Pulsatility

It has been recognized for quite some time that pressure and flow pulsatility can change with disease; this has been used as a diagnostic tool in a number of areas. These changes are mostly due to the dependence of volume change on mean pressure, as first described by Marmarou et al for brain tissue ¹⁾.

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Most clinical applications of pulsatility have been outside of the brain, and the cranium presents a unique challenge for measuring pulsatility as well as a unique biomechanical environment for pulsatility. The predominant theory of non-steady blood flow in the human body is the Windkessel model, in which the elastic arterial walls serve as a storage mechanism for flow pulsatility, transforming pulsatile arterial blood flow into steady peripheral flow ²⁾.

The brain is enclosed in a rigid container, and any transfer of pulsatility from the arterial walls into the surrounding tissue is felt almost instantaneously everywhere throughout the cranium. This leads to the observation that intraparenchymal and CSF pressure waveforms tend to be similar and independent of location. This is sometimes over generalized to suggest that pressures are everywhere equal intracranially, but this obviously does not apply to the very important arterial and venous compartments. Secondly, this leads to the interesting and potentially important phenomenon of measurable flow pulsatility in the microvasculature ³⁾ and in the venous system. In the brain, the substitute for tissue compliance, which dissipates arterial pulsations in non-cranial tissues, is the overall intracranial compliance. This compliance, is comprised of four main components: actual brain tissue compliance (which is small), arterial compliance, venous compliance (veins have highly compliant walls) and compliance of the spinal thecal sac (which communicates with the brain via the cerebrospinal fluid spaces). Traditionally, intracranial compliance is assumed to decrease primarily with increased ICP, due to the exponential pressure-volume relationship ⁴⁾.

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