Autism: Treatments
Hyperbaric Oxygen

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Hyperbaric Oxygen Therapy for Autism

Hyperbaric Oxygen Treatment

The logic for using hyperbaric oxygen treatment for developmental disorders relates to the auto-immune and/or viral theory of these conditions. Hyperbaric oxygen has been studied for auto-immune disorders and found to be helpful. [References for this will be posted soon]. Encephalitis, in this theory, is thought to be part of developmental disorders. The encephalitis can be initiated by viral infection, by exposure to vaccines, and/or by other auto-immune processes (the result of exposure to abnormal opioid-like substances from the opioid excess hypothesis, for example).

Treatment Study

Patients with viral encephalitis from ages 1 yr. to 11 yrs. were treated with hyperbaric oxygen therapy. The treated group consisted of 47 patients, 28 male and 19 female. The control group consisted of 45 patients, 24 male and 21 female. Viral encaphalitis presents a model for the inflammation that may be part of autism. Studies such as this provide a basis for the use of hyperbaric oxygen therapy for autism. Hyperbaric oxygen therapy was provided at 1.8 atm abs for 80 minutes daily for 10 days in a pure oxygen monoplace unit. The control group received supportive therapy with appropriate drugs.

Results:

Table 1. Comparison of the Curative Effect of the High Pressure Oxygen Treatment Group and the Control Group

Treatment Study					
Group	Cases #	Cured # (%)	Effective # (%)	Ineffective # (%)	Total Effective (%)
Treatment	47	18 (38.3)	25 (55.3)	3 (6.4)	93.6
Control	45	8 (17.8)	20 (44.5)	17 (37.8)	62.2

P Value		<0.05	N.S.	<0.0001	<0.01

Clearly, hyperbaric oxygen therapy is effective for the treatment of encephalitis in childhood.

Since we know of no data on the use of hyperbaric oxygen for developmental disorders, and, since patients are doing these therapies, we will endeavor to review what is known, so that parents can at least make informed choices. In Pennsylvania, the range of cost for hyperbaric treatments is from a low of $110 per treatment in Columbia, Pennsylvania, in the center of the state, to a high of $850 per treatment at St. Francis Hospital, in Pittsburgh. This range apparently exists around the country, with the most common charges being in the $225 to $280 range.

Encephalic Lesions

Hyperbaric-Oxygen Therapy (HBOT) has been used to treat encephalic lesions for over a decade.

Encephalitis can compromise blood supply to vital brain tissue, as can stroke, arterial narrowing, and arteritis (inflammation of the arteries). When blood supply of an area is compromised, a shadow like spread (called a penumbral zone) of damage occurs around the compromised area. HBOT can reduce damage to the areas of the brain that have reduced oxygen flow (ischemia) through improved bio-availability of oxygen in these ischemic areas, and indirectly through regulatory action of cerebral flow that improves perfusion in "critical" areas.

HBOT improves oxygen diffusibility throughout the brain, lowering lactates and raising ATP in affected areas; improving glucose metabolism by lowering production of substances, like aspartate and glutamate, which are responsible for over-response of the receptors; improving metabolic processes and perfusional flow distribution, bringing the generation of free radicals in the intracranial area under greater control.

As therapy can act only on areas not irreversibly damaged, treatment must occur as early as possible.

The Importance of Glial Cells

HBOT brings dissolved oxygen to the glial level that is important in treating brain damage. HBOT helps redistribute blood to the non-ischemic areas around the lesions, preventing these areas from being harmed. These areas are generally affected by the response of the brain to injury which is a vasoconstriction, making them more susceptible to ischemic damage also, thereby continuing to spread

the damage beyond its original area. HBOT prevents this "stealing" of blood, reducing intracranial pressure and raising tissue oxygen flow.

Glial cells are important and preventing their damage is necessary.

This press release from Stanford University illustrates this concept further:

Lowly Glia Strengthen Brain Connections

STANFORD -- Once dismissed as mere padding, cells known as glia may be essential for the correct wiring of the brain. This is the conclusion of a study reported in the Sept. 12 issue of Science by researchers from the Stanford University School of Medicine.

Postdoctoral fellow Frank Pfrieger and Dr. Barbara Barres, associate professor of neurobiology, used pure populations of nerve cells and glia to show that, by themselves, the nerve cells connected together poorly, but the combination of the two cell types resulted in strong connections between nerve cells.

In the brain, such connections allow nerve cells to pass along messages about our every sensation, thought and movement.

