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Commentary

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In the past two or three years a profound change has occurred in the understanding of intraventricular haemor-

rhage. It is now generally accepted that ischaemic periventricular lesions can be recognised in life and distinguished as separate from intraventricular haemorrhage, although they both may occur in the same brain. Rushton *et al* have provided further evidence for this and also stress that ischaemic lesions may occur well beyond the neonatal period in infants who develop surgical complications or severe seizures. They also confirm the poor prognosis as only two of their 10 infants surviving periventricular leucomalacia were normal at follow up.

The fundamental differences in aetiology between the popular notion of hyperperfusion leading to intraventricular haemorrhage and hypoperfusion causing periventricular leucomalacia makes it necessary to rethink our attitudes towards the prevention of the former. Uncomplicated subependymal haemorrhage may indeed prove to be a useful marker for those brains that have been well perfused and are at lower risk of subsequent neurodevelopmental handicap. Rushton and his colleagues have, however, extended their speculation well beyond accepted facts when they suggest that all parenchymal 'haemorrhage' in the preterm (and perhaps mature) brain is ischaemic in origin. The evidence for this is lacking but it is also true that evidence for direct parenchymal extension after intraventricular haemorrhage is equally poor. A mass of blood clot found at necropsy in the soft brain of a long dying infant makes elucidation of the precise pathophysiological process almost impossible. An ischaemic rim to the haemorrhagic lesion may be either primary or secondary. An *in vivo* study using positron emission tomography¹ showed extensive ischaemia in the hemisphere in which parenchymal haemorrhage had occurred but was not done early enough to determine the primary event.

How then can this fundamental question of ischaemia versus haemorrhage be answered? It is unlikely that either position emission tomography or nuclear magnetic resonance spectroscopy² can be used early or frequently enough to recognise the primary process. A portable and non-invasive technique is required to investigate cerebral haemodynamics prospectively from birth. At present Doppler ultrasound is the most promising tool. Modern combined real time and pulsed Doppler machines offer both safety and convenience. Further advances in our understanding of this important question may come from this direction.

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