with both periventricular hyperintensities (PVH) and DWMH suggesting that DWMH might relate to demyelination and PVH to neuronal axonal dysfunction.
CSF sulfatide could be a potential diagnostic marker in INPH and BD. Increased CSF NFL may be a marker of ongoing but still reversible axonal dysfunction in INPH. The diagnostic and predictive value of these markers is being investigated in a prospective study.

Hyperbaric oxygenation (HBO): a new therapeutic method in neurology

FW17–1

What is hyperbaric oxygenation? How is it administered, dose.

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Introduction Hyperbaric oxygenation therapy (HBOT) is the use of 100% oxygen at greater than atmospheric pressure. It adheres to all of the gas laws of physics. Edward Teller, father of the hydrogen bomb, stated that, with hyperbaric oxygenation, free molecular oxygen is delivered directly to the cell for immediate metabolic use without energy exchange, even with compromised circulation. No drug or combination of drugs could ever match these physiological properties.
Methods Administration may be either in a single monoplace chamber compressed with 100% oxygen or in a large chamber holding between 2 and 30 patients, where the compression takes place with air and oxygen delivered by mask or hood. There are specific doses for various problems, similar to insulin for the diabetic.
Results On a worldwide basis, the primary uses of hyperbaric oxygen are for decompression illness, carbon monoxide intoxication, and wound healing. Newer advances in the field indicate that it may play a significant role in the field of neurology because of the delivery of oxygen directly to the cells. This would be both in acute neurologic insults and in long-term neurorehabilitation.
Conclusions Hyperbaric oxygenation, a well-known treatment for select indications, is now expanding into the field of neurology for both the acute and the long-term cases. Further studies are required, and this may hold significant possibilities as a new modality for the neurologist in early intervention and long-term neurorehabilitation.

FW17–2

Effects of hyperbaric oxygenation on the central nervous system

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Introduction Hyperbaric oxygenation has significant effects in acute brain insults. It reduces cerebral edema, intracranial pressure, lactate toxic amine levels, and limits the ischemic cascade. Its use in acute ischemic thrombotic stroke is being evaluated. In acute brain insults and late neurorehabilitation, it reactivates the ischemic penumbra up to 12 years and enhances plasticity.
Methods Hyperbaric oxygenation has a specific dose for acute and long-term neurologic problems. Over 500 patients with brain injuries including stroke, traumatic injuries, and anoxic toxic encephalopathies, have been treated at the Ocean Hyperbaric Neurologic Center. In most cases, single photon emission computerized tomography (SPECT) scanning was utilized as a baseline and followed sequentially. Treatments ranged from 20 to 600 hours, at pressure of 1.1 in seizure disorder and 1.75 ATA. All patients were videotaped.
Results In acute brain insults, results are dramatic. In late cases, dormant, idling neurons may be reactivated and fire electrically for up to 12 years after ictus. PT, OT, speech, acupuncture, nutritional counseling, and herbal supplements encompass part of the program. There is a high correlation between the positive changes in the SPECT scans and the clinical changes in the patient. SPECT scanning in long-term stroke, traumatic brain injury, and anoxic ischemic encephalopathy will be presented.
Conclusions Hyperbaric oxygenation may play a major role in acute and long-term rehabilitation. An evolving role is feasible. Reductions in morbidity and mortality, as well as cost effectiveness, are major considerations.

FW17–3

Oxygen deficiency of the brain and hyperbaric oxygenation – in vitro and in vivo studies

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Background Hypoxia critically influences secondary brain lesions after stroke or injury, as is revealed by neuromonitoring in brain injured patients.
Increased tissue supply of oxygen using high pressure is the rationale behind hyperbaric oxygenation (HBO). Experimental and clinical data indicate a HBO benefit with traumatic or ischemic brain lesions.
Material and Methods Ischemia was simulated in rat brain slices in vitro. Anoxic depolarization (indicator of ischemic stress), evoked potentials (EP, a measure of functional integrity), and amino acid release measured by in vitro microdialysis were correlated.
Cerebral ischemia was induced for 30min using the Pulsinelli model in rats. Survival time and electrical brain activity of HBO treated animals were compared with controls.
HBO (≤1.5 ATA, 45min, 15 repetitions) was applied in every second of 99 patients with traumatic mid-brain syndrome.
Results With early reoxygenation of brain slices, EP recover partially without the release of excitotoxic transmitters, even though depolarization occurs. Longer ischemic periods lead to complete EP extinction.
Survival rate of Pulsinelli rats under HBO was eightfold higher compared to controls.
Survival time and survival rate of brain injured patients were better under HBO: 53% (HBO) vs. 74% of patients died or remained apallic. 33% (HBO) vs. 6% of patients completely recovered.
Conclusion In severe "energy crisis" of the brain HBO may prevent secondary damage. A randomized study in patients with severe brain lesions and bad prognosis revealed a distinct benefit from HBO especially in younger patients. Further investigations on HBO effects are promising.

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