Glia make up approximately 90 percent of the cells in the human brain, and yet researchers have assigned mainly passive functions to them. Some glia wrap around nerve cells and insulate them with a protein called myelin. Glia at synapses act both as a physical barrier that prevents crossed wires and as a disposal unit that mops up extra messenger molecules released by nerve cells.

The nerve cells chosen for the Stanford study -- retinal ganglion cells -- lead from the eyes deep into the brain. Barres is using them as representatives of a large class of nerve cells in the brain: those that use a chemical messenger called glutamate to send a positive, or excitatory, signal.

It is also possible, she said, that glia control the strength of synapses in the fully developed brain, beefing up some circuits and turning down others.

There are five types of glial cells:

*Oligodendroglia, which provide the insulation (myelin) to neurons in the central nervous system.

*Schwann Cells, which provide the insulation (myelin) to neurons in the peripheral nervous system.

*Astrocyte (Astroglia), star-shaped cells that provide physical and nutritional support for neurons:

1. to clean up brain "debris";
2. to transport nutrients to neurons;
3. to hold neurons in place;
4. to digest parts of dead neurons;
5. to regulate content of extracellular space

*Microglia, which like astrocytes, digest parts of dead neurons.

*Satellite Cells, which provide physical support to neurons in the peripheral nervous system

Hyperbaric Oxygen Therapy - Questions and Answers:

[from the Hyperbaric oxygen e-mail mailing list]

Q: Why is oxygen so important in the first place?

A: Every day an average adult consumes four pounds of food, two pounds of water and almost 6 pounds of oxygen. People need about the same amount of oxygen by weight compared to food and water combined! >From that six pounds of oxygen about 2 pounds gets into the blood for transport to tissue cells. We need this oxygen in order to complete the energy cycle that sustains life.

Q: Is hyperbaric oxygen different from natural oxygen?

A: Hyperbaric oxygen is natural oxygen, delivered in a pressurized chamber. The increased pressure does not change the molecular composition of oxygen. The increased pressure just allows extra oxygen to get into tissues above regular amounts.

Q: What do you feel inside a hyperbaric chamber?

A: Chamber atmosphere pressurization occurs slowly allowing you to adjust ear pressure changes. As the air pressure increases just yawn, swallow or "blow your nose" to clear pressure changes in your ears and you are "good to go". Other than this there are no unusual or different sensations.

Q: So what difference does extra pressure create?

A: Hemoglobin (in red blood cells) holds 97% of its maximum amount of oxygen from normal air or holds 100% when breathing pure oxygen. The limit that one gram of hemoglobin can combine with oxygen is 1.34 ml. Red blood cells can only deliver a limited level of oxygen to tissue cells, a pO2 of 40 mmHg or less. This is called oxygen tension (or oxygen partial pressure, "pO2") and is measured in units labeled "mmHg" (the amount of pressure able to raise the equivalent weight of a liquid mercury column, pretty heavy stuff).

Injuries, infections and diseases can drop this vital tissue oxygen level down to almost zero! As we age we can loose vital lung capacity and the ability to effectively obtain adequate oxygen. Some disease conditions impair oxygen utilization. Also, injuries or conditions with swelling can cause pressure that cuts off circulation flow. This loss of blood flow, called ischemia, cuts off oxygen circulation to the affected areas of the body. This problem drops the pO2 gravely low, destroys tissue, and slows healing.

Research has shown optimal tissue healing occurs if pO2 rises to 80 mmHg. Oxygen given in a normal room is not sufficient to raise tissue oxygen levels to 80 mmHg because red blood cells cannot carry the extra oxygen. The answer is to deliver the oxygen in a pressurized chamber to raise oxygen tension beyond red blood cell saturation.

Q: What is the difference between saturation and oxygen tension?

A: The problem we face in advocating proper useage of oxygen involves confusion between saturation and oxygen tension, 100% vrs. 100 mmHg. Only dissolved oxygen contributes to the tension (or partial pressure). Study the figures for oxygen transported by plasma (liquid) vrs. hemoglobin (one gram hemoglobin can only combine with 1.34 ml oxygen) - in 100ml of healthy blood there is 19ml oxygen as oxyhemoglobin and 0.3ml oxygen in liquid solution, here the hemoglobin is near maximum saturation (98%) and the pressure or tension of oxygen in the liquid solution is initially 95mmHg and downline tissue levels may only be 40mmHg. Breathing pure oxygen at 2.8 times atmospheric pressure increases the amount of oxygen in (plasma) liquid solution to almost 6.6 ml per 100ml blood. This increased oxygen volume measureably increases the oxygen tension and downline tissue levels can rise upwards of 200mmHg.

