Cerebrospinal Fluid Research

Oral presentation

Pressure phase relationships between carotid arterial pressure and intracranial pressure: the 'violin' analogy of intracranial pulsations

Michael Egnor*[1], Mark Wagshul[2], Erin McCormack[2], Pat McAllister[3], Joe Madsen[4], Rui Zou[4], Lili Zhen[5] and Jeff Peng[5]

Address: [1]Department of Neurosurgery, State University of New York at Stony Brook, Health Science Center, T-12 Room 080, Stony Brook, New York 11794-8122, USA, [2]Department of Radiology, State University of New York at Stony Brook, Health Science Center, Level 4, Room 109, Stony Brook, New York 11794-8460, USA, [3]Department of Neurosurgery, Wayne State University School of Medicine, 4201 St. Antoine, Suite UHC-6E, Detroit, Michigan 48201, USA, [4]Department of Neurosurgery, Harvard University, Boston, Massachusetts 02115, USA and [5]Department Mechanical Engineering, State University of New York at Stony Brook, Stony Brook, New York, USA

Email: Michael Egnor* - megnor@notes.cc.sunysb.edu

* Corresponding author

from 50th Annual Meeting of the Society for Research into Hydrocephalus and Spina Bifida
Cambridge, UK. 30 August – 2 September 2006

Published: 21 December 2006

Cerebrospinal Fluid Research 2006, **3**(Suppl 1):S14 doi:10.1186/1743-8454-3-S1-S14

Background

Many investigators have shown marked abnormalities of CSF and parenchymal pulse pressure in hydrocephalus, and Greitz, Bateman, and Egnor have proposed that abnormalities of pulsatility are at the root of the pathophysiology of hydrocephalus. Our work is aimed at a more rigorous understanding of pulsatility in the cranium. We have studied the phase relations between the carotid arterial pressure pulse and the intracranial pressure (ICP) pulse in dogs as a function of mean intracranial pressure.

Materials and methods

Carotid arterial pressure and intracranial pressure in 12 dogs were measured with progressive withdrawal and infusion of CSF to change intracranial pressure and compliance. The waveforms were recorded and synchronized in time. Phase between the carotid and ICP pulse was determined using the phase difference between the fundamental harmonics of the Fast Fourier Transform.

Results

The normal ICP pulse *led* the carotid arterial pulse by an about 50 degrees. With lowering of ICP, there was an increase in the lead of the ICP pulse with respect to the carotid pulse to about 100 degrees. Increasing ICP caused a progressive phase lag of ICP pulse with respect to carotid pulse, up to a maximum lag between arterial and intracranial pulse of approximately 30 degrees.

Conclusion

These data clearly show that there are conditions in which the ICP pulse *precedes* the carotid pulse. With raising and lowering of the mean ICP, the phase versus mean ICP curve is sigmoid-shaped, with the normal ICP phase is the inflection point of the sigmoid curve. The observation that the ICP pulse of the dog normally precedes the carotid arterial pulse is counterintuitive. It forces us to rethink our current understanding of the way in which the ICP pulse is generated. We propose that the ICP pulse is a standing wave, not a transmitted wave. Standing waves can be generated in chambers with elasticity, such as in the body of a violin. The vibrations from the violin strings excite the vibrations in the body of a violin, but the vibrations in the body of the violin can lead or lag the string vibrations. The concept that the ICP wave is a standing wave is important, because it suggests that the cranium can suppress (or accentuate) certain frequencies of arterial pulse entering it. This may shed new light on cerebral blood flow, the cerebral windkessel mechanism, and on the pathogenesis of hydrocephalus.