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Cardiac mediated cerebral blood flow changes in chronic hydrocephalus

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Background

Increased intracranial pressure (ICP), vascular compression as the result of enlarged cerebrospinal fluid (CSF) spaces, or impaired metabolic activity may be responsible for decreased cerebral blood flow (CBF) seen in hydrocephalus. Little attention has been given to the relationship between cardiac function and systemic blood flow in chronic hydrocephalus (CH).

Materials and methods

Using an experimental model of chronic obstructive hydrocephalus developed in our laboratory, we investigated the relationship between hydrocephalus duration and severity and cardiac output (CO), cerebral blood flow (CBF), myocardial tissue perfusion (MTP), and peripheral blood flow (PBF). Blood flow measures were obtained via microsphere injection method under controlled hemodynamic conditions in experimental CH (n = 23) and surgical control (n = 8) canines at baseline and 2, 4, 8, 12, and 16 weeks. CO measures were made using the Swan-Ganz thermodilution method. Intracranial compliance (ICC) via CSF bolus removal and infusion, and oxygen delivery in CSF and prefrontal cortex (PFC) were also investigated.

Results

We observed an initial surgical effect relating to 30% CO reduction and ~50% decrease in CBF, MTP, and PBF in both groups 2 weeks post-operatively that recovered in control animals but continued to decline further in CH animals at 16 weeks. CBF, which was positively correlated with CO ($p = 0.028$), showed no significant relationship with either CSF volume or pressure. Decreased CBF correlated with oxygen deprivation in PFC ($p = 0.006$). CO was inversely related with ventriculomegaly ($p = 0.019$), but did not correlate with ICP. Decreased CO corresponded to increased ICC as measured via CSF infusion ($p = 0.04$).

Conclusion

Our results suggest that CH may have more of an influence on CO and CBF in the chronic stage than in the early condition, which was dominated by surgical effect. The cause of this late deterioration of cardiac function in hydrocephalus is uncertain, but may reflect cardiac regulation secondary to physiological response or brain injury. The relationship between cardiac function and cerebral blood flow should be considered in the pathophysiology and clinical treatment of chronic hydrocephalus.