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A PERTURBATION THEORY APPROACH TO STUDY VARIABLE PERMEABILITY EFFECTS IN THE 1D CONSOLIDATION THEORY OF HYDROCEPHALUS

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Abstract. Hydrocephalus is a clinical condition that has afflicted human beings since time immemorial. Over the past two or three decades the interaction of mathematics and medicine has contributed to significant advances in the treatment of hydrocephalus. Recently a number of papers have applied poroelasticity theory to a simplified model of the brain. These have generally assumed that the permeability of the brain parenchyma is constant; however it has long been known that for other biological tissues (e.g. articular cartilage) that permeability decreases exponentially with compression. Thus a question of interest is how great an impact such variations might have on the pathogenesis and evolution of hydrocephalus. The assumption of variable permeability renders the governing equations nonlinear, and so we further simplify the cylindrical geometry to one dimension. Analytical approximations are obtained in this context for displacements, pressure and filtration velocity.

1 Introduction

Over the last three decades, considerable research effort has been directed towards the study of the mechanical properties of the human brain, and of its response to the hydrodynamic loading of the ventricular walls. This line of research has been driven (in large part) by the desire of clinicians and scientists to increase our understanding of the onset of hydrocephalus, as well as the consequences and repercussions of traumatic brain injury. In this paper, we will focus on the mathematical modelling of hydrocephalus, which is a condition that occurs as a congenital problem in a significant portion of live births. The condition arises as a result of the breakdown of the normal cycle of formation and drainage of cerebrospinal fluid (CSF), so that there is a build up of CSF in the central, lateral ventricles, resulting in increased pressure that compresses the brain tissue against the skull. In pediatric

cases, where the skull is not rigid this can frequently result in significant deformation of the skull itself, in addition to brain tissue compression.

To be aware of the difficulty of the problem and the limitations of the results, a more detailed knowledge of the condition itself is required. When the cranio-spinal system functions normally, CSF is formed primarily in the lateral ventricles and passes through channels into the third and fourth ventricles and then into the subarachnoid space surrounding the brain and spinal chord (see [2]). Although its specific functions are not well known, it is believed to play a multipurpose role in brain health and function, acting both as a shock absorber to cushion the brain, as well as to provide nutrients and remove waste from the cranio-spinal system. After circulation, the CSF reaches certain absorption sites and the system functions perfectly, provided the production rate is balanced by the absorption rate. Hydrocephalus occurs when some part of the circulatory system is obstructed, preventing the absorption of the fluid (or resulting in a reduced absorption rate). Obstruction most commonly arises as a result of congenital malformation, but may also result from an injury, a hemorrhage, or a tumor. Hydrocephalus occurs in about 1.5% of live births, often in conjunction with meningomydocele, the most severe form of spino bifida. Except in those rare cases in which the obstruction can be removed surgically, hydrocephalus is incurable and treatment is problematic. Tremendous advances have been made over the past five decades, and today the most common treatment is by way of a ventricular-peritoneal shunt. CSF shunts have been so successful in the treatment of hydrocephalus that currently there are over 500 types of shunts on the market. However, there are problems with their use, and a prospective multi-centre, randomized clinical trial led by the Hospital for Sick Children, at the University of Toronto, has shown that the shunt failure rate at two years is 50%, independent of the shunt type (cf [3]). It thus seems clear, that further progress will depend crucially on an improved understanding of the onset and development of hydrocephalus, which in turn must rely on progress in our understanding of the mechanics of the brain and the dynamics of CSF.

In contrast to ordinary materials of engineering interest, the brain is so fragile that standard engineering tests to determine mechanical properties cannot be used, which hinders the possibility of developing sound phenomenological models. A good illustration of the quandry we are faced with is offered by the numerous models of CSF hydrodynamics published over the past five decades (see [4], [5]). Although they have resulted in some criteria and measures (such as the pressure-volume index) useful to neurosurgeons, they all have the fundamental weakness of being unable to describe the distribution of stress and strain within the brain tissue or the configuration of the ventricular walls in a hydrocephalic brain, and it is information of this nature that is essential to make true progress in this field. Clearly, progress in hydrocephalus research will require much more realistic models. This means a greater number of factors must be taken into consideration.

