Discussion: Head injuries

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In the course of the previous papers and the resulting discussion, several people have suggested that cerebral hypoxia or ischaemia might contribute to brain damage in patients with head injuries. Professor Jennett has stressed the ultimate importance of the cerebral perfusion pressure which is dependent upon the mean arterial blood pressure and the intracranial pressure. It would appear from his observations that the cerebral perfusion pressure in a patient with an acute head injury might well fall below the critical level necessary to maintain the vitality of brain tissue. This, therefore, should lead to ischaemic brain damage. As I have become increasingly aware of the occurrence of cerebral infarcts in fatal cases of head injury, Dr D. I. Graham of the Division of Neuropathology in the Institute, and I have commenced a detailed study of large brain sections from an unselected series of fatal head injuries in an effort to establish, as part of the project, the frequency and the distribution of ischaemic and hypoxic brain damage. This investigation is still in its preliminary stages but it has already become apparent (1) that massive cerebral infarction is not uncommon in such cases and (2) that even when the brain has been adequately fixed before dissection, these infarcts may defy recognition with the naked eye.

I would like to illustrate this point by referring briefly to two representative cases. The first was a 15-year old boy who died eight days after a head injury. The intracranial pressure had been persistently elevated from the time he was admitted to the Division of Neurosurgery until death. There was no fracture of the skull and there was only one small contusion on the surface of the brain: on section the brain was rather pale and swollen but there was no obvious infarction. The examination of large celloidin sections, however, demonstrated massive asymmetrical infarction in the cerebral hemispheres involving both the cortex and the deep grey matter. The extent of this can be clearly seen in Figures 1 and 2.

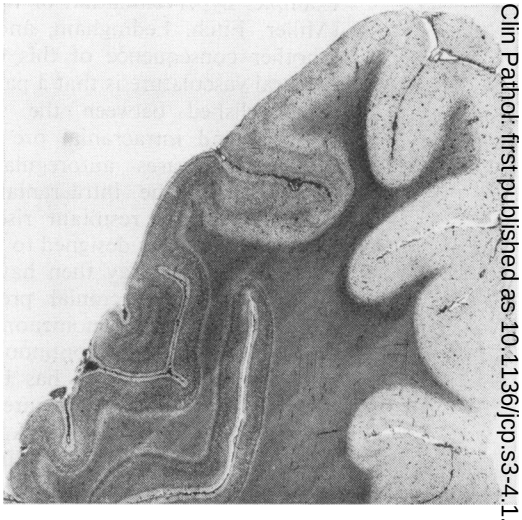

Fig. 1 *Frontal lobe: note the extensive necrosis (shown by pallor of staining) on the medial aspect of the hemisphere and at its superior angle. Normal cortex is seen in the lower part of the left lateral surface. Cresyl violet × 3.*

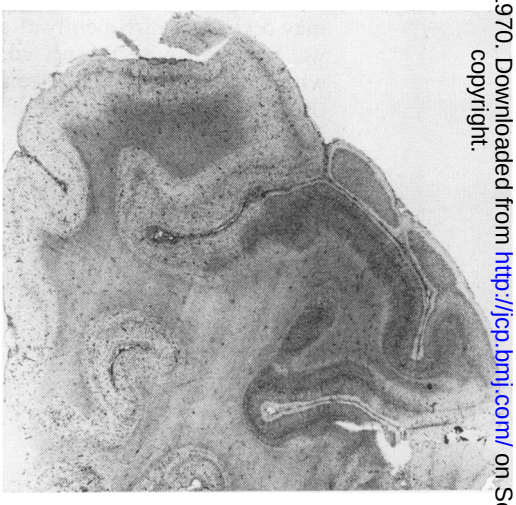

Fig. 2 *Occipital lobe: necrosis is of irregular distribution but normal cortex is seen in the middle third of the right lateral aspect of the hemisphere. Cresyl violet × 3.*

The second case was a 23-year old woman who died four days after a road traffic accident in which she had suffered a head injury and fractures of the tibia and fibula. The intracranial pressure is not known in this case. At necropsy there was a fracture of the skull and moderately severe conventional cerebral contusions. Although there was no naked eye evidence of ischaemic brain damage, microscopical examination demonstrated extensive necrosis of the cortex in one frontal lobe in the boundary zone between the anterior and middle cerebral arterial

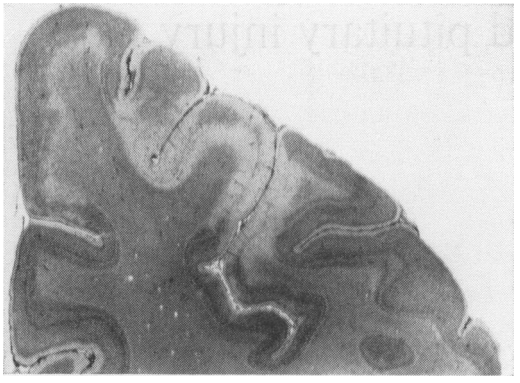

Fig. 3 *Frontal lobe: the pale zone of cortical necrosis immediately lateral to the superior angle of the hemisphere involves the boundary zone between the anterior and middle cerebral arterial territories. Cresyl violet* × *3.*

territories (Fig. 3). There was no histological evidence of fat embolism. Such boundary zone infarcts imply an abrupt and profound reduction in blood flow in the two adjacent arterial territories (Adams, Brierley, Connor, and Treip, 1966; Adams, 1967).

As there was no evidence of occlusive arterial disease in the neck or within the cranium, in either case, the ischaemic brain damage must be attributed to a reduced cerebral blood flow. These are very preliminary observations, and we hope to have much much more relevant information fairly soon, but they do suggest that reduced cerebral blood flow may be a more important factor contributing to brain damage in patients with acute head injuries than has hitherto been suspected.

References

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