Letter to the Editor

Critique for: Di Rocco, C; Di Trapani, G.; Pettorossi, V.E., and Caldarelli, M.: On the Pathology of Experimental Hydrocephalus Induced by Artificial Increase in Endoventricular CSF Pulse Pressure.

Dear Sirs,

Usually mean intracranial pressure and pulse amplitude vary directly with each other. The authors have designed an experiment which separates mean from amplitude. Their pathological findings would indicate that pulse amplitude rather than mean intracranial pressure is responsible for the increasing ventricular volume and the periventricular white matter destruction.

Chronically elevated intracranial pressure dissipated evenly throughout the intracranial compartment does not cause ventricular dilation inspite of a large pulse amplitude. The major events in this experience are the origin of the pulse within the ventricular system and the simulated lack of damping of pulse amplitude. Neurosurgeons have long been aware of the reverse situation in which a cerebrospinal fluid shunt system within one lateral ventricle, effectively damps the pulse within that ventricle resulting in that ventricle being smaller than the other lateral ventricle.

The mean intracranial pressure reflects the static state of the intracranial compensatory mechanisms. This fluctuates over a wide range of pressures reflecting position changes, peripheral venous pressures, central nervous system metabolic activity, and neural auto-regulation. Over a wide range of mean pressures, the pulse pressure generated by that portion of the cardiac output which gain entry into the intracranial compartment is small. Amplitude damping is the more dynamic portion of the compensatory mechanisms.

The fact that amplitude and mean pressure vary directly, but can be separated, would indicate the major compensatory mechanisms share both a rapid and slow compartment. Cerebrospinal fluid absorption, distention of the spinal subarachnoid space, and collapse of veins and sinuses respond rapidly, damping the cardiac pulse. Larger reductions in CSF volumes, decreases in extracellular volume, and vasoconstriction are rapid but not rapid enough to damp the cardiac pulse. Cell volume loss is the slowest and least acceptable compensatory mechanism.

Thus, ventricular dilation should occur if the ventricular pulse generated by the choroid plexus, is larger than the parenchymal pulse and rapid compensatory damping is impaired. The loss of elasticity of cerebral vessels from hypertension and/or aging may result in the transmission of the cardiac pulse more to the choroid plexus than cerebral tissues. This coupled with partial obstruction of the CSF pathways by fibrosis from sub-arachnoid hemorrhage could lead to normal mean CSF pressure but with a large pulse amplitude leading to ventricular enlargement.

The authors should be congratulated on this finding and encouraged to continue the study of this model to further illustrate the mechanisms involved in ventricular dilation at normal mean pressures.

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