Stroke Volume Ratio as a Measure of Intracranial Flow Dysfunction

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Abstract

A method for quantifying intracranial flow, which can potentially be used for distinguishing between normal and hyperdynamic flow in hydrocephalus patients, is described. The method is based on the measurement of the ratio of the stroke volume at the cerebral aqueduct to the stroke volume at the craniocervical junction, using quantitative phase contrast MRI. The method was tested in a group of healthy individuals and compared to two other measures of intracranial flow which have been used previously in characterization of dysfunctions in intracranial flow: aqueductal stroke volume and average flow rate. The method shows good consistency amongst a group of thirteen control subjects, and is expected to show less change in non-symptomatic patients with large ventricles while still demonstrating abnormal flow in symptomatic hydrocephalus patients.

Introduction

Disorders of intracranial flow, such as hydrocephalus, are often characterized by increased CSF flow in the cerebral aqueduct. While this fact is well documented [1] and accepted in clinical practice, the question of the most valuable flow measure indicative of intracranial flow disorder still lingers. Most important in this field, is the question of how to determine which patient is most likely to improve through ventricular shunting and which is not. For example, Bradley used the stroke volume to determine the likelihood of patients improving with shunting, recommending a threshold of 42 μl as the cutoff for shunt operations [2]. Luetmer *et al* [3], on the other hand, considered average flow rate as a more predictive measure of flow dysfunction and recommends a threshold of 18 ml/min to distinguish between normal and hyperdynamic flow in diagnosing normal pressure hydrocephalus. The difference between these two measures is the consideration of subject heart rate. In a recent publication, Egnor *et al* [4] developed a model of flow dysfunction in hydrocephalus which describes intracranial dynamics in terms of a tuned oscillator system. There are two important implications from this work which relate to the question at hand. First, because the intracranial cavity is modeled as a tuned system, heart rate is integral to the flow dynamics and can not be neglected from flow analyses of normal vs abnormal flow. Secondly, one must look at the entire intracranial cavity as a whole in order to gain insight into its function. Specifically, hydrocephalus which can lead to enlargement of the ventricles, may be due to a redistribution of intracranial pulsations between the subarachnoid and ventricular spaces. In communicating hydrocephalus, this redistribution could arise from changes in the compliance of the individual CSF spaces. Based on this model, we hypothesized that it would be useful to measure the ratio of stroke volume between the ventricular and subarachnoid spaces as an appropriate measure of flow dysfunction, rather than looking at the stroke volume of the aqueduct alone. The purpose of this work was to establish a baseline measure of stroke volume ratio in a group of healthy individuals, and to compare it to measures of aqueductal stroke volume and average flow rate.

Methods

Fifteen consenting healthy volunteers, ages 25 – 57 (mean age: 37 ± 12), were studied on a 1.5T Philips Edge scanner. The phase-contrast scans were single axial slices perpendicular to the aqueduct and at the level of the craniocervical junction (CC). Both sequences had the following parameters: craniocaudal encoding direction, Venc 4-6 cm/s, peripheral gating triggered on every other beat to acquire images over > 1 cardiac cycles, 2 k-space lines per cycle, 10-16 phases per cardiac cycle, 256x192 matrix, 2NEX. Aqueduct scans were obtained at a FOV of 14-16 cm, while images at CC used a FOV of 22 cm. Due to the low Venc and high spatial resolution, aliasing was often seen in the aqueduct and corrected in postprocessing via phase unwrapping.

Net flow waveforms were collected from the images, after a thresholding algorithm was applied. The algorithm used 1) pulsatile flow amplitude at the cardiac frequency (> than 3% of peak flow amplitude), 2) pulsatile flow amplitude relative to the background noise (> 2.5 * average flow amplitude above the cardiac frequency), and 3) flow phase at the cardiac frequency (within 40 degrees of average phase). The first two conditions were set empirically to exclude non-pulsatile regions, while the latter condition was used to exclude non-CSF flow, such as the epidural veins adjacent to the spinal subarachnoid spaces. Flow waveforms were then fit to a three harmonic sinusoidal function and baseline offsets, due to bulk flow or phase errors, were subtracted.

Stroke volume was calculated from the flow waveforms by summing the velocities in all valid pixels in the region of interest and multiplying by pixel area, producing net flow waveforms in ml/s. Net stroke volume (SV) was extracted as the integral of the flow waveform for all flow values above (for caudal flow) or below (for cranial flow) the baseline. Stroke volume ratio (SVR) was determined as the ratio of the aqueductal to the CC junction stroke volume. Average flow rate (AFR) was measured by taking the absolute value of the flow, and calculating the average flow value over one full cycle [3].

Results

Of the fifteen individuals studied, thirteen provided valid aqueduct flow data for analysis. The other two subjects demonstrated inadequate spatial resolution (< 8 pixels total) or poor image quality, so that reliable aqueductal flow waveform were not obtained. In the remaining thirteen subjects, valid flow data was obtained and analyzed.

	SV (μl)	SVR (%)	AFR(ml/min)
#1	33.2	6.05	3.99
#2	22.0	3.25	3.49
#3	62.1	8.02	8.09
#4	12.5	2.70	2.01
#5	29.8	4.67	4.45
#6	17.5	4.97	2.23
#7	36.7	4.51	4.86
#8	12.8	1.88	1.80
#9	27.5	4.61	4.70
#10	22.1	6.02	4.31
#11	45.8	7.42	6.25
#12	40.9	5.75	5.33
#13	38.1	6.05	4.36

Table 1: Data analysis results for all subjects.

Results for the group were:

Mean stroke volume ratio: 5.07 ± 0.49 % (mean ± SEM, range 1.88 – 8.02).
Mean aqueductal stroke volume: 30.9 ± 3.9 μl (range 12.5 – 62.1).
Mean flow rate: 4.28 ± 0.48 ml/min (range 1.80 – 8.08).

It should be noted that one outlier, subject #3, was noted to have larger ventricular volume than the group ($p < 0.001$), although from the neuroradiologist's reading the ventricle size was not outside of the normal range. While it is difficult to make judgments from a single subject, it is nonetheless interesting to note that this subject has a stroke volume and flow rate which are three standard deviations above the mean (when excluding this subject from the analysis), while the stroke volume ratio is within two standard deviations of the mean. By taking a ratio, the stroke volume is normalized to overall subarachnoid flow and may provide a more reliable measure of dysfunction.

Discussion

Idiopathic communicating hydrocephalus is a disease that has never been adequately explained and continues to be the source of much controversy in the neurosurgical community. The development of a diagnostic tool which can clearly and definitively distinguish between patients who will or will not benefit from shunt insertion will represent a major advance in this field. In addition, such analytical tools may also serve as diagnostic measures for evaluating new shunt technologies. We have presented an alternative to the standard measurement of hyperdynamic flow in the aqueduct, based on the assumption that flow dysfunction may be related not to absolute hyperdynamic flow, but to the redistribution of flow between the ventricular and subarachnoid spaces. The results show that this measure has a statistical variation slightly smaller than that of the measures of stroke volume or average flow rate, although with this small a sample size this difference may be insignificant. Nonetheless, the results present the range for this measure in healthy controls and we plan to move on to observe the changes in stroke volume ratio in various patient populations.

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