Q: How did you calculate that?

A: The solubility of oxygen in water (liquid of whole blood) is 0.0236mlO2/ml/atmPaO2. So, times 100ml (the relative unit) times the amount of atmospheric pressure and you get near the arterial partial pressure of oxygen, minus losses for dilution and transfer through the lungs.

Q: So how does being inside a pressurized chamber give us more oxygen?

A: When we sit inside a chamber pressurized at twice the normal air pressure it may not feel different, but we breathe double the number of molecules. Breathing pure oxygen in such a chamber gives us 10 times the regular amount of oxygen. In one hour we can inhale 2.4 pounds of oxygen! Red blood cells instantly fill with oxygen and the extra oxygen dissolves directly into the blood fluid. In a few minutes this extra oxygen builds up tissue oxygen levels far above normal. This action has been scientifically proven to stimulate healing. In order to raise tissue oxygen tension to 80 mmHg for optimal healing one must have oxygen delivered

under increased atmospheric conditions. This is not a plastic bag but a solid chamber certified to safely hold the high pressure. How high? The pressure against a 30" hyperbaric chamber hatch at 2 atmospheres has about 10 tons of pressure exerted against it!

Q: What is the history of HBO2?

A: The first pressurized room used to treat health problems was built by an Englishman named Henshaw in 1662; however, it was not until over a century later in 1788, that compressed hyperbaric air was put to large scale use in a diving bell for underwater industrial repairs of an English bridge. The first deep sea diving suit, invented in 1819 by August Siebe, used compressed air supplied to the helmet for generous underwater movement. A French iron shop in 1834 built the first hyperbaric tank under the direction of Dr. Junod. A copper sphere five feet in diameter with the appropriate viewports and compressed air fittings became the center of attraction for many patients. He reported wonderful recovery from a variety of debilitating conditions in the Bulletin of the Academe of Medicine. Hyperbaric enthusiasm spread among the European countries during the next forty years. Sick people came from America to try the new therapy. An enterprising Canadian built the first North American hyperbaric chamber in 1860.

Early French hyperbaric assisted surgery demonstrated that patients recovered with fewer complications. This interested the European medical profession. Dr. John S. Haldane studied the effects of compressed oxygen and taught at the University of Dundee in the early 1900's. He developed the first diving tables for the Royal Navy. His legacy gives him the title "Father of Oxygen Therapy" and physicians continue in his line of work to this day.

In 1918 Dr. Orval Cunningham considered the differences between people living or dying through the flu epidemic in the Rocky Mountains. He noticed people in the valley fared better than people in the mountains. He reasoned that denser air in the valley helped people fight the infection. He had an 8' diameter by 30' long hyperbaric chamber built next to his medical clinic. His good outcomes with patients suffering from pneumonia encouraged him to build other chambers. He built the world's largest functional hyperbaric chamber, a 64' steel sphere "hyperbaric medical hotel" with five floors of living space. The Great Depression in the 1930's ended his project and the giant chamber was scrapped for the war effort in the 1940's.

Harvard Medical School had a hyperbaric chamber built in 1928. It provided a tool for years of research. Public interest for HBO2 started to grow in the 1960's after publicity about its use on President Kennedy's sickly infant. In the last three decades great strides in HBO2 research has raised the value of this unique therapy. University studies have expanded the list of conditions usefully treated with compressed oxygen. Doctors used to ask, "Can it work?" Now they ask, "How much is needed to completely work?"

Q: Why are HBO2 treatment outcomes for stroke or other brain injuries so varied?

Q: Is there any reason to believe that the beneficial effects of HBO2 would continue beyond treatment endpoint? If so, approximately how long and why?

A: Barr and Perrins published some observations on this matter in the Proc.11th International Congress 1995 (ISBN 0-941332-44-6). Briefly, they showed tissue oxygen partial pressure measurements that rose from near zero to 50 mmHg after some months long course of HBO2 were retained without further treatment for at least three years! They tought they were witnessing a vascular 'medical disobliteration'. Whether this is due to recanalization of atrophied vessels or in-growth of neovasculature is open to question.

A: The ultimate clinical status and recovery with brain injuries depend upon: the size and location of the epicenter or core of irreversible destruction; the surrounding zone of dormant but recoverable neurons (ischemic penumbra) fanning out in varying rings from the center core; the asymmetry involved; the reorganization and plasticity of altered and nonimpaired sensory and motor brain functions; meshing of sensory and motor fibers at the brain stem cord junction; progression of temporal intervention with HBO2.

