Traumatic Brain Injury

Hyperbaric oxygen may induce angiogenesis in patients suffering from prolonged post-concussion syndrome due to traumatic brain injury.

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Sigal T1,2, Amir H3,1, Nadav B1,2, Efrat S4, Shai E3,1,5,6.

Recent clinical studies present convincing evidence that hyperbaric oxygen therapy (HBOT) may be the coveted neurotherapeutic method for brain repair. One of the most interesting ways in which HBOT can induce neuroplasticity is angiogenesis.The objective in this study was to assess the neurotherapeutic effect of HBOT in post TBI patients using brain perfusion imaging and clinical cognitive functions.

Retrospective analysis of patients suffering from chronic neuro-cognitive impairment from TBI treated with HBOT. The HBOT protocol included 60 daily HBOT sessions, 5 days per week. All patients had pre and post HBOT objective computerized cognitive tests (NeuroTrax) and brainperfusion MRI.

Ten post-TBI patients were treated with HBOT with mean of 10.3 ± 3.2 years after their injury. After HBOT, whole-brain perfusion analysis showed significant increased cerebral blood flow and cerebral blood volume Clinically, HBOT induced significant improvement in the global cognitive scores ($p=0.007$). The most prominent improvements were seen in information processing speed, visual spatial processing and motor skills indices.

CONCLUSION:

HBOT may induce cerebral angiogenesis, which improves perfusion to the chronic damages brain tissue even months to years after the injury.

Hyperbaric oxygen therapy can improve post concussion syndrome years after mild traumatic brain injury - randomized prospective trial.

Boussi-Gross R[1], Golan H, Fishlev G, Bechor Y, Volkov O, Bergan J, Friedman M, Hoofien D, Shlamkovitch N, Ben-Jacob E, Efrati S.

PLoS One. 2013 Nov 15;8(11):e79995. doi: 10.1371/journal.pone.0079995. eCollection 2013.

[1]The Institute of Hyperbaric Medicine, Assaf Harofeh Medical Center, Zerifin, Israel.

Traumatic brain injury (TBI) is the leading cause of death and disability in the US. Approximately 70-90% of the TBI cases are classified as mild, and up to 25% of them will not recover and suffer chronic neurocognitive impairments. The main pathology in these cases involves diffuse brain injuries, which are hard to detect by anatomical imaging yet noticeable in metabolic imaging. The current study tested the effectiveness of Hyperbaric Oxygen Therapy (HBOT) in improving brain function and quality of life in mTBI patients suffering chronic neurocognitive impairments.

The trial population included 56 mTBI patients 1-5 years after injury with prolonged post-concussion syndrome (PCS). The HBOT effect was evaluated by means of prospective, randomized, crossover controlled trial: the patients were randomly assigned to treated, or crossover groups. Patients in the treated group were evaluated at baseline and following 40 HBOT sessions; patients in the crossover group were evaluated three times: at baseline, following a 2-month control period of no treatment, and following subsequent 2-months of 40 HBOT sessions. The HBOT protocol included 40 treatment sessions (5 days/week), 60 minutes each, with 100% oxygen at 1.5 ATA. "Mindstreams" was used for cognitive evaluations, quality of life (QOL) was evaluated by the EQ-5D, and changes in brain activity were assessed by SPECT imaging. Significant improvements were demonstrated in cognitive function and QOL in both groups following HBOT but no significant improvement was observed following the control period. SPECT imaging revealed elevated brain activity in good agreement with the cognitive improvements.

CONCLUSIONS:

HBOT can induce neuroplasticity leading to repair of chronically impaired brain functions and improved quality of life in mTBI patients with prolonged PCS at late chronic stage.

"Hyperbaric oxygen in the treatment of elevated intracranial pressure after head injury."

Brown, J.A. et al.

PEDIATR NEUROSCI, 1988, 14(6): 286-90.

