

Enhancing "idling" neurons

SIR, Stroke, a global insult with focal neurological manifestations, in most cases clinically stabilises quite quickly. Evolutionary changes, local and global, have been observed with computerised tomographic scanning for up to three months. Newer tracers, measuring metabolic activity of grey-matter, have demonstrated changing tracer activity for up to six months.¹ Astrup and Symon have postulated a peri-infarctional zone of "idling neurons", which is viable but non-functional (2). Since idling neurons are not electrically active, measurement of this cellular component cannot be recorded. It is recovery of peri-infarctional zones that makes the initial prognosis in stroke difficult. In 1978 Roski reported recovery of vision after carotid bypass surgery nine years after stroke onset, suggesting long-term viability of a zone of idling neurons.³ Neubauer's report of recovery from coma after hyperbaric oxygen (HBO) also suggests a return of function of dormant cells.⁴ Using N-isopropyl-[¹²⁵I]-p-iodoamphetamine tracer with single photon emission computerised tomography (SPECT) we have demonstrated, in a stroke patient, peri-infarctional zones lasting up to 14 years; enhanced activity occurred after exposure to HBO.

In a 60-year-old white woman who had a right middle cerebral artery infarction 14 years previously, a magnetic resonance imaging (MRI) brain scan (fig 1) revealed right parietal lobe infarction with large areas of cystic transformation within the right parietal cortex and periventricular white-matter which were isointense with lateral ventricle CSF. Around the cystic area was an abnormal high-signal intensity consistent with gliosis and/or oedema secondary to ischaemia. (Gliosis or non-viable neuronal tissue cannot be differentiated on MRI from areas of cytotoxic oedema in viable cells.) SPECT brain scans were repeated within a four hour time frame both before and after a one hour exposure to oxygen at 1-5 atmospheres absolute (fig 2). Pre-HBO scans revealed a large metabolic defect in the right cerebral hemisphere corresponding to the area of signal drop-out seen on MRI. After HBO there was a sharp increase in tracer uptake in areas showing hypometabolism on the pre-HBO study. Areas with absent tracer uptake, corresponding to encephalomalacia on the MRI scan, remained unchanged. Increased tracer uptake occurred in peri-infarctional regions surrounding the encephalomalacia.

The post-HBO improvement in tracer activity suggests that a significant proportion of the MRI high signal intensity represented long-standing cytotoxic oedema rather than gliosis. The patient was then treated with continued HBO (averaging 5 consecutive treatments every other month), supplemented with surface oxygen, for a total of 60 HBO treatments. Reduced spasticity, improved ambulation and speech, and cessation of drooling were noted.

We suggest that idling neurons (peri-infarctional zones) may remain viable for a long time and that return of neuronal function



Fig 1-T2 weighted MRI brain scan (TR=2 s) before hyperbaric oxygen treatment.



Fig 2-Axial SPECT brain scan (through same region as fig 1) before and after one hour of hyperbaric oxygen therapy.

Colour bar=total metabolic activity in 16 segments from white (greatest) through red to blue (least). Small sagittal images at top right of each scan indicate relative position of axial view.

may be clinically significant. The interpretation of delayed imaging data with the SPECT tracer we used or possible enhancement with oxygen is difficult. Many variables must be considered besides grey-matter metabolism—ie, effects on the lungs, bone storage, changes in pH, reperfusion/redistribution phenomena, and leakage. However, control studies with surface oxygen did not reveal the same degree of uptake. Increased uptake suggests that hypometabolic peri-infarct tissue is viable. If this interpretation of neuronal reactivation is correct diagnostic and therapeutic measures in CNS dysfunction would have to be re-evaluated.

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Fig. 1 reversed