Hyperbaric Oxygen Improves Peripheral Nerve Regeneration

Several studies have documented the effectiveness of hyperbaric oxygen in models of acute and delayed crush injury. Intermittent exposure to hyperbaric hyperoxia serves to interrupt the injury cycle of edema, ischemia and tissue necrosis (1), as well as hemorrhagic hypotension (2), which in turn leads to former edema and ischemia. Tissue ischemia is countered by the ability of hyperbaric doses of oxygen to elevate tissue oxygen tensions (3).

Furthermore, edema is reduced, secondary to hyperoxia-induced arteriolar vasoconstriction (4), leading to improved tissue viability, thereby reducing necrosis (1). Hyperbaric oxygen has also been studied in models of peripheral nerve injury (5).

Researchers from the US Air Force School Aerospace Medicine and Louisiana State University recently sought to determine what, if any, morphologic changes are associated with hyperbaric oxygen treated peripheral nerve injury (6). Their model involved a crushed sciatic nerve in the rabbit. Exposure to hyperbaric oxygen across the range of current clinical dose schedules was compared to untreated, and pressure (hyperbaric air) controls. The extent of nerve regeneration was documented via morphologic analysis of electron micrographs, by a pathologist blinded as to group.

All of the animals exposed to hyperbaric doses of oxygen were reported to demonstrate advanced stages of a healed nerve, in contrast to both control groups.

As this research was limited to a determination of regeneration of morphology, the exact effects of hyperbaric oxygen were not known. The authors speculate, however, that there may be several suggesting increased myelination, decreased edema, reduced internal collagen and improvements in neurofilamentous material density. They conclude that this study provides additional evidence of a link between tissue oxygen levels and the health of peripheral nerves.

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