This study is the first to evaluate the effect of hyperbaric oxygen on elevated intracranial pressure after severe head injury during documented controlled ventilation, hypocapnea, and minute-by-minute ICP data collection. The authors studied the effect of HBO at 2 atmospheres absolute with 100% O2, on intracranial pressure in 2 patients, aged 5 and 21 years. Each patient had diffuse cerebral swelling after blunt trauma and after a gun shot wound, respectively. Both required controlled hyperventilation, osmotic diuretics and intracranial pressure monitoring. During pressurization the mean ICP dropped from 13 to 8 Torr, then returning to 12 Torr after HBO therapy. The authors conclude that HBO may lower ICP in head-injured patients with diffuse cerebral swelling during the first 15 minutes, or the pressurization phase of therapy. Lasting effects of treatment were not seen with 4 treatments. The effect of HBO deserves further careful study in those patients with severe enough injury to require intracranial pressure monitoring.

"Hyperbaric Medicine: An integral part of trauma care."

Camporessi, Enrico et al.

CRITICAL CARE CLINICS, 1990; 6(1): 203-219.

The author states that "hyperbaric medicine plays an integral role in comprehensive trauma care, from resuscitation to definitive therapy and subsequent recovery." Among his list of traumatic conditions include crush injury, exceptional blood loss, head injury and spinal cord injury.

"The Effect of hyperbaric oxygenation upon recovery of maze performance after experimental concussion."

Coe, John et al.

THE JOURNAL OF TRAUMA, 1971, 11(5): 436-439. (animal study).

Thirty rats were divided into 3 groups, were tested for errors in 10 consecutive maze runs and then subjected to either (a) nonlethal concussion, un-treated group, (b) concussion followed by 98% oxygen and 2% carbon dioxide at 3 atmospheres absolute for 1 hr, and (c) to the same hyperbaric oxygenation without concussion (control). The maze-running performance of the rats with concussion treated with hyperbaric oxygen recovered faster and equaled that of the control group by the fifth day after the concussion. The untreated group of animals showed a greater lag in performance persisting throughout the observation period.

"The effect of hyperbaric oxygen on glucose utilization in a freeze-traumatized rat brain."

Contreras, F.L. et al.

J NEUROSURG, 1998, 68(1): 137-41. (animal study).

Local cerebral glucose utilization was measured with the autoradiographic 2-deoxyglucose technique in rats injured by a focal parietal cortical freeze lesion then treated with hyperbaric oxygen. The cold lesion depressed glucose utilization in the contralateral as well as in the ipsilateral hemisphere. Treatment of lesioned animals with HBO at 2 atm for 90 minutes on each of 4 consecutive days tended to increase the overall cerebral glucose utilization measured 5 days after injury when compared to animals exposed to normobaric air. This improvement reached statistical significance in five of the 21 structure studied: the auditory cortex, medial genicuate body, superior olivary nucleus, and lateral geniculate body ipsilateral to the lesion, and the mammallary body. The data indicate that changes in lesioned rats exposed to HBO are not restricted to the period of time that the animals are in the hyperbaric chamber but are persistent.

"Effects of Hyperbaric Oyxgenation in Patients with Subarachnoid Hemorrhage."

Kawamura, S. et al.

JOURNAL OF HYPERBARIC MEDICINE, 1988; 3(4): 243-256.

Changes of N1-amplitude in somatosensory evoked potential (SEPs) were studied to evaluate the effects of hyperbaric oxygenation (HBO) in 26 subarachnoid hemorrhage patients. During HBO, significantly improved SEPs were seen in 57% of 21 records from 2 to 14 days after the onset of subarachnoid hemorrhage, in 34% of 35 records in cases where there was no or only mild brain swelling, and in 38% of 26 records in cases where there were free-to-mild neurologic symptoms. In cases of moderate swelling, and mild to severe neurologic deficits, significant improvement was recognized less frequently.

