## Cine MRI Flow-Analysis in Phantom Model of Cranio-Spinal Axis In-vivo Studies.

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**Introduction:** Considerable debate surrounds the genesis of cine flow curves at the level of the aqueduct and the foramen magnum. To understand the phase relationship between the arterial and CSF flow, we developed a phantom model and subjected it to MRI cine flow examination.

**Material and Method:** The model consisted of a volume plethysmograph based on the forearm with a chamber representing the ventricle with an outlet representing the aqueduct The plethysmograph was filled with water with an open tube on one side to represent the spinal canal. The pulsations of the radial artery acted as the force that moved the fluid from the ventricle through the aqueduct to the tube representing the spinal canal (Figure 1). Peripherally gated cine phase-contrast MRI scan was performed with 32 cardiac and TE ~ 9ms; FA=30°; Nex=1; FOV=18-16 cm ; Matrix = 256x128; thickness = 5 mm; rBW=15,63kHz; Head-Coil (2). Three velocity encodings values were chosen to match the flow of the cerebro-spinal fluid at the acqueductal level (venc = 10 cm/s), at the cervical level (*Venc* = 5 cm/s); and the blood flow at the cervical level (*Venc* = 80 cm/s)



Displacement of blood in cylinder can be visualized in terms of the pressure variations. The pressure in artery is maximal at the onset of systole (e.g. the blood flow velocity is least), and minimal during mid-systole (e.g. the velocity is greater). In fluid system having in parallel a resistance R, a capacitance C and an inductance L, which is pulsed by a driving pressure P, the general equation describing this model is P = Z.F where F is the flow rate  $[mm^3/s]$  and Z is the impedance (Ohm's law)  $Z^2 = R^2 + [L\omega - 1/(C\omega)]^2$ . The pressure exerted by pulsation of the artery is related to the flow rate  $(F_{art})$ . If the pulsatile blood flow rate is given by:  $F_{art} = K + F_{AB} \cos(\omega t)$  (K=constant,  $F_{AB}$  flow amplitude) then the driven pressure exerted on the CSF is  $P = P_0 \cos(\omega t-\varphi_\beta)$  and the consequent CSF flow is  $F_{CSF} = F_{CSFB} \cos(\omega t-\varphi_\beta-\varphi_1)$ . If the observed  $F_{CSF}$  and  $F_{art}$  are synchronous,  $\varphi_1=-\varphi_\beta$ , the system will be in resonance and  $\omega^2 = 1/(LC)$ .

**Results:** Flow curves in the radial artery, model aqueducts of different sizes and the model spinal canal were generated using flow analysis software (Advantage Windows, GE Healthcare). These were then compared to the curves generated on the intracranial carotid artery, aqueduct and foramen magnum. The flow curve patterns from the model were similar to those in vivo. Principle harmonics generated with Fast Fourier Analysis showed a phase lag of  $44^{\circ}$  in the phantom aqueduct and 8° at phantom cranio-cervical junction (CCJ) in relation to the phase of the arterial pulse. This corresponds to those reported in vivo. The observed phase difference between the arterial flow and the CSF flow at the level of the aqueduct is (-50°) and the foramen magnum is (+5°). The phase relationship of the aqueduct to the arterial flow was dependent on the cross section area of the model aqueduct and hence its natural frequency. With increasing cross-section area and therefore increasing natural frequency the phase lag from the arterial flow decreased



**Figure 2:** Pressure curves of the artery (A) and Foramen Magnum (B) and flow curves at the level of the aqueduct using two different diameters: (C)  $\emptyset = 3$  mm, and (D)  $\emptyset = 2$  mm, the phase difference between the artery and the aqueduct was  $\varphi = -48^\circ$ , and between the artery and the CCJ  $\varphi = -10^\circ$ .

**Conclusion**: Our study confirms the CSF-cerebrovascular relationships to be compatible with the inductive, capacitive and reactive circuits but fails to support existence of resonance between the cerebrovascular and CSF systems as has been proposed by Egnor et at.

Reference: [2] Baledent et al., JMRI 24(2006); [2] Bradley et al., JMRI 24 (2006); [3] Egnor et al., Ped Neuros 35(2001).