Summary of the effects of HBO2 both in acute and semi-acute and long-term neurologic conditions: HBO2 reduces any pressure within the brain caused by swelling, restoring the functions of the blood brain barrier and cell membrane; It neutralizes toxic products in the brain, and over a period of time, enhances growth of new blood vessels; It also acts as a scavenger of free radicals and promotes internal cleaning of debris; HBO2 also reduces the stickiness of blood products (white blood cells and platelets), and makes oxygen available for use without energy transfer (when the hemoglobin carries oxygen, it requires energy to deliver to the tissue spaces); With HBO2 the free oxygen is available immediately for metabolic use.

Hyperbaric Oxygen and Ischemia

Hyperbaric oxygenation has proven benefits in reversing the effects of ischemia.

Oxygen is the most essential substrate for metabolism. We only function by oxidative metabolism and the reason for restoring blood flow to the brain with CPR is to establish an oxygen supply.

Hypoxia (low oxygen levels in tissue) hinders healing. The sooner that tissue hypoxia is corrected the better the outcome. Many hypoxic tissues require hyperbaric pressure to achieve a significant increase in oxygen delivery because of poor oxygen solubility in blood. Although pure oxygen is easily available around the world why is the use of high dosage oxygen not accepted and used

despite many thousands of publications, including controlled trials, attesting to its value? Here are eleven reasons provided by Dr. Philip James:

I. Oxygen transport is determined by the percentage respired and the barometric pressure. In normal hospital practice barometric pressure is ignored and it is assumed that patients receiving 100% are being given the same amount. In the UK this is not important but in, for example, Denver, Colorado, which is at an altitude of over 5000 feet, the partial pressure is significantly lower than at sea level and a hyperbaric chamber is needed to give the same amount of oxygen as at sea level.

II. Tissue hypoxia may be present in the absence of cyanosis. Oxygen supplementation is accepted in the alleviation of cyanosis, where the absolute level of deoxygenated hemoglobin exceeds 5g /100 ml of blood. However, the presence of cyanosis requires blood to be present in the microcirculation of a tissue and there can be significant hypoxemia without cyanosis when the hematocrit is low or when there is microcirculatory closure.

III. Plasma oxygen transport is not limited by the saturation of hemoglobin. It is common for physicians to argue that blood is saturated with oxygen when a normal oxygen partial pressure (0.21 atm abs) is breathed at sea level. However it is not blood that is saturated, it is hemoglobin. The transport of oxygen by hemoglobin is finite as each of the ferrous receptor sites on the molecule can only bind one oxygen molecule. However, the plasma oxygen content increases directly as a function of the inspired partial pressure of oxygen. Breathing pure oxygen at twice atmospheric pressure, the plasma oxygen content is ten times the value of breathing air at sea level and life can be sustained without hemoglobin (continued consciousness may need higher pressure).

IV. Oxygen transport to tissue depends on the tension of oxygen in plasma. Severe tissue hypoxia can be present when arterial oxygen tensions are normal if local circulatory factors, such as arterial occlusion, closure of the microcirculation and edema are present. An increase in the water content of tissue limits oxygen transport. If inflammation, edema and the invasion of metabolically active inflammatory cells occur at the same time, we can have hypoxia even when the blood flow per unit volume of tissue is increased, hence hyperemic hypoxia. In hyperbaric conditions the oxygen plasma tension increases from values of 95mm Hg to over 2000 mm Hg increasing the gradient or the transfer of oxygen into tissues by 20 fold.

V. Normal blood flow does not ensure normal oxygenation. Oxygen delivery requires blood flow, although blood flow may be normal and the tissue still hypoxic. The only tissue that does not need blood flow for oxygenation is the lung.

VI. Oxygen is not "Hyperbaric". The use of the term "hyperbaric" may appear to imply that the oxygen delivered is different to the molecular oxygen available from the air. People may think of it as singlet oxygen, 01, or ozone, O3. The correct terminology is hyperbaric oxygenation or hyperoxia. The psychology of the word "hyperbaric" indicates a potential education or marketing problem.

VII. The adjunctive nature of most oxygen supplementation. Oxygen may be a primary treatment in some instances, but the impression is often given that oxygen therapy replaces other treatment. In most cases this is incorrect, other therapy is needed and optimal care is not a competition between therapies.

