

Relationship between hearing loss and body injuries caused by various firearms at a war site: A retrospective study

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ABSTRACT

BACKGROUND: This study aimed to investigate the relationship between trauma severe enough to cause physical injury and subsequent hearing loss in military personnel exposed to blast events.

METHODS: A retrospective review was conducted on 95 patients aged 30-39 who were admitted between 2015 and 2018 due to blast-related injuries and acoustic trauma. A control group of 51 military personnel without complaints was included. Patients were categorized based on the location of trauma, the energy level of the explosion, and the presence of tympanic membrane perforation. Hearing thresholds and clinical characteristics were compared between groups to evaluate the relationship between trauma patterns and auditory outcomes.

RESULTS: Patients with head-related injuries had significantly worse air and bone conduction thresholds at multiple frequencies compared to those with injuries in other body regions ($p<0.05$). Tympanic membrane perforation was significantly associated with eye injuries ($p=0.004$) and elevated air conduction thresholds ($p<0.05$), but not with bone conduction thresholds. Exposure to medium- and high-energy blasts was associated with elevated hearing thresholds across all frequencies compared to controls ($p<0.001$).

CONCLUSION: Blast-related acoustic trauma is associated with hearing loss across a range of frequencies. Tympanic membrane perforation contributes to air conduction hearing loss. Eye injury may be anatomically related to tympanic membrane damage. Hearing assessment should be integrated into the multidisciplinary care of trauma patients in war zones.

Keywords: Acoustic trauma; blast injury; battlefield audiology; hearing loss; tympanic membrane.

INTRODUCTION

Hearing loss resulting from acute acoustic trauma is a significant concern for both military personnel and affected civilians.^[1] Acute acoustic trauma refers to hearing loss caused by exposure to impulsive or blast noise.^[2] When sound intensity exceeds the elastic limits of the peripheral auditory system,

mechanical damage may occur immediately. Typically, acoustic trauma results from exposure to sound levels exceeding 130 dBA.^[3] Blast injuries can cause tympanic membrane perforation, ossicular disruption, and sensorineural, conductive, or mixed-type hearing loss.^[4]

Among auditory structures, the tympanic membrane is par-

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ticularly vulnerable to blast effects. Its perforation may present with otalgia, otorrhagia, and conductive hearing loss. Although spontaneous healing often occurs within eight weeks, with reported recovery rates ranging from 80% to 94%, this rapid resolution may lead to underreporting in clinical documentation.^[5]

While many previous studies have investigated the general effects of blast trauma on hearing, they have typically focused on either audiological or psychological consequences in isolation.^[6,7] To date, no published study has systematically examined the relationship between the anatomical site of blast-related bodily trauma and hearing thresholds in a battlefield context. This study aims to address that gap by analyzing audiometric outcomes in patients exposed to varying levels of blast energy and presenting with different trauma localizations. The findings may offer new insights into how the distribution and severity of trauma influence auditory outcomes in combat settings.

To our knowledge, this is the first clinical study to integrate trauma localization, tympanic membrane integrity, and blast energy exposure into a unified analysis of hearing thresholds in military personnel. This integrative approach provides a more anatomically and functionally comprehensive understanding of blast-induced auditory damage, contributing original clinical data to support trauma-informed otological care.

MATERIALS AND METHODS

This retrospective study was conducted in accordance with the principles of the Declaration of Helsinki. The requirement for informed consent was waived due to the retrospective nature of the study, and all data were anonymized to ensure patient confidentiality. The study was approved by the institutional ethical committee (2019/2-19/06). The manuscript was prepared in accordance with the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) checklist. Due to the retrospective nature of the study, written informed consent was not required.

We retrospectively enrolled 95 patients between the ages of 30 and 39 who were admitted to our hospital for acoustic trauma and other types of injuries between 2015 and 2018. Patients with any documented history of hearing loss prior to the traumatic event were excluded. This was verified through a review of hospital records, including pre-deployment audiometric evaluations, periodic health check-ups, and military enlistment medical files. Only individuals with confirmed normal hearing status prior to the incident were included. For the control group, we enrolled 51 patients between the ages of 30 and 39 who were admitted to the hospital for routine military health examinations and reported no complaints. Data collected included age, gender, type of explosion, explosion site, otologic examination findings, treatment protocols, and pure-tone thresholds. The type of explosion was catego-

rized into three groups: low energy, medium energy, and high energy. The low-energy group included exposures to light weapons, such as gunshots. The medium-energy group primarily included hand-made explosives. The high-energy group involved exposures such as artillery attacks and missile explosions. The energy groups were defined based on peak sound pressure levels associated with different types of explosive devices, as documented in the literature.^[8,9] Specifically, low-energy blasts, such as gunshots, typically produce sound levels of 140-160 dB sound pressure level (SPL); medium-energy explosives, including improvised devices, range between 160-180 dB SPL; and high-energy sources, such as artillery or missile explosions, exceed 180 dB SPL. This classification reflects the increasing destructive capacity and acoustic impact of the blast source. We also documented the injury sites in patients with multiple traumas. Trauma classifications were based on the anatomical location of injury, as recorded in medical files. Head-related injuries included brain trauma (e.g., concussion, intracranial injury), maxillofacial bone fractures, and eye injuries. Other injuries referred to trauma involving the thorax, trunk, arms, or legs. Trauma laterality was recorded as right-sided, left-sided, or bilateral, depending on the side(s) of the body affected. Patients were also categorized as having unilateral or bilateral trauma based on whether the injury was confined to one side or present on both sides of the body. These groupings were used for comparative analysis in the Results section. Relevant data were obtained from hospital records.

These classifications were applied in subgroup comparisons in the Results section to investigate associations with hearing thresholds and tympanic membrane perforation.

Audiometric testing was conducted in a double-walled booth using an Interacoustics AC40 audiometer (Interacoustics, Assens, Denmark). Supra-aural TDH-39 headphones (Telephonics, NY, USA) were used for conventional audiometry. Pure-tone air conduction thresholds were measured at 0.25, 0.5, 1, 2, 4, and 6 kHz. Bone conduction thresholds were measured at 0.5, 1, 2, and 4 kHz. Hearing loss types were classified according to standard audiological definitions consistent with American Speech-Language-Hearing Association (ASHA) guidelines. Conductive hearing loss was diagnosed when there was an air-bone gap of ≥ 15 dB at two or more tested frequencies, with normal bone conduction thresholds, indicating pathology in the middle or outer ear. Sensorineural hearing loss was defined by elevated bone conduction thresholds without an air-bone gap, reflecting dysfunction of the inner ear or nerve. Mixed hearing loss was identified when both air and bone conduction thresholds were elevated, along with an air-bone gap. Pure tone average was calculated using air conduction (AC) thresholds at 0.5, 1, 2, and 4 kHz. Thresholds were determined using the ascending method. The earliest audiometric evaluation was conducted three months after the incident.

