

REVIEW ARTICLE

Effects of hyperbaric oxygen therapy in the treatment of selected diseases of the nervous system

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ABSTRACT

Hyperbaric Oxygen Therapy (HBOT) has attracted increasing interest as a potential supportive intervention in neurological disorders. By increasing the amount of oxygen dissolved in blood plasma, HBOT enhances oxygen delivery to hypoxic tissues and may influence mechanisms involved in neuroplasticity, inflammation, and tissue repair. Experimental and clinical studies suggest that HBOT may support neurological recovery in selected conditions, particularly following ischemic or traumatic brain injury. However, the effectiveness of HBOT varies depending on the underlying disease, and further high-quality clinical studies are needed to establish its role in neurological rehabilitation.

The article was based on an analysis of data from Google Scholar and PubMed regarding the effects of hyperbaric therapy.

KEY WORDS: Alzheimer's disease, Parkinson's disease, stroke, hyperbaric oxygen therapy

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INTRODUCTION

Hyperbaric oxygen therapy (HBOT) is an established treatment modality that involves breathing 100% oxygen under increased atmospheric pressure, typically between 2.0 and 3.0 ATA. Initially developed for the management of decompression sickness, HBOT is currently used in several approved clinical indications, including carbon monoxide poisoning, chronic wound healing, radiation-induced tissue injury, and selected ischemic conditions [1].

The therapeutic effects of HBOT result from increased oxygen dissolution in blood plasma, which enhances oxygen delivery to hypoxic tissues independently of hemoglobin, in accordance with Henry's law. Elevated tissue oxygen tension stimulates angiogenesis, collagen synthesis, fibroblast proliferation, leukocyte antimicrobial activity, and stem cell mobilization while reducing tissue edema through vasoconstriction without compromising oxygen supply [2,3]. These mechanisms have generated increasing interest in the potential application of HBOT in regenerative and neurological medicine.

The brain is highly dependent on aerobic metabolism, consuming approximately 20% of the body's

oxygen despite representing only about 2% of total body mass. Adequate oxygen availability is essential for mitochondrial ATP production, neuronal signaling, and maintenance of redox homeostasis. Cerebral hypoxia and impaired perfusion contribute to oxidative stress, neuroinflammation, and neuronal dysfunction, which are common features of stroke, traumatic brain injury, vascular cognitive impairment, and other neurological disorders [4–8]. Experimental and clinical studies suggest that HBOT may improve tissue oxygenation and modulate cellular pathways involved in neuroplasticity and neurorepair, although the clinical evidence remains heterogeneous and requires further investigation.

Therefore, HBOT has emerged as a promising adjunctive therapy in neurological rehabilitation, warranting continued evaluation of its mechanisms of action and clinical effectiveness.

MATERIALS AND METHODS

The article was developed based on an analysis of data from Google Scholar and PubMed regarding the effects of hyperbaric therapy.

REVIEW AND DISCUSSION

THE STATE OF KNOWLEDGE

Hyperbaric oxygen therapy (HBOT) exerts its effects on the central nervous system by increasing both ambient pressure (typically 1.5–3.0 ATA) and oxygen concentration, thereby markedly enhancing oxygen solubility in blood plasma. This facilitates oxygen diffusion to hypoxic neural tissues independently of hemoglobin and improves tissue oxygenation in regions with impaired perfusion [9].

The neuroprotective effects of HBOT are mediated through several complementary mechanisms. Increased tissue oxygenation promotes cerebral vasoconstriction while maintaining adequate oxygen delivery, contributing to a reduction in intracranial pressure and cerebral edema. At the cellular level, HBOT supports mitochondrial respiration, enhances ATP synthesis, and limits excessive production of reactive oxygen species, thereby reducing neuronal apoptosis and necrosis following hypoxic or ischemic injury. In addition, hyperoxia activates signaling pathways involved in neurogenesis and angiogenesis, including the HIF-1 α /VEGF axis, and increases the expression of neurotrophic factors such as brain-derived neurotrophic factor (BDNF) and nerve growth factor (NGF), which are essential for neuronal survival and synaptic remodeling [9].

