Hyperbaric oxygen therapy: dramatic result reported in treating 'untreatable' long-term neurological damage

Two recent studies offer support for the use of hyperbaric oxygen therapy (HBOT) as a treatment for neurological disorders. The first, by Kenneth Stoller and colleagues, indicates that HBOT—an intervention being used increasingly for autistic children—can cause improvement even in children with long-standing structural brain damage. The second, by Stephen Thom and colleagues, indicates that HBOT mobilizes stem cells which can repair damage to the brain.

Stoller et al.: HBOT reverses some FAS symptoms

Kenneth Stoller used low-pressure HBOT to treat a 15-year-old boy with Fetal Alcohol Syndrome, a condition causing brain abnormalities leading to mental impairment and learning and behavioral problems. The boy underwent 40 HBOT sessions, followed seven months later by an additional 33 sessions, each lasting 60 minutes.

Stoller reports that following the first round of treatments, the boy showed improvement in all six categories on a neuropsychological test battery. He maintained his gains in verbal memory, and continued to exhibit lower levels of impulsive behavior, at a six-month follow-up after treatment. After 33 additional treatments, Stoller says, "[the subject's] verbal memory was 95 percent (pretreatment 55 percent), visual memory was 57 percent (pretreatment 38 percent), reaction time was 0.64 second (pretreatment 1.03 second), visual motor speed score was 20.1 (pretreatment 18.6 [higher score is better]) and all previously reported symptoms resolved."

Stoller says that his findings indicate that "it is time to revise the old concept that brain injury is a condition for which there is no treatment other than supportive measures." He cites research showing that stem cells in the adult brain can cause neural regeneration, a process that is oxygen-dependent. Stoller also says that the retinal damage that sometimes results from hyperbaric oxygen therapy should not be an issue in treating neurological disorders that do not stem directly from hypoxia, as it appears to be the hypoxia—rather than the HBOT itself—that sets the stage for this complication. He concludes, "Low-pressure hyperbaric oxygen therapy is a therapy with an extremely low risk profile and relatively low cost, with potential benefits that seem to be significant and measurable for a condition considered incurable."

Stoller's findings are consistent with the anecdotal reports of a number of physicians using HBOT, sometimes in conjunction with chelation, to treat autistic children. One of these physicians, Paul Harch, testified before the U.S. House of Representatives in 2004 that HBOT causes marked improvement in brain blood flow pattern, often leading to significant improvement. For instance, Harch testified, one child he treated with HBOT "was able to be weaned from the powerful psychoactive drugs Ritalin and Prozac, and improve his emotional outbursts, autistic behavior, ability to play sports and attend school." Harch testified that the combination of HBOT and chelation appears to be be more successful than the use of either therapy alone.

Thom et al.: HBOT mobilizes stem cells

In a study to be published in April, Stephen Thom et al. report that positive effects of HBOT may arise from its ability to mobilize stem cells. When mobilized, stem cells can move from the marrow and differentiate into different types of cells that can aid in repairing damage to the brain or other organs.

Thom et al. studied 26 patients undergoing HBOT, and found that a standard course of HBOT therapy increased by eight-fold the number of stem cells circulating in the human body. An analysis of mice exposed to HBOT revealed that the treatment increases synthesis of nitric oxide, which in turn triggers enzymes that mediate stem cell release.

"This is the safest way clinically to increase stem cell circulation," Thom says, "far safer than any of the pharmaceutical options."

Editor's Note: ARI, and its Defeat Autism Now! (DAN!) Project, have hosted several think-tanks and HBOT research planning meetings. Plans for conducting carefully designed HBOT evaluation studies are underway.

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"Quantification of neurocognitive changes before, during, and after hyperbaric oxygen therapy in a case of fetal alcohol syndrome," Kenneth P. Stoller, *Pediatrics*, Vol. 116, No. 4, October 2005, e586-e591. Address: Kenneth P. Stoller, 404 Brunn School Road #D, Santa Fe, NM 87505, hbotnm@netzero.net.

—and—

"Announcement of a new treatment protocol for autism spectrum disordres and other neurological impairments," Paul Harch, testimony on behalf of the International Hyperbaric Medical Association Foundation, presented at the May 6, 2004 United States House of Representatives Hearing on "Autism spectrum disorders: An update of federal government initiatives and revolutionary new treatments of neurodevelopmental diseases."

—and—

"Stem cell mobilization by hyperbaric oxygen," Stephen R. Thom, Veena M. Bhopale, Omaida C. Velazquez, Lee J. Goldstein, Lynne H. Thom, and Donald G. Buerk, *American Journal of Physiology-Heart and Circulation Physiology*, November 18, 2005 (epub in advance of publication). Address: Stephen R. Thom, Institute for Environmental Medicine, University of Pennsylvania, 1 John Morgan Building, 3620 Hamilton Walk, Philadelphia, PA 19104, sthom@mail.med.upenn.edu.

—and—

"Hyperbaric oxygen treatments mobilize stem cells," review article by Priya Saxena, *RxPG News*, January 1, 2006.

Anti-seizure effects of carnosine again reported

Two years ago, researcher Michael Chez and colleagues reported remarkable improvements in some autistic children taking the dietary peptide carnosine, as well as decreases in seizures (see ARRI 17/1). A new report supports Chez's finding that carnosine may have a significant anticonvulsant effect.

C. L. Jin and colleagues provoked amygdaloid seizures in rats, and injected them with varying doses of carnosine. The researchers found that the carnosine injections "significantly decreased seizure stage, after-discharge duration and generalized seizure duration, and significantly prolonged generalized seizure latency" in a dose-dependent manner. Further investigation indicated that carnosine works by both directly and indirectly activating histamine postsynaptic H-1 receptors. The researchers conclude that "carnosine may be an endogenous [naturally occurring] anticonvulsant factor in the brain and could be used as a new antiepileptic drug in the future."

DAN! researcher Jon Pangborn cites carnosine's anticonvulsant effects in *Autism: Effective Biomedical Treatments*, and notes that in Chez's study, autistic children with seizures benefited more than those without seizures. However, Pangborn also notes that some parents have reported to him that their children experienced significant adverse effects when taking carnosine. "One child became severely hyperactive, and the hyperactivity lasted for days after carnosine was discontinued," he notes. "Two became aggressive and destructive, and several lapsed deeper into isolation and noncommunication while taking carnosine." Pangborn suggests that physicians use caution in recommending carnosine if:

—Carnosine is already high according to urine amino acid analysis.

—Taurine is being wasted in urine, also according to amino acid analysis.

—Taurine is low in blood, according to plasma amino acid analysis.

—Blood copper-to-zinc ratio is excessive, or zinc is deficient.

—Creatine is deficient in blood.

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"Effects of carnosine on amygdaloid-kindled seizures in Sprague-Dawley rats," C. L. Jin, L. X. Yang, X. H. Wu, Q. Li, M. P. Ding, Y. Y. Fan, W. P. Zhang, J. H. Luo, and Z. Chen, *Neuroscience*, August 25, 2005 (epub ahead of print). Address: C. L. Jin, Department of Pharmacology and Neurobiology, School of Medicine, Zhejiang University, Hangzhou, China 310031.