A New Method for Detecting Phase Shifts of Pulsatile CSF Flow using Phase Contrast MRI

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Abstract:

We have used phase-contrast MRI to measure the amplitude and timing of CSF pulsations in the brain. As compared to earlier studies which utilized the peak systolic and peak diastolic positions as a measure of CSF flow timing, we have used custom data processing and fitting techniques in order to extract accurate flow timing parameters. In a group of healthy individuals, these measurements clearly show that CSF flow in the cerebral aqueduct lags behind the flow in the inferior portions of the CSF cranial and spinal subarachnoid spaces, as well as the arterial flow in the cranium. In addition, we have found a statistically significant lag of the flow in the prepontine cistern. We have also measured peak flow in the cerebral aqueduct and find a wide variety of these measurements, over the range of 77 - 208 ul/sec (133 ± 48 ul/sec) in healthy individuals.

Introduction:

Disorders of intracranial flow such as hydrocephalus have been viewed for many years as a product of dysfunction in the bulk flow of CSF through the cranium from its production sites in the choroid plexus to its reabsorption sites in the arachnoid villi. The current workhorse of therapeutic intervention in hydrocephalus, the shunt, is in fact based on this principle. However, over the past fifteen years, careful MRI studies have clearly shown that the majority of CSF flow in the brain is not one of bulk flow from point A to point B, but it is primarily pulsatile in nature, flowing back and forth between the ventricles and the cranial/spinal subarachnoid spaces (SAS) [2,3]. With this in mind, we have developed a new model of intracranial dynamics which views the cranium as a forced oscillator system, a system which is finely tuned to the cardiac pulsation rate with the forcing term being provided by the pulsatile arterial input [4,5]. In such a system, the resulting CSF flow patterns are oscillatory in nature and exhibit a very specific amplitude and phase relationship to the driving arterial forces. A number of earlier works have noted timing differences in certain portions of the CSF flow network. Specifically, Enzmann [6] and Bradley [7] have shown that the flow in the cerebral aqueduct typically lags behind the flow in the more caudal subarachnoid regions. However, no attempts were made in these studies to quantify the timing differences relative to the cardiac rate, or relative to the timing of the cranial arterial flow. The purpose of this work was to explore, in a healthy population, the specific phase relationships between CSF flow in various regions and arterial flow. This will establish a baseline with which we can begin to explore changes in these phase relationships in patients with hydrocephalus and other CSF flow disorders.

Methods:

Thirteen healthy volunteers were studied on a 1.5T Philips Edge scanner. The phase-contrast scans were acquired as single slices with the following orientations and parameters: 1) perpendicular to the cerebral aqueduct with Venc = 4-5 cm/s, FOV 15-18 cm and ST 5-6 mm, 2) axial at the level of C2 with Venc = 5 cm/s, FOV 22 cm and ST 5 mm, 3) sagittal midline slice with Venc = 5cm/s, FOV 22cm and ST 7 mm, and 4) perpendicular to the cerebral aqueduct with Venc = 60 cm/s, FOV 20-22 cm and ST 5mm to visualize intracranial carotid, basilar and sagittal sinus flows. The oblique axial studies allowed high resolution and accurate estimates of flow parameters, while the sagittal study suffered in terms of resolution but was used to visualize the entire length of the major CSF flow pathways. Other parameters: craniocaudal flow encoding, peripheral gating on every other beat to acquire images over 1.2 – 1.9 cardiac cycles, 2 k-space lines per cycle, 10-16 cardiac phases per cycle, 256x192 matrix, 2NEX. The low Venc and small FOV for the aqueduct images were crucial to obtain accurate estimates of flow timing and amplitude. Aliasing was often seen in the aqueduct but could always be corrected in postprocessing.

In our model of a tuned system, the amplitude and phase of the resulting flow patterns are a direct product of the coupling between the CSF and its driving system (the arteries). As an example, changes in the compliance of the system, as experienced in hydrocephalus, can have a significant impact on these parameters. To produce an accurate estimate of flow phase, it is important to view the entire flow pattern rather than looking at the timing of peak systolic and peak diastolic flows alone (although these are related to the overall flow pattern, there can be subtle differences which we sought to elucidate). Each flow waveform was therefore fit to a sum of the first and second harmonic sine waves at the cardiac frequency. However, the heart rate is an unknown and input into the model as a fit parameter. For waveforms containing < 1 cycle of data, this can lead to severe errors in the phase estimations due to the potential ambiguity between frequency and phase in the fit. Data were therefore accumulated over > 1 cardiac cycle, making the estimation of cardiac frequency in the fitting much more robust and the errors in phase were greatly reduced.
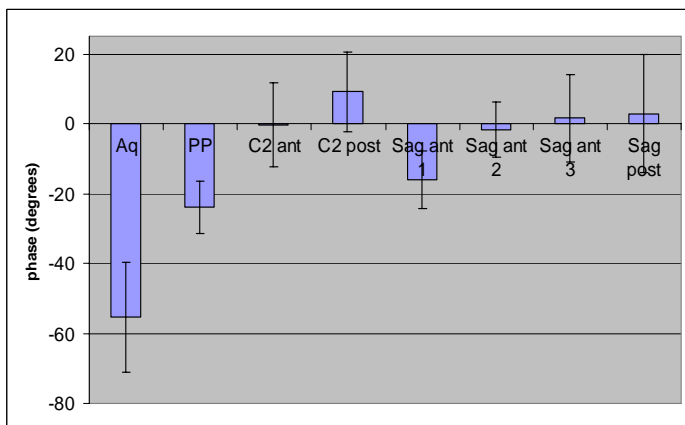

Fig 1. Flow phase relative to carotid flow in various CSF regions. Aq = aqueduct, pp = prepontine cistern, sag = sagittal study.

Results:

Excellent fits were obtained with our model in almost all cases. Figure 1 shows the flow phase, averaged for the entire group of subjects, in the various CSF compartments relative to the of the carotid pulse phase. The phase delay in the aqueduct, -55 ± 16 degrees, corresponds to a timing delay of about 150 ms. The unexpected result, a statistically significant phase delay in the prepontine cistern (PP), was corroborated in the sagittal studies where we divided the anterior SAS into three roughly equal sections. The first section, in the vicinity of the PP, clearly demonstrates the same phase delay. Peak flow in the aqueduct for the group was 133 ± 48 ul/sec (77 – 208 ul/sec).

Discussion:

If one views the cranium as an oscillator system, phase shifts between various compartments are of interest because they offer a method of directly measuring changes in compliance in the system. We have demonstrated here that there exist clear phase relationships between CSF and the arterial pulse in the cranium. In particular, there is zero phase difference in the caudal SAS, indicative of a resonant system where the driving arteries and the driven CSF beat in synchrony. In the more rostral spaces, this synchrony is lost, presumably due to compliance effects in the system. In future work, we hope to elucidate the source of these phase shifts, and to investigate the nature of the shifts in hydrocephalus patients where the compliance of the cranium is markedly disturbed from its normal state.

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