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The Effectiveness of Hyperbaric Oxygen Therapy in Acute Acoustic Trauma: A Narrative Review of Clinical Evidence and Treatment Considerations

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ABSTRACT

Background

Acute acoustic trauma (AAT) is a form of sensorineural hearing loss caused by exposure to sudden and intense noise. This condition results in mechanical damage to cochlear hair cells. It also leads to disturbances in cochlear microcirculation and tissue hypoxia. Due to the risk of permanent hearing impairment, rapid and effective treatment is essential. The aim of this review was to evaluate current therapeutic methods in AAT and to assess the potential role of hyperbaric oxygen therapy (HBOT) as an adjunctive treatment.

Methods

A literature search was conducted using PubMed and Google Scholar to identify studies evaluating the role of HBOT in the management of acute acoustic trauma. Both clinical and experimental studies were considered for background context, while the analysis focused primarily on clinical evidence. Relevant full-text articles were analyzed. The analysis focused on treatment protocols, timing of therapy initiation, and reported clinical outcomes.

Results

The rationale for HBOT in AAT is its ability to increase tissue oxygenation and counteract cochlear hypoxia resulting from microcirculatory disturbances following acoustic injury. Improved oxygen delivery may support cellular metabolism and limit secondary inflammatory processes. It may also reduce permanent hair cell damage. Available clinical studies suggest that HBOT may improve hearing outcomes, especially when administered early after injury and in combination with systemic corticosteroid therapy.

Conclusions

Current clinical evidence indicates that HBOT may represent a beneficial adjunctive therapy in the management of acute acoustic trauma. However, the methodological quality of available studies remains limited. Well-designed prospective randomized trials with standardized treatment protocols are needed. These studies should help define the optimal therapeutic strategy and the most effective therapeutic window for HBOT in AAT.

Keywords: noise-induced hearing loss, acute acoustic trauma, hyperbaric oxygen therapy, hearing loss

INTRODUCTION

Acute acoustic trauma (AAT) is defined as a sudden sensorineural hearing impairment due to sudden exposure to intense noise, which results in damage to hair cells and decreased cochlear blood flow. In addition to acute hearing impairment, AAT is often accompanied by tinnitus [1]. The individuals most at risk of AAT are professional soldiers, who are regularly exposed to high-intensity noise during firearms and explosive ordnance training. Despite the use of hearing protective devices, such injuries still occur, as evidenced by numerous publications in which soldiers are among the study participants [5-11, 14]. Furthermore, a number of individuals experience mild, temporary hearing loss and tinnitus in environments such as pop concerts or sporting events. [5].

AAT may only result in a Temporary Threshold Shift (TTS), where natural repair processes can lead to complete recovery. However, in more serious cases, the chance of complete recovery is rather low, and a Permanent Threshold Shift (PTS) may occur; therefore, due to the potential permanent clinical consequences, prompt and effective treatment is essential.

Despite the absence of a universally accepted treatment protocol for acute acoustic trauma, systemic corticosteroids remain the most commonly applied first-line therapy in clinical practice. The inclusion of such treatments is justified because they help to limit the acute inflammatory response [3] that occurs in the inner ear as a consequence of AAT. However, clinical outcomes following steroid therapy are variable, and some patients fail to achieve complete hearing recovery. Recently, it has been increasingly considered that Hyperbaric oxygen therapy (HBOT) should be routinely used as treatment for AAT to enhance the effect of steroid therapy. Nevertheless, standardized treatment regimens regarding timing, pressure, and number of HBOT sessions are lacking. Available studies have heterogeneous protocols and inconsistent outcome measures, and due to this, optimal management strategies remain unclear. As a result, despite the common use of both corticosteroids and HBOT, the general efficiency of current treatment approaches remains incompletely defined.