HBOT has also been shown to modulate neuroinflammatory processes by suppressing microglial activation and decreasing the production of pro-inflammatory cytokines, including tumor necrosis factor- α (TNF- α) and interleukin-1 β (IL-1 β). Furthermore, experimental studies suggest that hyperbaric oxygen may promote autophagy, facilitating the clearance of pathological protein aggregates and supporting synaptic plasticity [9,10].

Clinical and neuroimaging studies have demonstrated that HBOT may improve cerebral perfusion and functional recovery following traumatic brain injury, ischemic stroke, and selected neurodegenerative disorders. Improvements in cognitive performance and motor function have been reported after repeated treatment sessions; however, the magnitude of these effects varies across patient populations, and further well-designed randomized controlled trials are required to establish the long-term efficacy of HBOT in neurological rehabilitation [9,10].

INNOVATIVE APPROACHES TO HYPERBARIC OXYGEN THERAPY IN SELECTED MEDICAL CONDITIONS

ALZHEIMER'S DISEASE (AD)

Alzheimer's disease (AD) is the most common neurodegenerative disorder, characterized by progressive cognitive decline associated with extracellular accumulation

of β -amyloid (A β) plaques, intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein, synaptic loss, neuronal degeneration, and gliosis [11]. Despite significant advances in understanding its pathophysiology, no curative treatment is currently available, and therapeutic strategies remain primarily symptomatic.

Hyperbaric oxygen therapy (HBOT) has been proposed as an adjunctive intervention in AD, aiming to improve cerebral oxygenation, reduce hypoxic injury, and modulate neurodegenerative processes. Standard therapeutic protocols typically include 40–60 sessions lasting 60–90 minutes at pressures ranging from 1.5 to 2.0 ATA. Under these conditions, increased plasma-dissolved oxygen enables enhanced delivery to metabolically compromised brain regions, particularly the hippocampus and cerebral cortex, which are critically affected in AD.

Experimental and clinical data suggest that HBOT may exert multifactorial effects, including modulation of neuroinflammation, reduction of oxidative stress, and activation of pathways associated with neuroplasticity, such as BDNF and VEGF signaling. Additionally, HBOT has been associated with enhanced autophagic processes potentially involved in the clearance of pathological protein aggregates, including β -amyloid and tau.

Clinical studies have reported potential cognitive and functional benefits in patients with mild to moderate AD. In a randomized controlled trial by Harch et al. (2012), 40 HBOT sessions (1.5 ATA, 90 minutes) resulted in significant improvements in Mini-Mental State Examination (MMSE) and Alzheimer's Disease Assessment Scale–Cognitive Subscale (ADAS-Cog), accompanied by increased cerebral glucose metabolism in frontal brain regions assessed by PET imaging. Similarly, Shapira et al. (2020) observed cognitive improvement and reduced amyloid burden in patients undergoing HBOT, with corresponding neuroimaging changes.

Meta-analytical data from randomized controlled trials further indicate that HBOT may improve cognitive performance and activities of daily living in patients with AD and mixed dementia, with the most pronounced effects observed in protocols involving at least 40 sessions at 2.0 ATA. Reported adverse events are generally mild and primarily related to barotrauma of the middle ear, without evidence of severe oxygen toxicity in analyzed studies. However, heterogeneity of study designs and limited long-term follow-up necessitate cautious interpretation of results [12].

