

Parkinson's and HBOT

Parkinson's disease or PD is a disorder of the central nervous system that impairs motor skills, speech and other mental functions. PD belongs to a group called movement disorders. These disorders typically produce muscle rigidity, tremor and a general slowing of physical movement.

PD affects adults of all ages but is not considered fatal. However, late-stage Parkinson's may lead to choking, pneumonia or serious falls, all of which can cause death or disability.

What HBOT can do

The symptoms of Parkinson's can vary in type and severity therefore it can be difficult to predict results. Often HBOT has been discovered effective by "accident" as in the case where a diabetic patient tried HBOT for a foot ulcer but found that the treatment vastly improved his Parkinson's symptoms. After 50 years of HBOT treatment we do know that hyperbaric oxygen will not worsen PD. Numerous animal studies have shown that HBOT works as an anti-inflammatory and may be used in this way to alleviate the symptoms of PD.

Why is Parkinson's Disease amenable to oxygen therapy?

Extensive animal research has demonstrated a non-specific chronic inflammatory condition in the substantia nigra of the brain. Hyperbaric Oxygen Therapy (HBOT) has been shown to be an anti-inflammatory drug in many conditions. Anecdotal evidence of many patients with well established PD have been treated with HBOT for other conditions such as diabetic foot ulcers. For example, a patient with advanced PD who is confined to a wheelchair may get up and walk across the room after a series of HBOT.

What benefits can I expect from oxygen therapy for Parkinson's Disease?

Since every patient is different it is hard to predict the result in each individual case. However, we know from 50 years of experience that HBOT is safe and will not make the patient worse. The usual course of oxygen therapy is once daily, five days a week (M-F) for eight weeks. If a significant response is noted after 40 HBOT additional treatments may be helpful.

One-Two Punch: Glutathione and HBOT

Julian Whitaker, MD

Parkinson's disease is caused by the degeneration of neurons in the area of the brain that manufactures dopamine, a neurotransmitter that affects movement. As dopamine production drops, characteristic tremors and speech, balance, and motor problems develop. The primary treatment for Parkinson's is drugs that replace or mimic dopamine, and though these meds improve symptoms, they do not slow disease progression and their side effects increase with long-term use.

Although there's a lot that medical science does not know about Parkinson's, we do know that free-radical damage contributes to its progressive nature. That's why we use glutathione.

Glutathione/HBOT to the Rescue

Glutathione is a powerful natural antioxidant, and patients with Parkinson's have dangerously low levels of glutathione in the affected area of the brain. Boosting stores of this protective antioxidant not only guards against further damage, it also enhances the function of surviving neurons.

Unfortunately, oral glutathione has a hard time crossing the blood-brain barrier, so supplements aren't very helpful. When glutathione is infused intravenously, however, it hits its target. Most patients see dramatic improvements after just a handful of treatments—and many perk up after their first infusion. Even better, studies suggest that benefits last for two to four months after a treatment course.

Our patients with Parkinson's disease are also treated with hyperbaric oxygen therapy. HBOT is highly beneficial for stroke, multiple sclerosis, and brain injuries. It floods the brain with oxygen, slows neuronal degeneration, mobilizes rejuvenating stem cells, and enhances angiogenesis (the growth of new blood vessels that nurture damaged areas). It is the combination of these two treatments, working synergistically, that provides such remarkable results.

Serious Condition, Serious Intervention

Parkinson's is a serious condition that requires serious intervention. Coenzyme Q10, vitamin E, fish oil, curcumin, creatine, and vitamin D, along with N-acetyl-cysteine and vitamin C (both of which boost glutathione levels), show promise in improving symptoms and even slowing progression. I certainly recommend taking them. However, these supplements do not come close to approaching the therapeutic power of IV glutathione and HBOT.

Sadly, very few medical facilities offer these therapies. In fact, many physicians don't know a thing about them! Furthermore, although benefits are enduring, they don't last forever, and maintenance treatments are required for optimal function.

I don't know what it's going to take to get conventional physicians to embrace IV glutathione and HBOT, but don't hold your breath waiting for your doc to come around. I strongly urge you to find a treatment center near you, and if you can't, consider coming to the Whitaker Wellness Institute.

Recommendations

The suggested daily doses of supplements for Parkinson's disease are coenzyme Q10, a minimum of 1,200 IU; vitamin E, 400–800 IU; fish oil, 6–8 g; curcumin, 1,000–2,000 mg; creatine 10 g; vitamin D 2,000–4,000 IU; N-acetyl-cysteine 1,200–1,800 mg; and vitamin C, 1,000–2,000 mg. Look for them in your health food store or call (800) 810-6655 to order. Take in divided doses.

