

## D. Circuration and Metabolism of Cerebrospinal Fluid

### D-1. Cerebral Water and Electrolytes—Effect of Ischemia

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When local cerebral ischemia was produced in dogs by clipping the middle cerebral artery, water, sodium and potassium content remained within normal limits in the affected tissue for 24 hrs. Forty-eight hours after clipping of the artery, focal areas of necrosis, hemorrhage and staining by systemically injected Evans blue were seen in the anterior portion of the caudate nucleus, the internal capsule and occasionally in the overlying temporal-parietal cortex. Large areas of cortex, apparently ischemic on the basis of fluorescein angiography, were normal. In the tissue surrounding the infarctions changes in water, sodium and potassium characteristic of vasogenic edema were demonstrated. Subsequent experiments showed that clipping of the middle cerebral artery in the dog produces only relative ischemia. No blood flow could be demonstrated in the affected area with fluorescein angiography when the dye was injected through the lingual artery. However, injection of the fluorescein into the femoral vein demonstrated considerable collateral blood supply. Further, perfusion with carbon black resulted in uniform filling of vessels throughout the brain, including the areas normally supplied by the clipped artery.

In an attempt to induce more complete ischemia, following clipping of the middle cerebral artery dogs were subjected to hemorrhagic hypotension with blood pressure maintained at 50 mmHg for 60 min. Under these conditions fluorescein angiography by both routes revealed greatly delayed maximum-filling time but no alteration in the distribution of the fluorescein in the affected areas. Chemical analysis showed normal water and electrolytes at the end of the hypotensive period and the distribution of perfused carbon particles were normal. However, with restoration of the blood pressure, by reinjection of blood, dramatic changes occurred. A large area of abnormal and extremely delayed filling roughly corresponding to that supplied by the clipped artery became evident on angiography. The same area was clearly delineated as non-filling with carbon perfusion. The cortex showed progressive changes starting immediately after restoration of the blood pressure. These consisted of decrease in dry weight and in potassium and an increase in sodium, the latter approaching plasma levels within 24 hrs. The fall in potassium represented a net loss from the normal level of 511 meq/100 mgms dry weight at the end of the period of hypotension to 300 after 5 hrs of restoration of blood pressure and 187 after 24 hrs. Massive necrosis was demonstrated by conventional histological techniques in the affected area within 24 hrs. The changes in water, sodium and potassium content of the cortex in