In the simplest possible description capable of predicting stress and strain

distributions within the brain tissue, the human brain has been likened by Hakim (cf. [6]) to a sponge formed of an incompressible, elastic solid whose pores are filled with a viscous incompressible fluid (CSF). Adding to the challenge of developing an appropriate model is the difficult geometry of the brain, especially if we wish to accurately take into account the circulation of the CSF around the spinal chord. Nagashima *et al*[7] were the first to draw a parallel between Hakim's proposed representation of the brain and the problem of soil consolidation, the study of which was pioneered (decades earlier) by Biot [1]. One version or another of Biot's consolidation theory has been the foundation for a number of subsequent works. The numerical work of Nagashima *et al* [7] attempted to tackle the problem using a realistic geometry but used questionable values of the brain's physical parameters and the appropriate boundary conditions were not applied. Subsequently Tenti *et al* [9] and Stastna *et al* [8] attempted to address these problems analytically and in a simplified geometry. These latter works provide the direct motivation and basis for this paper. Since Biot's consolidation theory is well known, we forgo a detailed presentation of it here, and refer the reader to [10] instead.

2 Mathematical Model

Following [9], we introduce the governing equations for the system we will examine. Inertia forces associated with seepage are neglected since the typical Reynolds number based on pore size is much less than one. Body forces are also neglected so that in Cartesian coordinates, and using the Einstein summation convention, the equation of motion reduces to,

$$\frac{\partial T_{ij}}{\partial x_j} = 0 \text{ for } i, j = 1, 2, 3. \tag{2.1}$$

Here T_{ij} is the total stress tensor:

$$T_{ij} = -p\delta_{ij} + \tau_{ij} \tag{2.2}$$

where p represents the pore fluid pressure, and τ_{ij} is Terzaghi's effective stress, which in the linear approximation (for small strain and for incompressible constituents) is related to the strain by:

$$\tau_{ij} = \lambda e_{kk}\delta_{ij} + 2\mu e_{ij}. \tag{2.3}$$

The strain tensor is, in turn, given by

$$e_{ij} = \frac{1}{2}\left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i}\right) \tag{2.4}$$

where the u_i are the components of the displacement vector. Although the equations appear to be similar in form to the constitutive equations of classical linear elasticity theory, a caveat to be kept in mind is that the Lamé

like parameters λ and μ refer not to the properties of the solid matrix alone, but to the properties of the homogenized (hypothetically averaged) mixture.

Defining the volume flux vector (following Kenyon, [10]) as

$$V_i = \phi v_i^{(f)} + (1-\phi) v_i^{(s)} = W_i + v_i^{(s)} \tag{2.5}$$

where $v_i^{(f)}$ and $v_i^{(s)}$ are averaged components of fluid and solid velocities, respectively, ϕ is the pore fraction (pore volume divided by bulk volume) and W_i is the filtration velocity (flow relative to the solid). The continuity equation is then simply

$$\frac{\partial V_i}{\partial x_i} = 0 \tag{2.6}$$

where we have assumed that both solid and liquid constituents are each intrinsically incompressible. Note that the velocity of the solid is defined to be the rate of change of the solid displacement vector with respect to time, or

$$v_i^{(s)} = \frac{\partial u_i}{\partial t}. \tag{2.7}$$

The system of equations is closed by assuming the validity of Darcy's Law,

$$W_i = -\frac{k}{\eta}\frac{\partial P}{\partial x_i} \tag{2.8}$$

where k is the permeability of the medium and η is the fluid's coefficient of viscosity.

Analyses of the case k =constant were presented in Tenti *et al* [9] and Stastna *et al* [8] for both steady state and transient cases, respectively. The boundary conditions in both of these papers were taken to be

$$\begin{aligned} p(R) &= P_i \\ p(R+\Delta R) &= P_0 \end{aligned} \tag{2.9}$$

where R and $R+\Delta R$ represent the inner and outer radii of a porous cylinder, respectively, and P_i and P_o are prescribed. The pressure gradient drives flow through the porous, annular region. Stress equilibrium was also assumed to hold on the boundaries of the annular region, so that

$$\tau_{rr}(R) = \tau_{rr}(R+\Delta R) = 0. \tag{2.10}$$

The analyses of references [8] and [9] met with some success but raised questions as to the appropriateness of the assumption that the permeability k is constant. An increase in intraventricular pressure would compress the brain parenchyma and cause large, local strains initially. It seems reasonable to consider the possibility that such strains would affect the permeability of the bulk solid, in the case of the brain (although unfortunately there is no experimental evidence to corroborate this). Nevertheless this kind of behaviour has

been observed experimentally for human articular cartilage, where Holmes, Lai and Mow [11] have shown (in uniaxial compressive stress relaxation experiments) that permeability decreases exponentially with the magnitude of compression, specifically,

$$k = k_0 \exp(MI^*) \tag{2.11}$$

where k_0 is the apparent (strain–free) permeability of the medium, I^* is the first invariant of the infinitesimal strain tensor and M is a constant.