References

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Hyperbaric Oxygenation During the Complex Treatment of Parkinsonism

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Hyperbaric oxygenation (HBO2) was used to treat 64 patients suffering from parkinsonism of diverse etiology. HBO2 sessions were provided daily) 8-12 per course; treatment pressure ranged from 1.3 to 2 atm and exposure time ranged

from 40 to 60 minutes. A marked beneficial effect was noted in 55 patients. HBO2 treatment produced better results in the presence of vasillar parkinsonism, in patients under 65 years of age, and when the history of disease ranged from 1 to 5 years. The akineticorigid syndrome regressed to a greater extent, with HBO2 proving to be less effective when trembling hyperkinesia was present Submitted to the editorial office on 03 March 1988

Hyperbaric Oxygen Treatment on a Parkinson's Disease Patient: A Case Study

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INTRODUCTION

Parkinson's Disease (PD) is a chronic neurodegenerative disorder, which is characterized by the loss of dopaminergic neurons whose cell bodies are located in the substantia nigra pars compacta (SNpc) and project to the striatum. The initiation of this neuronal degeneration is not known, however the process of neuronal loss is suggested to occur via apoptosis rather than by necrosis (1). With the onset of the neurodegeneration of these neurons is the associated loss of the neurotransmitter, dopamine (DA), from its nerve endings and its subsequent release in the striatum. The major symptoms which are observed due to the progressive loss in function of the nigro-striatal dopaminergic neurons may be one or more of the following: resting tremor, rigidity, bradykinesia and/or postural instability. The actual clinical manifestation of the disease in any one patient is highly dependent upon the degree of severity of the neuronal loss, age of the patient and the length of time passed between the onset of the symptoms and the time of diagnosis. Early detection is important in order to institute a therapeutic strategy to relieve the symptoms and/or delay the progression of the disease state.

The major treatment strategy currently used is to affect the function of DA. Because systemically administered DA does not cross the blood-brain barrier; Levodopa (pro drug) is administered, which is taken up into the brain. Since Levodopa is metabolized both peripherally and centrally to DA by a DOPA decarboxylase, carbidopa an inhibitor of this enzyme is administered in combination with Levodopa to decrease its metabolism peripherally increasing its uptake into the brain. DA agonists and monamine oxidase-B (MAO-B) inhibitors are also administered as a monotherapy or as an adjunct to Levodopa-carbidopa (Sinemet) therapy, depending upon the clinical condition.

Taking a very different approach in the treatment of PD, Borromei et al. in 1996 showed that hyperbaric oxygen (HBO) therapy appeared to be effective in ameliorating many of the behavioral and motor deficits observed in PD patients (2). The objective of this study was to determine whether HBO therapy might enhance the effects of an antiparkinson treatment in a PD patient as an adjunct therapeutic modality.

METHODS

Brief patient history: A 72 year old male was diagnosed with idiopathic PD and placed on Sinemet (10/100) three doses 3 times daily. One year after diagnosis for PD the patient was diagnosed with total occlusion of the right coronary artery. A successful total occlusion angioplasty was performed and he was placed on Lopressor and Lipitor 10 mg daily. There were no complications from this surgical procedure. Eighteen months after being diagnosed as a PD patient he was treated with hyperbaric oxygen (HBO) at 1.9 ATA for 90 min. The patient was treated daily 5 times each week for 5 weeks (25 treatments). During the treatment the patient gradually reduced his dose of Sinemet until he was completely off of this medication between the 3rd and 4th week of HBO treatment. At this point his physician placed him on selegiline 10mg twice daily.

Clinical testing: The patient's voice and speech were evaluated by a speech-language pathologist, and the Jebsen-Taylor hand function test was performed by an occupational therapist prior to and after the end of the HBO therapy. The patient was informed of all aspects of hyperbaric oxygen therapy, including all risks of adverse effects according to the Declaration of Helsinki. The patient also signed an informed consent form detailing the treatment and the rights of the patient.

RESULTS

Voice and speech. There was little change in the overall evaluation of voice and speech after HBO therapy. Communication status changed very little. He appeared to be talking more and his rate was somewhat improved. He still had difficulty projecting his voice.

Jebsen-Taylor Hand Function Test. The results of this test are shown in Table 1. In testing the dominant hand there were small increments of improvement after HBO. The total improvement was more than 10%, while the improvement in the non-dominant hand was nearly 32%.

During the treatment period, the patient voluntarily reduced his Sinemet doses until he was completely off the drug after 3-4 weeks of HBO therapy, which was an unexpected result. He has continued to remain off of Sinemet therapy. No complications or adverse side effects such as myopia were observed. The long-term exposure of HBO was tolerated well by the patient.

DISCUSSION

PD is characterized by the loss of dopaminergic neurons of the nigro-striatal pathway. It is not clear how this neuronal degeneration is initiated, but there appears to be a number of potential ways in which this might occur in any one individual, including genetics, disease, drugs or other chemicals, oxidative stress and/or other environmental factors. However, once it is initiated there seems to be agreement that the degenerative process involves apoptosis and not necrosis.

