Hyperbaric Oxygen Therapy for Acute Acoustic Trauma: Blast versus Noise-Induced Hearing Loss

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ABSTRACT **Background:** Blast injuries impair hearing through several mechanisms that are distinct from other causes of acute acoustic trauma (AAT).

Objectives: To compare blast injured patients to those exposed to noise alone in their auditory response to hyperbaric oxygen (HBO) therapy with oral steroids.

Methods: Adult patients with evidence of a previously undocumented ≥ 30 dB pure-tone threshold within 30 days of AAT were treated with a combination of one 2.5 atm HBO therapy session for 90 minutes daily with oral prednisone. Exposure was classified by history as *blast* (for explosion-induced AAT) or *noise*. The change in high pure tone average (HPTA) was the primary outcome.

Results: Of 598 ears (387 patients) included in the final analysis, 259 were exposed to blast and 339 to noise. Before treatment, the blast injured patients had significantly more abnormal findings on otoscopy (87% vs. 95%, P = 0.003), higher pure-tone average (18 ± 11 dB vs 12 ± 9 dB; P < 0.001), and higher speech reception thresholds (16 ± 14 dB vs 10 ± 8 dB, P < 0.001). Following treatment, these patients exhibited a significantly smaller improvement in HPTA (6 ± 17 dB vs 10 ± 14 dB P = 0.022) with pure tone thresholds remaining significantly worse across all frequencies in the blast exposed group (mean difference ranging from 3.2 to 6.8 dB, all P < 0.05).

Conclusions: Blast injuries result in unique auditory characteristics and responses to HBO therapy compared to other causes of AAT.

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KEY WORDS: acute acoustic trauma (AAT), acute sensorineural hearing impairment (SNHL), blast injury, high pure tone average (HPTA), hyperbaric oxygen (HBO) therapy

Acute acoustic trauma (AAT), defined as an acute sensorineural hearing impairment (SNHL) caused by intense noise, is the leading cause of newly diagnosed preventable hearing disability in young adults [1]. Gunfire and explosions are the two main causes of AAT [2]. However, explosions differ from other noise sources both in their noise characteristics and in the presence of additional damage mechanisms (e.g., supersonic shock waves, debris, thermal injury) and widespread impact. This complex and multifaceted injury is often called blast injury [3]. Physical models, both using impulse peak insertion loss from two acoustic test fixtures and in vivo animal subjects, have revealed significant differences in the noise profiles contingent on the noise source [4]. While gunfire noise reaches peak sound pressure levels (SPL) of 154 dB, high explosives typically produce SPLs over 175 dB [5]. More importantly, the additional damage mechanisms often produce much more extensive damage and are exclusive to blast injuries [4].

Beyond these disparities in physical characteristics, the resulting injuries exhibit different pathophysiology [2]. Profound neurological damage is twice as common in patients with blast injuries compared to patients exposed to gunfire noise alone [6]. While exposure to gunfire noise typically results in a predominantly high frequency SNHL (particularly at 6000–8000 Hz), diffused (and significantly more clinically debilitating) injury across a wider range of frequencies seems to be the rule in blast injury [7]. Last, damage to both ears is more common in blast injury [8].

Therapeutic interventions thus far investigated for AAT included various glucocorticoids, antioxidants (including vitamin E), vasodilators, and hyperbaric oxygen (HBO) [5]. The combination of HBO therapy and glucocorticoids (either oral, intravenous, or intratympanic)

is by far the most studied and was consistently shown to be superior to either treatment alone. However, all these studies focused exclusively on gunfire-induced AAT [1,9-12]. Despite the physical, pathophysiological, and clinical peculiarities, there is little research about the specifics of auditory blast injury treatment and the effects of HBO therapy on blast-induced AAT [13].

We aim to fill this gap and study the response to the current best clinical practice: the combination of HBO therapy and glucocorticoids in AAT patients exposed to gunfire noise alone (noise) versus patients exposed to explosions (blast).