The assumption of a nonlinear dependence of the permeability of biological tissue upon strain renders the equations of consolidation theory nonlinear, with attendant mathematical complications.

3 One dimensional model of hydrocephalus

We begin by setting our equations in a one dimensional context (analogous to the three dimensional system we would ideally like to describe). In [12], Parker *et al* performed an experiment which models the kind of filtration behaviour we are interested in. Their experiment consisted of a highly flexible open celled polyurethane foam placed in a perspex cylinder and saturated with a mixture of glycerine and water (designed to produce large drag in the foam without development of turbulence). The foam was confined at the lower end by a filter composed of a perforated plate and a wire mesh, while the upper end was left free to move under the influence of fluid pressure. The rest of the apparatus was designed so that the pressure drop across the foam could be controlled, and the resulting compression of the foam was then observed. The physics should be closely analogous to what we believe happens in the brain in fully developed hydrocephalus. Hence, the steady-state results of such an experiment should be of interest to us, and we test our mathematical model to ascertain how well the results agree with Parker *et al*'s observations.

For ease of reference we repeat here the consolidation theory equations in one dimensional form. The co-ordinate system is defined such that $x = 0$ corresponds to the position of the lower end of the foam. The initial position of the top surface of the foam is taken to be $x = L$, while its position with advancing time is given by $x = h(t)$, see Figure 1. The equation of motion is

$$\frac{\partial p}{\partial x} = \alpha \frac{\partial^2 u}{\partial x^2} \tag{3.1}$$

where $\alpha = \lambda + 2\mu$ and the Lamé like parameters λ and μ have their usual (consolidation theory) meanings. This quantity is representative of the "stiffness" of the solid matrix (somewhat analogous to the spring constant).

The continuity equation, assuming incompressibility of the individual constituents, is given by

$$\frac{\partial}{\partial x}\left(w + \frac{\partial u}{\partial t}\right) = 0 \tag{3.2}$$

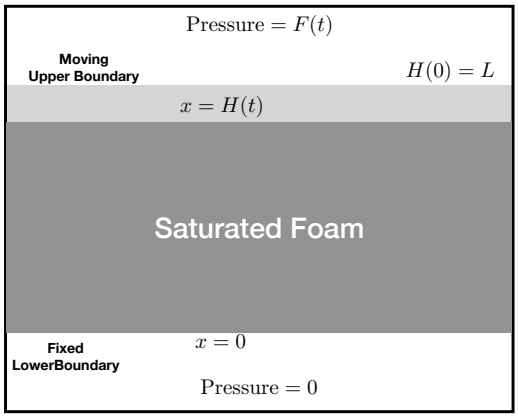

Figure 1: Foam Compression Test Experiment.

and Darcy's law

$$w = -\frac{k(x,t)}{\eta}\frac{\partial p}{\partial x} \tag{3.3}$$

where $k(x,t)$ represents the permeability of the medium, η the viscosity of the fluid and w, u and p represent filtration velocity, displacement and pore pressure, respectively. Equations (3.1), (3.2) and (3.3) represent the set of governing equations.

The initial condition is given by

$$u(x,0) = 0 \qquad 0 \leq x \leq L \tag{3.4}$$

since the filter is fixed we have

$$u(0,t) = 0 \text{ for all } t \geq 0. \tag{3.5}$$

Since there is no contact stress at the upper boundary of the foam, a second boundary condition is given by

$$\frac{\partial u}{\partial x}(h(t),t) = 0 \text{ for all } t \geq 0 \tag{3.6}$$

The controlled regulation of the pressure drop across the foam is given, without loss of generality by,

$$p(x,0) = 0 \tag{3.7}$$

$$p(0,t) = 0 \tag{3.8}$$

$$p(h(t),t) = F(t) \text{ for all } t \geq 0. \tag{3.9}$$

In the experiment of Parker *et al* only steady state data were recorded, thus it was not necessary to specify the forcing function $F(t)$. To investigate transient effects theoretically, before equilibrium is reached, we choose

$$F(t) = p_0 \left[1 - \exp(-\gamma t)\right] \tag{3.10}$$

for some limiting pressure p_0 and some value γ. Note that the function $h(t)$ is related to the governing equations by the requirement of conservation of volume, i.e.