VIII. The paradox: hypoxia causes oxygen free radical toxicity. Paradoxically it is hypoxia that mediates the release of oxygen free radicals. An inadequate oxygen supply to tissue results in the catabolism of ATP to adenosine and the creation of an electron donor, xanthine. When oxygen is made available the electron is accepted to form the superoxide anion 02. A cascade of interactions leads to the generation of the hydroxyl ion which is capable of damaging membranes and allowing calcium into the cell. It is important to recognize that this is caused by hypoxia. Correcting hypoxia will limit the extent of free radical formation.

IX. Hyperoxia and oxygen toxicity. It is well known that exposure to pure oxygen for a prolonged period, that is, in excess of 24 hours at 1 atm abs causes reversible damage to the endothelium of pulmonary capillaries. Short term exposure to very high oxygen pressures, for example, 3 ATA for 2 hours may cause convulsions resembling grande mal epilepsy. The time to convulsion is reduced by exercise or an increased metabolic rate. It is suspected that oxygen free radicals are involved in these toxic effects. However the

clinical use of hyperbaric oxygen uses well-defined exposure limits where neither of these effects are significant. The sites where autoregulation may fail to limit blood flow are the ends of fingers and toes. This is because arteriovenous shunts are present to return blood in vasodilatation and results in blood flow which is greatly in excess of tissue requirements. Toxicity to peripheral nerve endings is often manifest as parasthesia. Pre-existing epilepsy does not lower the threshold to oxygen toxicity, and epilepsy can be treated by HBOT.

X. Unfamiliar technology. Hyperbaric medicine is not generally familiar to most physicians because it is rarely taught in medical schools. Those who are involved have generally come from the fields of aviation or diving. As both of these disciplines use high technology, it is not surprising that hyperbaric oxygen itself is viewed in this light. However, the pressures used clinically, up to a maximum of 3 ATA, are very modest in comparison to the maximum human experimental pressurisation of 71.1 atm abs. Unfortunately even physicians familiar with hyperbaric medicine refer to "fitness to go under pressure," forgetting that we are all subject to normal atmospheric pressure. Also, it is outside our pharmaceutical paradigm in the west. In other cultures it has been more readily accepted. The

HBO2 approach has largely come after the tablet/injection approach was developed and therefore takes second place in healthcare. HBO2 has to jump higher "proof" hurdles to be accepted.

Xl. Finance. The use of increased pressure requires a hyperbaric chamber and therefore some financial investment. In the case of a walk-in multiplace chamber this can be considerable and there are usually building modifications required. There is no commercial promotion of oxygen in the pharmaceutical sense to make physicians aware of hyperbaric oxygenation's value. This will not change and is a major reason for the slow growth of oxygen as a therapy. No promotion without a patent! No matter how much scientific evidence we produce we need marketing and no one will make that investment without a return. We have more scientific evidence about actions and mechanisms supporting the correction of tissue hypoxia than any pharmaceutical product. Breathing oxygen actually reduces the volume of a pneumothorax by increasing the inherent unsaturation and gradient for nitrogen elimination.

Other reasons why HBO2 chambers are not in every doctor's clinic:

It is very clear there is a general failure to understand the fundamental importance of oxygen in human physiology. If this were not the case, HBO2 would already have become just another tool used in the day-to-day practice of medicine as are pills, surgical knives and injections. Perhaps a major barrier to gaining greater acceptance within the medical community at large is the persistence in referring to clinical HBO2 treatments as "dives".

Diving and clinical hyperbaric medicine are not the same thing. Diving relates to underwater military, commercial or amateur activities, recompression is necessary when things go wrong, it is not a choice if you wish to resolve a DCS problem. In clinical applications patients do not go anywhere near the water; they are pressurized for the specific purpose of increasing tissue oxygen tensions in order restore or assist the healing process. The term "fitness to dive" is another diving term and relates to the ability of an individual to deal with the physiological stress of deep diving and working underwater.

The whole objective of pressurizing a clinical patient is to increase tissue oxygen tensions in conditions where HBO2 is beneficial. This would not be necessary if they were "fit". A patient in a chamber breathing 100% oxygen is under less physiological stress rather than more because of the benefits derived from the oxygen. A simple risk analysis suggests that the risk of ear squeeze associated with hyperbaric treatment, is considerably less than risk associated with radical surgery, limb loss or death from multiple organ failure.