PATIENTS AND METHODS

POPULATION AND SETTING

Patients reporting exposure to loud noise (including unprotected or partially protected gunfire and/or explosions) as part of active military service were evaluated by an otolaryngologist and a speech therapist. If a pure-tone threshold above 30 dB was detected at 3000–8000 Hz, the patient was referred for further evaluation at our hyperbaric center. The study period was 1 January 2020 to 31 December 2024. All findings were considered new unless pre-injury audiograms were available and showed similar (to within 10 dB) findings.

To be included, patients had to: have been exposed to acute acoustic injury within 30 days before the initial evaluation, demonstrate a presumably new hearing impairment of ≥ 30 dB on a comprehensive audiometry performed ≥ 48 hours after injury [14], be deemed suitable for an HBO therapy course by a senior hyperbaric physician at the Israel Naval Medical Institute (INMI), be able to equalize during otoscopy, and have no significant otolaryngological or pulmonary pathology that would preclude hyperbaric treatment [15]. Exclusion criteria included patients who missed more than five consecutive therapy sessions, patients who refused HBO therapy before or during the therapy course, patients exposed to both gunfire and explosions, and patients with an unclear exposure history.

HYPERBARIC TREATMENT PROTOCOL

Patients who provided informed consent to therapy after an in-depth explanation by a senior hyperbaric physician were started on oral prednisone (60 mg/day for one week, tapered down over one additional week) and daily HBO therapy sessions. The HBO protocol consisted of gradual pressurization to 2.5 atm, followed by four intervals of pure oxygen breathing for 20 minutes each, separated by 5 minutes of air breathing, and a gradual depressurization with two decompression stops at 1.6 and 1.3 atm for 6 and 3 minutes, respectively.

Treatment sessions were administered daily, five days a week. Audiometry was performed every five sessions. Therapy continued until hearing was restored to within normal limits (i.e., 20 dB or less across all pure-tone thresholds examined), or hearing returned to pre-injury recorded thresholds (if available), or no significant (< 10 dB) change was observed in any of the pure-tone thresholds examined in two consecutive audiograms.

DIAGNOSTIC SETUP

All audiometry was performed by a board certified speech therapist at the INMI. The diagnostic setup consisted of an AC40 diagnostic audiometer audiometer (Interacoustics©, Middelfart, Denmark) in a sound-isolated, ISO 8253-1 compliant booth (DHA acoustic design, Tel Aviv, Israel). Testing was performed using supra-aural headphones at 250, 500, 1000, 2000, 3000, 4000, 6000, and 8000 Hz. Bone conduction with appropriate masking was performed whenever an abnormal finding was detected, at 500, 1000, 2000, 3000, and 4000 Hz. Speech reception thresholds (SRT) and speech discrimination scores were measured using the Hebrew Central Institute for the Deaf (CID) W-22 equivalent lists [2].

OUTCOMES MEASURED

The primary outcome was the change in the high-frequency pure-tone average (HPTA, defined as the arithmetic mean of pure-tone thresholds at 3000, 4000, 6000, and 8000 Hz) at the end of the HBO therapy course as compared to the HPTA at presentation. Secondary outcomes included the average change in SRT, discrimination, pure-tone average (PTA, the average thresholds of 500, 1000 and 2000 Hz), individual pure-tone frequencies, and the resolution of subjective symptoms. Symptoms were dichotomously defined as either present or absent by directly asking the patient before and after completing the therapy course. Safety outcome measures included the occurrence of sinus barotrauma, middle ear barotrauma (the modified TEED score), or any sign of central oxygen toxicity at any point during the therapy course.

STATISTICAL ANALYSIS

Standard descriptive statistics were used to summarize patient characteristics. We used Pearson's chi-square test for categorical variables and Student's *t*-test for numeric

variables. Categorical variables were described using proportions and percentages. A 2-sided $P < 0.05$ was considered statistically significant in all tests. Statistical analyses were performed using R Statistical Software, version 4.2.1 (R Foundation for Statistical Computing, Vienna, Austria).

ETHICS

This study was performed in accordance with the Declaration of Helsinki. The human study was approved by our institutional ethics committee (approval #2280-2021). The requirement for consent was waived by the ethics committee due to the retrospective nature of this study.

