Communicating Hydrocephalus: The Biomechanics of Progressive Ventricular Enlargement Revisited

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Summary

Background. This article investigates the physical mechanisms involved in the chronic ventricular enlargement that accompanies communicating hydrocephalus (CH) – including its normal and low-pressure forms. In particular, it proposes that this phenomenon can be explained by the combined effect of: (a) a reversal of interstitial fluid flow in the parenchyma, and (b) a reduction in the elastic modulus of the cerebral mantle.

Method. To investigate this hypothesis, these changes have been incorporated into a finite element computer simulation of CH, in which brain tissue is idealized as a sponge-like material. The fluid pressure in the lateral ventricles and the subarachnoid space has been set to 10 mmHg, while the fluid pressure inside the parenchyma has been set to 7.5 mmHg. The elastic moduli of white and gray matter have been set to the reduced values of 1 and 5 kPa, respectively.

Findings. The simulation revealed a substantial ventricular distension (6.5 mm mean outward displacement), which was accompanied by the appearance of stress concentrations in the cerebral mantle.

Interpretation. These results support the notion that a relative reduction in intraparenchymal fluid pressure coupled with low tissue elasticity can produce both a significant ventricular enlargement and periventricular solid stress concentrations.

Keywords: Communicating hydrocephalus; biomechanics; ventricular enlargement.

Introduction

Hydrocephalus presents many paradoxes including the distribution of periventricular tissue damage, association of ventricular dilatation with high blood pressure, specific neuropsychological deficits and lack of correlation in many cases between the size of ventricles, clinical symptoms and the efficacy of a shunt [25]. While it is easy to understand that ventricular distension in non-communicating hydrocephalus (NCH) can be attributed to a transmantine pressure gradient subsequent to obstruction of the aqueduct of Sylvius, it remains an unresolved question why in communicating hydrocephalus (CH) the ventricles continue to dilate despite free communication between ventricles and the subarachnoid space and a normalization of ICP [13].

Several authors have proposed explanations for ventricular enlargement, including: alterations in the viscoelastic properties of brain parenchyma [23, 33], raised mean ICP and intracranial pulse pressure [8], normal tissue stresses [16] and the existence of transmante pressure gradients [4, 38]. However, none of these studies have been able to convincingly integrate theoretical considerations, drawn usually from continuum mechanics, with experimental and clinical observations.

In this article we propose the hypothesis that the chronic ventricular distension that accompanies CH can be explained by the combined effect of: (a) a reversal of interstitial fluid flow into the parenchyma, and (b) a reduced tissue elasticity.

Methods

Biomechanics was introduced to the study of hydrocephalus by Hakim et al. [14, 15, 16]. In their pioneering work they proposed that brain parenchyma can be regarded as "an open cell sponge made of viscoelastic material". Nagai and Takaichi [22] formalized this concept in terms of the theory of poroelasticity [3], which studies the deformation of porous elastic materials. Since then, the view of brain tissue as a poroelastic material has been used in a number of studies [18, 24, 37]. The deformation mechanics of a poroelastic material is described by Biot's equations. As these equations have no general analytical solution, an approximate or numerical solution needs to be computed using finite element analysis [40]. The anatomical infor-
mation required to construct the FE mesh for our simulation was obtained from a T2-weighted, horizontally-orientated MR image of a brain from a normal volunteer.

Although the material properties of brain tissue are incompletely defined, experimental evidence suggests that the normal stiffness value of brain tissue is in the range 10 to 100 kPa [12]. In the hydrocephalic brain there is indirect evidence in the form of histological data from experimental models [28–30] and reduced PVI clinical data [6, 10, 36] which suggests that the brain tissue elasticity is reduced. In this study we have assumed isotropic material properties of 1 kPa and 5 kPa for the elastic moduli of white and gray matter, respectively. A Poisson’s ratio of 0.30 and an hydraulic conductivity of $10^{-11}$ m$^2$ N$^{-1}$ s$^{-1}$ has also been assumed. The fluid pressure in the lateral ventricles and the subarachnoid space has been defined at 10 mmHg, within the normal range of ICP.