"Clinical analysis of diffuse axonal injury."

Mao, B. et al.

HUA HIS I KO TA HSUEH HSUEH PAO, 1996, 27(4): 422-5.

Sixty cases of diffuse axonal injury were analysed in this paper. All cases were caused by traffic accident; the mortality was 53.12%. Clinical manifestations were post-traumatic immediate and continuous coma with severe dysfunction of the brain stem. Pathological findings included the diffuse axonal injury of cortical white matter, corpus callosum, brain stem, and focal hemorrhage and infarction. The early use of hyperbaric oxygen combined with neuro-growth factor as an effective therapy is recommended.

"Hyperbaric oxygen for treatment of closed head injury."

Neubauer, R.A. et al.

SOUTHERN MEDICAL JOURNAL, 1994, 87(9): 933-6.

Traumatic and vascular brain injuries consist of acute episodes followed by development of chronic components of varying magnitude and duration whose potentials for recovery differ. The authors discuss a case of closed head injury in which interventional hyperbaric oxygen (HBO) with single photon emission computed tomography were used as aids in determining the presence of recoverable neurons, to follow therapeutic progress, and to determine the end point of therapy. This case also shows the successful use of intensive HBO as a therapeutic modality.

"Effect of hypoxia or hyperbaric oxygen on cerebral edema following moderate fluid percussion or cortical impact injury in rats."

Nida, TY et al.

J NEUROTRAUMA, 1995, 12(1): 77-85. (animal study).

This study was designed to evaluate the production of cerebral edema following moderate fluid percussion and cortical impact injury in rodents. To determine the effects of asecondary systemic insult, hypoxia (13% oxygen for 30 minutes) was added to some experimental groups immediately after head injury. To determine the effects of hyperbaric oxygen (HBO) on injured cortical tissue, additional animal groups were exposed to HBO (1.5 atm, for 60 minutes) beginning 4 hours after head trauma. HBO reduced the water content of the trauma site in animals that had received fluid percussion but not in animals receiving cortical impact injury. The authors conclude that both fluid percussion and cortical impact appear to produce focal cerebral edema at the site of trauma. Hypoxia does not worsen the edema. HBO appears to reduce edema produced by fluid percussion but the number of treatments and the ata were not enough to reduce the effects from cortical injury.

"Hyperbaric oxygen therapy in the Hokkaido University Hospital."

Okamura, A. et al.

MASUI, 1994, 43(6): 947-50.

The authors surveyed hyperbaric oxygen therapy during the past seven years in the Hokkaido University Hospital. The mean number of patients was 27 per year. The average number of the therapy was 328 per year. There were neither complications nor accidents attributable to the hyperbaric oxygen therapy. Three

representative diseased states: hypoxic brain damage, sudden deafness and occlusion of retinal arteries showed remarkable recovery by this therapeutic modality.

Hyperbaric oxygen therapy in the clinical treatment of mental disorders accompanying severe cranio-cerebral trauma.

Tishchenko AT.

The author examined 20 patients with severe brain damage. Under the influence of hyperbaric oxygen therapy there was a more rapid restitution of consciousness and a relatively short development of soporous and comatose conditions. Disorders of the Korsakoff syndrome type in such conditions had an abortive development as well (4--7 days). In cases of developing delirious states the use of hyperbaric oxygen therapy demonstrated a clearing of consciousness and a disappearance of psychotic disturbances, already following the first session.

Hyperbaric oxygenation in the acute period of craniocerebral injuries.

Isakov IV, Anan'ev GV, Romasensko MV, Aide KB.

