CEREBROVASCULAR RESPONSIVENESS IN BRAIN INJURY AND OEDEMA

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SUMMARY

Brain injuries, traumatic, ischaemic, hypoxic or inflammatory are a common cause of death or severe handicap. Clinical assessment and treatment may be hampered by difficulty in predicting and measuring the complex changes initiated by an injury and occurring within the enclosed cranium.

Clinicians seeking to increase perfusion and limit brain damage have advocated such methods as increasing arterial pressure or hypercapnia. However cerebral blood flow measurements in patients have shown that the responses of the cerebral vessels to these physiological stimuli may be quite variable. Several studies have reported constant cerebral blood flow with arterial hypertension in patients with severe brain damage, even though experimental studies have shown that physiological autoregulation may be impaired by quite minor injuries.

The present study analysed the changes in cerebral blood flow, intracranial pressure, brain tissue water and electrolyte content following an acute cryogenic injury in baboons. This model of a focal necrotic injury with oedema resembles a cerebral contusion or infarction, and the oedema is also similar to that which develops around inflammatory or neoplastic lesions. In the first series of experiments, cerebral blood flow and cerebral vascular reactivity were studied before and after cryogenic injury. Cerebral blood flow was measured by intracarotid ^{133}Xenon clearance. Following injury, arterial hypertension was accompanied by a marked increase in cerebrovascular resistance in half the animals studied. Comparison with animals which did not show this response clearly demonstrated that such an increase in cerebrovascular resistance was not physiological autoregulation, but rather indicated a severe degree of brain damage.

The second series of experiments studied changes occurring 24 hours after cryogenic injury. At this time the effects of the primary injury could be separated from those of the secondary oedema. Cerebral blood flow was measured in small areas in both cerebral hemispheres by hydrogen clearance. A methodological study established the accuracy of this technique by comparing it with the intracarotid ^{133}Xenon method.

In areas with oedema, resting cerebral blood flow was low and failed to increase with arterial hypertension, even though other parameters of cerebrovascular reactivity were impaired. In animals with injury but no oedema, cerebral blood flow increased in a pressure-passive manner indicating loss of autoregulation. Furthermore, focal injury with and without oedema produced widespread changes in resting cerebral blood flow and cerebrovascular reactivity in both hemispheres.

These experiments demonstrate that clinical studies of patients with brain damage which report intact autoregulation based on the cerebrovascular response to arterial hypertension must be interpreted with care. They also provide an explanation why apparently intact autoregulation may not correlate with the outcome from injury.

Increasing cerebrovascular resistance with arterial hypertension in areas of injury appeared to depend upon the presence of oedema. However, changes in cerebral blood flow and cerebrovascular responsiveness also occurred in grey and white matter throughout both hemispheres, and were not related to changes in tissue fluid. These widespread effects of a focal injury may underly the clinical phenomenon of diaschisis.

Clearly, the responses of the cerebral vasculature in areas of injury are quite variable. In severe injuries, particularly those associated with oedema, arterial hypertension and hypercapnia may not lead to increased tissue perfusion, but may indeed increase tissue damage.

Methods of reducing oedema and lowering cerebrovascular resistance, perhaps by reducing tissue pressure, are an essential part of any attempt to increase perfusion in areas of brain damage.

A clinical study of patients with brain lesions and oedema, measured blood flow by the inhalational ^{133}Xenon method in areas of reduced X-ray density recorded by computerized axial tomography (CAT scanning). This study showed the value of CAT scanning in detecting oedema and monitoring therapy. Cerebral blood flow was low in areas of verified oedema, and appeared to increase with steroid therapy.