The only exclusion criterion was the absence of an audio-

metric evaluation at least three months after the traumatic incident. Although temporary threshold shifts (TTS) are typically expected to resolve within 10 days to several weeks, any sensorineural hearing loss persisting beyond eight weeks is generally considered permanent (permanent threshold shift, PTS).^[5] To ensure the stability of threshold measurements and avoid the inclusion of reversible hearing loss, a three-month interval was chosen as a conservative and standardized cutoff across all patients.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 22.0 (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to assess the normality of data distribution. As most variables did not meet the assumption of normality, non-parametric methods were applied. For pairwise comparisons, the Mann-Whitney U test was used, and for comparisons involving more than two groups, the Kruskal-Wallis H test was employed. When significant differences were found using the Kruskal-Wallis tests, post-hoc pairwise comparisons were conducted with Bonferroni correction to adjust for multiple testing. A p value of <0.05 was considered statistically significant.

In addition to statistical significance, effect sizes were calculated to assess the magnitude and clinical relevance of group differences, particularly given the non-parametric data distribution and unequal sample sizes. Effect sizes were interpreted according to Cohen's guidelines: values around 0.1 were considered small, 0.3 medium, and 0.5 or above large effects.^[10]

RESULTS

The mean age of the patients was 32.62 ± 2.52 years. All patients were male. Fourteen patients (14.7%) were exposed to low-energy explosions, 53 (55.8%) to medium-energy, and 28 (29.5%) to high-energy explosions. There was no significant age difference between the acoustic trauma and control groups (Mann-Whitney U test, $p=0.075$).

The Relationship Between Hearing Loss and Bodily Injuries

A total of 68 patients (71.6%) had a history of multiple traumas. Among these, 36 patients (52.9%) had unilateral trauma (involving only one side of the body), while 32 patients (47.1%) had bilateral trauma. Right-sided trauma was present in 52 patients (76.4%), and left-sided trauma in 48 patients (70.6%).

Seven patients (10.3%) sustained brain injuries, and 30 patients (44.1%) had maxillofacial trauma. Upper extremity trauma was observed in 38 patients (55.9%): 13 (19.1%) had right-sided, 16 (23.5%) had left-sided, and six (8.8%) had bilateral upper extremity trauma. Lower extremity trauma occurred in 37 patients (54.4%): 12 (17.6%) were right-sided, eight (11.8%) were left-sided, and 15 (22.1%) were bilateral. Eye injuries were present in 17 patients (25%): seven (10.3%)

Table 1. Injury sites in patients with multiple trauma

| | n | % |
|-----------------------|----|------|
| Brain | 7 | 10.3 |
| Maxillofacial | 30 | 44.1 |
| Upper Extremity (Arm) | 38 | 55.9 |
| Right | 13 | 19.1 |
| Left | 16 | 23.5 |
| Bilateral | 6 | 8.8 |
| Lower Extremity (Leg) | 37 | 54.4 |
| Right | 12 | 17.6 |
| Left | 8 | 11.8 |
| Bilateral | 15 | 22.1 |
| Eye | 17 | 25 |
| Right | 7 | 10.3 |
| Left | 3 | 4.4 |
| Bilateral | 7 | 10.3 |
| Thorax | 5 | 7.4 |
| Trunk | 20 | 29.4 |
| Anxiety Disorder | 30 | 44.1 |

had right-sided, three (4.4%) had left-sided, and seven (10.3%) had bilateral eye injuries. Thoracic trauma was reported in five patients (7.4%), and scars on the trunk were documented in 20 patients (29.4%). Additionally, 30 patients (44.1%) were diagnosed with anxiety disorder (Table 1).

Patients with trauma histories were grouped based on trauma localization into two categories: head-related injuries (including brain concussion, maxillofacial bone fracture, and eye injury) and other injuries (including trauma to the arms, thorax, trunk, and legs). There were 30 ears (from 15 patients) in the head-related injury group and 58 ears (from 29 patients) in the other injury group. A statistically significant difference was found between these two groups in air conduction thresholds at 0.25 kHz, 1 kHz, and 2 kHz, as well as in bone conduction thresholds at 0.5 kHz, 1 kHz, and 2 kHz. These thresholds were significantly higher in patients with other injuries (Mann-Whitney U test, $p>0.05$) (Table 2). These results are also presented visually in Figure 1 to facilitate comparison of threshold shifts across trauma groups.

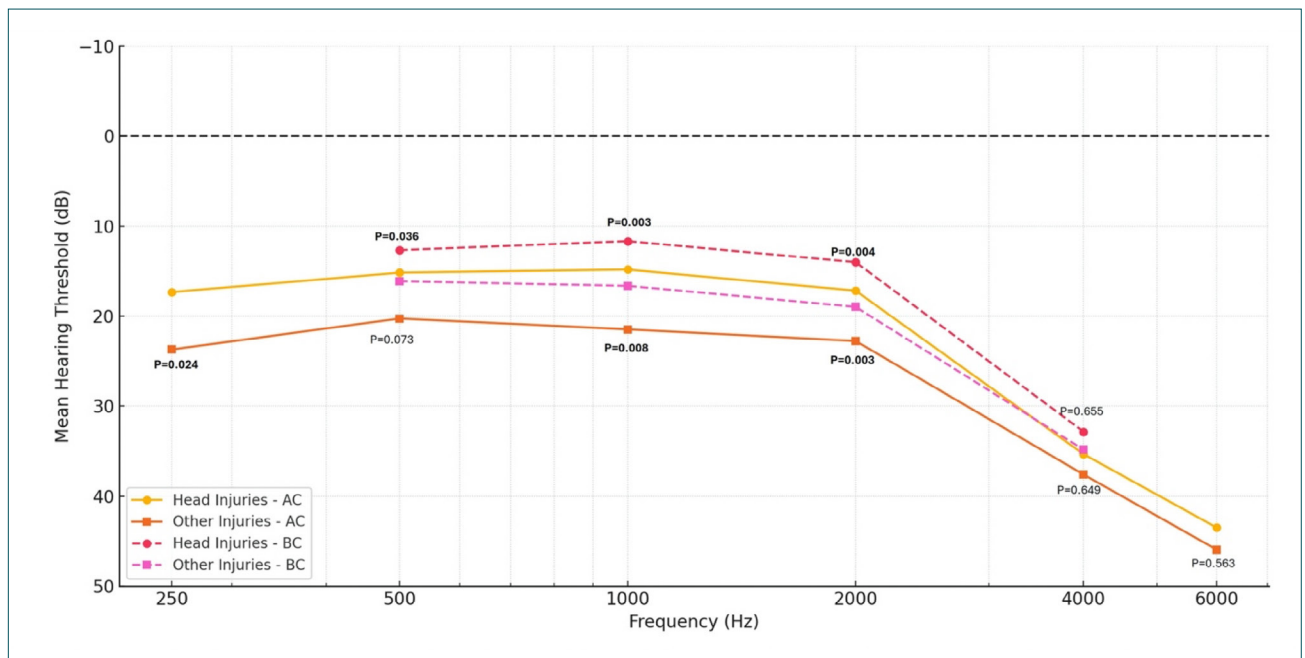
The Relationship Between Tympanic Membrane Perforation and Trauma Sites