HBOT increases the delivery of oxygen to the body by providing pure oxygen in an enclosed space with higher-than-normal air pressure, resulting in increased availability of tissue oxygenation. Exposure to intense acoustic stimuli has been shown to impair cochlear microcirculation and induce hypoxia within the inner ear. During this acute phase of injury,

sensory hair cells may enter a transitional state in which cellular dysfunction is potentially reversible before progression to irreversible cell death. By enhancing oxygen delivery to the cochlea, HBOT may support cellular metabolism and limit secondary injury processes following acoustic trauma. Pathophysiological mechanisms of acute acoustic trauma and the potentially reversible nature of early cochlear damage have resulted in growing clinical interest in the use of HBOT as a therapeutic option. Despite the increasingly frequent use of HBOT in clinical practice, its exact role has not yet been clearly established, which justifies further evaluation of the available clinical evidence. The existing literature on the use of hyperbaric oxygen therapy in acute acoustic trauma remains heterogeneous. One major source of inconsistency is the lack of standardized treatment protocols across centers, leading to substantial variation in applied pressure, the number of sessions, and the duration of exposure in the hyperbaric chamber. In addition, there is ongoing debate about the optimal timing of HBOT initiation after AAT, as well as the time point beyond which such treatment may no longer provide clinical benefit. Further discrepancies emerge from differences in outcome assessment, as no uniform definition of therapeutic success has been established. Studies use diverse audiometric parameters and criteria to define positive or negative treatment effects, limiting comparability of results. Moreover, for ethical reasons, most available studies lack untreated control groups, which complicates distinguishing between treatment-related improvement and spontaneous recovery driven by natural reparative processes. These factors hinder the ability to draw definitive conclusions regarding the effectiveness of HBOT in AAT and impede the development of standardized, evidence-based treatment guidelines.

This review is focused on evaluating the effectiveness of hyperbaric oxygen therapy in the treatment of acute acoustic trauma based on the currently available clinical evidence. In addition, it aims to determine whether existing data allow for more uniform clinical conclusions regarding the role of HBOT.

METHODS

Original clinical studies evaluating the effects of hyperbaric oxygen therapy on hearing outcomes in patients with acute acoustic trauma were identified through searches of PubMed and Google Scholar. The literature review included studies published between 1985 and 2025 that investigated the treatment of acute acoustic trauma using hyperbaric oxygen therapy.

Additional full-text articles were accessed via SpringerLink and ResearchGate when available.

To ensure comparability of clinical outcomes, only human clinical studies, including retrospective analyses, case series, and controlled clinical investigations, were considered. We focused on studies where acute acoustic trauma was the primary condition investigated and hyperbaric oxygen therapy was used as part of the therapeutic intervention in at least one of the analyzed patient groups. Experimental studies based on animal models, single case reports, and publications addressing related but distinct conditions, such as idiopathic sudden sensorineural hearing loss, were excluded from comparative analysis.

PATHOPHYSIOLOGY

Exposure to intense acoustic stimuli can result in either temporary or permanent threshold shifts through multiple pathophysiological mechanisms. In the injured inner ear, acoustic trauma induces a combination of mechanical and metabolic damage, accompanied by disturbances in cochlear microcirculation. Because sensory hair cells are anchored between the basilar and tectorial membranes, they are particularly vulnerable to mechanical injury caused by intense or prolonged noise exposure [2].

Experimental studies have demonstrated that acoustic overstimulation is associated with increased intracellular calcium levels in the cochlea. This promotes the generation of reactive oxygen species (ROS) and may directly trigger cell death pathways [1]. The release of intracellular components into the extracellular environment initiates an inflammatory response. Alterations in ionic homeostasis, characterized by elevated K^+ in the endolymph and increased Na^+ in the perilymph, result in cellular edema and structural damage [2]. Additionally, noise exposure induces the expression of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α). These have been associated with hair cell loss [1].

Reactive oxygen species (ROS) can be detected in cochlear cells shortly after acoustic exposure, even before overt morphological changes become apparent. ROS contribute to damage of cellular components such as DNA, proteins, and membrane lipids, and promote lipid peroxidation, leading to apoptosis and impaired cochlear blood flow [2]. Moreover, ROS

participate in the amplification of inflammatory signaling by stimulating further cytokine release.

