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# Is intracranial pressure monitoring always accurate? A case report

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## 1. Introduction

The theoretical "Kellie-Monroe" model, which is a closed system model, has allowed physicians and scientists to understand many of the underlying mechanisms of ICP regulation in physiologic and pathophysiologic conditions [1]. However, how to accurately monitor ICP has been subject of debate for the last few decades [2]. The closed system model focuses solely on the pressure gradients between left and right hemispheres, or supra and infra-tentorial compartments [1]. However, within the intracranial compartment there are multiple gradients that interact through a complex mechanism that cannot be studied accurately using a model that considers ICP dynamics alone. Schaller et al. propose different patterns of ICP values to better understand the varying physiologic ICP signals and better quantify the inter-compartmental ICP differences [1]. These patterns consist of 1) transient gradients between compartments, 2) true bicompartmental gradients, 3) different compliance, and 4) uni-compartmental space [1]. In this paper the authors present a case in which ICP readings using EVD did not match the clinical examination and imaging studies.

### 1.1. Case report

A 45-year-old right-handed female was admitted with a history of severe, sudden headaches, confusion, and meningismus. Neurological examination showed right sided drift and facial droop. The blood pressure was elevated, and she was confused, but able to follow commands. A non-contrast CT scan of the head showed massive subarachnoid hemorrhage with blood clots in the left frontal and temporal lobes (Fig. 1). The patient was intubated, and an EVD was placed in the right frontal horn. The ICP recording was in the high 20's mm Hg. With mannitol and ventricular drainage, the ICP was reduced to high teen's mm Hg.

An angiogram showed 4 mm saccular aneurysm in the left middle cerebral artery, which was coiled. The patient was started on nimodipine; and systolic blood pressure was maintained at approximately 145 mm Hg. The next day, the patient became more cooperative, and therefore was extubated. She moved all her extremities, left greater than right, and the ICP stayed in the mid-to-high teens. Eight days after admission, she started to worsen and the ICP rose to 30 mm Hg. Mannitol was given along with ventricular drainage to lower the ICP. The following day, she was awake and her ICP was measured at 20 mm Hg. On hospital day 10, she worsened clinically and had to be intubated. A non-contrast CT scans showed increase mass effect (Fig. 2); however, the ICP remained around 14 mm Hg. Over the next 4 days, she continued to worsen despite the ICP hovering around 10 mm Hg. Intra-arterial verapamil was injected twice with the hope reducing the vasospasm that had set in. On hospital day 14, the patient expired.

## 2. Discussion

In this report we document a case in which the declining neurological status of the patient did not correlate with ICP readings; although EVD, which is the gold standard for ICP recording was used (3). Despite CT scans showing mass effect with midline shift and worsening of the neurological status, ICP remained within normal limits. This suggests, that in this case, the ICP readings did not reflect the true intracranial pressure.

Our patient's presentation and recent research findings suggest that

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**Fig. 1.** CT scan on the day of admission shows subarachnoid bleeding centered around left Sylvian fissure with intra-cerebral bleed in the frontal and temporal lobes.

**Fig. 2.** CT scan a day prior to death shows intracranial bleed in the left hemisphere with massive midline shift. The tip of the ventricular catheter is seen at the bottom of the right frontal horn.

intracranial pressure may in fact exist as a much more complex and delicate multisystem environment that allows for the formation of ICP gradients. As mentioned previously, Schaller and Graf present an anatomical explanation for observed ICP gradients whereby the cerebral falx and tentorium, in addition to other anatomical structures, subdivide the cranial vault into at least five smaller systems that could experience pressure effects without affecting the other divisions [1]. Various animal studies have further added to the burden of proof that ICP gradients can exist. In Ganz et al.'s epidural bleeding experiment in swine pressure gradient was recorded between the lateral ventricle and basal cistern [3].

Mass effect created by subarachnoid and intracerebral hemorrhages, and the surrounding edema in our patient did displace the surrounding tissue and adjacent structures to create a midline shift of the brain to the right. This should ordinarily cause increase in ICP readings. This did not happen in the last several days prior to her death when her neurological status was progressively worsening.

We have several hypotheses to explain the observed outcome in our patient. Firstly, it is possible that the ventricular wall in this patient was tougher than usual, which would reduce the effect of external pressure; though, early recording did show elevated ICP. The other possibility, is, that, the collapse of the right ventricle in which the ventricular catheter was placed, could have reduced the ICP readings. However, this is less likely; because, CSF drainage through the catheter was unaffected at all time. Another possibility is that, since the catheter was in the right ventricle and an inter-hemispheric pressure gradient existed, the ICP reading was low. Yet, another possibility is that, the falx and/or the solid clots may have acted as “damping points” for pressure transmission; resulting in low ICP readings.

## 3. Conclusion

In conclusion, we cannot rely on ICP monitoring entirely to assess progression of ICP. Therefore, clinical examination in conjunction with imaging studies remain the gold standard in the management of intracranial hypertension.

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