There are many precautions that can be taken to reduce the risk associated with treatment in any medical modality, clinical HBO2 is no different. Slow down rate of pressure change, insert grommets and give vitamin E are just a representative

sample. It is not "fitness to dive" that is the issue, just responsible medical practice. The rate of impending or actual aural barotrauma (ear pain) requiring aborting of a treatment on compression is less than 3% of total attempted treatments, and mostly due to people with a history of head and neck irradiation and eustachian tube dysfunction, complex head and neck surgery and those with residual CNS depression from drugs. Calling hyperbaric sessions "dives" does contribute to the underuse of HBO2 therapy.

Dr. Philip James' notes that in Great Britain their hyperbaric facilities have safely done over 1.2 million patient sessions without incident. He says that, "Engineering standards are of primary importance but adequate training for the operation of chambers in a non-acute setting requires only basic information. It is sensible to supplement the growth period in children having brain damage as there is a syndrome of delayed deterioration after birth injury."

For information on hyperbaric oxygenation in brain injured children contact Mrs Linda Scotson, at the Hyperbaric Oxygen Trust for Brain Injured Children, Ryton House, Forest Row, East Sussex England.

Vitamin Use in Hyperbaric Oxygen Therapy:

Dr. Richard Kelton, hyperbaric physician at the Greater Baltimore Medical Center, believes that vitamins A, C, and E should be used in conjunction with hyperbaric therapy to treat central nervous system conditions. There is some debate about this. These vitamins are free radical scavengers.

On one side of the debate are those who believe that treatment of necrotizing infections should be advanced by the release of free radicals, and that vitamins should not be used. On the other hand, these vitamins may reduce oxygen toxicity in the central nervous system or the lungs. Some believe that vitamin E should be withheld when wound healing is desired, since it is known to impede collagen formation.

Unfortunately, as most acknowledge, all of this is conjecture, since there is no data upon which to rely. Nevertheless, for hyperbaric therapy of autism and cerebral palsy, adjunctive use of vitamins seems desirable. Dr. Kelton can be reached at rkelton@gbmc.org .

Published papers regarding Hyperbaric Oxygenation for Anoxic Encephalopathy and Coma:

1. Neubauer RA. The effect of hyperbaric oxygen in prolonged coma. Possible identification of marginally functioning brain zones. Medicina Subacquea ed Iperbarica. 1985: (3) 75-79.

17 cases of vegetative coma for 1 - 22 months. 40 - 120 exposures over 20 - 90 days. Glasgow Coma Scale improved in all. Complete recovery of 5.

2. Eltorai I, Montroy R. Hyperbaraic Oxygen Therapy leading to recovery of a 6-week comatose patient afflicted by anoxic encephalopathy and post-traumatic edema. J. Hyperbaric Medicine 1991: (3) 189-198.

90 mins HBO2 od. After 24 sessions, started talking and ate meals. Gradually mobilised to a wheelchair.

3. Harch PG, et al. SPECT brain imaging and low pressure HBO2 in the diagnosis and treatment of chronic traumatic, ischaemic, hypoxic and anoxic encephalopathies. Undersea and Hyperbaric Med. 1994 (Supp)

4/5 showed improvement in focal cortical & deep grey matter deficits.

4. Shn-rong Z. Hyperbaric Oxygen Therapy for Coma - report of 336 Cases. In Proc XI Intnl Cong Hyperbaric Med. Best, Flagstaff. 1995; 279-285

HBO2 is effective in acute brain hypoxia and oedema and can hasten recovery of consciousness, including prolonged coma.

5. Neubauer RA, Gottlieb SF, Pevsner NH. Long-anoxic ischaemic encephalopathy: predictability of recovery. In Proc XII Intnl Cong Hyperbaric Med. Best, Flagstaff. 1996. (In press).

8 long-term patients with severe anoxic ischaemic encephalopathy between 3 months and 12 years. Improvement in all cases, both clinically and on SPECT scans. Until the introduction of SPECT scanning there has been no diagnostic technique providing evidence that any treatment would be effective.

6. Quinly C, Shaoji Y. Nursing of Brain-Stem injury with HBO2. Ibid.

39 patients treated with HBO2. Decreased mortality and increased awake rate.

7. Zhi Y, et al. Assessment of the efficacy of HBO2 in patients with a persistent vegetative state. Ibid.

8 patients in coma, longest 281 days prior to HBO2. 20 - 86 daily sessions. All resumed consciousness.