RESULTS

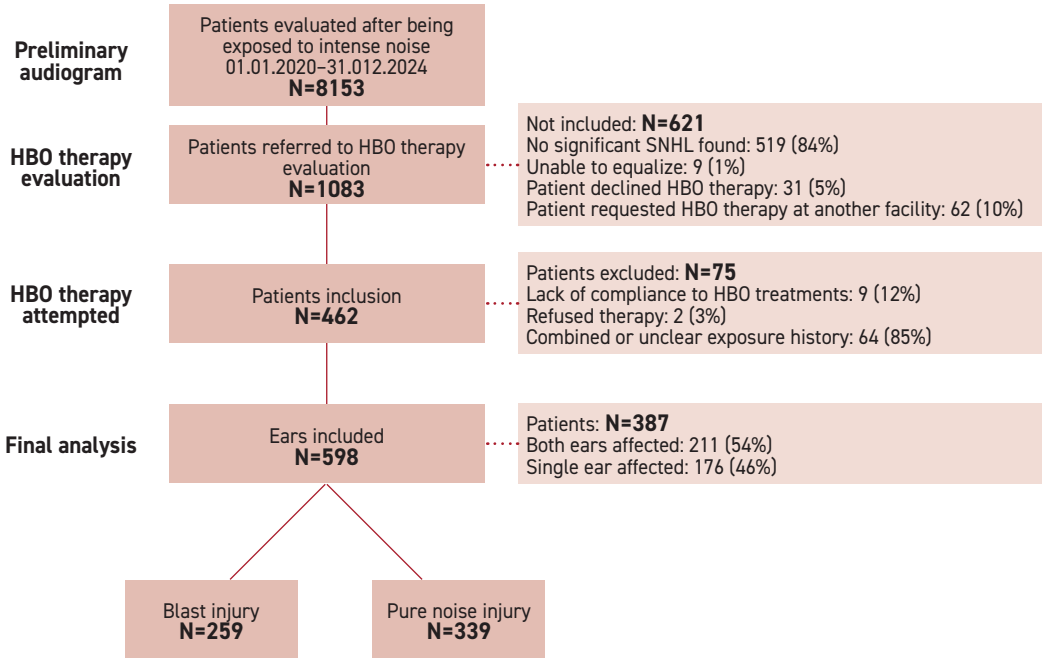
Of 1083 patients referred for our evaluation, 462 met the criteria for HBO therapy. After excluding 75 patients (64 of whom had combined or unclear exposure histories, nine missed more than five consecutive HBO sessions and two refused therapy), 387 were included in the analysis. Due to bilateral involvement in 211 patients, 598 ears were included in the final analysis, of whom 259 ears were exposed to blast injury and 339 exposed to noise alone. The patient selection process is outlined in Figure 1.

Patients in the blast group were slightly older than those in the noise group (median age 28 vs. 25 years; mean difference 1.8 years, 95% confidence interval [95%CI] 0.23–3.5, $P = 0.025$). There were no significant differences between groups in the side affected, the time elapsed from injury to therapy initiation, or the spoken word discrimination. Otoscopy revealed a lower rate of normal exams in the blast group compared to the noise group (87% vs. 95%, $P = 0.003$). Other otoscopic abnormalities (e.g., myringosclerosis, perforations, hemotympanum) were infrequent and did not differ significantly between groups.

Pre-treatment hearing thresholds were consistently and significantly worse in the blast group from 250 Hz to 4000 Hz, with mean differences ranging from 3.2 to 6.0 dB ($P < 0.01$). In contrast, at the high frequencies, these differences did not reach statistical or clinical significance (medians 1.2 and 3.2 dB, $P = 0.5$ and $P = 0.85$, respectively). Pre-treatment SRT were significantly higher (worse) in the blast group (median 10 dB, mean 16 ± 14) compared to the noise group (median 10 dB, mean 10 ± 8; mean difference 5.9 dB, 95%CI 3.7–8.1, $P < 0.001$). Concurrently, subjective hearing loss was more commonly reported in the blast group (64% vs. 54%, absolute difference 11%,

Figure 1. Study design, according to the STROBE guidelines

HBO = hyperbaric oxygen, INMI = Israel Naval Medical Institute, SNHL = sensorineural hearing impairment



95%CI 1.4%–20%, $P = 0.023$). There were no significant group differences in speech discrimination scores or in the prevalence of tinnitus, auricular fullness or pain, hyperacusis, or vertigo [Table 1].

