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Blast-Induced Auditory Injury: From Peripheral Pathology to Central Neurodegeneration

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Abstract

Introduction:
Blast wave exposure represents a primary cause of complex auditory and neurological injuries. The ear acts as a sensitive pressure transducer, making it exceptionally vulnerable to the intense mechanical forces of a blast. This review provides an overview of the multidimensional damage to the auditory system following blast events.

Materials and Methods:
We conducted a systematic literature search across PubMed, Scopus, Web of Science, and Google Scholar (January 2000–January 2025) using comprehensive search terms. After screening 462 unique records, 36 original research studies (27 animal, 8 human, 1 computational) met the inclusion criteria and underwent quality assessment.

Results:
Evidence suggests that blast exposure causes widespread auditory impairment, ranging from cochlear synaptopathy to central neurodegeneration. Blast-related injuries involve distinct biomechanical forces that differ from conventional noise trauma. Furthermore, cumulative damage from repeated exposures leads to chronic functional impairments, including tinnitus and difficulty understanding speech in noisy environments.

Conclusion:
Effective management of blast-induced hearing loss requires a multidisciplinary approach. Future research needs to focus on validating sensitive biomarkers for early diagnosis and developing targeted neuroprotective therapies.

Keywords: Blast Injuries, Cochlear Synaptopathy, Noise-Induced, Hearing Loss, Tinnitus, Auditory System.

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Introduction

A blast wave is a sudden increase in atmospheric pressure caused by the rapid conversion of an explosive to gas, producing very intense wave It has serious effects on body structures, especially the auditory system. These high-pressure gases expand outward from the point of explosion, compressing the surrounding air. The forces associated with these factors can cause severe injuries to people in the vicinity of the blast (1). In contrast to conventional sound, which is typically quantified in decibels sound pressure (dB SPL), blast waves are frequently measured in units of pressure, such as Pascals (Pa) or pounds per square inch (psi). These units can reach levels as high as 250 dB SPL. It is estimated that a blast wave with a pressure of 5 psi can severely damage the eardrum and delicate structures of the inner ear. Pressures of about 15 psi can have a devastating effect on the lungs, and pressures above 30–40 psi can lead to rapid death (2).

The loudness and distance of the sound waves from a blast determine the type of hearing damage. Sound waves exceeding 120 to 130 dB SPL, typical of combat explosions, surpass the human ear's safety threshold. They can cause serious damage. This includes rupturing the tympanic membrane, damaging the outer hair cells (OHCs), and impairing the function of the auditory nerve (3,4). The intensity and amplitude of sound pressure determine the severity of the impairment. Sound waves above 120 dB SPL exceed the normal tolerance threshold. They can cause devastating damage. This includes perforation of the eardrum and damage to inner hair cells (IHCs). These problems often lead to sensorineural hearing loss and tinnitus. These conditions are often persistent and irreversible. The blast wave not only produces a very loud sound but also exerts high physical pressure on the ear. This damages the outer, middle, and inner ear simultaneously (5). The auditory system is particularly vulnerable to the initial blast wave. This damage is not confined to the peripheral auditory system. It also extends throughout the central auditory pathways. This results in a complex set of functional deficits (6,7). Blast pressures also damage the vestibular system, causing balance disorders. The severity of injury decreases sharply with increasing distance from the explosion site. Specifically, doubling the distance from the explosion leads to a fourfold decrease in sound intensity (8). Although blast waves share similarities with impact noises, they represent a distinct class of acute hearing disorders due to their unique pathological mechanisms. Given their much higher intensity, the specific physical effects of blast waves must be examined independently from conventional noise. These distinctions have significant clinical importance for determining protective, diagnostic, and therapeutic strategies. Therefore, the primary goal of this review is to synthesize current data on blast-induced hearing damage. We specifically differentiate blast pathology from conventional noise exposure and examine the impact of repeated blasts. Additionally, we evaluate current strategies for diagnosis, prevention, and rehabilitation to highlight the need of tailored management strategies. This review aim to enhance clinical care standards and advance research in managing acute blast-induced hearing damage.