8. News article: "High pressure chambers could be used in preventing paralysis"

Washington (May 10, 1998) - High pressure chambers used to treat divers who rise too fast underwater could he used to help prevent paralysis in people with damaged spinal cords, researchers said Sunday. "It may mean the difference

between significant disability and no disability," Dr. Philip James of the University of Dundee in Scotland said in a statement. James said the high pressure chambers forced healing oxygen into the tissues of a damaged spine. If the blood flow is not restored quickly, cells die, resulting in permanent damage.

A consultant to the North Sea diving industry, James told a conference in Washington that hyperbaric chambers used to treat or prevent the "bends" from decompression sickness in divers would also benefit patients who had spinal injuries. "Most trauma centers do not have hyperbaric chambers, which is a tragedy, and most physicians don't understand the need to increase the dissolved oxygen in the plasma of the blood," said James, who presented his ideas to the Space and Underwater Research Group of the World Federation of Neurology.

9. "Hyperbaric oxygen and reflex sympathetic dystrophy: a case report", Peach G. Hyperbaric Medicine Department, Shock Trauma Center, University of Baltimore Medical Center, Baltimore, Maryland. "Hyperbaric oxygen and the reflex sympathetic dystrophy syndrome; a case report". Undersea Hyperbaric Medicine 1995; 22(4):407-408.

A patient suffering from acute smoke inhalation also had a long medical history that included reflex sympathetic dystrophy syndrome of the left foot and ankle. The entire foot and ankle were tender and cool to palpation; range of motion was severely reduced. Shewas referred for hyperbaric oxygen therapy , and 15 minutes into the firsttreatment (46 min at 60 swf) she reported a lessening of the pain in her foot; moreover, the foot was less cyanotic and warmer to the touch. Subsequent treatments continued to improve her conditions and for longer periods of time.

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10. "Hyperbaric Oxygen in the Treatment of Sudeck's Syndrome", G.Lovisetti, L.Lovisetti, A.Favelli; Istituto di Terapia Iperbarica; Como, Italy.

Summary: The decrease in tissue hypoxia obtained with Hyperbaric Oxygenation (HBO2) counteracts the effects of reflex vasomotor disturbances caused by an injury in post-traumatic Sudeck's syndrome.

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Bakker (1988). Amputation rate 8% cf 50-60% without HBO2. In: Problem Wounds - role of Oxygen. Elsevier pp 153-172.

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Pallotta et al. (1986) 8 year follow-up of relapses in 11+11 Paris MS & HBO2 Symposium.

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Rockswold & Ford. (1985). With a ‘Coma score’ of 4-6 two died with HBO2, seven without. Minn Med 68:533-538.

5. Myasthenia Gravis.

Li et al. (1987). Clinical improvement and reduction in IgA & IgM 89% with HBO2, 45% with steroids. Proc IX Intnl Cong. Sydney.

6. Myocardial Infarction.

Thurston et al. (1973). Mortality in high-risk patients reduced by half. Proc. IV Intnl Cong. Sapporro.

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Yefuni et al (1986) HBO2 shortened healing time by 28 days. Proc 1st Swiss Symposium.

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Information about using HBOT for auto-immune diseases:

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6. Wallace, D.J., Silverman, S., Goldstein, J., and Hughes, D. Use of hyperbaric oxygen in rheumatic diseases: case report and critical analysis. Lupus. 4(3):172-175, 1995. Notes : Department of Medicine, Cedars-SinaiMedical Centre, Los Angeles, CA, USA 0961-2033 English England

Hyperbaric oxygen has been used in patients with rheumatic disease for many years without reports of untoward or unusual complications for a variety of non-rheumatic indications. Recent evidence that hyperbaric oxygen inhibits the actions of certain cytokines, acts as an immune modulator and may help cognitive dysfunction has resulted in a re-examination of its potential role in rheumatic diseases. A case report of a lupus scleroderma crossover patient is presented whose cognitive dysfunction improved after hyperbaric oxygen therapy. The history of hyperbaric oxygen and its physiology are related, along with a focused review of its effects on the immune and central nervous systems.

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Hyperbaric oxygenation treatment of systemic scleroderma has a favorable effect on microcirculatory changes whose positive dynamics can be demonstrated by conjunctival biomicroscopy. These changes include accelerated blood flow and decrease in the degree of erythrocyte aggregation. The method can be used or the objective assessment and for prognosis of the effectiveness of hyperbaric oxygenation treatment in patients with systemic scleroderma.