While post-treatment thresholds remained significantly worse in the blast group at all frequencies from 250 Hz to 8000 Hz (medians 4.2 to 6.8 dB, all $P \leq 0.021$), the change in thresholds because of treatment was not significant in the lower frequencies. PTA values mirrored the patterns seen at individual lower frequencies, with the blast group exhibiting a significantly worse PTA compared to the noise group, both before (18 ± 11 dB vs. 12 ± 9 dB; $P < 0.001$) and after (15 ± 12 dB vs. 9 ± 7 dB; $P < 0.001$). Changes did not differ significantly between the groups (Δ Md -0.31 dB, 95%CI -2.1–1.5, $P = 0.5$).

Conversely, the improvement in the higher pure-tone thresholds was significantly more pronounced in the noise group, particularly at 3000 Hz (Md 2.8 dB, 95%CI 0.24–5.4, $P = 0.032$) and 6000 Hz (Md 6.2 dB, 95%CI 2.5–9.8, $P < 0.001$). The improvement in HPTA following HBO therapy was significantly more pronounced in the noise group (10 ± 14 dB) compared to the blast group (6 ± 17 dB; mean difference 3.8 dB; 95%CI 0.94–6.6; $P = 0.022$). Analysis of individual pure-tone thresholds showed that the blast group had consistently worse hearing across all pure-tone frequencies examined (Md ranging from 3.2 to 6.8 dB, all $P < 0.05$) [Figure 2].