Materials and Methods

Search Strategy

We conducted a comprehensive literature search to identify studies examining blast-induced auditory injury. The search was performed in January 2025 across four electronic databases: PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar. The search period covered publications from January 2000 through January 2025. This 25-year timeframe was selected to capture contemporary research. This period follows increased recognition of blast-related injuries in modern military conflicts and civilian incidents involving improvised explosive devices. We employed combinations of keywords and subject headings relevant to blast injury and auditory pathology. Core search terms included: "blast wave," "blast injury," "blast trauma," "blast-induced hearing loss." These were combined with "hearing loss," "auditory damage," "sensorineural hearing loss," "cochlear injury," "cochlear synaptopathy," "hidden hearing loss," "central auditory processing disorder," "CAPD,"

"auditory neurodegeneration," "tinnitus," and "vestibular dysfunction." Boolean operators (AND, OR) were used to combine terms. For example, a representative PubMed search string was: ("blast wave" OR "blast injury" OR "blast trauma") AND ("hearing loss" OR "auditory damage" OR "cochlear injury" OR "synaptopathy"). Additional studies were identified through manual screening of reference lists from key articles.

Study Selection Criteria

Studies were considered for inclusion if they met the following criteria. (1) They were published in English in peer-reviewed journals. (2) They investigated physiological, pathological, cellular, molecular, or functional effects of blast exposure on auditory or vestibular structures. (3) They utilized human subjects, animal models, or computational simulations. (4) They reported quantitative or qualitative outcome measures related to auditory function or structure. (5) They provided sufficient methodological detail to assess study quality. Exclusion criteria were: (1) non-English language publications; (2) review articles, systematic reviews, meta-analyses, and narrative reviews (although their reference lists were screened for additional primary studies); (3) studies focused solely on traumatic brain injury without specific auditory or vestibular outcome data; (4) non-peer-reviewed materials including editorials, commentaries, letters, and conference abstracts; (5) duplicate reports of the same patient population or experimental data; (6) studies with insufficient methodological detail; and (7) publications outside the specified date range.

Screening Process and Study Flow

The literature search yielded 624 records from electronic databases. An additional 15 records identified through manual screening of reference lists. This totaled 639 records. After removing 177 duplicates, 462 unique records remained for screening. Title and abstract screening excluded 402 records for the following reasons: no auditory outcome data (n=180), review articles or meta-analyses (n=95), non-English language (n=65), and conference abstracts or editorials (n=62). The remaining 60 records underwent full-text evaluation. Of these, 24 were excluded due to: duplicate reporting of patient populations or experimental data (n=12), insufficient methodological detail to assess quality (n=8), and failure to meet peer-review standards (n=4). This systematic process resulted in 36 original research studies meeting all inclusion criteria. The complete study selection process is illustrated in Figure 1 (PRISMA flow diagram).