The relationship between tympanic membrane perforation and various trauma sites was analyzed. No significant association was found between tympanic membrane perforation and the following conditions: brain injury (Pearson's chi-square test, $p=0.139$), maxillofacial trauma (Pearson's chi-square test with Yates' continuity correction, $p=0.660$), thoracic trauma (Pearson's chi-square test, $p=0.733$), trunk trauma (Pearson's chi-square test with Yates' continuity correction $p=0.433$),

Table 2. Hearing thresholds in patients with head-related (brain, maxillofacial, and eye trauma) versus other types of injuries (arms, thorax, trunk, and legs)

| | Head-Related Injuries (n=30) | | Other Injuries (n=58) | | Test Statistic | p | Cohen's d |
|----------|------------------------------|------------------|-----------------------|------------------|----------------|-------|-----------|
| | Mean±SD | Median (min-max) | Mean±SD | Median (min-max) | | | |
| AC | | | | | | | |
| 0.25 kHz | 17.33±9.35 | 15 (5-45) | 23.71±14.97 | 20 (10-80) | -2.253 | 0.024 | -0.478 |
| 1 kHz | 14.83±12.90 | 10 (5-65) | 21.47±13.92 | 15 (5-65) | -2.648 | 0.008 | -0.488 |
| 2 kHz | 17.17±19.24 | 10 (5-70) | 22.76±15.22 | 20 (5-70) | -2.921 | 0.003 | -0.335 |
| BC | | | | | | | |
| 0.5 kHz | 12.67±6.66 | 10 (5-25) | 16.12±7.78 | 15 (5-40) | -2.102 | 0.036 | -0.465 |
| 1 kHz | 11.67±11.90 | 10 (5-65) | 16.64±10.32 | 15 (5-45) | -2.986 | 0.003 | -0.470 |
| 2 kHz | 14.00±14.29 | 15 (5-60) | 18.97±12.38 | 15 (5-60) | -2.916 | 0.004 | -0.380 |

*Mann-Whitney U Test. SD: Standard deviation; min: Minimum; max: Maximum; AC: Air conduction, BC: Bone conduction.

**Figure 1.** Graphical representation of mean air conduction (AC) and bone conduction (BC) hearing thresholds (250-6000 Hz) across different trauma groups.

anxiety disorder (Pearson's chi-square test with Yates' continuity correction, $p=0.166$), upper extremity injury (Pearson's chi-square test with Yates' continuity correction, $p=0.166$), or lower extremity injury (Pearson's chi-square test with Yates' continuity correction, $p=0.060$). However, a significant relationship was observed between eye injury and tympanic membrane perforation (Pearson's chi-square test with Yates' continuity correction, $p=0.004$), with 56.3% of patients with eye injuries exhibiting eardrum perforation, while 43.8% did not. The relationship between trauma sites and tympanic membrane perforation is presented in Table 3.

The Relationship Between Tympanic Membrane Perforation and Hearing Thresholds

Tympanic membrane perforation was observed in 32 patients (33.7%). Of these, eight patients (8.42%) had bilateral perforations, 16 patients (16.84%) had right tympanic membrane perforation only, and eight patients (8.42%) had left tympanic membrane perforation only.

Patients were grouped into two categories: those with tympanic membrane perforation and those without (Table 4). In total, there were 40 ears with tympanic membrane perforation.

Table 3. Relationship between bodily injuries and tympanic membrane perforation

| | Non-Perforated (n=90) | Perforated (n=44) | Total (n=136) | p |
|------------------------|-----------------------|-------------------|---------------|--------------------|
| Brain Injury | | | | |
| No | 78 (63.9%) | 44 (36.1%) | 122 (89.7%) | 0.139 ^a |
| Yes | 12 (85.7%) | 2 (14.3%) | 14 (10.3%) | |
| Maxillofacial Trauma | | | | |
| No | 52 (68.4%) | 24 (31.6%) | 76 (55.9%) | 0.660 ^b |
| Yes | 38 (63.3%) | 22 (36.7%) | 60 (44.1%) | |
| Upper Extremity Injury | | | | |
| No | 48 (72.7%) | 18 (27.3%) | 66 (48.5%) | 0.166 ^b |
| Yes | 42 (60%) | 28 (40%) | 70 (51.5%) | |
| Lower Extremity Injury | | | | |
| No | 38 (57.6%) | 28 (42.4%) | 66 (48.5%) | 0.060 ^b |
| Yes | 52 (74.3%) | 18 (25.7%) | 70 (51.5%) | |
| Eye Injury | | | | |
| No | 76 (73.1%) | 28 (26.9%) | 104 (76.5%) | 0.004 ^b |
| Yes | 14 (43.8%) | 18 (56.3%) | 32 (23.5%) | |
| Thoracic Trauma | | | | |
| No | 84 (66.7%) | 42 (33.3%) | 126 (92.6%) | 0.733 ^a |
| Yes | 6 (60%) | 4 (40%) | 10 (7.4%) | |
| Trunk Trauma | | | | |
| No | 66 (68.8%) | 30 (31.3%) | 96 (70.6%) | 0.433 ^b |
| Yes | 24 (60%) | 16 (40%) | 40 (29.4%) | |
| Anxiety Disorder | | | | |
| No | 46 (60.5%) | 30 (39.5%) | 76 (55.9%) | 0.166 ^b |
| Yes | 44 (73.3%) | 16 (26.7%) | 60 (44.1%) | |

^aPearson's chi-square test; ^bPearson's chi-square test with Yates' continuity correction.

ration and 150 ears without. Air conduction thresholds at 0.25, 0.5, 1, 2, and 4 kHz were significantly higher in ears with tympanic membrane perforation (Mann-Whitney U test, $p < 0.05$). However, there was no significant difference in bone conduction (BC) thresholds between the two groups (Mann-Whitney U test, $p > 0.05$) (Table 4).