Table 1. Baseline characteristics

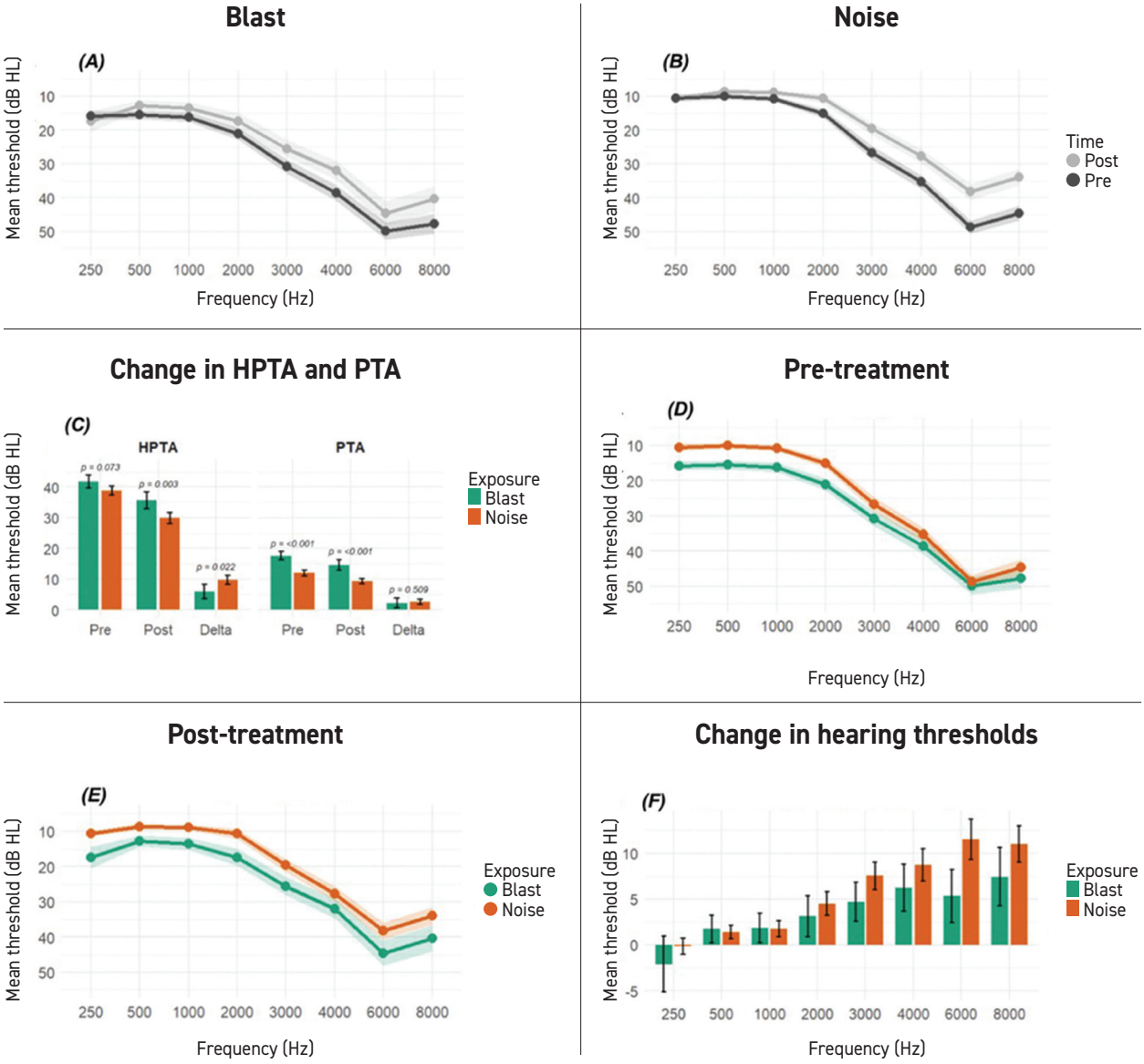
Characteristic	**Overall**, N=598	**Blast**, n=259	**Noise**, n=339	***P*-value**
Age in years, Median (IQR)	27 (20–35)	28 (21–37)	25 (20–33)	0.020
Mean ± SD	29 ± 9	29 ± 9	28 ± 9	
Female	12 (2%)	5 (1.9%)	7 (2.1%)	0.8
Left ear	320 (54%)	136 (53%)	184 (54%)	0.7
Both ears (overall number of patients)	211/387 (54.5%)	84/165 (50.9%)	127/222 (57.2%)	0.26
Days to steroid initiation	6 (4–13)	6 (3–13)	6 (4–13)	0.65
Days to HBO initiation	8 (5–14)	8 (4–14)	8 (5–14)	0.47
Total number of HBO sessions	10 (5–11)	10 (5–11)	10.0 (5–11)	0.3
Findings before HBO initiation				
Otoscopy				
Normal examination	437 (91%)	226 (87%)	211 (95%)	0.002
Bullous myringitis	1 (0.2%)	1 (0.4%)	0 (0%)	0.9
Hemotympanum	5 (1.0%)	3 (1.2%)	2 (0.9%)	0.9
Mild redness	7 (1.5%)	5 (1.9%)	2 (0.9%)	0.5
Single perforation	7 (1.5%)	6 (2.3%)	1 (0.5%)	0.13
Multiple perforations	5 (1.0%)	5 (1.9%)	0 (0%)	0.5
SOM	2 (0.4%)	1 (0.4%)	1 (0.5%)	0.9
SRT dB, median (IQR)	10 (5–15)	10 (5–20)	10 (5–10)	< 0.001
SRT dB, Mean ± SD	13 ± 12	16 ± 14	10 ± 8	
Discrimination (%)	100.0 (96.0–100.0)	100.0 (92.0–100.0)	100.0 (96.0–100.0)	0.13
Symptoms				
Tinnitus	273 (57%)	144 (56%)	129 (58%)	0.5
Auricular fullness	203 (42%)	100 (39%)	103 (47%)	0.077
Subjective hearing loss	286 (60%)	167 (64%)	119 (54%)	0.018
Auricular pain	116 (24%)	71 (27%)	45 (20%)	0.072
Hyperacusis	212 (44%)	113 (44%)	99 (45%)	0.8
Vertigo	74 (15%)	47 (18%)	27 (12%)	0.073

HBO = hyperbaric oxygen, IQR = interquartile range, SD = standard deviation
All numbers are median (IQR) or count (proportion) unless otherwise specified

