interface is about 10 Angstroms. An other problem of Weinbaum et al. model is the accepted 6 to 7 nM spacing of their glycocalyx. It would "filter" the flow in tracer studies and prohibit the movement of ferritin into the lacunar-canalicular spaces, which will cause serious disagreement with extensive experimental results by many authors (Dillaman, [13]).

NEW MATHEMATICAL MODEL

To design an appropriate new osteonal model we extend the Kufahl and Saha [4] model by accepting fluid flow within the microporosity and explore the Finite Volume (FV) Method of formulating the problem. The fluid balance within the \( N^{th} \) computational domain is presented schematically on Figure 2. In addition to accepting fluid flow within the microporosity compartment, we introduce an effective hydraulic canalicular radius, instead of an anatomical radius to take into account that the open portion of the canalicular cross section is neither uniform nor fully open for fluid flow. Also, for the case of step loading we use the value of the effective lacunar bulk modulus estimated by Petrov[5]. The master equations of the new osteonal model are:

\[
\frac{d}{dt} \{P\} + \frac{1}{\tau} [S]\{P\} = \frac{\bar{V}_0}{V_0^{cl}} \left( \{\bar{P}\} - \{P\} \right) \\
\frac{d}{dt} \{\bar{P}\} = -\frac{\{\bar{P}\} - \{P\}}{\tau}, \quad t \in (0, t_0]
\]

where \( \{\bar{P}\} \) and \( \{P\} \) are vectors composed of the domain's (Fig. 2) microporosity and lacunar pressures \( \bar{P}^k \) and \( P^k \) respectively. The values \( V_0^{cl}, \bar{V}_0, R_{eff}^{exch}, C^{exch} \) are lacunar-canalicular porosity and microporosity volume fractions, effective canalicular radius and the permeability of the canalicular fluid - matrix interface.

The new osteonal model of stress generated fluid flow is able very precisely to fit the experimental data of Pienkowski [3] and Otter et al. [9]. This is demonstrated on Figure 3 for Otter's experimental data. As one can see, the Kufahl and Saha limit of our theory, namely for

![Figure 2: FV computational domain (\( J_{exch} \)-exchange flux between the microporosity and

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canaliculi; \( J_{cl} \) - lacunar-canalicular input-output fluxes; \( J_m \) - microporosity input-output fluxes (accepted as neglectful)
the case of no flow in the microporous matrix and no exchange flow, is capable of fitting experimental curves only for small times. In Table 1 are presented the fitting parameters determined by minimization of the "quadratic difference" between theoretical and the experimental curves.

**Table 1:** Model parameters

<table>
<thead>
<tr>
<th>Data from</th>
<th>Model</th>
<th>( \tau ) (sec)</th>
<th>( \bar{\tau} ) (sec)</th>
<th>( \bar{V}_o / V_0^{cl} )</th>
<th>( R_{eff} ) (nm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pienkowski, (1982)</td>
<td>new model</td>
<td>0.02</td>
<td>1.1</td>
<td>1.2</td>
<td>14.0</td>
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<tr>
<td>sample BT3-90-7</td>
<td>Kufahl and Saha limit</td>
<td>0.03</td>
<td>( \infty )</td>
<td>0.0</td>
<td>13.0</td>
</tr>
<tr>
<td>Pienkowski, (1982)</td>
<td>new model</td>
<td>0.03</td>
<td>2.2</td>
<td>1.1</td>
<td>14.0</td>
</tr>
<tr>
<td>Sample BT3-90-8</td>
<td>Kufahl and Saha limit</td>
<td>0.04</td>
<td>( \infty )</td>
<td>0.0</td>
<td>12.0</td>
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<tr>
<td>Otter et al., (1992)</td>
<td>new model</td>
<td>0.05</td>
<td>2.1</td>
<td>1.6</td>
<td>11.0</td>
</tr>
<tr>
<td>in vitro</td>
<td>Kufahl and Saha limit</td>
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<td>( \infty )</td>
<td>0.0</td>
<td>10.0</td>
</tr>
<tr>
<td>Otter et al., (1992)</td>
<td>new model</td>
<td>0.04</td>
<td>2.1</td>
<td>0.6</td>
<td>12.0</td>
</tr>
<tr>
<td>In vivo</td>
<td>Kufahl and Saha limit</td>
<td>0.05</td>
<td>( \infty )</td>
<td>0.0</td>
<td>11.0</td>
</tr>
</tbody>
</table>

**RELAXATION TIMES PROBLEM**

By eliminating \( \{\vec{P}\} \) in equations ( 18 ) we obtain for lacunar-canalicular pressure:
\[ \frac{d^2}{dt^2} \{P\} + \left( \frac{1}{\hat{t}} [S] + \frac{\left( 1 + \frac{\tilde{V}_0}{V_{0c}} \right) [I]}{\hat{t}} \right) \frac{d}{dt} \{P\} + \frac{1}{\hat{t} \hat{\tau}} [S] \{P\} = 0 \] 

where \([I]\) is the unit matrix.