The results of this study suggest that HBO might be a possible new modality of treatment for PD because it appeared to be able to replace Sinemet as a therapeutic regimen. The mechanism by which the HBO effect might be occurring may be partly due to an anti-apoptotic effect. It has been shown that HBO increased the expression of Bcl-2 protein, a major anti-apoptotic protein, in treating forebrain cerebral ischemia in gerbils (3). The Bcl-2 protein has also been elevated by repeated HBO treatment in normal gerbils (4). So it is possible that HBO in this study inhibited the apoptotic pathway involved in the progressive neuronal degeneration by stimulating the expression of the Bcl-2 proteins.

Other possible HBO effects should not be discounted such as improved oxygen perfusion due to increased extravascular oxygen diffusion and to possible angiogenesis (5). Axonal repair and regeneration and/or synaptogenesis could occur due to increased expression of neurotrophin(s), since HBO has been shown to increase vascular endothelial growth factor (6) and act synergistically with platelet derived growth factor and transforming growth factor-beta (7).

The results of this case study agree with much of the results observed in the clinical study by Borromei and his coworkers. It is not clear from their study whether some of their patients were concurrently being treated with anti-parkinson drugs. In our study, HBO replaced the Sinemet therapy and appeared to improve the clinical condition. Thus, results from this case study suggest that HBO therapy might be a potential therapeutic modality in treating patients suffering from PD without causing untoward side effects such as dyskinesia observed in long-term Sinemet therapy.

In conclusion, we suggest that HBO therapy might be neuroprotective in nature to the nigro-striatal neurons by acting as an antiapoptotic process. This could stabilize neuronal function, thereby potentially decreasing the progression of the neurodegeneration observed in Parkinson's Disease.

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Table 1. Jebsen-Taylor Hand Function Test.

Clinical Testing : Time (in sec)	Pre-HBO	Post-HBO
Dominant Hand		
Writing	14	12
Card Turning	7	6
Manipulating Small Objects	11	11
Simulated Feeding	11	10
Stacking Small Objects	8	5
Lifting Large Light Objects	11	10
Lifting Large Heavy Objects	8	6
Total	70	60
Non Dominant Hand		
Writing	41	28
Card Turning	8	4
Manipulating Small Objects	11	7
Simulated Feeding	12	12
Stacking Small Objects	13	8
Lifting Large Light Objects	9	6
Lifting Large Heavy Objects	7	5
Total	101	70

The Second Wave Of Oxygen: Hyperbaric Oxygen therapy (HBOT)

There is growing interest in the use of HBOT to treat the neurotoxicity induced by long-term alcohol and psychostimulant abuse. Even in the absence of double-blind studies, there is enough positive anecdotal information and case studies to warrant intensive investigation in residential treatment. An example is the case of a 19-year-old serious drug abuser with a preliminary brain SPECT scan that looked as bad as the scan of a demented 74-year-old man. The scan of the drug abuser showed a marked improvement in blood flow after just one HBOT session (Harch, 2007). It is conjectured that the 'mechanism of benefit' involves supplying additional oxygen to hasten the combustion of neurotoxins that accumulate in the brain from the excess intake of alcohol. Previous research from our laboratory showed that raising endorphin levels in the brain increases blood flow in the reward site of the brain (Blum et al., 1985). Synaptose raises brain levels of endorphins by preventing their breakdown. Coupled with an enhanced oxygenation by HBOT, the synergy should translate to improving brain health in addicts. Brain healing is necessary in order to overcome neurological deficits that result from SUD. HBOT has been used to successfully treat a myriad of other neurologically-based disorders including: traumatic brain injuries, cardiovascular accidents, post-traumatic stress disorder, dementia and Parkinson's Disease.

Harch PG and Mccoullough, "The Oxygen Revolution" Hatherleigh Press, 2007 , NewYork page 151–152.

Hyperbaric Oxygenation Therapy

In an Italian study, 55 of 63 patients showed significant improvement after hyperbaric oxygenation therapy. This therapy is beginning to be widely used in neurodegenerative conditions, particularly movement disorders. More and more research is being published on the benefits of HBOT in Parkinson's disease/syndrome.

Research suggests that glutathione, a critically important brain chemical, is deficient in Parkinson's patients and may play a significant role in the treatment of this disease. Glutathione is a powerful antioxidant, and helps to prevent free radical damage to brain tissue. So far, the intravenous use of glutathione has shown promising results in reducing tremors and improving movement and balance.

Ongoing clinical trials suggest that multi-modality therapy, combining intravenous glutathione with Hyperbaric Oxygenation Therapy (HBOT) and nutritional supplementation, may be more effective than glutathione alone. HOC is currently conducting clinical trials on combination therapy for Parkinson's disease and is looking for participants. Please contact Dr. Tasreen Alibhai, ND at 604-520-3941 for further information.

Video References

<http://www.youtube.com/watch?v=ECncQzGXrC4>

http://www.youtube.com/watch?v=nbFs9NN_Mk&feature=related

<http://www.youtube.com/watch?v=78PXByMI5J8>