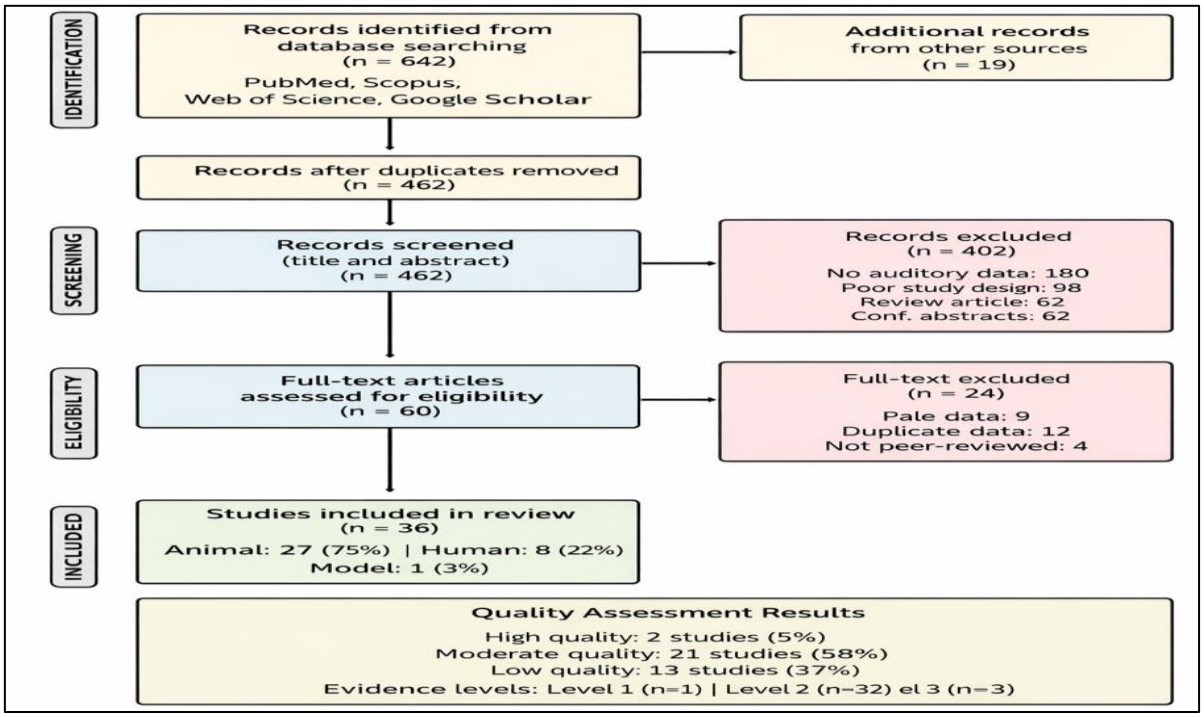

Fig 1. PRISMA diagram for systematic literature search and study selection process

Study Characteristics and Quality Appraisal

Study quality was systematically evaluated using criteria adapted from established methodological quality assessment frameworks (9,10). These criteria assessed: (1) clarity and reproducibility of methodology; (2) appropriateness of study design and controls; (3) adequacy of sample size; (4) validity of outcome measures; (5) appropriateness of statistical analysis; and (6) acknowledgment of limitations. Studies meeting ≥5 criteria were classified as "high quality." Those meeting 3-4 criteria were classified as"moderate quality." Risk of bias was evaluated according to study design, following principles adapted from established frameworks. For animal experimental studies, we applied key domains from SYRCLE's risk of bias tool. We examined randomization, blinding, outcome completeness, selective reporting, and baseline characteristics (11). For human observational studies, we evaluated cohort selection, exposure ascertainment, outcome assessment, and follow-up adequacy. Complete quality and bias assessment results, including evidence levels per Oxford Centre for Evidence-Based Medicine (OCEBM) criteria (12), are presented in Table 1.

Table 1. Characteristics and Quality of Included Studies

Study Design	N (%)	Evidence Level	Quality Rating	Main Contributions
Animal experimental studies	27 (75%)	Level 2	High: 24 Moderate: 3	Cochlear and central damage echanisms; molecular pathways; temporal progression
Human clinical/observational studies	8 (22%)	Level 2-3	High: 7 Moderate: 1	Functional deficits; CAPD; vestibular dysfunction; clinical outcomes
Computational modeling	1 (3%)	Level 3	High: 1	Blast wave transmission mechanics
Total	36 (100%)	-	High: 32 (89%) Moderate: 4 (11%)	-

Evidence levels based on Oxford Centre for Evidence-Based Medicine (Level 1: RCT; Level 2: cohort studies and well-designed animal experiments; Level 3: case series and modeling). Quality ratings based on methodological rigor, clarity of methods, appropriate controls, adequate sample size, valid outcome measures, appropriate statistical analysis, and acknowledgment of limitations.

Results

Pathophysiology of Hearing Damage Caused by Blast Waves

Blast-induced hearing loss involves complex molecular and cellular cascades triggered by mechanical force. In the initial phase, sudden mechanical damage to hair cells in the cochlea and synaptic connections impairs neural signaling. This initial damage also leads to disruption of cell membranes and ion channels. Subsequently, the release of excess glutamate causes neuronal excitotoxicity (13).