$$u(h(t), t) = h(t) - L. \tag{3.11}$$

3.1 Steady State

We follow the analysis of Barry & Aldis [13] as preparation for the more complicated problem to be considered subsequently. The equilibrium equations are given by

$$\frac{d^2u}{dx^2} = \frac{1}{\alpha}\frac{dp}{dx} \tag{3.12}$$

$$\frac{dw}{dx} = 0 \tag{3.13}$$

$$w = -\frac{k(x)}{\eta}\frac{dp}{dx} \tag{3.14}$$

From (3.13) it follows that $w(x) = c_1$ for some constant c_1, and so by equation (3.14)

$$c_1 = -\frac{k(x)}{\eta}\frac{dp}{dx}. \tag{3.15}$$

The initial boundary conditions reduce to

$$u(0) = 0 \tag{3.16}$$

$$p(0) = 0 \tag{3.17}$$

$$\frac{du}{dx}(h) = 0 \tag{3.18}$$

$$p(h) = p_0 \tag{3.19}$$

where

$$h = \lim_{t\to\infty} h(t)$$

and finally the conservation of volume condition becomes

$$u(h) = h - L.$$

Substituting from (3.14) into (3.12) we obtain

$$\frac{d^2u}{dx^2} = -\frac{\eta c_1}{\alpha k(x)}. \tag{3.20}$$

We let

$$k(x) = k_0 g(x)$$

where k_0 is the intrinsic permeability of the medium, corresponding to a state with zero deformation.

For the case of constant permeabilty (i.e. $g(x) = 1$), it is straightforward to show that the full solution reads

$$u(x) = \frac{p_0}{2\alpha h}x^2 - \frac{p_0}{\alpha}x = \frac{p_0}{\alpha}\left(\frac{x^2}{2h} - x\right) \tag{3.21}$$

$$p(x) = \frac{p_0}{h}x \tag{3.22}$$

$$h = \frac{L}{1 + \frac{p_0}{2\alpha}}. \tag{3.23}$$

It is also easy to check and confirm through nondimensionalization that our results coincide with those of Barry & Aldis [13] . We now introduce variable permeability and see how well our theory matches the experimental results of Parker *et al.*

3.2 Variable Permeability

We assume now that permeability varies exponentially with compression

$$g(x) = \exp\left(M\frac{\partial u}{\partial x}\right)$$

i.e.

$$k(x) = k_0 \exp\left(M\frac{\partial u}{\partial x}\right).$$

With this form of the permeability, equation (3.20) becomes

$$\frac{d^2u}{dx^2} = -\frac{\eta c_1}{k_0\alpha}\exp\left(-M\frac{du}{dx}\right). \tag{3.24}$$

Viewing this as a first order, separable DE for $\frac{du}{dx}$ we can solve the governing equations to obtain, after nondimensionalization:

$$u(x) = \left(\frac{x}{M} - \frac{h}{M[1-\exp(Mp_0)]}\right)\ln\left[\exp(-Mp_0) - \frac{(\exp(-Mp_0)-1)}{h}x\right] - \frac{x}{M} - \frac{p_0 h}{1-\exp(Mp_0)} \tag{3.25}$$

$$p(x) = \frac{1}{M}\ln\left[\exp(-Mp_0) - \frac{(\exp(-Mp_0)-1)}{h}x\right] + p_0 \tag{3.26}$$

$$h = \left[1 + \frac{1}{M} + \frac{p_0}{1-\exp(Mp_0)}\right]^{-1} \tag{3.27}$$

and in this case

$$w = \frac{1}{Mh}\left[1 - \exp(-Mp_0)\right] \tag{3.28}$$

Substituting from (3.28) into (3.25), the displacement can be written in terms of the filtration velocity w instead of the pressure as

$$u(x) = \left(\frac{x}{M} - \frac{1}{M^2 w} - \frac{h}{M}\right)\ln\left[1 + Mw(x-h)\right] - \frac{x}{M} + \frac{Mhw - 1}{M^2 w}\ln\left[1 - Mhw\right] \tag{3.29}$$

which is in agreement with the results of Barry & Aldis [13].

