Neuropathy and Hyperbaric Oxygen Therapy

A study of peripheral neural conduction, motor and sensory, in diabetic patients treated with hyperbaric oxygenation.

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INTRODUCTION: There are some occlusive disorders in the vasa nervorum and metabolic changes disminishing oxygen liberation by erytrocites at the capillary blood vessels, and these disturbances lead to endoneural microhypoxia. Hyperbaric oxygen reverts hypoxia in the diabetic neuropathy. OBJECTIVE: We studied motor and sensitive peripheric neuroconduction in nine diabetic patients, with distal symmetrical polyneuropathy, during normoglycemia. Four of them were insulin dependent and five were non insulin dependent. PATIENTS AND METHODS: The electrophysiological studies were done before treatment with hyperbaric oxygen, in a week, three and six months later. The abnormal electrophysiological parameters detected in diabetics were terminal latencies (enlarged), velocities of conduction (slowed) and distal amplitudes of compound action potentials (reduced). RESULTS: Neither distal latencies nor distal amplitudes and conduction velocities in peroneal nerve showed significative changes in the statistical analysis. We observed slower conduction velocities in the motor fibers of the median nerve in the examination performed six months after treatment. There was an increase of distal latency and retardation of the velocity of conduction six months later after treatment in the sensitive fibers of median nerve, whereas the amplitudes of sensitive action potentials decreased progressively. These changes suggest large diameter peripheral fibers didn't receive benefit with hyperbaric oxygen treatment.

CONCLUSIONS: In all patients disappeared all symptoms of dysesthesias, paresthesias, distal pains and cramps in the legs and arms, suggesting functional changes in small unmyelinated fibers which we can't test with conventional techniques to prove it.

Experimental ischemic neuropathy: salvage with hyperbaric oxygenation.

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Hyperbaric oxygenation is effective in augmenting the delivery of oxygen to tissue, but also causes oxidative stress. As part of our focus on improving peripheral nerve salvage from ischemic fiber degeneration, we evaluated whether hyperbaric oxygenation rescues peripheral nerve, rendered ischemic by microembolization, from ischemic fiber degeneration. The supplying arteries of rat sciatic nerve were embolized with microspheres of 14 microns diameter at moderate (2 x 10(6)) and high (5.6 x 10(6)) doses. Rats were randomized to receive hyperbaric oxygenation treatment (2.5 atm 100% oxygen for 2 hours/day for 7 days beginning within 30 minutes of ischemia), or room air. End points for the embolized limb were (1) behavioral scores (0-11 in increasing levels of limb function), (2) nerve action potential of sciatic-tibial nerve, (3) nerve blood flow, and (4) histological grade as percentage of fibers undergoing ischemic fiber degeneration (0 = < 5%; 1 = 5-25%; 2 = 26-50%; 3 = 51-75%; 4 = > 76%). Nerve blood flow and nerve action potential were uniformly absent and more than 90% of fibers had degenerated in both control and treatment groups receiving high doses. Control and treatment groups receiving moderate doses were well matched by level of ischemia (8.5 +/- 0.3 [N = 18] vs 7.7 +/- 0.4 ml/100 gm/min [N = 18], $p > 0.05$) but were significantly different by behavior score (5.6 +/- 0.7 vs 9.2 +/- 0.5 [N = 19], $p < 0.001$), nerve action potential (1.4 +/- 1.0 vs 3.9 +/- 0.5 [N = 6], $p < 0.05$), and histology (2.4 +/- 0.4 [N = 5] vs 0.8 +/- 0.5 [N = 4], $p < 0.05$). On single teased fiber evaluation, the predominant abnormality was E (axonal degeneration). We conclude that hyperbaric oxygenation will effectively rescue fibers from ischemic fiber degeneration, providing the ischemia is not extreme.

Effect of hyperbaric oxygenation on normal and chronic streptozotocin diabetic peripheral nerves.

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Hyperbaric oxygenation is known to affect energy metabolism and endothelial cell structure and function, but its effects on peripheral nerve have not been reported. We investigated whether it would (i) reverse established streptozotocin-induced diabetic neuropathy, a condition in which endoneurial hypoxia exists; (ii) affect energy metabolism in nerve; and (iii) alter the blood-nerve barrier. Sprague-Dawley rats that had been diabetic for 3 months and age-matched controls were used in these studies. One diabetic group and one control group were treated with hyperbaric oxygenation (2 atm for 2 h, 5 days/week) for 4 weeks. Identical groups remained in room air. Sciatic nerve adenosine triphosphate (ATP), creatine phosphate, lactate, and glucose concentrations showed similar changes at rest in both room air and after hyperbaric oxygenation. Nerves of control and diabetic groups exhibited increased lactate production and increased utilization of glucose, ATP, and creatine phosphate after 15 min of anoxia. The albumin blood-nerve barrier index was increased in control and diabetic nerves after hyperbaric treatment. Nerve conduction velocity was reduced in the diabetic-room air group and not improved by hyperbaric oxygenation. Caudal nerve action potential, which was significantly reduced in this group, was normalized after hyperbaric treatment. Resistance to ischemic conduction failure was increased in untreated diabetic nerve but not significantly different from controls after hyperbaric exposure. These findings indicate that treatment with hyperbaric oxygenation will partially reverse the neuropathy encountered in chronic diabetes. The biochemical changes are suggestive of enhanced nerve energy metabolism induced by hyperbaric oxygenation. The altered albumin blood-nerve barrier index presumably results from the action of free radicals on endothelial cells.

The effectiveness of intermittent hyperbaric oxygen in relieving drug-induced HIV-associated neuropathy.

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This 3-month study evaluated the effects of hyperbaric oxygen on drug-induced neuropathies in 22 patients with human immunodeficiency virus. All patients included in the study had been taking an antiretroviral medication for at least 12 months and had subjective symptoms of numbness or tingling, lethargy, and a decrease in deep tendon reflex. Patients with an active substance abuse history or Kaposi's sarcoma were excluded. Of the 20 patients who completed the series, 17 had significant improvement, 2 had a demyelinating disorder that may have affected the outcome, and 1 had no change.

Effect of hyperbaric oxygen therapy on nerve regeneration in early diabetes.

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Nerve regeneration in diabetes is essential for reversal of neuropathy as well as the recovery of nerves from injury due to acute nerve compression and entrapment. Endoneural hypoxia due to hyperglycemia-induced blood flow reductions is observed early in the course of diabetes, and the resultant ischemia plays a role in the diminished neural regeneration. Hyperbaric oxygen therapy is capable of producing tissue hyperoxia by raising

oxygen tensions in ischemic tissues, and was shown to be beneficial in the reversal of experimental ischemic neuropathy. In this study, an experimental diabetes model was used to evaluate the functional and histomorphological effects of hyperbaric oxygen therapy on early diabetic nerve regeneration. Our results indicate that there is significant histomorphological impairment of nerve regeneration, even in very early stages of diabetes. However, no beneficial effects of hyperbaric oxygen therapy could be demonstrated at this stage.