A study was made of the effect of hyperbaric oxygenation (HBO) on the clinical progress of craniocerebral trauma by means of a comparative analysis of the time course of the clinical symptoms and the occurrence of complications seen in the main group of patients given HBO (103 subjects) and the control group not given HBO (also 103 subjects). Some of the parameters of external respiration and central hemodynamics were examined in these patients during HBO. It was disclosed that HBO exerts a prophylactic action as regards the development of mental disorders in the acute period of brain trauma and some complications (meningitis, suppuration of operative wound, bedsores, pneumonias). HBO had no noticeable effect on the rate and degree of the recovery of motor and speech functions. In part of cases, HBO eliminated the hyperventilation syndrome and the pathological rhythm of respiration and myocardial hypodynamia.

Effects of hyperbaric oxygenation therapy on cerebral metabolism and intracranial pressure in severely brain injured patients.

Rockswold SB, Rockswold GL, Vargo JM, Erickson CA, Sutton RL, Bergman TA, Biros MH.

Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, Minnesota 55415-1829, USA.

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.

Intracranial pressure responses during hyperbaric oxygen therapy.

Kohshi K, Yokota A, Konda N, Kinoshita Y, Kajiwara H.

Department of Hyperbaric Medicine, University of Occupational and Environmental Health, Fukuoka.

The responses of intracranial pressure (ICP) to hyperbaric oxygen (HBO) therapy and arterial gas pressures were investigated. ICP was measured through a ventricular or spinal drainage catheter in patients with brain tumor or cerebrovascular disease. Changes in ICP, heart rate (HR), arterial blood pressure (ABP), and transcutaneous partial pressure of carbon dioxide (PtcCO2) or oxygen (PtcO2) were recorded continuously during air or 100% O2 breathing at 1 and 2.5 atmospheres absolute (ATA). HR and PtcCO2 decreased and mean ABP was unchanged during HBO inhalation. ICP was reduced at the beginning and tended to increase gradually during HBO inhalation. The change from air to O2 without altering respiratory frequency and volume caused a gradual increase of ICP and PtcCO2 with a transient ICP reduction in an artificially respirated patient. Intentionally reduced respiration to maintain PtcCO2 at the value at 2.5 ATA with air caused the ICP to return to near the value at 2.5 ATA with air even during HBO inhalation. These findings suggest that reduced ICP is initially due to direct cerebral vasoconstriction caused by hyperoxia and is maintained mainly by induced hypocapnia during HBO inhalation. Care is required when giving HBO therapy to patients with a high ICP and/or who are respirated artificially.

Hyperbaric oxygen therapy for the adjunctive treatment of traumatic brain injury.

Bennett MH[1], Trytko B, Jonker B.

Cochrane Database Syst Rev. 2012 Dec 12;12:CD004609. doi: 10.1002/14651858.CD004609.pub3.1

Department of Anaesthesia, Prince ofWales Hospital, Randwick, Australia. m.bennett@unsw.edu.au

Traumatic brain injury is a common health problem with significant effect on quality of life. Each year in the USA approximately 0.56% of the population suffer a head injury, with a case fatality rate of about 40% for severe injuries. These account for a high proportion of deaths in young adults. In the USA, 2% of the population live with long-term disabilities following head injuries. The major causes are motor vehicle crashes, falls, and violence (including attempted suicide). Hyperbaric oxygen therapy (HBOT) is the therapeutic administration of 100% oxygen at environmental pressures greater than 1 atmosphere absolute (ATA). This involves placing the patient in an airtight vessel, increasing the pressure within that vessel, and administering 100% oxygen for respiration. In this way, it is possible to deliver a greatly increased partial pressure of oxygen to the tissues. HBOT can improve oxygen supply to the injured brain, reduce the swelling associated with low oxygen levels and reduce the volume of brain that will ultimately perish. It is, therefore, possible that adding HBOT to the standard intensive care regimen may reduce patient death and disability. However, a concern for patients and families is that using HBOT may result in preventing a patient from dying only to leave them in a vegetative state, entirely dependent on medical care. There are also some potential adverse effects of the therapy, including damage to the ears, sinuses and lungs from the effects of the pressure and oxygen poisoning, so the benefits and risks of the therapy need to be carefully evaluated.

