Increased pressure in the dural venous sinuses has been proposed as the cause of increased intracranial pressure in the condition known as idiopathic intracranial hypertension (IIH). This hypothesis has received further support from manometry of the dural venous sinuses, showing a substantial proximal-to-distal pressure gradient, and from reports of improvement of IIH following stenting of the dural sinuses. Increased intracranial venous pressure has also been proposed as the cause of IIH in morbid obesity through increased abdominal pressure that is transmitted through the thorax to the cerebral draining veins. Although these hypotheses are intriguing, neither has enough scientific support to be endorsed yet. Moreover, dural venous sinus stenting should not be adopted as a therapeutic procedure in IIH until larger clinical trials attest to its safety and efficacy [1].

Traumatic brain injury and stroke are both characterized by an ischemic core surrounded by a penumbra of low to hyperemic flows. The underperfused ischemic core is the focus of edema development, but the source of the edema fluid is not known. We hypothesized that flow of edema fluid into the tissue is derived from cerebral venous circulation pressure, which always exceeds intracranial pressure (ICP). As a first step toward testing this hypothesis, the aim of the current study was to determine whether cerebral venous pressure in the normal brain is always equal to or higher than ICP. In studies on 2 pigs, cerebral cortical venous, intracranial (subarachnoid), sagittal sinus, and central venous pressures were monitored with manipulation of ICP by raising and lowering a reservoir above and below the external auditory meatus zero point. The results show that cerebral venous pressure is always higher than or equal to ICP at pressures of up to 60 mmHg. On the basis of these observations, we hypothesize that increased cerebral venous pressure initiated after traumatic brain injury and stroke drives edema fluid into the tissue, which thereby increases ICP and a further increase in cerebral venous pressure in a vicious cycle of brain edema [2].

1)
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