Overall, current evidence suggests that HBOT may represent a promising adjunctive therapy in Alzheimer's disease, potentially improving cognitive function and metabolic brain activity. Nevertheless, it should be

Table 1. Meta-analysis of the effects of hyperbaric oxygen therapy on cognitive function, motor symptoms, and sleep parameters in Parkinson’s disease

Scala/Parametr	Change in the group HBOT (MD/SMD, 95% CI)	p- value	Heterogeneity (I ²)
MoCA (cognitive functions)	MD=2,45(1,23-3,67)	<0,001	28%
MMSE	SMD=0,68(0,15-1,21)	0,012	42%
PDSS (sleep quality)	MD=4,72(2,10-7,34)	<0,001	15%
PSQI	MD=-2,18(-3,45 to -0,91)	0,001	35%
Kubota swallowing test	SMD=0,82(0,29-1,35)	0,002	22%
UPDRS III (Motor Symptoms)	MD=-5,63(-8,92 to -2,34)	<0,001	51%
Total sleep time	MD=45 min(22-68 min)	<0,001	18%
Sleep efficiency	MD=8,2%(4,1-12,3%)	<0,001	29%

considered experimental in this indication, and further large-scale randomized controlled trials are required to determine optimal treatment parameters and long-term efficacy [12,13].

PARKINSON’S DISEASE (PD)

Parkinson’s disease (PD) is a progressive neurodegenerative disorder characterized primarily by the degeneration of dopaminergic neurons in the substantia nigra, associated with motor and non-motor symptoms. Key pathogenic mechanisms include oxidative stress, mitochondrial dysfunction, neuroinflammation, and abnormal accumulation of α-synuclein, which contributes to neuronal dysfunction and loss.

Hyperbaric oxygen therapy (HBOT) has been proposed as a supportive intervention in PD, with the aim of reducing oxidative damage, improving mitochondrial function, and modulating neuroinflammatory processes. Experimental data suggest that increased tissue oxygenation may support the survival of dopaminergic neurons, reduce glial activation, and attenuate the production of pro-inflammatory cytokines involved in disease progression. In addition, HBOT may influence autophagic pathways, potentially facilitating the clearance of misfolded proteins such as α-synuclein, which plays a central role in PD pathophysiology.

Clinical evidence regarding the efficacy of HBOT in Parkinson’s disease remains limited but emerging. A meta-analysis by Pan et al. (2025), published in *Clinical Rehabilitation*, included four randomized controlled trials (n=152 patients) evaluating HBOT in PD, primarily focusing on non-motor symptoms. The pooled results indicated statistically significant improvements in clinical outcomes, with moderate to large effect sizes (SMD > 0.5). No significant differences in safety outcomes were observed between HBOT and control groups (RR = 1.12; 95% CI 0.78–1.61; p = 0.54).

However, considerable heterogeneity was noted across included studies, particularly in treatment protocols (pressure ranging from 1.5 to 2.4 ATA and 30–40 treatment sessions), which limits direct comparability of results. The overall quality of evidence was rated as moderate according to GRADE criteria. Despite promising findings, HBOT is not currently included in standard clinical guidelines for Parkinson’s disease (e.g., ECHM/ ECGP recommendations), and its use remains experimental in this indication.

In summary, HBOT shows potential as an adjunctive therapy in Parkinson’s disease, particularly in relation to non-motor symptoms, through mechanisms involving neuroprotection, angiogenesis, and neuroplasticity. Nevertheless, due to limited high-quality evidence and lack of protocol standardization, further large-scale randomized controlled trials are required to confirm its clinical utility [14].

THE EFFECT OF HYPERBARIC OXYGEN THERAPY ON STROKE OUTCOMES

Clinical analyses of hyperbaric oxygen therapy (HBOT) indicate its considerable therapeutic potential in promoting neuroplasticity and functional recovery in patients with chronic neurological deficits following ischemic stroke and traumatic brain injury (TBI). Intensive HBOT protocols, most commonly consisting of 40–60 sessions at pressures ranging from 1.5 to 2.0 ATA, have been associated with statistically significant improvements in cognitive performance, motor function, and health-related quality of life, even in the late stages of neurological impairment traditionally regarded as stable and resistant to further recovery.