3.3 Transient Case

Although the work in the previous sections are of interest to us in the study of hydrocephalus, in the initial stages of increased fluid pressure there is reason to believe that the brain tissue would behave differently than the foam experiment of Parker *et al.*

In the steady state analysis, we assumed both boundaries of the solid matrix to be permeable. In the initial stages we assume that the ependyma is essentially impermeable to CSF. As pressure builds up in the lateral ventricles the resulting compliance of the surrounding brain tissue is primarily attributable to the displacement of blood flowing out of the brain through the vascular system, while the interior acts as a movable wall (we consider this loss of blood to be represented by filtration through the outer boundary).

Assuming no flow at the outer boundary implies that $w(h,t) = 0$ and, using Darcy's law, this can be written as

$$\frac{\partial p}{\partial t}(h,t) = 0. \tag{3.30}$$

Furthermore treating the top surface as an impenetrable membrane the applied load must be shared by both interstitial pressure and the contact stress. The total stress (in consolidation theory), in one dimension, is given by:

$$T = -p + (\lambda + 2\mu)\frac{\partial u}{\partial x}. \tag{3.31}$$

Thus the second boundary condition at $x = h(t)$ is given by

$$-F(t) = -p(h,t) + \alpha\frac{\partial u}{\partial x}(h,t) \tag{3.32}$$

where

$$F(t) = p_0[1 - \exp(-\gamma t)]$$

and

$$\alpha = \lambda + 2\mu.$$

We now try to solve equations (3.1), (3.2), (3.3) subject to conditions (3.4), (3.5), (3.6), (3.7), (3.8), (3.11), (3.30) and (3.32). Integrating (3.1) we obtain

$$\alpha \frac{\partial u}{\partial x}(x,t) - p(x,t) = c_1(t) \tag{3.33}$$

and applying the boundary condition gives

$$c_1(t) = -F(t). \tag{3.34}$$

Notice that equation (3.2) implies

$$w + \frac{\partial u}{\partial t} = c_2(t) \tag{3.35}$$

and using this to eliminate w in (3.35) we obtain

$$-\frac{k(x,t)}{\eta}\frac{\partial p}{\partial x} + \frac{\partial u}{\partial t} = c_2(t). \tag{3.36}$$

Differentiating this with respect to x yields

$$-\frac{1}{\eta}\frac{\partial}{\partial x}\left(k(x,t)\frac{\partial p}{\partial x}\right) + \frac{\partial}{\partial t}\left(\frac{\partial u}{\partial x}\right) = 0 \tag{3.37}$$

and substituting for the permeability, this reduces to

$$\frac{\partial}{\partial t}\left(p - F(t)\right) = \kappa_0^0 \frac{\partial}{\partial x}\left(\exp\left[\frac{M}{\alpha}(p - F(t))\right]\frac{\partial p}{\partial x}\right) \tag{3.38}$$

where

$$\kappa_0^0 = \frac{k_0 \alpha}{\eta}.$$

We attempt to solve this using perturbation theory. Define

$$f(t) = 1 - \exp(-\gamma t)$$

then we can write

$$F(t) = p_0 f(t)$$

and nondimensionalize as

$$p(x,t) \to \frac{p(x,t)}{p_0}$$

$$x \to \frac{x}{L}$$

$$\tau \to \frac{\kappa_0^0 t}{L^2}$$

It follows that (3.38) can now be written as

$$\frac{\partial}{\partial \tau}\left(p - f(\tau)\right) = \frac{\partial}{\partial x}\left[\exp\left(\frac{Mp_0}{\alpha}(p - f(\tau))\right)\frac{\partial p}{\partial x}\right]. \tag{3.39}$$

Let

$$v(x,\tau) = p(x,\tau) - f(\tau),$$

then

$$\frac{\partial v}{\partial \tau} = \frac{\partial}{\partial x}\left[\exp\left(\frac{Mp_0 v}{\alpha}\right)\frac{\partial v}{\partial x}\right] \tag{3.40}$$

with boundary conditions

$$p(x,0) = 0 \qquad \Rightarrow \qquad v(x,0) = 0 \tag{3.41}$$

$$p(0,\tau) = 0 \qquad \Rightarrow \qquad v(0,\tau) = -f(\tau) \tag{3.42}$$

$$\frac{\partial p}{\partial x}(h,\tau) = 0 \qquad \Rightarrow \qquad \frac{\partial v}{\partial x}(h,\tau) = 0 \tag{3.43}$$

$$h(\tau) = 1 + u(h,\tau) \tag{3.44}$$

where

$$u \to \frac{u}{L}.$$

Carrying out a short time analysis ($\tau \to 0$), we look for a perturbation solution in terms of

