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Cerebrospinal fluid osmolality

Because the specific gravity of serum and CSF is known to be 1.024–1.028 and 1.004–1.007, respectively, the estimated average of osmolarity (mOsm/L) in the serum and CSF covered exactly the same range (i.e., 290.5–291.5 mOsm/L).

Interstitial fluid movement in the brain parenchyma has been suggested to contribute to sustaining the metabolism in the brain parenchyma and maintaining the function of neurons and glial cells. The pulsatile hydrostatic pressure gradient may be one of the driving forces of this bulk flow. However, osmotic pressure-related factors have not been studied until now. In this prospective observational study, Akaishi et al. wanted to elucidate the relationship between osmolality (mOsm/kg) in the serum and that in the cerebrospinal fluid (CSF), they simultaneously measured the serum and CSF osmolality of 179 subjects with suspected neurological conditions. Serum osmolality was 283.6 \pm 6.5 mOsm/kg and CSF osmolality was 289.5 \pm 6.6 mOsm/kg. Because the specific gravity of serum and CSF is known to be 1.024-1.028 and 1.004-1.007, respectively, the estimated average of osmolarity (mOsm/L) in the serum and CSF covered exactly the same range (i.e., 290.5-291.5 mOsm/L). There was a strong correlation between CSF osmolality and serum osmolality, but the difference in osmolality between serum and CSF was not correlated with serum osmolality, serum electrolyte levels, and protein levels, or quotient of albumin. CSF osmolarity was suggested to be equal to serum osmolarity. Osmolarity is not one of the driving forces of this bulk flow. Other factors such as hydrostatic pressure gradient should be used to explain the mechanism of bulk flow in the brain parenchyma. This study was approved by the Institutional Review Board of the Tohoku University Hospital (approval No. IRB No. 2015-1-257) on July 29, 2015 1.

The idiopathic normal pressure hydrocephalus etiology is currently unknown. With no visible obstructions, altered cerebrospinal fluid dynamics may explain the accumulation of ventricular fluid. Oernbo et al. hypothesized that elevated osmolality in the Cerebrospinal fluid of iNPH patients could potentiate the formation of ventricular fluid and thereby cause the disease progression and/or predict the surgical outcome. To address this hypothesis, they determined the lumbar and ventricular CSF osmolality of iNPH patients at different disease stages and compared it with lumbar CSF samples obtained from control subjects.

The osmolality of CSF was determined on a total of 35 patients at diagnosis and at the subsequent treatment with shunt surgery (n = 20) and compared with the CSF osmolality from 20 control subjects. Simultaneously collected lumbar and ventricular CSF samples from experimental pigs were used to evaluate the compatibility between CSF from different compartments.

They found no evidence of increased osmolality in the CSF of iNPH patients upon diagnosis or at the time of shunt treatment months after the diagnosis, compared with control individuals. CSF tapped from the lumbar space could be used as a read-out for ventricular CSF osmolality, as these were similar in both the patient group and in experimental pigs. We further observed no correlation between the CSF osmolality in iNPH patients and their responsiveness to shunt surgeries.

The osmolality of lumbar CSF is a reliable reflection of the ventricular CSF osmolality and is not elevated in idiopathic normal pressure hydrocephalus patients. iNPH, therefore, does not appear to arise as a function of osmotic imbalances in the CSF system and CSF osmolality cannot serve as a idiopathic normal pressure hydrocephalus biomarker or as a predictive tool for shunt responsiveness ²⁾.

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