While it is generally agreed that there is fluid exchange between the CSF and brain via perivascular spaces, the magnitude and direction of flow remains controversial [26]. Intraventricular dye-injection experiments have demonstrated that the brain parenchyma acts as a CSF sink during hydrocephalus [20] while ventricle perfusion experiments suggest that there is a reversal of transcapillary flow of interstitial fluid from CSF to brain [27]. More recently it has been established that perivascular spaces serve as conduits to transport cerebral interstitial fluid (ISF) to both blood and lymph [5]. In order to simulate this behavior, a reduced fluid pressure inside the parenchyma has been defined at 7.5 mmHg. Intravascular pressure at different points of the cerebrovascular bed will differ from adjacent interstitial fluid pressure as the result of the interposed compliance of the vascular wall [7]. Therefore, in this study interstitial fluid pressure refers to the extracellular space fluid pressure and not to venous pressure.

Results

In accordance with clinical and experimental observations, the simulation of brain deformation during hydrocephalus, as defined by a sequence of FE meshes demonstrated progressive ventricular expansion. The magnitude of this enlargement at steady-state is illustrated in Fig. 1. The outward movement or displacement of the ventricular wall associated with this distension was heterogeneous along the antero–posterior direction of the ventricle. The maximum displacement occurred in the region of the thalamus with a value of almost 9 mm (Fig. 1, point B). This was followed by displacements of 8 and 9 mm in the anterior and posterior cingulate gyri, respectively (Fig. 1, points A & C, respectively) and two regions of very small displacements near the anterior and posterior horns (4.5 and 5.0 mm, respectively). The overall mean outward displacement of the ventricles was 6.5 mm.

Discussion

Several studies have emphasized the importance of various physiological mechanisms in the development of chronic ventricular enlargement during CH [4, 8, 16]. Our FE simulation enabled us to investigate the effects of intraparenchymal fluid pressure and tissue elasticity on this process. Our theoretical analysis confirms that realistic alterations in these variables can result in both a significant ventricular enlargement and periventricular solid stress concentrations. One of the early theories of normal pressure hydrocephalus (NPH) was that of Fishman [9] and Guinane [13] who proposed that it was not the absolute ventricular pressure, but rather the difference between ventricular pressure and the pressure over the cerebral convexity (the so-called transmantle pressure), that was the physiological determinant for ventricular dilatation. However, the existence of such a pressure gradient has been difficult to confirm. For example, Hoff and Barber [17] found an elevated transmantle pressure in three of four hydrocephalic patients and Conner [4] detected a gradient of 0.5 kPa (3.4 cm H$_2$O) in the cat. However, Shapiro et al. [32] failed to measure any pressure gradient. Regardless of these data, the measured values of Conner et al. are too small to create any significant ventricular distention, given that they are, respectively, 2 and 5% of the typical range of the stiffness modulus of brain parenchyma. Hakim et al. [16] who also postulated the existence of a transmantle pressure gradient, proposed that one of the laws of mechanics could be applied to the understanding of hydrocephalus: $P = F/A$ where $P$, $F$, and $A$ are
pressure, force and area, respectively. However, while this reference to Pascal’s law enables the computation of changes in pressure, force and area on the surface on the brain, it provides no information about the deformation sustained inside the parenchyma. Finally, Pang and Altschuler [23] suggested that ventricular enlargement is related to an alteration of the viscoelastic modulus of the brain, secondary to expulsion of extracellular water from the brain parenchyma and to structural changes in brain tissues. However, changes in the mechanical properties of tissue without the presence of a pressure gradient cannot produce ventricular distension.