Disturbances in cochlear blood flow and the resulting hypoxia represent additional key factors in the pathophysiology of acute acoustic trauma. Physiological studies have demonstrated reduced cochlear perfusion, vessel constriction, and hypoxic changes following intense noise exposure. Both inflammatory mediators and oxidative stress contribute to vascular dysfunction, while noise-induced inhibition of cyclooxygenase (COX) activity reduces prostaglandin E_2 (PGE2) production, further limiting vasodilation and aggravating cochlear ischemia [3].

Importantly, sensory hair cells that cease to function following acoustic trauma may persist in a prolonged transitional state between recovery and irreversible cell death, highlighting the potential reversibility of early cochlear injury.

MECHANISM AND RATIONALE OF HBOT

Hyperbaric oxygen therapy increases the partial pressure of oxygen in blood and tissues, thereby enhancing oxygen delivery to hypoxic regions of the inner ear following acute acoustic trauma. Under typical therapeutic conditions, the partial pressure of oxygen dissolved in plasma may reach levels more than 20 times higher than those achieved while breathing room air at normal atmospheric pressure [4]. The cochlea is particularly vulnerable to hypoxic injury due to its high metabolic demand and limited tolerance to reductions in blood flow; therefore, these pathophysiological features suggest the existence of a limited therapeutic window during which restoration of tissue oxygenation may be biologically most relevant. In conditions associated with capillary vasoconstriction and subsequent cochlear hypoxia, hyperbaric oxygen therapy facilitates increased diffusion of oxygen into cochlear tissues. By improving tissue oxygenation and supporting aerobic cellular metabolism, HBOT may help preserve the viability of sensory hair cells and prevent hypoxia-induced cell death.

Beyond ischemic injury, acoustic trauma initiates secondary cellular processes that may continue after cessation of the acoustic stimulus. Reactive oxygen species (ROS) are detected in cochlear tissues shortly after noise exposure, and oxidative stress may persist for several days following acute acoustic trauma, thereby contributing to progressive cellular injury [1].

Increased ROS production has been associated with the upregulation of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α), which may further amplify cochlear damage [1]. Prolonged activation of inflammatory pathways may result in permanent hearing loss after acoustic trauma, highlighting the clinical relevance of limiting secondary injury processes. Therefore, the coexistence of hypoxia and inflammation following acute acoustic trauma provides a biological rationale for therapeutic strategies aimed at early restoration of tissue oxygenation. By improving oxygen availability at the tissue level, hyperbaric oxygen therapy may indirectly modulate oxidative stress by limiting hypoxia-driven mitochondrial dysfunction and subsequent excessive ROS generation. Although systemic corticosteroid therapy remains the cornerstone of anti-inflammatory treatment in acute acoustic trauma, emerging evidence suggests that its therapeutic effects may be enhanced when combined with hyperbaric oxygen therapy.

CLINICAL EVIDENCE

Overview of available studies

Although the use of hyperbaric oxygen therapy (HBOT) in acute acoustic trauma (AAT) is not a novel concept — with the first clinical reports dating back to the 1980s — the topic has gained renewed attention in recent years. A growing number of publications over the past decade reflect increasing clinical interest in HBOT as a potential therapeutic strategy for AAT. In the present review, conclusions regarding the role of HBOT were based primarily on retrospective clinical studies as well as more recently published prospective investigations. Most available studies involve military personnel who sustained acoustic trauma during firearms training, which reflects the typical epidemiological context of severe impulse noise exposure.

Despite the growing number of available data, definitive conclusions remain difficult to establish due to the limited number of randomized controlled trials and significant heterogeneity in treatment protocols, including differences in pressure settings, number of sessions, and timing of initiation. Furthermore, the interpretation of outcomes is complicated by variability in recovery definitions and the possibility of spontaneous partial hearing improvement. Current literature increasingly focuses on two key aspects: the relationship between the timing of HBOT initiation and treatment outcomes, and the role of HBOT as an

adjunctive therapy in combination with systemic corticosteroids, compared with corticosteroid monotherapy.