To assess the vulnerability of the inner ear, right ear thresholds in patients with right tympanic membrane perforation were compared with left ear thresholds in those with left tympanic membrane perforation. No significant difference was found between the groups across any frequency (Mann-Whitney U test, $p > 0.05$).

Hearing Characteristics Among Explosion Groups

The explosion groups and the control group were compared in terms of air and bone conduction thresholds. Descriptive statistics for the pure-tone thresholds are presented in Table 5.

For the 0.25 kHz AC threshold in the right ear, there was no significant difference among the low-, medium-, and high-energy explosion groups. However, when compared to the

control group, the low-energy group showed no significant difference, while both the medium- and high-energy groups had significantly higher thresholds than the control group (Kruskal-Wallis test, $p < 0.001$). At all other AC frequencies, there were no significant differences between the low-, medium-, and high-energy groups. However, the control group had significantly lower thresholds than all three explosion groups (Kruskal-Wallis test, $p < 0.001$). Although no significant differences were found among the low-, medium-, and high-energy groups in any of the BC thresholds evaluated for the right ear, the control group showed significantly lower thresholds than all three (Kruskal-Wallis test, $p < 0.001$).

For the 0.25 kHz and 0.5 kHz AC thresholds in the left ear, there was no significant difference among the low-, medium-, and high-energy groups. When compared with the control group, there was no significant difference for the low-energy group, whereas the medium- and high-energy groups had significantly higher thresholds (Kruskal-Wallis test, $p < 0.001$). At all other AC frequencies, no significant differences were found among the low-, medium-, and high-energy groups. However,

Table 4. Relationship between air conduction (AC), bone conduction (BC) thresholds, and tympanic membrane perforation

| | Perforated (n=40) | | Non-Perforated (n=150) | | Test Statistic | p | Cohen's d |
|----------|-------------------|------------------|------------------------|------------------|----------------|--------|-----------|
| | Mean±SD | Median (min-max) | Mean±SD | Median (min-max) | | | |
| AC | | | | | | | |
| 0.25 kHz | 37.75±20.88 | 35 (5-90) | 19.7±12.93 | 15 (5-110) | -5.646 | <0.001 | -12.089 |
| 0.5 kHz | 33±20.59 | 32.5 (5-85) | 17.3±13.36 | 15 (5-120) | -4.803 | <0.001 | -10.366 |
| 1 kHz | 31.25±20.65 | 30 (5-85) | 17.5±16.34 | 10 (5-120) | -4.578 | <0.001 | -0.7937 |
| 2 kHz | 34.13±22.18 | 30 (5-95) | 20.87±19.66 | 15 (5-120) | -4.176 | <0.001 | -0.6561 |
| 4 kHz | 45.5±25.41 | 45 (5-95) | 37.3±28.37 | 30 (5-120) | -1.971 | 0.049 | -0.2951 |
| 6 kHz | 58±26.69 | 55 (10-115) | 46.53±29.66 | 42.5 (5-120) | -2.333 | 0.020 | -0.3944 |
| BC | | | | | | | |
| 0.5 kHz | 16.13±10.83 | 12.5 (5-55) | 15.83±12.8 | 15 (5-120) | -0.268 | 0.789 | -0.0235 |
| 1 kHz | 12.75±9.54 | 10 (5-55) | 16.03±15.67 | 10 (5-120) | -0.907 | 0.364 | 0.2248 |
| 2 kHz | 19.25±13.66 | 15 (5-75) | 19.7±18.95 | 15 (5-120) | -1.160 | 0.246 | 0.0250 |
| 4 kHz | 31.13±21.32 | 27.5 (5-85) | 36.17±27.95 | 30 (5-120) | -0.557 | 0.577 | 0.1887 |
| AC-PTA | 35.97±20 | 32.5 (5-82.5) | 23.24±16.49 | 21.3 (5-120) | -4.038 | <0.001 | -0.7366 |
| BC-PTA | 19.81±11.37 | 17.5 (5-61.25) | 21.93±15.9 | 20 (5-120) | -0.337 | 0.736 | 0.1407 |

*Mann-Whitney U Test. SD: Standard deviation; min: Minimum; max: Maximum; PTA: Pure tone average of four frequencies.

Table 5. Descriptive statistics by energy group

| | Right Ear | | | | | | |
|----------------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|
| | PTA | 0.25 kHz | 0.5 kHz | 1 kHz | 2 kHz | 4 kHz | 6 kHz |
| Low Energy (n=14) | 25.36±14.87 | 19.29±9.78 | 17.86±9.75 | 19.29±16.51 | 23.57±19.56 | 40.71±30.63 | 45.71±32.57 |
| Medium Energy (n=53) | 24.93±17 | 22.83±15.8 | 20.47±14.72 | 20.09±16.22 | 23.21±19.86 | 35.94±27.28 | 48.3±27.61 |
| High Energy (n=28) | 24.6±17.43 | 23.75±17.46 | 20±16.67 | 18.93±16.29 | 21.43±18.6 | 38.04±28.2 | 46.43±31.91 |
| Control (n=51) | 8.43±3.02 | 12.35±4.93 | 8.53±3.78 | 6.57±3.08 | 6.86±4 | 11.37±6.33 | 15.1±7.78 |
| | Left Ear | | | | | | |
| | PTA | 0.25 kHz | 0.5 kHz | 1 kHz | 2 kHz | 4 kHz | 6 kHz |
| Low Energy (n=14) | 22.32±12.35 | 20±9.61 | 15.36±9.5 | 14.64±8.87 | 20±15.19 | 39.29±25.93 | 46.79±25.77 |
| Medium Energy (n=53) | 26.86±21.8 | 24.62±18.39 | 21.79±19.02 | 22.08±22.92 | 25±25 | 38.58±30.53 | 49.34±30.92 |
| High Energy (n=28) | 29.42±16.95 | 26.25±19.47 | 23.21±19.4 | 22.68±18.08 | 26.07±20.56 | 45.71±24.22 | 54.64±28.83 |
| Control (n=51) | 8.63±2.78 | 12.45±4.62 | 9.41±3.69 | 6.57±3.81 | 6.86±3.87 | 11.27±5.99 | 13.82±6.29 |

*PTA: Pure tone average of four frequencies; Hz: Hertz. Values are given as mean ± standard deviation.

the control group had significantly lower thresholds than all three explosion groups (Kruskal-Wallis test, $p<0.001$). Similarly, for all BC thresholds in the left ear, there were no significant differences among the explosion groups, while the control group showed significantly lower thresholds than all of them (Kruskal-Wallis test, $p<0.001$). The comparisons between energy groups and the control group are presented in Table 6.