The proposed mechanisms underlying the regenerative effects of HBOT are primarily related to the concept of the “hyperoxic–hypoxic paradox”. Repeated exposure to elevated oxygen concentrations followed by normox-

ic conditions activates adaptive molecular pathways responsible for angiogenesis, endogenous stem cell mobilization, mitochondrial function enhancement, and attenuation of chronic neuroinflammation within ischemic penumbra regions. HBOT-mediated stimulation of hypoxia-inducible factors (HIFs) promotes the expression of genes involved in tissue repair and neuroplasticity. Furthermore, neuroimaging studies using single-photon emission computed tomography (SPECT) and perfusion magnetic resonance imaging (MRI) have demonstrated increased cerebral metabolism and blood flow in previously impaired cortical regions, supporting the hypothesis that HBOT may reactivate metabolically compromised but viable neurons and facilitate functional reorganization of neural networks [15][16].

A systematic review by Marcinkowska et al. (PMC8888529) analyzed data from multiple clinical studies, including randomized controlled trials involving patients with chronic neurological deficits after cerebral injury and ischemic stroke. The reviewed evidence demonstrated significant improvements in neuropsychological outcomes, particularly in verbal and visual memory, sustained attention, and executive functions. Improvements were reported in standardized assessments such as the Rey Auditory Verbal Learning Test, Trail Making Test, and Stroop Test. These effects have been attributed to enhanced tissue oxygenation, stimulation of angiogenic pathways involving vascular endothelial growth factor (VEGF), improved cerebral perfusion, and reduction of ischemic tissue edema [15][16][17].

The study identified by PMID 31985478 evaluated the effects of an intensive HBOT protocol consisting of 40 sessions at 2.0–2.5 ATA in patients with chronic cognitive impairment following cerebral hypoxic injury. The intervention resulted in measurable improvements in global cognitive function, including increases in Mini-Mental State Examination (MMSE) scores and improvements in episodic memory and spatial orientation. The persistence of these effects during follow-up suggests that HBOT may induce long-term neuroplastic changes through mechanisms involving enhanced brain-derived neurotrophic factor (BDNF) activity, synaptic remodeling, and reduced neuronal apoptosis [16].

Further evidence was provided by the study reported in PMID 35821512, which investigated 63 patients in the chronic phase following ischemic stroke (>6 months after the acute event). After completion of 40 HBOT sessions, patients demonstrated approximately 20% improvement in global neurocognitive performance measured by the Neuropsychological Assessment Battery, including domains such as information processing

speed and verbal fluency. Improvements in quality of life assessed using the SF-36 questionnaire were also observed. The treatment was generally well tolerated, with mild barotrauma reported in approximately 5% of participants [17].

The molecular and physiological mechanisms of HBOT were further examined in the study associated with PMID 33847854. Treatment protocols involving exposure to 2.4 ATA for 90 minutes per session were shown to reduce inflammatory biomarkers, including interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α), while improving cerebral perfusion parameters assessed by MRI. Reported increases in cerebral blood flow (CBF) of approximately 12–18% were associated with improved performance in executive function tests among patients recovering from both ischemic and hemorrhagic stroke [16].

Overall, current evidence suggests that HBOT may represent a valuable non-invasive adjunctive therapy in post-stroke rehabilitation, particularly for patients with persistent cognitive and functional deficits. The beneficial effects appear to result from a combination of increased oxygen availability, stimulation of angiogenesis and neurogenesis, modulation of inflammatory processes, and enhancement of neural network plasticity.

However, interpretation of existing findings requires caution due to several methodological limitations, including relatively small study populations (frequently fewer than 100 participants), limited availability of blinded randomized controlled trials, and significant heterogeneity in treatment protocols, including variations in pressure parameters (2.0–2.5 ATA), session duration, and total number of treatments (40–60 sessions). Therefore, large-scale multicenter randomized controlled trials with extended follow-up periods exceeding 12 months are required to determine optimal HBOT protocols and confirm the long-term sustainability of clinical benefits.