To assess the effects of adjunctive HBOT for traumatic brain injury.

We searched CENTRAL, MEDLINE, EMBASE, CINAHL and DORCTHIM electronic databases. We also searched the reference lists of eligible articles, handsearched relevant journals and contacted researchers. All searches were updated to March 2012.

Randomised studies comparing the effect of therapeutic regimens which included HBOT with those that did not, for people with traumatic brain injury.

Three authors independently evaluated trial quality and extracted data.

Seven studies are included in this review, involving 571 people (285 receiving HBOT and 286 in the control group). The results of two studies indicate use of HBOT results in a statistically significant decrease in the

proportion of people with an unfavourable outcome one month after treatment using the Glasgow Outcome Scale (GOS) (relative risk (RR) for unfavourable outcome with HBOT 0.74, 95% CI 0.61 to 0.88, P = 0.001). This five-point scale rates the outcome from one (dead) to five (good recovery); an 'unfavourable' outcome was considered as a score of one, two or three. Pooled data from final follow-up showed a significant reduction in the risk of dying when HBOT was used (RR 0.69, 95% CI 0.54 to 0.88, P = 0.003) and suggests we would have to treat seven patients to avoid one extra death (number needed to treat (NNT) 7, 95% CI 4 to 22). Two trials suggested favourably lower intracranial pressure in people receiving HBOT and in whom myringotomies had been performed. The results from one study suggested a mean difference (MD) with myringotomy of -8.2 mmHg (95% CI -14.7 to -1.7 mmHg, P = 0.01). The Glasgow Coma Scale (GCS) has a total of 15 points, and two small trials reported a significant improvement in GCS for patients treated with HBOT (MD 2.68 points, 95%CI 1.84 to 3.52, P < 0.0001), although these two trials showed considerable heterogeneity (I(2) = 83%). Two studies reported an incidence of 13% for significant pulmonary impairment in the HBOT group versus 0% in the non-HBOT group (P = 0.007).In general, the studies were small and carried a significant risk of bias. None described adequate randomisation procedures or allocation concealment, and none of the patients or treating staff were blinded to treatment.

CONCLUSIONS:

In people with traumatic brain injury, while the addition of HBOT may reduce the risk of death and improve the final GCS, there is little evidence that the survivors have a good outcome. The improvement of 2.68 points in GCS is difficult to interpret. This scale runs from three (deeply comatose and unresponsive) to 15 (fully conscious), and the clinical importance of an improvement of approximately three points will vary dramatically with the starting value (for example an improvement from 12 to 15 would represent an important clinical benefit, but an improvement from three to six would leave the patient with severe and highly dependent impairment). The routine application of HBOT to these patients cannot be justified from this review. Given the modest number of patients, methodological shortcomings of included trials and poor reporting, the results should be interpreted cautiously. An appropriately powered trial of high methodological rigour is required to define which patients, if any, can be expected to benefit most from HBOT.

bubbles to a much smaller size and the delivery of high doses of oxygen to ischemic brain tissue.

Hyperbaric oxygen therapy reduces neuroinflammation and expression of matrix metalloproteinase-9 in the rat model of traumatic brain injury.

Vlodavsky E, Palzur E, Soustiel JF.

Neuropathol Appl Neurobiol. 2006 Feb;32(1):40-50.

Institute of Pathology, Rambam Medical Center, Haifa 31096, Israel. e_vlodavsky@rambam.health.gov.il