In summary, no significant differences were observed among the low-, medium-, and high-energy groups at any frequency in either the right or left ear. Mean hearing thresholds by energy level are visually summarized in Figure 2.

The most prominent differences in hearing thresholds were observed at higher frequencies (particularly at 4 and 6 kHz), where blast-exposed patients—especially those exposed to medium- and high-energy explosions—showed significantly

Table 6. Air conduction (AC) and bone conduction (BC) pure tone thresholds by group

| | Low Energy (n=14) | Medium Energy (n=53) | High Energy (n=28) | Control (n=51) | Test Statis-tic | p* | ε ² |
|------------------|----------------------------|----------------------------|---------------------------|-------------------------|-----------------|--------|----------------|
| Right Ear | | | | | | | |
| AC | | | | | | | |
| 0.25 kHz | 20 (10-40) ^{ab} | 15 (5-80) ^b | 20 (10-75) ^b | 10 (5-25) ^a | 21.27 | <0.001 | 0.147 |
| 0.5 kHz | 12.5 (10-35) ^b | 15 (5-70) ^b | 15 (5-85) ^b | 10 (0-20) ^a | 36.881 | <0.001 | 0.254 |
| 1 kHz | 12.5 (5-65) ^b | 15 (5-65) ^b | 12.5 (5-75) ^b | 5 (0-15) ^a | 39.016 | <0.001 | 0.269 |
| 2 kHz | 20 (5-70) ^b | 15 (5-80) ^b | 15 (5-70) ^b | 5 (0-20) ^a | 37.912 | <0.001 | 0.261 |
| 4 kHz | 40 (5-90) ^b | 30 (5-120) ^b | 35 (5-95) ^b | 10 (0-25) ^a | 32.809 | <0.001 | 0.226 |
| 6 kHz | 40 (5-95) ^b | 45 (5-120) ^b | 47.5 (5-115) ^b | 15 (0-30) ^a | 45.36 | <0.001 | 0.313 |
| PTA | 23.75 (8-58) ^b | 23.75 (5-79) ^b | 19.38 (5-80) ^b | 9 (3-16) ^a | 49.852 | <0.001 | 0.357 |
| BC | | | | | | | |
| 0.5 Hz | 10 (10-35) ^b | 15 (5-50) ^b | 15 (5-55) ^b | 10 (0-20) ^a | 26.69 | <0.001 | 0.184 |
| 1 kHz | 10 (5-65) ^b | 10 (5-50) ^b | 10 (5-45) ^b | 5 (0-15) ^a | 27.636 | <0.001 | 0.191 |
| 2 kHz | 20 (5-70) ^b | 10 (5-80) ^b | 12.5 (5-60) ^b | 5 (0-20) ^a | 36.849 | <0.001 | 0.254 |
| 4 kHz | 40 (5-90) ^b | 20 (5-90) ^b | 32.5 (5-90) ^b | 10 (0-20) ^a | 28.193 | <0.001 | 0.194 |
| PTA | 21.88 (8-58) ^b | 15 (5-65) ^b | 17.5 (5-46) ^b | 9 (3-16) ^a | 32.329 | <0.001 | 0.245 |
| Left Ear | | | | | | | |
| AC | | | | | | | |
| 0.25 kHz | 17.5 (10-40) ^{ab} | 20 (5-110) ^b | 20 (5-90) ^b | 10 (5-25) ^a | 28.467 | <0.001 | 0.196 |
| 0.5 kHz | 12.5 (5-40) ^{ab} | 15 (5-120) ^b | 15 (5-80) ^b | 10 (5-20) ^a | 32.063 | <0.001 | 0.221 |
| 1 kHz | 15 (5-30) ^b | 15 (5-120) ^b | 15 (5-80) ^b | 5 (0-20) ^a | 48.234 | <0.001 | 0.333 |
| 2 kHz | 17.5 (5-50) ^b | 15 (5-120) ^b | 22.5 (5-85) ^b | 5 (5-20) ^a | 51.863 | <0.001 | 0.358 |
| 4 kHz | 42.5 (5-75) ^b | 25 (5-120) ^b | 45 (10-90) ^b | 10 (0-25) ^a | 47.781 | <0.001 | 0.330 |
| 6 kHz | 50 (10-90) ^b | 45 (5-120) ^b | 52.5 (5-120) ^b | 15 (5-30) ^a | 60.281 | <0.001 | 0.416 |
| PTA | 24.38 (5-43) ^b | 21.25 (5-120) ^b | 25 (8-83) ^b | 7.5 (4-14) ^a | 59.741 | <0.001 | 0.422 |
| BC | | | | | | | |
| 0.5 kHz | 12.5 (5-40) ^b | 15 (5-120) ^b | 15 (5-30) ^b | 10 (0-20) ^a | 28.66 | <0.001 | 0.198 |
| 1 kHz | 15 (5-30) ^b | 10 (5-120) ^b | 15 (5-45) ^b | 5 (0-20) ^a | 43.592 | <0.001 | 0.301 |
| 2 kHz | 17.5 (5-50) ^b | 15 (5-120) ^b | 15 (5-75) ^b | 5 (0-20) ^a | 51.525 | <0.001 | 0.355 |
| 4 kHz | 42.5 (5-75) ^b | 25 (5-120) ^b | 35 (10-90) ^b | 10 (0-25) ^a | 43.031 | <0.001 | 0.297 |
| PTA | 24.38 (5-43) ^b | 18.75 (5-120) ^b | 20.63 (8-48) ^b | 7.5 (3-14) ^a | 59.695 | <0.001 | 0.422 |

*Kruskal-Wallis Test. No statistical difference exists between groups sharing the same letter (a, b, or ab), whereas groups labeled with different letters show a statistically significant difference in pairwise comparisons. PTA: Pure tone average of four frequencies. Values are given as median (minimum-maximum). ε²: Effect size.

elevated air conduction thresholds compared to controls. These effects were most pronounced in the left ear at 6 kHz, where the difference exceeded 35 dB. Additionally, pure tone average (PTA) values were significantly higher across all blast groups, indicating widespread auditory dysfunction extending beyond isolated high-frequency loss.

