These results demonstrate that the revascularization of the epileptogenic zone and neighboring areas can reduce or abort complex partial seizures and confirm our previous clinical experiences, as well as of other authors. Thus, placement of the omental tissue can improve the function of neurons and/or axons in the epileptic foci in ischemia, ischemic penumbra, and hypoxia if the blood flow is restored through the omentum and, later, because of axonal regeneration. Therefore, interictal hyperfusion and hypometabolism in the epileptogenic zone are normalized. Likewise, extracellular concentration of glutamate and aspartate as well as neuronal hyperexcitability are reduced.

In summary, these clinical results with this new surgical technique, we can reduce the neuronal hyperexcitability of the epileptic foci. This is contrary to treatment by surgical ablation of the epileptogenic zone, which is currently performed at many neurosurgical centers.

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References

RESPONSE: We thank Drs. Rafael and Mego for their interest in our paper regarding intractable epilepsy following functional hemispherectomy. Based on the ictal phenomenon in our patient, high-resolution reconstructed magnetic resonance images and the ictal single-photon emission CT scan showing enhancement in the caudate and frontobasal region, we hypothesized that residual cortical dysplasia of the frontobasal area was the cause of the refractory seizures in our patient, which were devastating. This is not a patient who showed any signs of ischemia or seizures after stroke as is suggested in the letter by Rafael and Mego. The pathology specimen obtained at the last surgery confirmed cortical dysplasia, and it is therefore very unlikely that such a patient would have responded to a revascularization procedure. Cortical dysplasia, especially when extensive, as in our patient, is an extremely difficult problem to manage surgically as most centers with significant experience in seizure management can attest. Our patient remains seizure free. His diencephalic syndrome has stopped and his feeding tube has been removed. He is not receiving any medication. The thrust of our paper was to outline management of refractory seizures following hemispherectomy. Knowing what we now know would likely have modified our hemispherectomy technique to include the frontobasal tissue at the time of the first intervention.

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Effects of Hyperbaric Oxygenation


Abstract
Object. Hyperbaric oxygenation (HBO) therapy has been shown to reduce mortality by 50% in a prospective randomized trial of severely brain injured patients conducted at the authors’ institution. The purpose of the present study was to determine the effects of HBO on cerebral blood flow (CBF), cerebral metabolism, and intracranial pressure (ICP), and to determine the optimal HBO treatment paradigm.

Methods. Oxygen (100% O2, 1.5 atm absolute) was delivered to 37 patients in a hyperbaric chamber for 60 minutes every 24 hours (maximum of seven treatments/patient). Cerebral blood flow, arteriovenous oxygen difference (AVDO2), cerebral metabolic rate of oxygen (CMRO2), ventricular cerebrospinal fluid (CSF) lactate, and ICP values were obtained 1 hour before and 1 hour and 6 hours after a session in an HBO chamber. Patients were assigned to one of three categories according to whether they had reduced, normal, or raised CBF before HBO.

In patients in whom CBF levels were reduced before HBO sessions, both CBF and CMRO2 levels were raised 1 hour and 6 hours after HBO (p < 0.05). In patients in whom CBF levels were normal before HBO sessions, both CBF and CMRO2 levels were increased at 1 hour (p < 0.05), but were decreased by 6 hours after HBO. Cerebral blood flow was reduced 1 hour and 6 hours after HBO (p < 0.05), but CMRO2 was unchanged in patients who had exhibited a raised CBF before an HBO session. In all patients AVDO2 remained...
constant both before and after HBO. Levels of CSF lactate were consistently decreased 1 hour and 6 hours after HBO, regardless of the patient’s CBF category before undergoing HBO (p < 0.05). Intracranial pressure values higher than 15 mm Hg before HBO were decreased 1 hour and 6 hours after HBO (p < 0.05). The effects of each HBO treatment did not last until the next session in the hyperbaric chamber.

Conclusions. The increased CMRO₂ and decreased CSF lactate levels after treatment indicate that HBO may improve aerobic metabolism in severely brain injured patients. This is the first study to demonstrate a prolonged effect of HBO treatment on CBF and cerebral metabolism. On the basis of their data the authors assert that shorter, more frequent exposure to HBO may optimize treatment.

We had originally demonstrated reduction in intracranial pressure (ICP) during HBO. Increased cerebral PO₂ continues after decompression. This contributes to the understanding of the prolonged effect of HBO in the treatment of traumatic encephalopathy. Previous studies have indicated the beneficial effect of HBO on cerebral metabolism as outlined in our book chapter and are supported in the study of Rockswold, et al.

In our book chapter on the role of HBO in neurosurgery, we recommend that patients be given treatments every 4, 6, 8, or 12 hours, depending on the clinical picture and results of monitoring and that the protocol must consist of exposure to HBO at between 1.5 and 2 atmospheres absolute (ATA) on an intermittent basis. In our original study we noted that ICP varied little when the patients were in the hyperbaric chamber; however, occasional significant elevation of ICP has been reported by others. Perhaps the fact that we treated all patients within 6 hours and every 8 hours at 2 ATA, instead of once a day at 1.5 ATA, might explain the differences in initial ICP response to HBO. Additionally, all of our patients were given an intramuscular dose of phenobarbital 30 minutes before entering the hyperbaric chamber.

The correlation by Rockswold, et al., between HBO, cerebral blood flow (CBF), and cerebral metabolic rate is important. These authors clearly demonstrate that ICP responds better to HBO when the rate of CBF is lower. It has been well demonstrated that HBO causes arterial vasodilatation reducing the intracranial volume and therefore the pressure. It must be understood that cerebral vasomotor activity must be preserved for HBO to exert this effect. This can be tested by the patient’s ICP response to hyperventilation and computer analysis of arterial blood pressure and ICP. Intact cerebral vasomotor activity is essential.

The essence of the paper by Rockswold and colleagues, as well as other previous reports is clear; oxygen is a drug and therefore dosimetry is essential. Each patient must be treated individually. The frequency, duration, and atmospheres of HBO treatment will depend on monitoring and clinical response. The treatments must begin as early as possible. The frequency is determined by clinical ICP, metabolic, and CBF results. Similarly, the 1.3 to 2 ATA depends on these parameters. In this manner the true therapeutic effects of HBO can be realized. If CBF is low and ischemia is present, ICP will likely be reduced by HBO because of its ability to combat ischemia. If CBF is elevated, ICP is high because of hyperemia and HBO will be able to reduce the CBF and therefore lower ICP. Salutary effects on cerebral metabolism can also be part of HBO’s therapeutic effect. In all instances the proper dosimetry must be determined by the clinical and monitoring results.

Rockswold and colleagues have taken a significant step in supporting the use of HBO as an important part of the therapeutic armamentarium for traumatic encephalopathy.

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References

RESPONSE: We would like to thank Dr. Sukoff for his comments regarding our recent publication in the Journal of Neurosurgery, as well as for his contributions to the field. We would like to make several observations regarding the potential use of HBO in the treatment of severe