Hyperbaric Oxygen for Treatment of Closed Head Injury

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ABSTRACT: Traumatic and vascular brain injuries consist of acute episodes followed by development of chronic components of varying magnitude and duration whose potentials for recovery differ. We discuss a case of closed head injury in which interventional hyperbaric oxygen (HBO) with single photon emission computed tomography were used as aids in determining the presence of recoverable neurons, to follow therapeutic progress, and to determine the end point of therapy. This case also shows the successful use of intensive HBO as a therapeutic modality.

BRAIN INJURIES, **regardless of their cause, share common pathophysiologic pathways' that result in the destruction of neurons, and, to a varying extent, formation of idling neurons.**[2-5] **Diagnosis, prognosis, and treatment of central nervous system dysfunction requires the ability to differentiate between viable idling neurons and dead ones. To provide effective therapy for brain injury, the clinician must consider the locations and extent of irreparably damaged cells as well as ischemic penumbral zones.**[2,3,6] **This case study suggests that single photon emission computed tomography (SPELT) imaging, used in conjunction with interventional hyperbaric oxygen (HBO) therapy, is useful, not only in identifying potentially recoverable brain tissue, eg, idling neurons, in cases of stroke' and hypoxic encephalopathy,**[3] **but also in treating cases of closed head injury. It may be used to monitor the effectiveness of therapy and to determine the end point of therapy.**

METHODS

Initial and delayed SPELT brain imaging was done using N-isopropyl-[^{113}1]-p-iodoamphetamine (iofetamine hydrochloride 1123, IMP [SPECTamine, Medi-Physics Inc, Paramus, NJ]) and the techniques outlined by Neubauer et al.[2,3] **IMP imaging was done using an ADAC ARC 3300 computer interfaced to a Phillips rotating gamma camera. Unlike scans in previous studies**[2,3] **the final scan in this case was done using technetium Tc 99m dl-hexamethylpropyleneamine oxime (exametazime, dl HM-PAO [Ceretec, Amersham Corp, Amersham, England]) complex and a Picker SX 300 SPELT camera. Tracer change was unavoidable because the supplier stopped manufacturing IMP (last commercially available IMP source, April 4, 1991). Technetium scans were done using a split dose protocol; half the dose was given for the initial scans, and half was given after an hour of exposure to HBO (4 to 6 hours after the first injection).**

CASE REPORT

In December 1989 a 40-year-old white man was involved in a single-car accident. He was unconscious at the scene and was improperly intubated. He was subsequently transferred to a hospital, where he was reintubated. Computed tomography (CT) revealed fracture of the zygomatic bone; it also showed air in the intercranial region. After 2 weeks in the intensive care unit, the patient was transferred to a coma management program. At that time, he did not respond to commands or open his eyes spontaneously. The arms moved randomly, and there was marked spasticity of all extremities. He remained comatose for 28 days, eventually advancing from level 2 to level 7 on the Los Ranchos Amigos Coma Scale. When discharged in June 1990, he required total life support and care despite the fact that he had received intensive physical and cognitive rehabilitation with neuropsychologic and nutritional support. His wife was told that because of severe motor, memory, speech, language, and cognitive impairments, no further improvement could be expected and was advised to place him in a nursing home.

In June 1990, he was brought to us for further rehabilitative efforts using HBO. Detailed independent neuropsychologic and physical examinations confirmed the disabilities while providing objective baseline data.

SPELT-IMP scans (Fig 1) revealed a marked defect of the right posterior temporoparietal cortex and a diffusely diminished cerebral cortex. After a single 1-hour exposure to 1.5 atmospheres absolute (ATA) oxygen, repeat injection of tracer agent, and repeat scan, there was filling of the right defect and increased tracer uptake in the left parieto-occipital cortex (Fig 2).