The Relationship Between Site of Acoustic Trauma and Hearing Thresholds

Fifteen patients were bilaterally affected by the explosion. In total, 38 patients were affected in the right ear, and 42 pa-

tients were affected in the left ear.

When ears affected by acoustic trauma were compared with unaffected ears, all air and bone conducted thresholds were significantly higher on the affected side (Table 7). The mean hearing thresholds for affected and unaffected ears are illustrated in Figure 3. Speech discrimination scores were also significantly lower on the affected site ($Z=-5.837$, $p<0.001$).

A summary of mean air conduction thresholds across blast energy exposure groups, averaged for both ears, is provided in Table 8.

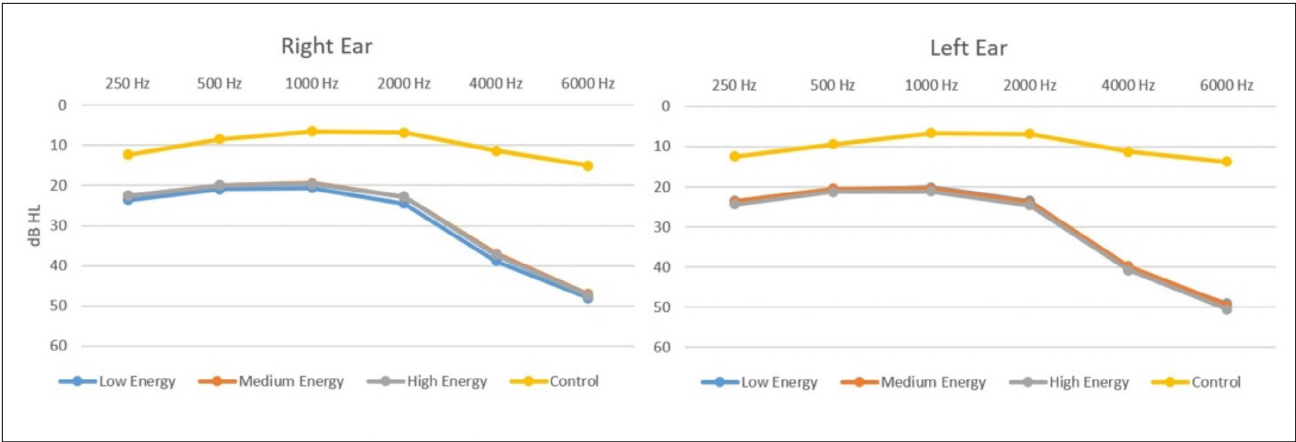


Figure 2. Comparison of hearing thresholds between patient and control groups.

| Table 7. Comparison of hearing thresholds between blast-affected and unaffected ears | | | | | | |
|--|--------------|------------------|----------------|------------------|--------|--------|
| | Affected Ear | | Unaffected Ear | | Z | p* |
| | Mean±SD | Median (Min-Max) | Mean±SD | Median (Min-Max) | | |
| AC | | | | | | |
| 0.25 kHz | 28.68±18.86 | 25 (5-110) | 16.38±9 | 15 (5-65) | -5.505 | <0.001 |
| 0.5 kHz | 26.27±18.85 | 20 (5-120) | 12.81±6.93 | 10 (5-40) | -6.255 | <0.001 |
| 1 kHz | 26.91±20.63 | 20 (5-120) | 11.44±7.88 | 10 (5-45) | -6.567 | <0.001 |
| 2 kHz | 31.95±23.04 | 25 (5-120) | 12.25±9.07 | 10 (5-60) | -7.113 | <0.001 |
| 4 kHz | 48.64±27.44 | 45 (5-120) | 25.81±22.8 | 15 (5-90) | -5.893 | <0.001 |
| 6 kHz | 59.64±27.68 | 60 (5-120) | 34.25±25.07 | 25 (5-110) | -6.032 | <0.001 |
| BC | | | | | | |
| 0.5 kHz | 18.86±14.85 | 15 (5-120) | 11.81±5.81 | 10 (5-40) | -4.209 | <0.001 |
| 1 kHz | 18.55±17.52 | 15 (5-120) | 10.94±7.38 | 10 (5-45) | -3.794 | <0.001 |
| 2 kHz | 25.23±20.64 | 20 (5-120) | 11.88±8.8 | 10 (5-60) | -5.775 | <0.001 |
| 4 kHz | 42.14±27.16 | 40 (5-120) | 25.44±22.95 | 15 (5-90) | -4.640 | <0.001 |
| SDS | 82.4±19.87 | 88 (0-100) | 94.9±7.61 | 100 (64-100) | -5.837 | <0.001 |
| *Mann-Whitney U test. AC: Air Conduction; BC: Bone Conduction; SDS: Speech Discrimination Score. | | | | | | |

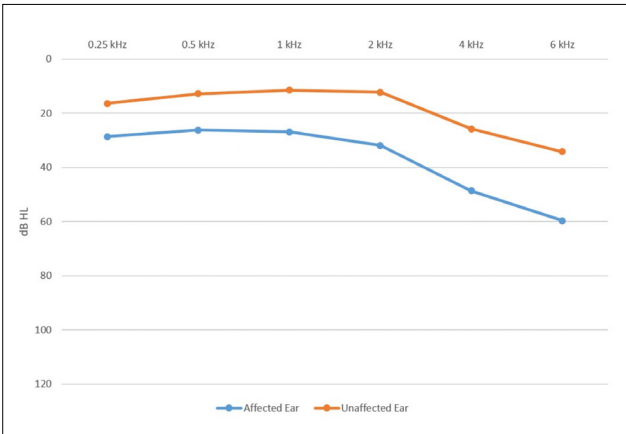


Figure 3. Air conduction hearing thresholds of affected versus unaffected ears.

