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A MATHEMATICAL INVESTIGATION OF THE ROLE OF INTRACRANIAL PRESSURE PULSATIONS AND SMALL GRADIENTS IN THE PATHOGENESIS OF HYDROCEPHALUS

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Abstract. Cerebrospinal fluid (CSF) pulsations have been proposed as a possible causative mechanism for the ventricular enlargement that characterizes the neurological condition known as hydrocephalus. This paper summarizes recent work by the authors to anaylze the effect of CSF pulsations on brain tissue to determine if they are mechanically capable of enlarging the cerebral ventricles. First a poroelastic model is presented to analyze the interactions that occur between the fluid and porous solid constituents of brain tissue due to CSF pulsations. A viscoelastic model is then presented to analyze the effects of the fluid pulsations on the solid brain tissue. The combined results indicate that CSF pulsations in a healthy brain are incapable of causing tissue damage and thus the ventricular enlargement observed in hydrocephalus. Therefore they cannot be the primary cause of this condition. Finally, a hyper-viscoelastic model is presented and used to demonstrate that small long-term transmantle pressure gradients may be a possible cause of communicating hydrocephalus in infants.

Key words. Biomechanics, Hydrocephalus, Poroelasticity, Viscoelasticity, Fractional Calculus.

1. Introduction

Hydrocephalus is a condition characterized by the accumulation of cerebrospinal fluid (CSF) in the ventricles of the brain. Elevated intracranial pressure (ICP) and elevated intracranial pressure wave amplitudes are often observed in some manifestations of hydrocephalus. Non-communicating hydrocephalus occurs when a blockage in CSF flow causes an accumulation of fluid in the ventricles and an increase in ICP. In communicating hydrocephalus, where there is no blockage in CSF flow, large pressure gradients are not observed across the brain parenchyma. Thus, the standard explanation of how ventricles enlarge and why fluid accumulates no longer applies.

The relationship between intracranial pressure, intracranial compliance, and hydrocephalus is complex. Experimental evidence indicates that in hydrocephalus patients with a reduced compliance, the amplitude of ICP pulsations increases but then decreases back to normal upon shunt insertion [33, 38]. Other experiments demonstrate the synchrony between arterial and CSF pressure pulsations [20] and the effect these pulsations seemingly have on ventricular enlargement [3, 49].

Measurements of CSF pressure clearly indicate the pulsatile nature of CSF flow [26]. Some theories for the development of hydrocephalus postulate a link between these pulsations and ventricular enlargement [3, 28, 31, 49]. One such theory, proposed by Egnor et al. [11], suggests that the cranial compartment is in a natural state of resonance and that deviation from this state leads to a breakdown of the windkessel effect and a loss of normal cerebral blood and CSF dynamics. Intracranial pressure, however, is pulsatile in healthy brains and the frequency of these pulsations changes with the heart rate, leading one to ask "if CSF pulsations cause hydrocephalus, why don't we all have this condition?"

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One possible mechanical explanation is that large amplitude CSF pulsations cause tissue damage in the periventricular regions of the brain. This hypothesis for the onset of hydrocephalus proceeds as follows. Each influx of arterial blood generates a CSF pressure pulse felt by the ventricle walls. Periodic pressurization of the walls causes the brain tissue to periodically compress and expand and CSF to oscillate in and out of the brain tissue. When these oscillations are large they generate large shear strains which damage the periventricular tissue. Finally, the damaged tissue allows fluid to penetrate further into the brain, propagating tissue damage and leading to ventricular expansion. We call this mechanistic theory the pulsation-damage hypothesis for hydrocephalus.

Mathematical models have been used to study hydrocephalus since the pioneering work of Hakim et al. [21, 22] in the 1970's, in order to further the understanding of how ventricular enlargement occurs and how treatments can be improved. In the literature, there are two main approaches to mathematically model hydrocephalus. The first approach uses time-dependent compartment models such as pressure volume models and analogous electrical circuit models [11, 12, 27, 37]. The second approach uses time- and space-dependent models such as poroelastic [29, 39, 40, 42, 43, 50] or viscoelastic [9, 10, 35, 36, 41, 46, 47] models. Both poroelastic and viscoelastic models are useful when modelling brain tissue and in this paper we will discuss the application of both classes of models to analyze the effect of CSF pulsations on brain tissue with regards to the development of hydrocephalus.

Biological tissues are composed of both fluid and solid phases and, in addition, the brain has 4 interconnected fluid compartments or ventricles through which the CSF circulates around the subarachnoid region of the brain and spinal cord areas. Thus, ventricular CSF pulsations affect the brain via the periodic loading and unloading of the ventricle walls and via the fluid exchange that occurs between the ventricular and interstitial spaces of the tissue. In Section 2 we present a poroelastic model and analyze the interactions that occur between the solid and fluid phases of brain tissue resulting from this fluid exchange. In Section 3 we present a viscoelastic model and analyze the effect of ventricular CSF pulsations on the solid brain. And in Section 4 we present a hyper-viscoelastic model and show that small long-term transmantle pressure gradients are capable of causing ventricular expansion in infants.

2. Brain Tissue as a Fluid-Solid Composite

We begin by presenting a poroelastic model to analyze the effects of periodic fluid exchange between the ventricles and the interstitial space of brain tissue resulting from CSF pulsations. The goal is to determine if the tissue strains and shear stresses produced in this way are sufficient to cause tissue damage and hence hydrocephalus.

2.1. Poroelastic Model Formulation. A simplified view of brain parenchyma is to think of the brain as a porous linearly elastic solid saturated in a viscous incompressible fluid. Such biphasic materials behave according to Biot's theory of consolidation [4]. Following the work of Tenti et al. [42] we assume a simple model geometry for which analytic solutions can be found. Thus, we model the brain as a thick walled cylinder: the interior representing the ventricles, the exterior representing the subarachnoid space (SAS) and skull, and the thick wall representing the brain parenchyma, see Figure 1. This tethered cylindrical geometry allows the assumption of planar strain and results in a simplification of the governing equations to one spatial dimension as a result of the radial symmetry.