By substituting \( \exp(-\frac{t}{\tau_{(m)}}) \{g^{(m)}\} \) in equation (6) for \( \{P\} \), where \( \{g^{(m)}\} \) is the \( m \)-th eigenvector of the matrix \([S]\), we obtain the characteristic equation for the relaxation time spectrum, namely:

\[ \left( \frac{1}{\tau_{(m)}} \right)^2 - \left( \frac{\lambda^{(m)}}{\hat{t}} + \frac{1 + \tilde{V}_0}{V_{0c}} \right) \frac{1}{\tau_{(m)}} + \frac{\lambda^{(m)}}{\hat{\tau} \hat{\tau}} = 0 \quad m = 1, 2, \ldots, M \]

where

\[ \lambda^{(m)} = 4\sin^2 \left( \frac{2m-1}{2} \right) \frac{\pi}{M+1} \]

is the \( m \)-th eigenvalue of the matrix \([S]\).

Solving equation (7) for \( \tau_{1,2}^{(m)} \):

\[ \frac{1}{\tau_{1,2}^{(m)}} = \frac{1}{2} \left( \frac{\lambda^{(m)}}{\hat{t}} + \frac{1 + \tilde{V}_0}{V_{0c}} \right) \pm \sqrt{\frac{1}{4} \left( \frac{\lambda^{(m)}}{\hat{t}} + \frac{1 + \tilde{V}_0}{V_{0c}} \right)^2 - \frac{\lambda^{(m)}}{\hat{\tau} \hat{\tau}}} \]

where the subscript 1,2 refers to the + and – sign in equation (9) respectively. The relaxation time spectrum for the Kufahl and Saha limit follows from equation (9) inserting in it \( \frac{\tilde{V}_0}{V_{0c}} = 0 \) and \( \hat{\tau} \to \infty \). It is seen from equation (9), that our anatomical model of 5 lacunae connected in a chain \((M=5)\) is described with ten relaxation times. The relaxation process of drainage of the fluid from the cement line to the Haversian Canal corresponds to the first eigenvalue, \( \lambda^{(1)} \), and creates the only two experimentally visible relaxation times in the above mentioned studies of Otter, Pollack and Korostoff.

Table 2: Comparison between the theoretical and experimental relaxation times; slow and fast experimental relaxation times \( \tau^s, \tau^f \); (in sec)

<table>
<thead>
<tr>
<th></th>
<th>( \tau_1^s, \tau_1^f )</th>
<th>( \tau_2^s, \tau_2^f )</th>
<th>( \tau^s, \tau^f )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pienkowski, (1982), sample BT3-90-7</td>
<td>1.46, 0.19</td>
<td>1.4, 0.20</td>
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<tr>
<td>Pienkowski, (1982), sample BT3-90-8</td>
<td>2.67, 0.30</td>
<td>3.3, 0.35</td>
<td></td>
</tr>
<tr>
<td>Otter, (1992) in vitro</td>
<td>3.30, 0.40</td>
<td>3.3, 0.60</td>
<td></td>
</tr>
</tbody>
</table>
Otter, (1992) in vivo

\[ \tau_1 = 2.47 \quad \tau_2 = 0.42 \quad \tau' = 2.5 \quad \tau'' = 0.50 \]

Inserting in (9) the values of parameters identified by computational analysis (Table 1) we obtain the relaxation time spectrum. The respective values are presented in Table 2 along with the relaxation times computed directly from experimental curves.

CONCLUSIONS

First, it was demonstrated for relaxation type experiments that the model is capable of an excellent fit to the multi-time-constant experimental data. Second, the model avoids the use of the arbitrary assumptions of impermeability of the canalicular and lacunar wall and the existence of a structured glycocalyx. As an alternative, the present report introduces an effective hydraulic canalicular radius whose size we have estimated to be of order of 10 nm. Third, this model provides the opportunity to calculate fluid flow and transport using an anatomical configuration that could lead to a better understanding of complex tracer studies. Finally, a model did not exist up to now that simultaneously and effectively accounts for fluid flow, both in live bone and dead bone, and therefore permits a new approach to rigorously studying bone adaptation phenomena related to fluid flow.

REFERENCES