Stroke Treatment

Sir, - The pathophysiology of acute stroke (your May 11 editorial) suggests a logical approach for pharmacological intervention. Although ischaemia was mentioned 36 times in your editorial and oxygen deprivation was identified as the fundamental cause of pathophysiological changes, oxygen was not mentioned as a potential therapy. Organic compounds do not enhance oxygen delivery; nor do they diffuse any significant distance into poorly perfused, oedematous tissue.

Hyperbaric oxygen (HBO) efficiently increases the diffusional driving force for oxygen, thereby increasing tissue oxygen availability. This overcomes ischaemia/hypoxia and so reduces cerebral oedema, restores integrity to the bloodibrain barrier and cell membranes, neutralises toxic amines, promotes phagocytosis, scavenges free radicals, stimulates angiogenesis, and reactivates idling neurons. Astrup, et al.' suggest that idling neurons are metabolicly lethargic and electrically non-functional - but remain viable in the ischaemic penumbra because of critical low tissue oxygen availability. By correcting oxygen deficiencies, idling neurons may be metabolically stimulated to regain electrical function.

Discussion of treatments must take into account that outcome in stroke depends on the site and extent of ischaemic penumbral zones. Outcome may be predicted, to some extent, by the volume affected. Computerised tomographic volume is one predictor: but comparative functional volume obtained by single photon emission tomography (SPET) often indicates a larger region of potentially recoverable oedematous and hypoxic tissue'. Pathophysiological considerations dictate use of HBO or, at least, isobaric oxygen. Chronic neurological deficits may benefit from HBO therapy'. Therapy should be directed at potentially reversible brain tissue. We question the limited 3-4 h interval for therapeutic intervention you cite since most patients are not assessed within that time frame. Transient ischaemic attacks may not be differentiated from cerebral thrombosis for at least 24 h. SPET and CT scans indicate positive evolutionary changes for months. That idling neurons are longlasting and found in all types of head injury is attested to by our studies on stroke and other brain injuries4 showing reversal of peri-infarctional/peri-injury zones even after 15 years.' Roski's restoration of vision, years after its loss, by extracranial-intracranial anastomosis5 and spontaneous arousal from long-term coma can also be explained by reactivation of idling neurons, rather than by brain regeneration.

We suggest that stroke and other brain injuries share a common pathophysiology: therefore, management should include evaluation by initial and delayed SPET (or PET) hyperbaric imaging. Identification of potentially recoverable brain tissue warrants every effort at restoring it.

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