DISCUSSION

This study aimed to explore how different types of blast-related trauma influence hearing thresholds in military personnel. We specifically examined the associations between trauma localization, tympanic membrane status, and blast energy levels, as these variables are frequently encountered in battlefield injuries but are rarely analyzed together. Our findings showed that tympanic membrane perforation was associated with elevated air conduction thresholds but did not appear to protect cochlear function. No significant differences in hearing thresholds were observed among low-, medium-, and high-energy explosion groups. Patients with head-related trauma demonstrated lower hearing thresholds, which may reflect a survivorship bias related to the severity of the trauma. Additionally, we identified a statistically significant asso-

Table 8. Mean air conduction hearing thresholds (Mean \pm SD) by blast energy exposure, averaged across right and left ears at each test frequency

| Frequency (Hz) | Head Injuries (Mean \pm SD) | Other Injuries (Mean \pm SD) | Perforated (Mean \pm SD) | Non-Perforated (Mean \pm SD) | Low Energy (Avg) | Medium Energy (Avg) | High Energy (Avg) | Affected Ear | Unaffected Ear |
|----------------|-------------------------------|--------------------------------|----------------------------|--------------------------------|-------------------|---------------------|-------------------|-------------------|-------------------|
| 0.25 kHz | 17.33 \pm 9.35 | 23.71 \pm 14.97 | 37.75 \pm 20.88 | 19.7 \pm 12.93 | 19.64 \pm 9.7 | 23.73 \pm 17.09 | 25.0 \pm 18.46 | 28.68 \pm 18.86 | 16.38 \pm 9 |
| 0.5 kHz | 15.17 \pm 8.56 | 20.26 \pm 12.99 | 33 \pm 20.59 | 17.3 \pm 13.36 | 16.61 \pm 9.62 | 21.13 \pm 16.87 | 21.61 \pm 18.04 | 26.27 \pm 18.85 | 12.81 \pm 6.93 |
| 1 kHz | 14.83 \pm 12.90 | 21.47 \pm 13.92 | 31.25 \pm 20.65 | 17.5 \pm 16.34 | 16.96 \pm 12.69 | 21.09 \pm 19.57 | 20.8 \pm 17.18 | 26.91 \pm 20.63 | 11.44 \pm 7.88 |
| 2 kHz | 17.17 \pm 19.24 | 22.76 \pm 15.22 | 34.13 \pm 22.18 | 20.87 \pm 19.66 | 21.79 \pm 17.38 | 24.11 \pm 22.43 | 23.75 \pm 19.58 | 31.95 \pm 23.04 | 12.25 \pm 9.07 |
| 4 kHz | 35.33 \pm 26.59 | 37.59 \pm 26.78 | 45.5 \pm 25.41 | 37.3 \pm 28.37 | 40.0 \pm 28.28 | 37.26 \pm 28.91 | 41.88 \pm 26.21 | 48.64 \pm 27.44 | 25.81 \pm 22.8 |
| 6 kHz | 43.50 \pm 30.88 | 45.95 \pm 27.87 | 58 \pm 26.69 | 46.53 \pm 29.66 | 46.25 \pm 29.17 | 48.82 \pm 29.27 | 50.53 \pm 30.37 | 59.64 \pm 27.68 | 34.25 \pm 25.07 |

Note: Values represent the average of bilateral thresholds for participants exposed to low-, medium-, and high-energy blasts. All thresholds are measured in decibels hearing level (dB HL).

ciation between eye injuries and tympanic membrane perforation, likely due to their close anatomical proximity.

The ear is one of the most vulnerable organs in the body during blast. Since military personnel are most frequently exposed to blast trauma, acoustic trauma is predominantly observed in this population.^[4] Acute acoustic trauma is caused by exposure to excessive noise, typically in the range of 90-130 dB, lasting for approximately one millisecond. In addition to the previously mentioned effects, acute acoustic trauma can also lead to vasospasm and hypoxia, which damage the hair cells.^[11] Excessive release of neurotransmitters can cause swelling of the synapses between hair cells and auditory nerve fibers. Ultimately, free oxygen radicals and inflammatory cytokines formed in the inner ear cause more microcirculation defects, further exacerbating inner ear hypoxia.^[12] The 4000 Hz frequency is the most susceptible to acoustic trauma due to the resonance frequency of the ear canal. The basal cochlea is the most vulnerable part of the inner ear due to its high metabolic activity.^[13] Additionally, antioxidant glutathione levels are higher in the apical outer hair cells compared to those in the basal cochlea.^[14] In our study, low-frequency thresholds appeared to be better preserved in the low-energy group, likely due to the higher antioxidant activity at the cochlear apex.

The amount of energy transmitted through the cochlea is another important factor in hearing loss. In a study conducted by Tlikoski in the Finnish Army, it was found that greater energy transfer through the cochlea correlated with more significant hearing loss. He concluded that explosions caused more damage than rifle fire.^[15] However, in our study, no significant differences in hearing thresholds were observed among the different explosive energy groups. According to the inverse square law, sound pressure is inversely proportional to the square of the distance from the source.^[16] Due to the nature of this study, the exact distance between the sound source and each patient is unknown. This may explain

why no differences were observed between the explosion groups in terms of hearing thresholds. However, it is well established that exposure to medium- or high-energy explosions affects the cochlea across all frequencies.

Tympanic membrane perforation is thought to offer some protective effects to the cochlea by inducing conductive hearing loss and reducing the amount of energy transmitted to the inner ear.^[17] Several animal studies have sought evidence to support this theory.^[18-20] In a recent Turkish study, Taşlı et al. (2021) concluded that while tympanic membrane perforation does not prevent inner ear damage, it may offer partial protection to cochlear structures—a hypothesis worthy of further investigation.^[21] Similarly, Kurioka et al. (2022), using a murine blast model, demonstrated that tympanic membrane perforation may reduce peripheral cochlear synaptic disruption but does not prevent damage to the central auditory pathway.^[22] However, there are also publications that do not support the protective effect of tympanic membrane perforation.^[23,24] In our study, we did not observe any such protective effect. When comparing hearing thresholds across all frequencies between perforated and non-perforated right ears, we found that all air conduction thresholds between 250 and 4000 Hz in the right ear were elevated due to conductive hearing loss. In the left ear, a similar pattern was observed between 250 and 2000 Hz. In the left ear, the bone-conduction threshold at 500 Hz was significantly higher in the non-perforated group compared to the perforated group. However, since the same finding was not observed in the right ear, it is not possible to conclude a protective effect of tympanic membrane perforation. This result aligns with the pathophysiological understanding that blast waves can transmit energy beyond the tympanic membrane. As described by Bukowski (2023), air-filled structures such as the middle and inner ear are highly susceptible to overpressure due to their low acoustic impedance. Consequently, tympanic membrane perforation does not necessarily prevent inner ear trauma, as the energy from the blast wave may still reach and damage cochlear structures.^[25]