$$\delta = \frac{P_0}{\alpha} \ll 1.$$

The problem we seek to solve can then be written as

$$\frac{\partial v}{\partial \tau} = \frac{\partial}{\partial x}\left(\exp(M\delta v)\frac{\partial v}{\partial x}\right) \tag{3.45}$$

subject to

$$v(x,0) = 0 \tag{3.46}$$

$$v(0,\tau) = \exp(-\sigma\tau) - 1, \text{ where } \sigma = \frac{\gamma L^2}{\kappa_c^0} \tag{3.47}$$

$$\frac{\partial v}{\partial x}(1,\tau) = 0. \tag{3.48}$$

Substituting the perturbation expansion

$$v(x,\tau) = v_0(x,\tau) + \delta v_1(x,\tau) + \delta^2 v_2(x,\tau) + ...$$

in equation (3.45), we obtain

$$\frac{\partial v_0}{\partial \tau} + \delta\frac{\partial v_1}{\partial \tau} = \frac{\partial}{\partial x}\left[\frac{\partial v_0}{\partial x} + \delta\left(\frac{\partial v_1}{\partial x} + Mv_0\frac{\partial v_0}{\partial x}\right)\right] + \mathcal{O}\left(\delta^2\right). \tag{3.49}$$

The $\mathcal{O}(1)$ problem is simply the diffusion equation

$$\frac{\partial v_0}{\partial \tau} = \frac{\partial^2 v_0}{\partial x^2} \tag{3.50}$$

subject to

$$\begin{aligned} v_0(x,0) &= 0, \\ v_0(0,\tau) &= \exp(-\sigma\tau) - 1 \text{ where } \sigma = \frac{\gamma L^2}{\kappa_c^0}, \\ \frac{\partial v_0}{\partial x}(1,\tau) &= 0. \end{aligned} \tag{3.51}$$

Using Laplace transforms, it is reasonably straight forward to show that

$$v_0(x,\tau) \sim -(4\sigma\tau) i^2 \text{erfc}\left(\frac{x}{2\sqrt{\tau}}\right) \text{ as } \tau \to 0 \tag{3.52}$$

where erfc is the complementary error function.

The $\mathcal{O}(\delta)$ problem is easily seen to be

$$\begin{aligned} \frac{\partial v_1}{\partial \tau} &= \frac{\partial}{\partial x}\left[M v_0 \frac{\partial v_0}{\partial x}\right] + \frac{\partial^2 v_1}{\partial x^2} \\ &= \frac{\partial^2 v_1}{\partial x^2} + M\left[\left(\frac{\partial v_0}{\partial x}\right)^2 + v_0 \frac{\partial^2 v_0}{\partial x^2}\right] \end{aligned} \tag{3.53}$$

subject to

$$\begin{aligned} v_1(x,0) &= 0, \\ v_1(0,\tau) &= 0, \\ \frac{\partial v_1}{\partial x}(1,\tau) &= 0. \end{aligned} \tag{3.54}$$

Using (3.52), together with a standard property of the iterated complementary error function given by:

$$\frac{d}{dz}(i^n erfc(z)) = -i^{n-1} erfc(z), \text{ for n=0,1,2...} \tag{3.55}$$

we obtain:

$$\frac{\partial v_0(x,\tau)}{\partial x} \sim -(2\sigma\sqrt{\tau}) i \text{ erfc}\left(\frac{x}{2\sqrt{\tau}}\right) \text{ as } \tau \to 0 \tag{3.56}$$

$$\frac{\partial^2 v_0(x,\tau)}{\partial x^2} \sim -\sigma \text{ erfc}\left(\frac{x}{2\sqrt{\tau}}\right) \text{ as } \tau \to 0 \tag{3.57}$$

and substituting into equation (3.53), the PDE for the first order correction reads:

$$\frac{\partial v_1}{\partial \tau} = \frac{\partial^2 v_1}{\partial x^2} + G(x,\tau). \tag{3.58}$$

where

$$G(x,\tau) = 4M\sigma^2\tau\left(i\ erfc\left(\frac{x}{2\sqrt{\tau}}\right)\right)^2 + 2M\sigma^2\tau\left(i^2 erfc\left(\frac{x}{2\sqrt{\tau}}\right)\right)\left(erfc\left(\frac{x}{2\sqrt{\tau}}\right)\right) \tag{3.59}$$