Based on these data, an intensive course of HBO therapy in a Vickers monoplace chamber was instituted as follows: 1.5

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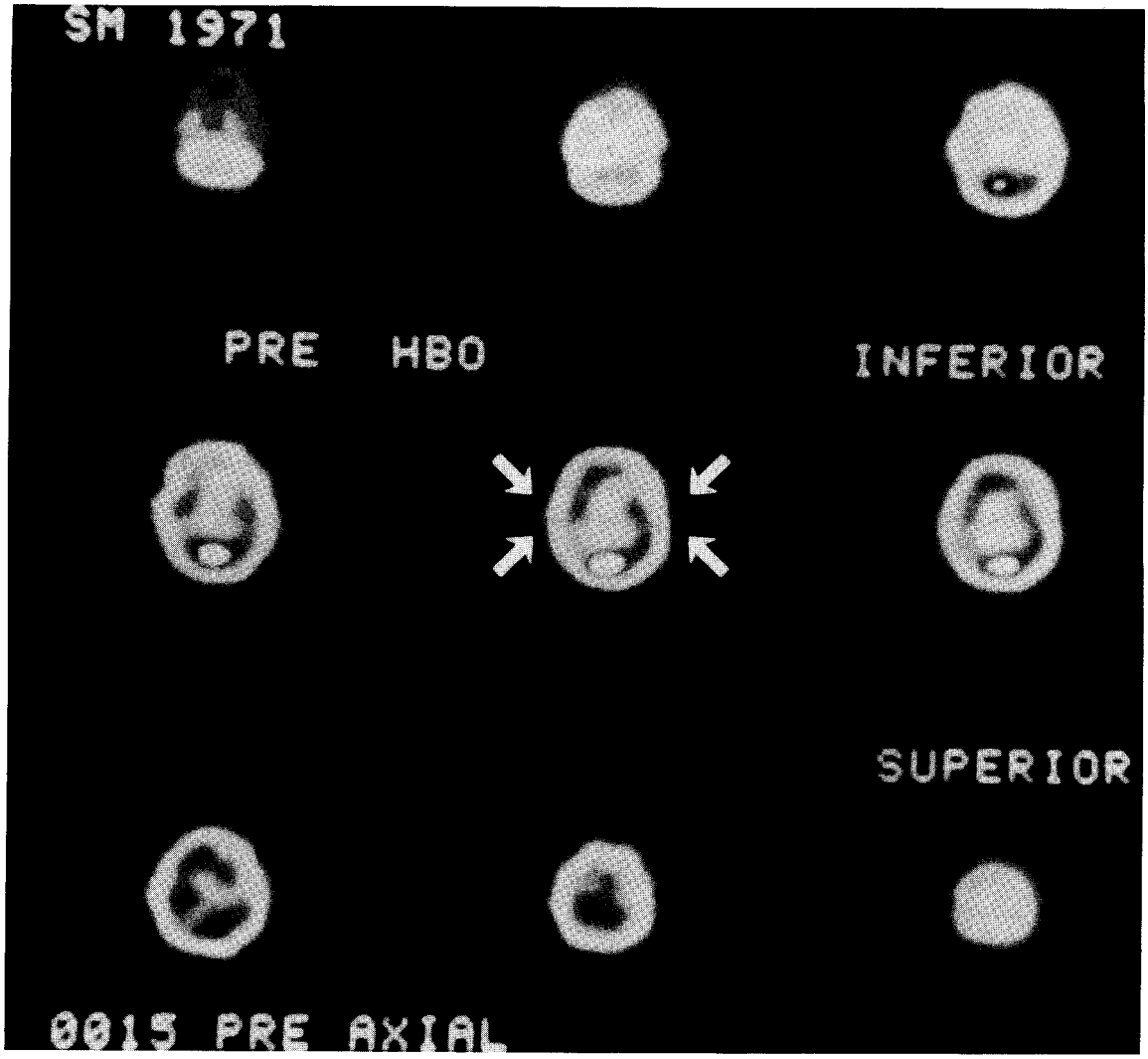

FIGURE 1. Initial SPECT/IMP scan, axial view (one atmosphere pressure). Note perfusion/metabolism defect.