THE INFLUENCE OF HYPERBARIC OXYGEN THERAPY ON COGNITIVE FUNCTIONS IN HEALTHY ELDERLY INDIVIDUALS

Hyperbaric oxygen therapy (HBOT) has demonstrated potential as a non-pharmacological intervention capable of enhancing neuroplasticity and improving cognitive performance not only in patients with neurological disorders but also in healthy older adults experiencing age-related cognitive decline. Recent evidence suggests that repeated exposure to hyperbaric oxygen may optimize cerebral metabolism, improve vascular function, and stimulate adaptive mechanisms associated with brain aging.

In a randomized controlled trial involving 63 healthy adults over 64 years of age without a history of stroke or major neurological disease, a protocol consisting of 60 HBOT sessions (100% oxygen at 2 ATA for 90 minutes, five sessions per week, with intermittent air breaks) resulted in statistically significant improvements in global cognitive performance. The most pronounced effects were observed in domains associated with attention, information processing speed, and executive functions. The reported net effect sizes were 0.745 for attention and 0.788 for information processing speed, indicating a clinically meaningful improvement following the intervention [18].

Neuroimaging analysis using magnetic resonance imaging (MRI) and perfusion-based methods demonstrated significant increases in cerebral blood flow (CBF) following HBOT, particularly within regions involved in higher cognitive processing, including the middle and superior frontal gyri, prefrontal cortex, parietal cortex, and motor-related cortical areas. These findings suggest that cognitive enhancement following HBOT is associated with improved cerebral perfusion and metabolic activation of brain regions vulnerable to age-related functional decline.

The observed effects may be explained by several biological mechanisms, including stimulation of angiogenesis, improved mitochondrial efficiency, increased oxygen availability to neural tissue, and activation of pathways associated with neuroplasticity. Similar to the mechanisms described in neurological rehabilitation, the “hyperoxic–hypoxic paradox” induced by repeated oxygen fluctuations may activate hypoxia-inducible factors (HIFs), promote vascular remodeling, and support neuronal adaptation.

Importantly, the magnitude of cognitive improvement reported in this study suggests that HBOT may provide benefits beyond conventional lifestyle interventions, including physical exercise-based approaches, although direct comparisons between HBOT and aerobic training require further investigation. These findings indicate that HBOT may represent a promising therapeutic strategy for maintaining cognitive function and delaying age-associated neurological decline in healthy elderly populations.

Nevertheless, further large-scale, multicenter randomized controlled trials are required to confirm the reproducibility of these findings, establish optimal treatment protocols, and determine the long-term clinical relevance of HBOT-induced cognitive enhancement in aging populations [18].

CLINICAL CASE REPORTS DEMONSTRATING THE EFFECTS OF HYPERBARIC OXYGEN THERAPY AFTER STROKE

Several clinical case reports published in the available literature describe functional and neurophysiological

improvements following hyperbaric oxygen therapy (HBOT) in patients with chronic neurological deficits after stroke. Although individual case reports cannot provide definitive evidence of treatment efficacy, they offer valuable insights into possible mechanisms of recovery and highlight the potential role of HBOT as an adjunctive intervention in long-term neurorehabilitation.

One reported case involved a 45-year-old Arabic male who developed a left-sided hemorrhagic stroke resulting in persistent neurological impairment 15 months after the acute event. At baseline evaluation, the patient presented with right-sided hemiparesis, severe gait impairment requiring wheelchair mobility, and cognitive deficits. The patient received 83 HBOT sessions over a period of 16 weeks using a protocol of 2.0 ATA for 90 minutes per session with intermittent air breaks. Clinical assessment included neurological and cognitive evaluation, while structural and functional brain changes were examined using diffusion tensor imaging (DTI) and single-photon emission computed tomography (SPECT).

Following completion of the HBOT protocol, substantial functional improvements were observed. The patient demonstrated increased muscle strength, reduced spasticity, improved postural balance, and progression from wheelchair dependence to independent ambulation with the assistance of a quadraped cane. Cognitive assessment revealed improvements in attention, verbal memory, and information processing speed. Neuroimaging findings supported these clinical changes: DTI demonstrated increased fractional anisotropy within major white matter tracts, suggesting improved structural connectivity, while SPECT revealed significant increases in cerebral perfusion within the right motor cortex (+15.83%) and right frontal lobe (+15.92%) [19].