HBOT as adjunctive therapy

Systemic corticosteroid therapy remains the widely accepted first-line treatment for acute acoustic trauma. Most published clinical studies have compared corticosteroid monotherapy with combined corticosteroid and HBOT, while investigations evaluating HBOT as the only treatment are relatively uncommon. In the majority of cases, HBOT was initiated alongside systemic steroid therapy or used as an escalation strategy in patients who did not demonstrate expected effects following initial corticosteroid treatment. This variability in therapeutic strategies is methodologically relevant, as it directly influences the interpretation of reported clinical outcomes.

Variability of HBOT protocols

To date, no universally accepted or standardized HBOT protocol has been established for the treatment of acute acoustic trauma. Individual studies have applied treatment regimens based on institutional practice or investigator preference. Reported pressure levels range from approximately 1.35 ATA [9] to 2.5 ATA, with most authors employing pressures around 2.0–2.5 ATA. The duration of a single session typically varies between 80 and 90 minutes, whereas the total number of sessions shows considerable variability, ranging from as few as 6 compressions [4] to more than ten sessions depending on clinical response [7]. This variability in therapeutic protocols limits direct comparison between studies and complicates attempts to formulate uniform conclusions regarding the effectiveness of HBOT in AAT.

Timing

Another clinically relevant aspect referred to in the literature is the relationship between the timing of HBOT initiation and final hearing recovery. Several authors have specifically focused on this issue by comparing subgroups according to the interval between acoustic trauma and treatment initiation. In most studies, early intervention was defined as treatment initiated within 48 hours [6,8,11] or within seven days [7,14,15] after injury. Conversely, some investigations evaluated delayed HBOT, with a mean treatment initiation time of

approximately 24 days following acoustic trauma [10]. Despite the growing number of studies addressing this problem, there is a lack of strong evidence to clearly define the most beneficial therapeutic window.

Outcome definitions

Considerable heterogeneity exists not only in treatment protocols but also in the definitions of therapeutic success. Hearing improvement was assessed using different audiometric measures, including pure-tone average (PTA) and/or high-frequency pure-tone average (HPTA). In addition, some authors took into account subjective evaluation of hearing improvement and associated symptoms such as tinnitus into their outcome assessment. Several studies reported treatment efficacy as mean threshold gain [5,6,8], whereas others defined recovery categorically, most commonly using a cut-off of ≥10 dB improvement at affected frequencies [10,13,14]. More rarely, therapeutic success was defined as complete restoration of hearing to pre-trauma levels [9]. The absence of standardized recovery thresholds may lead to over- or underestimation of HBOT efficacy.

Reported Clinical Outcomes and Efficacy

The currently available literature suggests that HBOT may provide a potential therapeutic benefit in patients with acute acoustic trauma. Several clinical studies have reported greater hearing improvement when HBOT was administered in combination with systemic corticosteroids compared with corticosteroid therapy alone. In selected investigations, HBOT monotherapy was also associated with improved audiometric outcomes when compared with normobaric oxygen exposure. However, treatment response was not uniform across all studies, and not all patients demonstrated measurable improvement following HBOT. In some cases, observed differences did not reach statistical significance. Reported benefit varied depending on treatment timing, protocol characteristics, and outcome definitions. The following section provides a more detailed analysis of individual study findings.

In comparative clinical analyses, patients receiving combined HBOT and systemic corticosteroids demonstrated greater hearing improvement than those treated with corticosteroids alone. Both absolute hearing improvement (dB) and relative hearing improvement (%) were significantly higher in the combination therapy group compared with

corticosteroid monotherapy [5]. The most noticeable differences were observed in patients with high-frequency hearing loss (4–6 kHz), which is characteristic of acute acoustic trauma. Similar conclusions were reported by other authors who measured hearing recovery specifically at the frequencies showing the greatest post-traumatic threshold shifts (2, 4, and 8 kHz). In their analysis, both HBOT-treated groups demonstrated significantly greater improvement compared with the non-HBOT groups ($p < 0.05$) [8]. In contrast, when lower frequencies (0.5–2 kHz) were analyzed, between-group differences were less marked and did not consistently reach statistical significance [5].

