Cerebrospinal Fluid Pulsatility Model and its Validation

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Introduction: It is widely believed that abnormal behaviour in CSF flow is due to changes in ventricular size, aqueductal diameter, brain compliance, and cerebral blood flow. In diseases such as dementia, Alzheimer's disease, and hydrocephalus, the normal equilibrium between blood and CSF flow is disturbed, and it is important to know the mechanisms of these flows and their relationship. Studies on normal subjects suggested that volumetric flow capacitance must be provided by a comparable volume of fluid (blood) or tissue displacement since the brain is not compressible and there is no net CSF flow in or out. We have developed a physical model of cerebral blood and CSF flows, based on the identification of an equivalent electrical circuit [2], and estimated model parameters using MRI data obtained from normal subjects.

Methods: We would expect all mechanisms relating to net production of CSF to be negligible within a single heart cycle and model-able as a linear function. We can analyse the circuit using conventional means by specifying current flows and the equations describing current and voltage, Fig 1. This yields the frequency domain constraint equation $\gamma I_3 - \alpha I_1 + \beta I_4 = 0$ where α, β, γ can be found via application of the Likelihood based variational method. We can say that the complex residual on the constraint for each Fourier amplitude ω in the measured currents is $F_\omega = \gamma I_{3\omega} - \alpha I_{1\omega} + \beta I_{4\omega}$. Assuming equal random independent Gaussian errors on each of the measured Fourier amplitudes on each current, σ_i we get the likelihood that we should be minimising: $-Log(P) = \Sigma_\omega F_\omega^* F_\omega / var(F_\omega)$, where $var(F_\omega) = \alpha^* \alpha \sigma_1^2 + \beta^* \beta \sigma_4^2 + \gamma^* \gamma \sigma_3^2$. We have 24 normals (18 male, average age 35 and 6 female, average age 24.17) and acquired velocity encoded PC-cine MR images of CSF at AQ (cerebral aqueduct) & FM (foramen magnum), arterial blood flow images at BA (basilar) & CA (carotid) for each subject (Fig 2). We fitted a quadratic function over the cerebral aqueduct in each time point in the cardiac cycle, and integrating the area under this curve for positive values of the fitted function. The BA, CA, FM flow was estimated from the sum of flow values within the region of the arteries, defined by thresholding the modulus images to find the ROI.

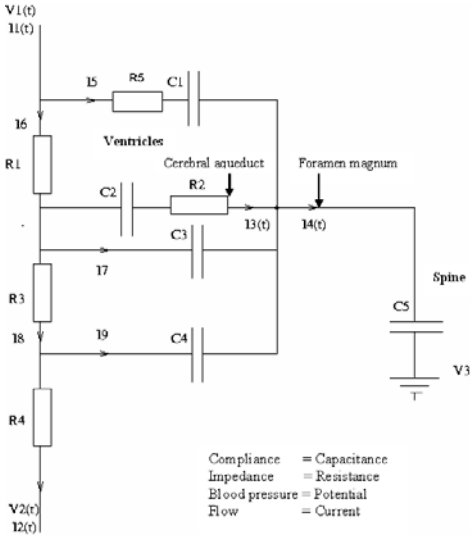

Fig 1.Electrical circuit model

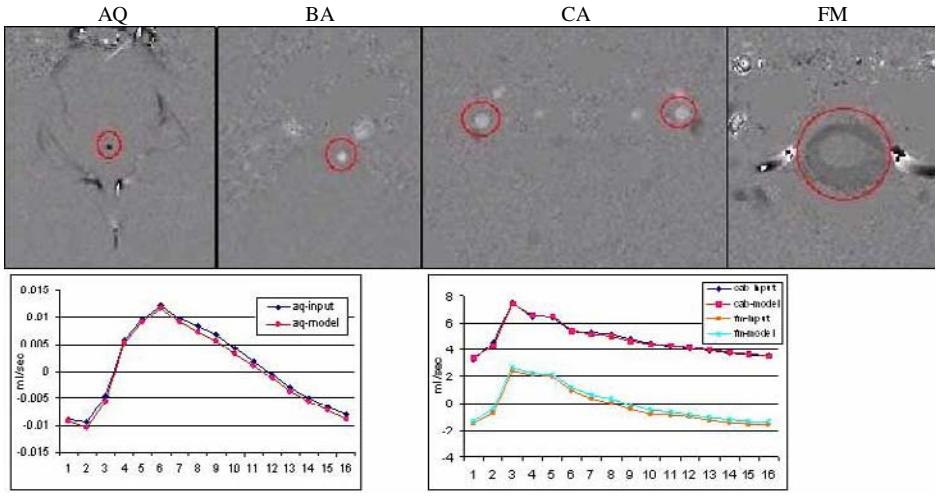

Fig 2. MR images and flowrate graphs obtained from them and error corrected model outflow.

Results: Fitting parameters to each individual showed that there are often not enough significant Fourier terms to allow estimation of the parameters. Therefore, we attempted to identify a subset of our normal group (in this case 16 from 24 individuals) which appeared to be consistent with one set of parameters. Using this approach any differences between observed flow curves for the selected subset are assumed to be due to differences in the systemic behaviour (or input flow curves). The adequacy of the model was tested by how well the model regenerated the input data sets (Fig 2) and the stability of derived parameters was tested by plotting the shape of the Likelihood function (Fig 3).

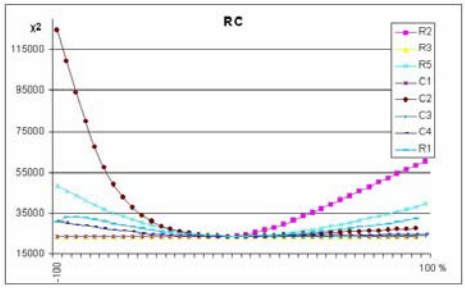

Fig 3. Parameter fitting

Parameter	Physical interpretation	Value
R1	impedance of arterial capillaries	1.0
R2	impedance of cerebral aqueduct	~ 0.0
R3	impedance of venous capillaries	~ 0.0
R5	impedance of arteries	0.01
C1	elastic capacitance of arteries	~ Large
C2	elastic capacitance of ventricles	4.11
C3	elastic capacitance of capillaries	~ 0.0
C4	elastic capacitance of veins	271.017

Tab 1. Parameter values obtained

Conclusion: Previous work has suggested the use of equivalent circuit models for the description of CSF pulsatility [1, 2]. However, our work is first to validate such a model with MR data from normal control subjects. The model is capable of describing a large subset of our normal group with one set of parameters. We hope in the future to extend and test the model for patient groups and test predictions from the model such as venous output.

References: [1] M. Egnor et al. A model of pulsations in communicating hydrocephalus. *Pediatric Neurosurgery*, 36:281-303, 2002.

[2] M. Ursino and M. Giulioni. Quantitative assessment of cerebral autoregulation from transcranial doppler pulsatility: a computer simulation study. *Medical Engineering and Physics*, 25:655-666, 2003.