Magnitude and Temporal Characteristics of Lateral Ventricle Contraction and Expansion

D. C. Zhu¹, M. Xenos², A. A. Linninger², R. D. Penn³

¹Radiology, University of Chicago, Chicago, IL, United States, ²Chemical Engineering, University of Illinois at Chicago, Chicago, IL, United States, ³Neurosurgery, University of Chicago, Chicago, IL, United States

Introduction

Disturbances of the cerebrospinal fluid (CSF) flow in the brain leads to hydrocephalus. Considerable controversy exists about fluid and pressure dynamics, and about how the brain responds to changes in flow patterns and compression. In an attempt to clarify this controversy, a new hydrodynamic model based on the first principles of fluid mechanics was introduced recently (1). Reported here are our measurements of the volumetric change of the lateral ventricle and its temporal relationship with CSF flow based on MR cine phase contrast techniques. These measurements will play important roles in validating the first principle models.

Methods

Two-dimensional cine phase contrast techniques (2) were applied to collect CSF flow data from five healthy brains on a 3T GE Signa system equipped with a standard bird-cage head coil. Three-direction velocity data at 16 equidistant time frames were acquired at a slice location at the middle of the lateral ventricle to investigate the contraction and expansion of this ventricle. Velocity data in the superior-inferior direction at 32 equidistant time frames were acquired at a slice location between the aqueduct of Sylvius and the 4th ventricle to measure CSF flow. For all studies, the other parameters are listed: flow compensation, peripheral gating, 5 cm/sec VENC (corresponding to a phase shift of 180°), TE = 8.4 ms, TR = 18 ms, flip angle = 20°, field of view = 24 cm, slice thickness = 5 mm, matrix size = 256×192, number of excitation = 2, 75% phase field of view to achieve an effective matrix resolution of 256×256 . To reduce the possible spatially dependent offset velocity due to eddy currents or head motion, the velocity at a pixel at CSF was corrected through subtraction by the time-average "velocity" of a nearby solid brain tissue "background" within a $29mm\times29$ mm region with this pixel at the center (3, 4). In calculating the velocity at solid brain tissue, the velocity at each pixel was corrected through subtraction by the time-average "velocity" of this pixel at the center (3, 4). In calculating the velocity at solid brain tissue, the velocity at each pixel was corrected through subtraction by the time-average "velocity" of this pixel itself. The volumetric flow at the CSF pathway was estimated by multiplying the average velocity at the cross section and the corresponding area.

The brain tissue movement through the cardiac cycle is often just a small fraction of a pixel based on measurements from other group (3) as well as from our group using a cine phase contrast technique. We developed a velocity-based technique to measure the lateral ventricle contraction and expansion at sub-pixel level, similar to the one developed by Oyre et al. (5). The starting ventricle edge positions were manually drawn and then the position offsets were calculated based on the velocity on the same plane. The expected position of each original ventricle edge pixel was estimated by adding the original position with the position offset. The points at the ventricle edge at each cardiac frame, including the original ones manually drawn, were connected together with spline interpretation. After the area of the enclosed region at each cardiac time frame was calculated, the fractional area change (f_A) of the enclosed region was calculated as the ratio between the maximum-minimum area difference and the average area through the cardiac cycle. Assuming the lateral ventricle contracted and expanded uniformly across the whole ventricle,

the fractional volumetric change (f_V) of the lateral ventricle from maximum to minimum was estimated based on this equation, $f_V = \left(1 + \frac{f_A}{4}\right)^3 - \left(1 - \frac{f_A}{4}\right)^3$. The

volume of the lateral ventricle was estimated from inversion prepared T_1 -weighted volumetric images, where CSF was suppressed. The volumetric change of the lateral ventricle was estimated from the ventricle volume and f_V . The temporal relationship between the lateral ventricle size change and the CSF pathway was also measured. The mid-point of the CSF flow at either direction and the mid-point of the larger or smaller half of the lateral ventricle size were estimated based on temporal weighted average. The CSF flow-direction switching points were estimated to be at the midle of the mid-points of the two flow directions.

Results and Discussion

For the five normal subjects studied, the maximum displacement of all the pixels at the edge of the lateral ventricle was 0.113 ± 0.024 mm on the same scanning plane, and was 0.150 ± 0.023 mm with all three spatial directions considered. The volumetric change of the lateral ventricle was estimated to be 0.745 ± 0.420 %. The mean oscillatory flow at the junction of the aqueduct of Sylvius and 4^{th} lateral ventricle was $4.53 \pm 1.33 \text{ ml/min}$. As demonstrated in Figure 1, similar pulsatile pattern of lateral ventricle contraction and expansion was seen across all subjects. Four subjects but one showed similar temporal relationship with CSF flow (Figure 1): The lateral ventricle began to contract ahead of the switch from the flow direction of inferior-to-superior (Ito-S) to that of superior-to-inferior (S-to-I) by 15.7% cardiac cycle, and began to expand ahead of the switch from S-to-I flow direction to that of I-to-S by 17.3% cardiac cycle. Assuming the change of the lateral ventricle size was one of the driving forces of the CSF flow, there was a delayed response. The lateral ventricle had a volume of 18.8 ± 4.0 ml. The amount of volumetric change due to contraction or expansion was estimated to be 0.151 ± 0.113 ml. The amount of oscillatory flow (the average of S-to-I and I-to-S flows) in one cycle was 0.0335 ± 0.0179 ml. Only about 27.0 ± 11.9 % of the lateral ventricle volumetric change was needed to drive the oscillatory flow, assuming all the volume of the CSF flow was due to the lateral ventricle volumetric change. This suggests that some CSF might have seeped into the brain parenchyma during contraction and was released from the brain parenchyma during expansion. Further investigation is needed to estimate the overall under or over estimation of volumetric change of the ventricle and CSF flow.





References

Linninger AA, Tsakiris C, Zhu DC, Penn R. IEEE Transactions on Biomedical Engineering (in press), 2004.
Dumoulin CL, Souza SP, Walker MF, Yoshitome E. Magn Reson Med. 1988;6:275-286.
Enzmann DR, Pelc NJ. Radiology 1992;185:653-660.
Poncelet BP, Wedeen VJ, Weisskoff RM, Cohen MS. Radiology 1992;185:645-651.
Oyre S, Ringgaard S, Kozerke S, Paaske WP, Scheidegger MB, Boesiger P, Pedersen EM. Magn Reson Med. 1998;40:645-655.