Figure 2. Pure-tone thresholds

[A] Mean pure-tone thresholds before and after completing HBO therapy for patients with blast injury, **[B]** Patients with pure noise induced acoustic trauma, **[C]** Change in HPTA (average of the pure-tone thresholds of 3000–8000 Hz) and PTA (500–2000 Hz) presented by group, **[D]** Pre-treatment, **[E]** Post-treatment, **[F]** Difference between the individual pure-tone thresholds

dB = sound pressure level decibels of pure-tone threshold, HPTA = high (3000–8000 Hz) pure-tone average, PTA = pure (500–2000 Hz) tone average

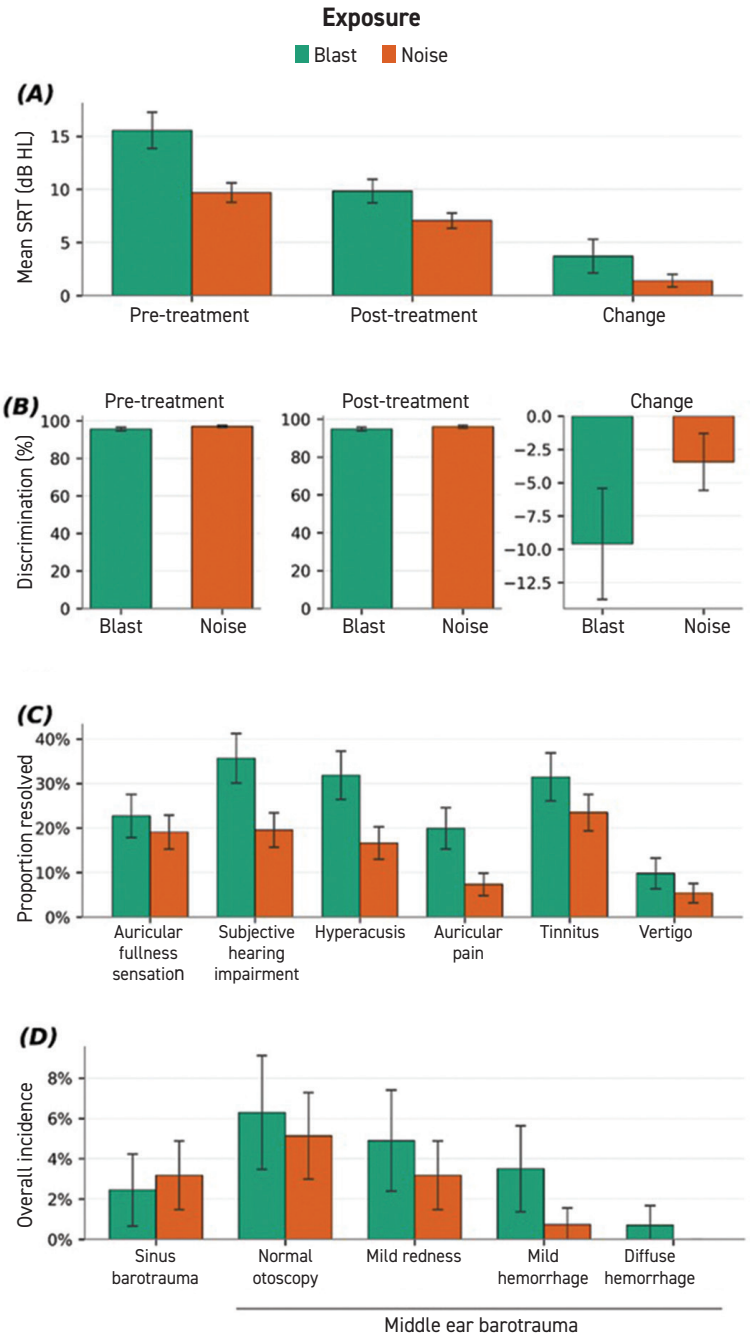


SRT followed a similar pattern to PTA. The blast group had higher pre-treatment and post-treatment values (both $P \leq 0.001$) and significantly more improvement following HBO (Md 2.6 dB, 95%CI 0.68–4.5, $P = 0.008$). Discrimination scores were comparable between groups before treatment, but the blast group experienced a significantly larger decline at the post-treatment examination (mean change in discrimination scores -11 ±33% vs. -3 ± 18% in the noise group; $P < 0.001$). Symptomatic improvement was more common in the blast group, particularly regarding tinnitus (32% cases resolved vs 24% in the noise group, $P = 0.04$), subjective hearing loss (36% vs 16%, $P < 0.001$), auricular pain (20% vs. 8.3%, $P < 0.001$), and hyperacusis (32% vs 19%, $P < 0.001$) [Figure 3].