ATA for 1 hour twice a day for the first 28 treatments, 1.75 ATA for the next 106 treatments, and 1.5 ATA for the final 54 treatments, for a total of 188 treatments. After the 93rd treatment, the patient showed good motor, cognitive, linguistic, and speech improvement as documented by video taping, brain jogging computer using the Parrot software program and neuropsychologic examinations. Images depicting SPECT/IMP scans 24 hours after the 160th treatment (Fig 3) indicated frontal and left parietal deficits but no longer showed the large right posterior parietal defect. Images made after the 161st treatment (Fig 3) showed further improvement in tracer uptake, suggesting the continued presence of potentially recoverable tissue and thereby providing scientific justification for further HBO therapy.

The conclusion of all therapeutic departments was that HBO therapy and rehabilitative intervention resulted in significant improvement in all areas of previously identified deficits. The clinical psychologist summarized his conclusions as follows: "During this time [the patient] has had markedly dramatic improvement in many of his cognitive functions. He has become ambulatory, acquired good communication skills with others again, has become independent once more in his self-help skills, and regained much of his short-term and long-term memory. He seems to have responded to the hyperbaric treatment programs."

The final SPECT scan revealed normalized images with intact cortical uptake (Fig 4). The patient was fully ambulatory (although he occasionally used a cane), was self-sufficient, and required only minimal care; he returned to his home city. It was recommended that he obtain some additional cognitive-linguistic rehabilitation as well as vocational rehabilitation. He planned to return to work.

DISCUSSION

Currently, opinions vary in regard to assessment, prognostication, and treatment of acute or chronic neurologic deficits resulting from traumatic or vascular brain injury or from toxic and anoxic episodes. Brain injury imposes a tremendous expense and personal burden on involved parties and society. This case report shows that SPECT imaging before and after HBO therapy is useful in identifying potentially recoverable brain tissue, in monitoring the effectiveness of

