A BIPHASIC HYPERELASTIC MODEL FOR HYDROCEPHALUS

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***Abstract*— A biphasic hyperelastic model with spherical symmetry is presented to study hydrocephalus. The model can take into account the biphasic nature of brain tissue, non-linear stress-strain curves through an Ogden-type compressible strain energy function, a nonlinear variation of hydraulic conductivity with deformation, a constant production of cerebrospinal fluid (CSF) as well as a fluid absorption rate proportional to pressure. The biphasic equations were implemented in an updated Lagrangian finite element code where a novel procedure was devised to consider the constant generation of CSF in the ventricles. Results of a non-communicating model showed strains in the range of 35–45% in the peri-ventricular area and pore pressure and radial displacement distributions that were not significantly affected by material nonlinearities. High circumferential stresses documented in acute hydrocephalus suggest that the tissue may suffer damage at the ventricular surface while a communicating model was not capable of reproducing normal pressure hydrocephalus. Overall results suggest that normal pressure hydrocephalus cannot be explained with models that consider an elastic law for the solid phase of the material.**

***Keywords*— Acute Hydrocephalus; Normal Pressure Hydrocephalus; Porohyperelastic Model; Hyperelastic Biphasic Model.**

I. INTRODUCTION

The cerebrospinal fluid (CSF) present in the central nervous system plays an important role in the physiological activities and protection of the brain. This fluid is produced at a constant rate in the choroid plexuses of the lateral and third ventricles and flows within the Sylvius aqueduct to the fourth ventricle, whence it goes through small passages to the subarachnoid space. Disruptions of the CSF flow lead to different forms of a disease known as hydrocephalus, characterized by a significant increment of the ventricular space. In acute hydrocephalus the Sylvius aqueduct is blocked and ventricular pressure is greatly increased. In contrast, normal pressure hydrocephalus (NPH) is characterized by a significant deformation of the ventricles without an increase of the transmantle pressure or evidence of aqueduct blockage. It is important to study the biomechanics of hydrocephalus in order to help understanding the etiology of the disease, which may lead to better treatments or the development of more precise diagnostic tools.

In addition to several studies aimed to understand problems associated with the deformation of the brain (Wittek *et al.*, 2007), many mechanical models have been proposed to analyze hydrocephalus. Some of these models have considered brain tissue as a single phase elastic or viscoelastic material (Sivaloganathan *et al*., 2005; Drapaca *et al*., 2006). Beginning with the work of Nagashima *et al.* (1987), other models have simulated brain tissue as a poroelastic material, including the work of Kaczmarek *et al.* (1997) for a cylindrical geometry, of Taylor and Miller (2004) for a planar geometry, and of Smillie *et al.* (2005) and Sobey and Wirth (2006) for a spherical symmetric geometry.

All these models have generally given good correlations of clinical observations for acute hydrocephalus, however they have not considered the nonlinear stress-strain response that has been experimentally documented for brain tissue under finite deformations (Miller, 1999; Franceschini *et al*., 2006). Considering that displacements occurring during acute hydrocephalus can be significantly large, it would appear that nonlinear stress-strain curves under finite deformations should be taken into account, as recently proposed by Dutta-Roy *et al*. (2008) in a patient-specific non-communicating model that was unable to explain NPH. Whereas this model represents an important advance from the geometric point-of-view, it includes a pressure gradient condition not consistent with the constant generation of CSF and does not include the effect of CSF flow from the ventricles to the subarachnoid space through the Sylvius aqueduct.

While a physical explanation for acute hydrocephalus seems to be well agreed upon, many attempts have been made to explain NPH. The primary issue is that a communicating (unblocked) aqueduct minimizes the secondary mechanism of flow of CSF to the subarachnoid space through the parenchyma. Recent analysis by Levine (1999) and Sobey and Wirth (2006) suggest that large displacements in NPH can be explained by a combination of fluid absorption in the parenchyma and small values of hydraulic conductivity, however these models did not consider nonlinear stress-strain response of brain tissue.

A mechanical model intended to explain both communicating and non-communicating hydrocephalus is presented here that can take into account the biphasic