The theories discussed above provide insufficient explanations for ventricular distension. This is evident if we frame the discussion in terms of the fundamental principle established by Hooke in 1675 on the relationship between deformation and an applied force: \( dL/L = (P/A)/E \), where \( dL \) is the change in length of the sample, \( L \) is the original length of the sample, \( P \) is the applied force, \( A \) is the cross-sectional area of the sample, and \( E \) is a proportionality constant known as Young’s modulus [11]. Relative deformation or strain (i.e. \( dL/L \)) is directly proportional to an applied stress (i.e. \( P/A \)) and inversely proportional to the elasticity modulus \( (E) \) of the material. It follows that a substantial deformation of the cerebral mantle may be produced by the combined application of an increased pressure gradient together with a reduction in tissue elasticity. The application of this simple principle has

**Non-communicating Hydrocephalus**

**Communicating Hydrocephalus**

![Diagram](image)

Fig. 2. This diagram illustrates the interaction of the various mechanisms involved in the development of non-communicating (left) and communicating (right) hydrocephalus. The arrows represent the interstitial CSF flow. The open dot represents sources of CSF, the closed dot represents sinks. The square represents the sagittal sinus (SS). The cross represents an obstruction to CSF flow. The broken line represents the aqueduct of Sylvius. LV stands for lateral ventricles. SAS for subarachnoid space. In the case of NCH, due to the obstruction of the aqueduct, a transmantle pressure (shown in black) is established between the LV and the SAS. CSF follows this gradient moving across the parenchyma to be finally absorbed at the SS. In the case of CH, there is no obstruction to the aqueduct but rather at the sagittal sinus. An intramantle pressure gradient (shown in black) is produced, as the CSF is absorbed by the parenchyma. In both situations, ventricular distension occurs...
led us to formulate the hypothesis that the chronic ventricular distension that accompanies CH can be explained by the combined effects of two factors: (a) a reversal of interstitial fluid flow in the parenchyma with a subsequent absorption of CSF by the tissue, and (b) a low tissue elasticity, brought about by a series of pathological changes.

Concerning the first factor, there is some convincing evidence for transepndymal absorption of CSF. Early work on the pathways for CSF absorption postulated an alternative transventricular route [39] and these alternative pathways were demonstrated to be pressure dependent in the experimental hydrocephalic dog [2]. Subsequent studies substantiated the idea of transventricular absorption after demonstrating uptake of label into the parenchyma of various experimental models of hydrocephalus [1, 35].

Concerning the second factor, while the Young’s modulus of bovine brain has been measured in vitro [12] no equivalent clinical or experimental in vivo hydrocephalic data exists. However many studies using the PVI as a measure of CSF compliance do provide some indirect evidence to support the notion that the stiffness modulus of tissue is reduced in CH. For example, the PVI was increased from the predicted normal value in a group of hydrocephalics at the time of shunt malfunction [10], in NPH patients [36], in low pressure hydrocephalus [23], infants [31] and in the experimental hydrocephalic cat when compared to controls [34]. The markedly increased PVI in these studies is presumably related to an alteration in the mechanical properties of the brain parenchyma, since the potential for deformation of the cranium is limited, especially in adults, given that cartilage is one hundred times stiffer than brain parenchyma [21]. Similarly, the vasculature represents only 7% of the total intracranial volume so that a substantial contribution to the changes in PVI is unlikely. Finally, the reduction in the stiffness modulus of the brain, from a physical point of view is also inferred from the major histopathological changes, such as neuronal injury, reactive astrocytosis and myelin degradation, in both experimental [19] and clinical hydrocephalus [30].

The crucial point in our theory is that the reversal of CSF transepndymal flow implies that the fluid pressure is smaller in the parenchyma than in the surrounding CSF spaces. As a consequence of the movement of fluid into the tissue, a pressure gradient is established between the CSF spaces and the cerebral mantle: an intramantle pressure gradient.

Acknowledgments

AP is in receipt of a Wellcome Trust Training Fellowship in Mathematical Biology, and NGH of a Merck, Sharp & Dohme Research Fellowship.

References


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