as $\tau \to 0$, we make use of the asymptotic expansion for $i^n erfc(z)$, to obtain:

$$v_0(x,\tau) \sim -\frac{8\sigma\sqrt{\tau^5}}{\sqrt{\pi}x^3}\exp\left(\frac{-x^2}{4\tau}\right)\left(1 - \frac{12\tau}{x^2} + \frac{180\tau^2}{x^4} + ...\right), \tag{3.60}$$

$$\frac{\partial v_0}{\partial x} \sim \frac{4\sigma\sqrt{\tau^3}}{\sqrt{\pi}x^2}\exp\left(\frac{-x^2}{4\tau}\right)\left(1 - \frac{6\tau}{x^2} + \frac{60\tau^2}{x^4} + ...\right) \tag{3.61}$$

$$\frac{\partial^2 v_0}{\partial x^2} \sim \frac{2\sigma\sqrt{\tau^3}}{\sqrt{\pi}x}\exp\left(\frac{-x^2}{4\tau}\right)\left(1 - \frac{4\tau}{x^2} + \frac{12\tau^2}{x^4} + ...\right) \tag{3.62}$$

The $\mathcal{O}(\delta)$ problem is rather difficult to handle, however since to $\mathcal{O}(\tau^2)$ we have $v_1(x,\tau) = 0$ for practical purposes (for any fixed x with $\delta \ll 1$ and $\tau \to 0$) we have

$$\frac{\partial v_1}{\partial \tau} = -(4\sigma\tau)i^2\mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right) + \mathcal{O}\left(\delta\frac{\tau^3}{x^4}\exp\left[-\frac{x^2}{4\tau}\right]\right). \tag{3.63}$$

Since

$$v(x,\tau) = p(x,\tau) - f(\tau)$$

this implies

$$p(x,\tau) = 4\sigma\tau i^2\mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right) + 1 - \exp(-\sigma\tau) + \mathcal{O}\left(\delta\frac{\tau^3}{x^4}\exp\left[-\frac{x^2}{4\tau}\right]\right) \tag{3.64}$$

where

$$\sigma = \frac{\gamma L^2}{\kappa_c^o}.$$

To find the filtration velocity $w(x,\tau)$, we use Darcy's law

$$w(x,\tau) = -\delta\exp\left(M\frac{\partial u}{\partial x}\right)\frac{\partial p}{\partial x} \tag{3.65}$$

where w has been nondimensionalized as

$$w \to w\frac{L}{\kappa_c^o}.$$

Notice that (3.65) suggests that to leading order (even with a positive pressure gradient) there is no filtration, which is nonphysical. This suggests the existence of a boundary layer. Furthermore, since the boundary condition at $x = 1$ requires zero flow, we deduce that the boundary layer must be located near $x = 0$. Stretching the boundary layer, by introducing

$$\xi = \frac{x}{\delta}$$

and rewriting (3.65) in terms of ξ we obtain, to leading order

$$w_0(\xi, \tau) = -\frac{\partial v_0}{\partial \xi} \tag{3.66}$$

while the first order correction (recalling that $v_1(\xi, \tau) = 0$) is

$$w_1(\xi, \tau) = -M v_0 \frac{\partial v_0}{\partial \xi}. \tag{3.67}$$

Thus, in terms of the original spatial variable x, the filtration velocity in the boundary layer is given by

$$w(x, \tau) = -\delta \frac{\partial v_0}{\partial x} - \delta^2 M v_0 \frac{\partial v_0}{\partial x} + \mathcal{O}\left(\delta^3\right) \tag{3.68}$$

or using equations (3.60) and (3.61)

$$w(x, \tau) \sim -2\delta\sigma\sqrt{\tau} i\mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right) - 8\delta^2 M \sigma^2 \tau^{3/2} i \left[\mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right)\right]^2 \tag{3.69}$$

for $\delta \ll 1$ and $\tau \to 0$, it can be shown using a standard asymptotic expansion for

$$i\mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right)$$

that the second term is in fact $\mathcal{O}(\delta^2\tau^4)$, making it even smaller (asymptotically) than the v_1 term (assumed to be zero in (3.67) above). Thus our solution is better expressed as

$$w(x, \tau) \sim -2\delta\sigma\sqrt{\tau} i\mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right) + \mathcal{O}\left(\delta^2 \frac{\tau^3}{x^4} \exp\left[-\frac{x^2}{4\tau}\right]\right). \tag{3.70}$$