The acute inflammatory response plays an important role in secondary brain damage after traumatic brain injury (TBI). Neutrophils provide the main source of matrix metalloproteinases (MMPs) which also play a deleterious role in TBI. Numerous preclinical studies have suggested that hyperbaric oxygen therapy (HBOT) may by beneficial in various noncerebral and cerebral inflammatory diseases. The goal of this study was to evaluate the effects of HBOT on inflammatory infiltration and the expression of MMPs in correlation with secondary cell death in the rat model of dynamic cortical deformation (DCD). Twenty animals underwent DCD with subsequent HBOT (2.8 ATA, two sessions of 45 min each); 10 animals: DCD and normobaric oxygenation (1 ATA), 10 animals: not treated after DCD. Cell death was evaluated by TUNEL. Neutrophils were revealed by myeloperoxidase staining. Immunohistochemical staining for MMP-2 and -9 and tissue inhibitors of MMP-1 (TIMP-1) and -2 was also performed and the results were quantitatively evaluated by image analysis. In the animals treated by HBOT, a significant decrease in the number of TUNEL-positive cells and neutrophilic inflammatory infiltration was seen in comparison with nontreated animals and those treated by normobaric oxygen. The expression of MMP-9 was also significantly lower in the treated group. Staining for MMP-2 and TIMP-2 did not change significantly. Our results demonstrate that HBOT decreased the extent of secondary cell death and reactive neuroinflammation in the TBI model. The decline of MMP-9 expression after HBOT may

also contribute to protection of brain tissue in the perilesional area. Further research should be centred on the evaluation of long-term functional and morphological results of HBOT.

Effects of hyperbaric oxygenation.

Sukoff MH.

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.

Cerebral Blood Flow Changes and Cognitive Improvement in Chronic Stable Traumatic Brain Injuries Treated with Hyperbaric Oxygen Therapy

2009. Kevin F. Barrett, Brent E. Masel, Galveston, TX, Paul G. Harch, New Orleans, LA Fred Ingram, Kevan P. Corson, Jon T. Mader, Galveston, TX.

Objective: To determine if hyperbaric oxygen therapy in patients with chronic stable traumatic brain injuries can produce: 1) neurological and cognitive improvements; 2) changes in cerebral blood flow to ischemic penumbral areas as determined by ECD-Tc99m SPECT brain scanning.

Background: Numerous studies have shown that cognitive improvement after severe traumatic brain injury (TBI) is most dramatic in the first six months after injury, and is fairly static after 18 months. Anecdotal reports exist that attest to the efficacy of hyperbaric oxygen therapy (HBOT) to improve post traumatic neurologic deficits by increasing blood flow in the ischemic penumbra despite protocol differences. Cerebral blood flow, speech, neurological and neuropsychometric testing have not been studied serially in patients undergoing HBOT for chronic stable TBI.

Design/Methods: Five patients with TBI, at least three years post injury, underwent 120 HBOT's at 1.5 atmospheres absolute of oxygen for 60 minutes. They received a set of 80 HBOT's, a five month rest, and a second set of 40 HOBT's. Patients were studied sequentially to determine HOB's effects on: cerebral blood flow, speech fluency, neurologic, neuropsychometric and progressive exercise testing. Six TBI controls were not treated with HBOT, but underwent serial SPECT scanning to study time related alterations in cerebral blood flow. Five non-TBI controls underwent SPECT scanning, one HBOT, and a repeat scan to study HBOT influence on cerebral blood flow in normal subjects. SPECT brain scans were performed serially on the HBO treated group. Scans were spatial and intensity normalized and subjected to statistical parametric mapping.

Results: Serial SPECT imaging showed: TBI controls had no significant consistent change in CBF over time; non-TBI controls had essentially no influence from one HBOT upon CBF; the treated TBI patients had permanent increases in CBF to penumbral areas and a reqression to a mean CBF range. In the HOB treated group, no changes were seen in progressive exercise and neurologic testing. Speech fluency improved in all cases, as did group mean scores of memory, attention, and executive function. Improvement peaked at 80 HBOT, suggesting a possible maximum length of treatment between 80 and 120 HBOT.

Conclusions: Our pilot study findings suggest that HBOT at 1.5 ATA is a promising therapy for permanently improving penumbral brain blood flow and cognitive function in chronic stable TBI where no improvement would have been expected.