Current literature primarily focuses on tinnitus and the psychological effects of acoustic trauma in war zones. In this context, this study might be the first to evaluate both whole-body injuries and hearing loss simultaneously. We compared hearing thresholds between patients with head-related injuries and those with other types of trauma. Interestingly, patients with head-related trauma exhibited lower hearing thresholds. One possible explanation is that individuals with more severe head injuries may have been underrepresented in our cohort due to higher mortality or the inability to undergo audiometric testing. Therefore, our findings may reflect a survivorship bias and should be interpreted with this limitation in mind. As Wells et al. noted, "As is the case in most studies of hearing following head trauma, this study included only patients who survived their injuries. Thus, the results may underrepresent the extent of auditory damage in the most severe trauma cases."^[26] Similar to our interpretation, previous studies have also acknowledged that blast-induced auditory injuries associated with head trauma may be underestimated due to the exclusion of fatal cases. A recent study emphasized that survivors included in such analyses likely represent a biased sample of less severe injuries, potentially underreporting the extent of damage to auditory structures in high-impact trauma cases.^[27]

We also evaluated the relationship between tympanic membrane perforation and trauma sites. The only relationship found was between eye injury and tympanic membrane perforation, which may be explained by their close anatomical proximity. This finding is consistent with previous reports from mass casualty events, where injuries to the head, face, and ear frequently co-occur due to anatomical closeness and shared exposure to blast pressure waves.^[28]

Hearing loss is often underestimated in military personnel hospitalized with multiple injuries sustained on the battlefield. As a result, access to timely medical treatment is frequently delayed, reducing the likelihood of hearing recovery. This study also aims to raise awareness of this issue.

An important limitation of this study is the absence of data on the exact distance between the injured individual and the blast source. In battlefield conditions, it is rarely feasible to objectively measure or retrospectively estimate this distance due to the chaotic nature of combat environments. Furthermore, relying on subjective self-reports from patients is unreliable, as acute stress, confusion, and sensory overload during such traumatic events typically impair accurate recall. This lack of spatial data introduces a significant confounding factor, as proximity to the blast is a well-established factor influencing the severity of auditory damage. Future prospective studies should incorporate objective spatial measurements whenever possible to better elucidate the relationship between blast intensity and hearing outcomes. A secondary limitation of this study is that the number of individuals who died at the scene is unknown, which prevents calculation of the mortality rate. Another limitation is the lack of consistent documenta-

tion regarding the localization and size of tympanic membrane perforations. These parameters were not uniformly recorded in patient files due to the retrospective design and, therefore, could not be included in the analysis. Future prospective studies may help clarify whether perforation characteristics influence hearing thresholds.

Unlike prior research that has focused predominantly on the psychological sequelae and tinnitus following blast exposure, our study uniquely investigates the relationship between the physical localization of injuries and hearing thresholds. This novel approach provides clinically relevant insights for both otologists and trauma teams working with combat-exposed individuals.

CONCLUSION

Our findings indicate that tympanic membrane perforation is associated with increased air conduction thresholds, without evidence of cochlear protection. Although some animal studies have suggested partial protective effects, our clinical data did not support these findings. The observed relationship between non-head-related trauma and higher hearing thresholds may reflect a survivorship bias, in which individuals with severe head trauma did not survive to undergo evaluation. Notably, eye injuries were significantly associated with tympanic membrane perforation, suggesting a shared anatomical vulnerability. Despite comparable hearing thresholds across different blast energy groups, all blast-exposed patients exhibited worse hearing than controls. These results highlight the importance of comprehensive auditory evaluation in military personnel with blast injuries and offer insight into the anatomical and mechanical contributors to hearing loss in combat settings.

Ethics Committee Approval: This study was approved by the Health Sciences University Ethics Committee (Date: 17.01.2019, Decision No: 2019/2-19/06).

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Authorship Contributions: Concept: C.K., B.S.; Design: C.K., N.D.Ö., K.Ş., B.S.; Supervision: C.K., B.S.; Resource: B.S., K.Ş.; Materials: C.K., B.S.; Data collection and/or processing: C.K., N.D.Ö., K.Ş., B.S.; Analysis and/or interpretation: C.K.; Literature review: C.K., B.S.; Writing: C.K.; Critical review: B.S.

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ORJİNAL ÇALIŞMA - ÖZ

Savaş alanında gör  len yaralanmalar ve i  tme kaybı arasındaki ili  ki: Retrospektif   alışma

AMA  : Bu   alışma, patlayıcı etkisine maruz kalan askeri personelde fiziksel yaralanmaya neden olacak d  zeydeki travma ile sonrasında geli  en i  tme kaybı arasındaki ili  kiyi ara  tırmayı ama  lamaktadır.

GERE   VE Y  NTEM: 2015-2018 yılları arasında patlama kaynaklı yaralanmalar ve akustik travma nedeniyle ba  vuran, ya  ları 30-39 arasında de  i  en 95 hasta retrospektif olarak incelendi.   ik  yeti olmayan 51 askeri personelden olu  an bir kontrol grubu da   alışmaya d  hil edildi. Hastalar, travmanın yeri, patlamanın enerji d  zeyi ve timpanik membran perforasyonunun varlı  ına g  re sınıflandırıldı. Travma desenleri ile i  itsel sonu  lar arasındaki ili  kiyi de  erlendirmek amacıyla, gruplar arasında i  tme e  ikleri ve klinik   zellikler kar  ıla  tırıldı.

BULGULAR: Kafayla ili  ekli travma ge  iren hastalarda, di  er travma t  rlerine sahip hastalara kıyasla bir  ok frekansta hava ve kemik yolu e  iklerinin anlamlı   ekilde daha d    k oldu  u bulundu ($p<0.05$). Timpanik membran perforasyonu, g  z yaralanmasıyla anlamlı   ekilde ili  kiliydi ($p=0.004$) ve hava yolu e  iklerinin y  kselmesine neden oluyordu ($p<0.05$); ancak kemik yolu e  ikleriyle ili  ki g  stermedi. Orta ve y  ksek enerjili patlamalara maruz kalan hastalarda, t  m frekanslarda kontrol grubuna kıyasla e  ikler anlamlı   ekilde daha y  ksekti ($p<0.001$).

SONU  : Patlamaya ba  lı akustik travma, farklı frekanslarda i  tme kaybı ile ili  kilidir. Timpanik membran perforasyonu, hava yolu kaynaklı i  tme kaybına katkıda bulunmaktadır. G  z yaralanmaları, anatomik yakınlık nedeniyle kulak zarı hasarıyla ili  ekli olabilir. Savaş alanındaki travma hastalarının multidisipliner de  erlendirmesine i  tme testlerinin de entegre edilmesi gerekti  i d    n  lmektedir.

Anahtar s  zc  kler: Akustik travma; i  tme kaybı; patlama yaralanması; savaş alanı odyolojisi; timpanik membran.

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