The Cerebral Windkessel
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Introduction
The cranial contents are enclosed in a rigid vault. The normal windkessel mechanism by which capillary beds are protected from the arterial pulse by dissipation of pulsatility into the surrounding soft tissues requires modification in the cranium. The modification is a system of reflection and venting, by which the arterial pulse is coupled via the CSF to the intracranial veins and is thus reflected back to the right heart. This reflection and venting is the windkessel mechanism in the cranium. Optimal venting of the arterial pulse in the cranium depends on a precise coupling of the arterial pulse to the ICP pulse.

Methods
Over the past two decades, our team and other investigators have explored the phase, pressure and flow relationships associated with intracranial pulsatility and the cerebral windkessel using laboratory animals and flow sensitive MRI studies on animals and humans.

We studied the intracranial dynamics of 12 dogs using intracranial pressure monitoring, carotid artery pressure monitoring and transfer function analysis of the arterial pressure (input) and ICP (output), under varying conditions of intracranial pressure and compliance accomplished by injection and removal of CSF from the lumbar subarachnoid space.

Results
We identified:

1) With normal ICP and normal intracranial compliance, a notch filter was present in the frequency domain at the heart rate, which is a hallmark of a tuned pulsation absorber accomplished by high impedance resonance in the cranium.

2) With low intracranial pressure and high compliance, there was a progressive lead of the ICP pulse with respect to the arterial pulse. This is consistent with the behavior of a notch filter as predicted by the windkessel model.

3) With high intracranial pressure and low compliance, there was a progressive lag of the ICP pulse with respect to the arterial pulse. This is also consistent with the windkessel model.

4) With high intracranial pressure and low compliance, there was attenuation of the windkessel notch at the frequency of the heart rate. This represents impairment of the windkessel effect in the cranium caused by low intracranial compliance and excessive elastic reactance with loss of high impedance resonance.

Conclusion
The windkessel is the ability of the vasculature to dissipate arterial pulsatility and provide smooth capillary blood flow. Our investigations suggest that the windkessel is enabled in the cranium by a system of pulsation absorption. Using transfer function analysis, normal intracranial dynamics appears to be associated with a windkessel notch at the frequency of the heart rate. Abnormal intracranial dynamics, for example increased intracranial pressure, is associated with ablation of the notch and shifting of the phase relationships between the arterial pulse and the ICP pulse. The pulsation absorber model of the cerebral windkessel provides insight into pulsatile intracranial dynamics and suggests potential novel approaches to treatment of disorders of intracranial dynamics.

Learning Objectives
By the conclusion of this session, participants should be able to describe the nature and importance of the cerebral windkessel 2) discuss, in small groups, the evidence supporting the pulsation absorber model of the cerebral windkessel.

References