Our final task is to determine the displacement. Substituting from (3.63) into

$$\frac{\partial u}{\partial x} = \delta v(x, \tau) \tag{3.71}$$

we obtain

$$\frac{\partial u}{\partial x} = -4\delta\sigma\tau i^2 \mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right) + \mathcal{O}\left(\delta^2 \frac{\tau^3}{x^4} \exp\left[-\frac{x^2}{4\tau}\right]\right) \tag{3.72}$$

subject to

$$u(0,\tau) = 0.$$

Integrating yields

$$u(x,\tau) = \left(8\delta\sigma\tau^{3/2}\right) i^3\mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right) + C + ... \tag{3.73}$$

and since

$$i^3\mathrm{erfc}(0) = \frac{1}{6\sqrt{\pi}}$$

we find

$$C = -\frac{4}{3\sqrt{\pi}}\delta\sigma\tau^{3/2} + \mathcal{O}\left(\delta^2\frac{\tau^3}{x^4}\exp\left[-\frac{x^2}{4\tau}\right]\right).$$

Thus

$$u(x,\tau) = \left(8\delta\sigma\tau^{3/2}\right) i^3\mathrm{erfc}\left(\frac{x}{2\sqrt{\tau}}\right) - \frac{4}{3\sqrt{\pi}} + \mathcal{O}\left(\delta^2\frac{\tau^3}{x^4}\exp\left[-\frac{x^2}{4\tau}\right].\right) \tag{3.74}$$

In conclusion, equations (3.64), (3.70) and (3.74) constitute an asymptotic solution of the governing system of PDEs and boundary conditions (3.40)-(3.43).

4 Conclusion

We have applied Biot's consolidation theory to the human brain described in a simplified manner as a composite of a viscous fluid saturating and filtrating through a solid matrix. While there has been some previous progress in this direction, the established results are based on the assumption that the permeability of the solid matrix remains constant. On physical grounds it seems reasonable to assume that the permeability of the solid matrix should decrease with compression. The aim of this paper has been to investigate the role that these types of variations might play in the dynamics of such biphasic materials. We assume, analogous to work on cartilage, that the permeability varies exponentially with the strain [11], however this renders the resulting governing equations nonlinear with associated difficulties, in any attempt to solve them. Thus we have limited ourselves in this paper to a study of an analogous one dimensional problem using perturbation methods. Along these lines, we have applied the theory to a thought experiment on pressure driven compression of brain tissue using the experiment of Parker *et al* [12] as the basis for the steady state part in our analysis. The boundary conditions were based on the full 3D problem, assuming the ventricular wall to be impermeable initially during compression, but permeable in the steady state.

It is interesting to compare the transient surface stresses for the cartilage experiment [11] to our own results since the $\sqrt{t}$ behaviour of the displacement-driven experiment has been verified experimentally in [11], our finding of t

behaviour for the pressure driven experiment is an indication of a fundamental difference between the two situations. This discrepancy between the asymptotic transient behaviour of the model brain and the model cartilage merits further study. If confirmed experimentally, this would challenge the commonly held assumption that most soft hydrated biological tissues display qualitatively similar responses to mechanical or hydrodynamic loading. At present, however, it is difficult to reach any definitive conclusion due to the lack of appropriate experimental data for the brain.

Another source of difficulty is that the solution of (3.45) entails dealing with a nonlinear parabolic PDE; however given the straight forward boundary conditions (3.46)-(3.48) one might expect to be able to find good approximate solutions using perturbation theory. Instead, even the leading order solution (see (3.52)) and the first order correction (see (3.53)) have to be obtained by means of a further asymptotic expansion. It is clear that regular perturbation fails and singular perturbation theory techniques have to be applied in this context. One could try to obtain a fully numerical solution, but then much of the physical insight obtained through the use of analytical techniques would be lost. Moreover, the fundamental problem of choosing appropriate values of the mechanical parameters of the brain still remains unresolved. A numerical/graphical comparison of the analytic approximations with fully numerical solutions of the boundary value problem would be useful and informative, and will be the subject of future work. While the mathematical challenges are worthy of study in themselves, improvements in technology in the near future may facilitate experiments on the brain to generate appropriate and relevant data, which in turn can be used to inform our mathematical models. Although the mathematical modelling of the brain presents a formidable challenge, there is reason to hope that the continuing synergy between the mathematical and biomedical sciences will lead to dramatic advances in our understanding of brain biomechanics.

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