Treatment of Children's Epilepsy by Hyperbaric Oxygenation:

STUDY:

Treatment of Children's Epilepsy by Hyperbaric Oxygenation: Analysis of 100 Cases. Hyperbaric Oxygen Treatment Centre, Zhou Gulan, EEG Lab, Zhujiang. Proceedings of the Eleventh International Congress on Hyperbaric Medicine; President: Wen-ren Li, M.D., Fuzhou, People's Republic of China; Secretariat: Frederick S. Cramer, M.D., San Francisco, California, U.S.A.

Gender and age:

The whole group included 100 patients (72 males, 28 females), ages 4 days to 14 years. 84% of them were between 1 month and 9 years old.

Causes of disease:

23 patients cause unknown (primary epilepsy), others had the following causes: cerebral lesion due to birth injury in 55 patients; encephalitis in 14 patients; high fever in 2 pediatric patients; anoxic cerebropathy in 4 children; brain tumor in 1 child; cerebrovascular malformation in 1.

Neuropsychiatric manifestation:

Intelligence was impaired in 68 patients: 45 children had mental symptom and personality change; local neurosystemic signs were detected in 47 patients. Patterns of Seizures: Grand mal 32 - Psychomotor seizures 12 - Petit mal 10 - Focal seizures 44 - Autonomic symptoms 2. EEG examination: All patients in this group had an EEG test. lt was found that 92 patients had abnormal EEGs; 66 patients had focal sparkle or sharp wave; 10 patients had paroxysmal sparkle-slow wave and sharp-slow wave; 6 patients had paroxysmal cerebral dysrhythmias; 10 patients had confusing abnormal EEGs; 3 patients had normal EEG&s; 5 other patients had boundary EEGs.

CT and MRI scanning:

Seventy-six patients were proved abnormal, including ventricular enlargement due to atelencephalia, focal encephalatrophy, tumours and local low density pathy, skull fracture. The other 24 patients were normal. Seizure frequency: 21 patients seizured every week; 18 patients did every month; 23 patients did every two months, the other 38 patients seizured more than twice a year.

TREATMENT:

Anticonvulsant medication:

39 patients were treated systematically; 20 patients could be controlled by little diazepam and r-amino butyric acid; 41 patients received no anticonvulsant because of their parents' objection, since they thought the children were too young; some individuals were controlled by luminal intramuscular injection on convulsion.

Hyperbaric oxygenation treatment:

The private hyperbaric oxygen chamber was manufactured by Ninpo Hyperbaric-Oxygen Chamber Factory. ln the chamber, the pure oxygen pressure is 1.7-2.0 atmospheres. The patients were treated for 80 minutes every day. A course was 15-30 days. Some patients had therefore been treated 35-45 times.

Outcome:

The treatment was found effective in 82 patients (82%), significantly effective ln 68 patients (68%). It showed that the seizures greatly diminished, and the EEG

was improved. Forty-three patients had stopped anticonvulsant medication, while in other patients the amount of antiepileptic was decreased.

After hyperbaric oxygenation treatment, 82 patients' intelligence, personality, and mentalities were improved; 51 children studied very well; 10 primary and 4 secondary epilepsy children had no change after being treated 30 times.

Electroencephalogram (EEG): After hyperbaric oxygenation treatment, 45 patients had normal EEGs; 28 patients had focal abnormal EEGs; 3 patients had paroxysmal sharp-slow wave and another 20 patients' EEGs were slightly abnormal; 4 patients had boundary EEGs.

Follow-up: Seventy-six patients had been observed for more than 3 years. Forty children had been completely free of anticonvulsants. Three children had 1 or 2 slight attacks every year. Twenty-five patients were administered a little anticonvulsants and their seizures diminished a lot. The attacks did not change in 11 children with systemic therapy.

DISCUSSION:

Mechanism of treatment of children's epilepsy with HBO2:
Hyperbaric oxygenation could improve the cerebral circulation, provide the brain with more oxygen, and reduce edema. Hyperbaric oxygen could also promote the energy metabolism of cerebral cells and improve the recovery of epileptic foci.

Reduction of handicapped children due to epilepsy:
Epilepsy often impairs the children's intelligence and personality; hyperbaric oxygenation could not only control the attacks of epilepsy but also prevent the occurrence of intelligence impairment and abnormal personality, so as to diminish the ratio of handicapped children due to illness.

The way of gaining a good effect in the treatment:
The effect is good in the cases whose causes are known, especially those caused by brain damage due to birth injury. As to the period of treatment, most patients need 2-3 years. If the infants do not have high fever or respiratory inflammation, the treatment can begin from several days after birth. Fifteen to 20 days make a course, and 2 courses a year.

[excerpted from: the Hyperbaric Oxygen Trust website]