Another case described a 73-year-old Romanian female with a medical history of hypertension, atrial fibrillation, and cerebellar ischemic stroke occurring on December 25, 2019. The patient was evaluated approximately one year after stroke onset. Perfusion MRI demonstrated reduced cerebral blood flow in the region of the left cerebellar lesion and surrounding cerebral tissue. Clinically, the patient exhibited severe balance impairment affecting activities of daily living, requiring caregiver assistance for indoor mobility and wheelchair use outdoors. Neurological examination revealed left-sided muscle strength of 4/5, preserved range of motion in all extremities, a motor Functional Independence Measure (FIM) score of 13/21, and a positive Romberg test. Before treatment, the patient was unable to independently perform sit-to-stand (STS), 10-meter walking, dual-task walking, or the Timed Up and Go (TUG) test.

Following HBOT, the patient demonstrated clinically meaningful improvements in balance, mobility, and functional independence. She progressed from wheelchair dependence to assisted walker ambulation indoors and improved her ability to perform previously impossible functional assessments, including STS repetitions, Romberg balance maintenance for 30 seconds under supervision, 10-meter walking, dual-task walking, and the TUG test. Follow-up perfusion MRI demonstrated increased cerebral blood flow in both cerebellar hemispheres, while the core of the previous lesion remained unchanged, suggesting functional recovery through improved perfusion and activation of surrounding neural networks rather than structural regeneration of the infarcted tissue [20].

These case reports support the hypothesis that HBOT may promote recovery in chronic post-stroke conditions through mechanisms involving enhanced cerebral perfusion, improved white matter integrity, modulation of neuroplastic processes, and functional reorganization of remaining neural circuits. However, due to the observational nature and limited number of reported cases, these findings should be interpreted as preliminary evidence requiring confirmation in larger controlled clinical trials.

CONCLUSIONS

Hyperbaric oxygen therapy (HBOT) represents a promising supportive approach for modulating nervous system function through mechanisms associated with enhanced tissue oxygenation, improved neuronal metabolism, and stimulation of neuroplasticity. Available experimental and clinical evidence suggests that exposure to elevated oxygen concentrations under hyperbaric conditions may promote adaptive changes within the central nervous system, includ-

ing improved mitochondrial activity, regulation of inflammatory responses, reduction of oxidative stress, and activation of pathways involved in neuronal survival and repair.

The clinical effects reported in the literature indicate that HBOT may contribute to improvements in selected neurological outcomes, including cognitive performance, attention, information processing, sleep quality, and reduction of neuropsychiatric symptoms. In patients with chronic neurological deficits following stroke or traumatic brain injury, HBOT has been associated with functional recovery, improved cerebral perfusion, and enhanced neurocognitive performance, suggesting a potential role in long-term neurorehabilitation. Furthermore, preclinical studies provide evidence that HBOT may exert neuroprotective effects by regulating mitochondrial function, reducing apoptosis, and supporting the preservation of vulnerable neuronal structures.

Despite these encouraging findings, the current evidence base remains limited by methodological challenges, including variability in treatment protocols, differences in patient populations, relatively small sample sizes, and an insufficient number of large-scale randomized controlled trials. Therefore, further multicenter studies with standardized HBOT protocols and long-term follow-up are required to establish optimal therapeutic parameters and define the populations most likely to benefit.

Overall, the available evidence supports the potential value of HBOT as an adjunctive therapy in neurological rehabilitation and disorders associated with cognitive decline. When integrated with established treatment strategies, HBOT may represent a valuable therapeutic modality for enhancing nervous system recovery and maintaining neurological function, particularly in conditions characterized by impaired neuroplasticity and progressive cognitive dysfunction.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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