A mathematical model of blood, cerebrospinal fluid and brain dynamics

Andreas A. Linninger · Michalis Xenos · Brian Sweetman · Sukruti Ponkshe · Xiaodong Guo · Richard Penn

Received: 13 June 2008 / Revised: 13 January 2009 © Springer-Verlag 2009

Abstract Using first principles of fluid and solid mechanics a comprehensive model of human intracranial dynamics is proposed. Blood, cerebrospinal fluid (CSF) and brain parenchyma as well as the spinal canal are included. The compartmental model predicts intracranial pressure gradients, blood and CSF flows and displacements in normal and pathological conditions like communicating hydrocephalus. The system of differential equations of first principles conservation balances is discretized and solved numerically. Fluid–solid interactions of the brain parenchyma with cerebral blood and CSF are calculated. The model provides the transitions from normal dynamics to the diseased state during the onset of communicating hydrocephalus. Predicted results were compared with physiological data from Cine phase-contrast magnetic resonance imaging to verify the dynamic model. Bolus injections into the CSF are simulated in the model and found to agree with clinical measurements.

Keywords Cerebrospinal fluid · Communicating hydrocephalus · Intracranial pressure · Mathematical modeling · Computational fluid dynamics

A. A. Linninger · M. Xenos · B. Sweetman · S. Ponkshe Laboratory for Product and Process Design (LPPD), Department of Bioengineering and Chemical Engineering, University of Illinois at Chicago, Chicago, USA

A. A. Linninger (✉) Department of Bioengineering and Chemical Engineering, University of Illinois at Chicago, Science and Engineering Offices (SEO), Room 218 (M/C 063), 851 S Morgan St, Chicago, IL 60607-7052, USA e-mail: linninge@uic.edu

X. Guo · R. Penn Department of Neurosurgery, University of Chicago, Chicago, USA

Published online: 15 February 2009
Mathematics Subject Classification (2000)  74F10 · 76Z05 · 92B05

Abbreviations

cAr Carotid artery
Ar Arteries
Al Arterioles
Cp Capillaries
Vl Veinules
V Veins
vSinus Venous sinus
Lv Lateral ventricle
3V Third ventricle
4V Fourth ventricle
SAS Cranial subarachnoid space
sp.canal Spinal subarachnoid space
br Brain
exf Extracellular fluid
$x^L,R$ Signifying two equations, one for the left brain hemisphere and one for the right brain hemisphere
$xx^R$ Right compartment
$xx^L$ Left compartment
$f_{xx_{in}}$ Flow into the compartment
$f_{xx_{out}}$ Flow out of the compartment

1 Introduction

1.1 Motivation

A variety of central nervous system diseases alter intracranial dynamics and changes in dynamics may in turn result in changes to the brain. An important example is hydrocephalus in which the cerebral ventricles enlarge, thus in effect displacing and compressing brain tissue. This condition is well described clinically, but its fundamental dynamic principles are poorly understood. The goal of our research is to provide such an understanding and by doing so point the way to new treatment based on this knowledge.

Current mathematical models do not incorporate the interaction between the cerebral vasculature, parenchyma and cerebrospinal fluid (CSF) during the cardiac cycle, and many models do not account properly for conservation of the fluid volume (Lakin et al. 2003; Sorek et al. 1988a). According to the Monro-Kellie doctrine, the cranium is a closed system, enclosing the brain, CSF and cerebral blood; but for intracranial dynamics to be described correctly the spinal canal and its pulsating CSF displacements need to be included. The flow of CSF with each cardiac pulse into and out of the spinal subarachnoid space is well known by clinicians and
A mathematical model of blood, cerebrospinal fluid and brain dynamics

has been measured by Cine phase-contrast MRI (Pelc et al. 1991; Loth et al. 2001; Raksin et al. 2003; Zhu et al. 2006). As we will show, it is critically important in accounting for CSF flow patterns inside the ventricular and subarachnoidal systems.

1.2 Background

Some early models of the brain vasculature have simplified the dynamics by lumping numerous compartments (Sorek et al. 1988a,b, 1989; Ursino and Lodi 1997; Stevens 2000). Other approaches use bundles of tubes to represent different types of cerebral blood vessels (Zagzoule and Marc-Vergnes 1986). Monro's first model of the intracranial cavity consisted of two compartments, brain and blood. This model was expanded by Karni to contain several fluid structures, including arterial, capillary, venous, venous sinus, jugular bulb, and CSF pathways (Sorek et al. 1988a,b). To refine the model, Karni et al., added an additional component, brain tissue, to the previous six compartments model. Piechnik and collaborators developed a mathematical model to study autoregulation and cerebral species transport in the human brain (Piechnik et al. 2001). Marmarou derived a widely used mathematical model that describes intracranial pressure (ICP) dynamics (Marmarou et al. 1978). However, his model does not explicitly incorporate brain vasculature or the porous parenchyma in the calculations. Many researchers have based their experimental work on this model which correlates well with experimental data, but does not predict blood alterations or brain water content change (Czosnyka et al. 2004).

For the current study, a more complete dynamic model consisting of the bi-phasic brain, arteries, arterioles, capillaries, veinules, veins, venous sinus, ventricles, subarachnoid space and the spinal canal will be described and compared to experimental results obtained from Cine phase-contrast MRI measurements. This multi-compartment mathematical model accounts for cerebral hemodynamics, the expansion or compression of the parenchyma and the CSF flow dynamics. The expansion of the vasculature is linked with the volumetric change of the brain parenchyma. In turn, changes in cerebral volume affect the space available to the ventricles and the CSF. Due to the full coupling of the distensible blood vessels, CSF spaces, and the brain parenchyma, it is possible to solve dynamic force and mass balances of the entire system. The model is used to simulate normal and pathological conditions to determine the temporal change in ICPs and volumes for the various brain structures. The brain parenchyma pressure is a function of the force interaction with the embedded cerebral vasculature and CSF. Hence, elevation of ICP is not an input to the model, but is calculated by solving the model equations for the brain interacting with CSF and blood. Similarly, ventricular expansion is only possible as a function of force balances and flow equations. The objective is to describe and quantify the dynamic interactions between blood flow, ICP, extensions of the cerebral vasculature and brain parenchyma during the cardiac cycle, and how it is changed in pathological conditions. The modeling results should consistently describe the transient force interactions between blood, CSF and brain. Predictions of accurate states are a secondary objective.