Figure 3. Speech reception thresholds, discrimination, symptomatic improvement and adverse events

[A] Speech reception thresholds, **[B]** Discrimination presented by exposure history before (pre-treatment) and after completing HBO therapy (post-treatment), with the average difference (change) presented on the right. **[C]** Percentage of patients reporting a complete resolution of specific symptoms are presented by exposure history, **[D]** Complications and adverse effects associated with hyperbaric oxygen therapy are presented at the bottom

dB = sound pressure level decibels of pure-tone threshold, SRT = speech reception threshold



Complications and adverse events associated with HBO were mild and infrequent. There were no reported cases of myoclony, sensory disturbances, decreased consciousness, convulsions, or any other sign of central oxygen toxicity. No respiratory symptoms or evidence of pulmonary oxygen toxicity were recorded. Mild sinus barotrauma, manifested as sinus pain without epistaxis, was reported in 2.5% of patients. Ear pain during pressurization without any otological abnormality (TEED 0), with diffused redness (TEED 1), mild hemorrhage (TEED 2), or diffuse hemorrhage (TEED 3) were reported in 5.9%, 4%, 2.2%, and 0.3% of patients, respectively. No tympanic perforations (TEED 4 and TEED 5) were reported. There was no significant difference in these findings between the groups [Figure 3].

DISCUSSION

To the best of our knowledge, this is the largest systematic study to date of blast-induced hearing loss [16,17]. It was made possible primarily due to the small and centralized nature of our military healthcare and operations, which ensured that all acoustic trauma cases evaluated by any caregiver were referred to our care.

Patients exposed to blast tended to have significantly worse pre-treatment lower-frequency audiological findings, including SRT and PTA. This finding is consistent with the current understanding of the injury mechanisms of blast injury, which is more diffused across the various hearing organs (including conductive and neurological structures) [3]. This understanding is also supported by the overall increased incidence of abnormal otoscopy in the blast group noted in our data.

Blast injured patients were also less responsive to HBO therapy, a finding most pronounced in higher frequencies and reflected in the significant difference observed in our primary outcome of ΔHPTA. In animal models, blast exposure leads to mechanical disruption of entire cochlear structures, hair cell loss, synaptopathy, tympanic membrane rupture, and some signs of central auditory pathway damage [18]. More limited human histopathological studies corroborate this understanding of a more pronounced and diffused damage, including outer hair cell loss, synaptic ribbon loss (cochlear synaptopathy), and spiral ganglion neuron degeneration [9].

This multifocal and severe damage could hamper local blood supply and disrupt the main therapeutic pathway of HBO, the temporary increase in oxygen content delivered to the affected organs. The involvement of higher

processing stations, including ganglionic and supra-ganglionic pathways, could explain why speech discrimination trends did not mirror those observed when testing for simpler auditory outcomes, such as pure-tone thresholds and averages or SRT [2]. This reasoning also correlates with our observation of greater symptomatic improvement in the blast group.

LIMITATIONS

The retrospective nature of this study limits our ability to infer causality. Both groups were predominantly male (98%). The short-term outcomes we describes must be corroborated by longer follow-up. Last, our scope was limited to auditory outcomes. In view of the mounting evidence of the potential efficacy of HBO therapy in traumatic brain injuries and neuroregeneration [19,20]. Further studies are needed to elucidate the full therapeutic potential of HBO in blast injury.

CONCLUSIONS

While auditory improvement was noted following HBO therapy with steroids across both exposure groups, this treatment is associated with greater improvement in patients not exposed to blast injury. Further long-term studies are needed to quantify the lasting auditory effects of blast injured patients undergoing HBO therapy.

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Time engraves our faces with all the tears we have not shed.
Natalie Clifford Barney (1876–1972), American poet, playwright, and novelist

Certainly none of the advances made in civilization has been due to counterrevolutionaries and advocates of the status quo.
Bill Mauldin (1921–2003), American editorial cartoonist who won two Pulitzer Prizes for his work