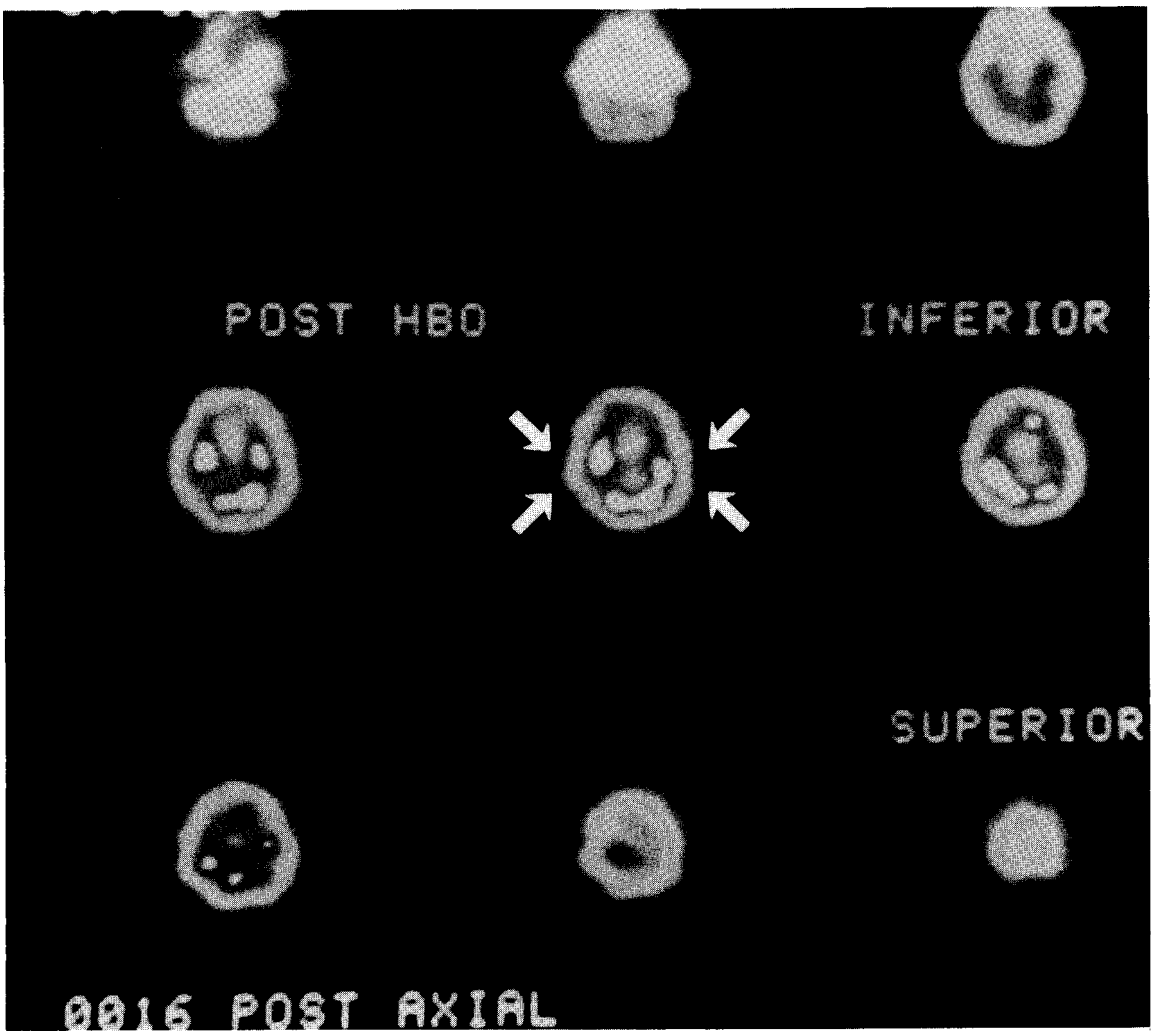

FIGURE 2. SPECT/IMP scan, axial view, taken after 1 hour's exposure to 1.5 atmospheres absolute oxygen. Scan was done within 4 hours of first scan (Fig 1). Note improvement in perfusion/metabolism in previously deficient areas.

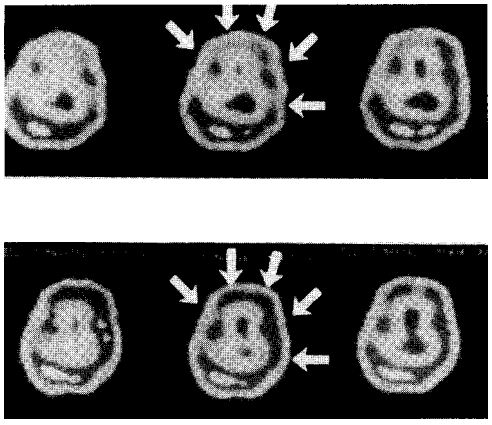

FIGURE 3. (Upper images) SPECT/IMP scans, axial view, taken 24 hours after 160th hyperbaric treatment and immediately before 161st treatment showing frontal and left parietal perfusion/metabolism deficits; large right posterior parietal deficit is no longer seen. (Lower images) Scans taken after 161st hyperbaric treatment showing oxygen-induced improvement in tracer uptake and, therefore, potential for further healing with continued therapy.

therapy, and in helping to identify the end point of therapy. CT scans and magnetic resonance ¡maging (MRI) provide anatomic information concerning brain injury. Sequential SPECT imaging with HBO intervention (SPECT/HBO) provides functional information,' especially early in the evolution of defects, thereby allowing earlier intervention than other imaging techniques. SPECT/HBO imaging, in delineating a defect volume greater than that revealed by either CT or MRI, may be indicating tissue with recoverable idling neurons. The motor and cognitive

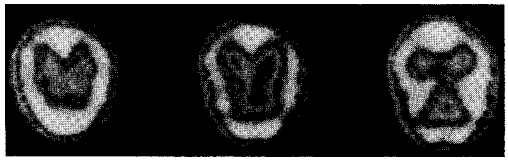

FIGURE 4. SPECT/dl HM-PAO scan, transaxial view, after 188 hyperbaric treatments. Note normal perfusion/metabolism.

improvements could be correlated with recovery of specific previously hypometabolic brain areas. Also, our data support the hypothesis that traumatic, vascular, and anoxic brain injuries and long-standing ischemic hypoxia have a common pathophysiology and may include a penumbra of recoverable tissue. We propose that HBO therapy be used routinely as an early diagnostic tool and as an adjunct to physical rehabilitation for patients with brain injuries. We also believe that research in this area would be promising.

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