The timing of therapy initiation has become one of the most frequently discussed topics, with most authors suggesting similar conclusions and advocating earlier initiation of treatment [6,16,17]. In a timing-focused cohort, as in Bayoumy AB et al. (2021) publication, initiation of HBOT within two days of injury was associated with significantly greater mean hearing improvement compared with initiation after two days. Absolute hearing gain across affected frequencies was approximately 23 dB in the early-treatment group versus 12 dB in the delayed group ($p = 0.007$), with relative recovery rates of 56% and 31%, respectively ($p = 0.004$) [6]. More recent analyses further support the importance of treatment latency, suggesting that initiation of HBOT within seven days of injury is associated with more favorable outcomes, whereas delays exceeding three weeks are linked to reduced therapeutic response [14]. These findings are consistent with the concept of a clinically relevant therapeutic window.

Comparative analyses of different HBOT protocols (TT5 vs TT9) also demonstrated a significant difference in recovery grade ($p = 0.016$) in favor of TT9, despite TT5 being a more intensive treatment [10]. This observation raises the possibility that cumulative oxygen exposure and total treatment dose may be more relevant than the intensity of a single session.

Studies evaluating HBOT as monotherapy have likewise reported beneficial effects following AAT. Compared with normobaric oxygen therapy, HBOT was associated with significantly greater recovery, including higher rates of complete hearing normalization (70% vs 40%, $p < 0.01$) and superior high-frequency recovery ($p < 0.001$) [9].

In contrast, Richard Holy et al. reported that although the HBOT group demonstrated greater overall threshold improvement compared with the non-HBOT group, statistically significant

differences were observed only at 500 Hz ($p < 0.01$) and 2000 Hz ($p < 0.05$). At frequencies typically most affected in acute acoustic trauma (4–6 kHz), inter-group differences did not reach statistical significance ($p > 0.05$) [7]. An important limitation of this study is that HBOT was administered primarily to patients who did not respond successfully to first-line therapy, wherefore the "HBOT vs no HBOT" groups weren't directly comparable.
Furthermore, early initiation of HBOT shortly after injury may complicate differentiation between treatment-related improvement and spontaneous recovery processes, including the existence of temporary threshold shift (TTS). This may lead to overestimation of therapeutic benefit in some cases. Additionally, isolated reports have suggested potential negative effects associated with very early initiation of HBOT [19].

Taken together, the available evidence suggests that HBOT may enhance clinical recovery in selected patients, particularly when administered early and in combination with systemic corticosteroids. However, the current body of literature does not allow for definitive conclusions regarding the optimal therapeutic window or the most effective treatment protocol. Large, prospective, randomized controlled trials are still needed to establish clear recommendations for the use of HBOT in acute acoustic trauma.

CONCLUSIONS

Recently, there has been increasing support for the use of HBOT in hearing impairment caused by intense noise. There is a biological justification for such therapy, as HBOT may limit cochlear hypoxia and indirectly modulate inflammatory processes, thereby potentially enhancing the effects of systemic corticosteroid therapy. Available studies suggest that the most favorable outcomes are observed when HBOT is administered as an adjunctive therapy and initiated early after injury. Greater improvement has been reported, particularly in cases involving high-frequency hearing loss, which is characteristic of acute acoustic trauma.
The methodological quality of the existing literature remains limited. The predominance of retrospective study designs, lack of randomized controlled trials, absence of standardized treatment protocols, and varying definitions of treatment success preclude definitive conclusions regarding treatment efficacy. Furthermore, there is still a lack of long-term data to determine whether observed hearing improvements are sustained over time.
Despite the growing number of recent publications investigating the use of HBOT in the treatment of AAT, our conclusions remain largely consistent with those of earlier review

studies [16,18]. This narrative review, based on available clinical evidence, supports the idea that the use of HBOT in the treatment of AAT may have a positive therapeutic effect. However, well-designed prospective randomized trials with standardized protocols and clearly defined outcome measures are required to establish evidence-based recommendations and to define the optimal therapeutic window for HBOT in AAT.

Disclosure

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The authors declare no conflict of interest.

Declaration of AI use

During the preparation of this manuscript, generative AI (Chat GPT and Grammarly) tools were used solely for translation and language editing. The authors reviewed and edited the generated text and take full responsibility for the final content of the manuscript.

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