As a result, cellular signaling pathways such as caspase activation induce apoptosis and cell necrosis. This process leads to neuronal inflammation and destruction of neurons in the brainstem and auditory cortex. This causes persistent impairments in auditory information processing (1).

Peripheral Auditory System

The peripheral auditory system comprises the structures of the outer, middle, and inner ear, responsible for converting sound waves into neural signals. When exposed to a blast wave, the initial damaging effects occur in the outer and middle ear. The blast wave typically ruptures the tympanic membrane, which is a common initial injury. However, studies indicate that serious damage can be transmitted to inner ear structures even without tympanic perforation. This significantly impairs hearing function (14).

Sensorineural hearing loss originates in the inner ear, particularly the cochlea. Intense blast

pressure causes mechanical damage to the stereocilia bundles on outer hair cells (OHCs). As OHCs are vital for amplifying and separating sound, their damage compromises hearing sensitivity and clarity (15). Stereocilia damage initiates OHC dysfunction. At high sound pressure levels, this progresses to apoptosis or necrosis, ultimately causing OHC death (16). OHCs are notably more vulnerable than inner hair cell (IHCs), which often remain relatively intact. This injury pattern mirrors age-related changes, noise-induced damage, and ototoxicity. For example, a mouse study reported a reduction of less than 4% in IHCs compared to a 28–53% decrease in OHCs at 21 days post-blast (2). This differential vulnerability is attributed to differences in energy processing and reactive oxygen species production. Tonotopic variations in OHC calcium homeostasis may also play a role (17,18).

Pathologically, stereocilia destruction reduces the cochlea's nonlinear amplification capacity. This results in diminished frequency sensitivity and dynamic range. Such processes lead to sensorineural damage that is typically irreversible, with limited treatments available to restore cell function. Consequently, blast exposure involves both direct biomechanical damage to the middle ear and internal damage to cochlear hair cells. Current research on blast-induced hearing loss prioritizes the protection and regeneration of OHCs (17).

Beyond direct hair cell damage, a complex condition known as cochlear synaptopathy has emerged as a key indicator of blast injury (19). Cochlear synaptopathy refers to the permanent loss or dysfunction of ribbon synapses between IHCs and auditory nerve fibers, which preferentially affects low-spontaneous-rate, high-threshold nerve fibers while outer hair cells remain intact (20,21). This synaptic damage results in "hidden hearing loss". This is auditory dysfunction characterized by normal pure-tone audiometric thresholds. However, it involves suprathreshold deficits in processing. Patients particularly have difficulty understanding speech in noise. Behavioral detection thresholds remain unchanged until neural loss exceeds 80-90% (22). This condition involves the loss of synaptic ribbons connecting IHCs to auditory nerve fibers. Synaptic ribbons are specialized presynaptic structures within IHCs. They store and release neurotransmitters to ensure rapid, continuous, and accurate signal transmission to spiral ganglion neurons. Damage to these ribbons reduces the number of active synaptic connections, resulting in "hidden hearing loss" (15). In cochlear synaptopathy, OHCs typically remain intact, yet their neural connections to the brain are disrupted. Consequently, electrical signals are not properly generated or are insufficiently conveyed through nerve fibers. This functional decline disrupts the transmission of auditory information to the brain's processing centers (23).

Cochlear synaptopathy is considered a primary factor in auditory deficits, particularly difficulty understanding speech in noisy environments (24). While the dominant frequency of the blast spectrum determines the specific area of cochlear damage, synaptopathy is the most common pathology observed. It occurs regardless of the blast's frequency range (23).

Central Auditory System

The impact of blast waves is not limited to the peripheral system. Biomechanical forces can be transmitted directly through the external auditory canal or via cranial bone conduction to the central nervous system (CNS). This transmission causes significant damage to the central auditory system, even in the absence of peripheral injury (25). Notably, even when ear protection blocks the pressure wave from entering the ear canal, significant neurodegeneration has been observed in the brainstem. Studies have shown that following exposure to single or repeated blasts, both acute and chronic neurodegeneration occur in the cochlear nucleus of the brainstem (7). This central damage manifests as a decrease in the number of synapses and loss of myelin, which is directly related to the development of tinnitus. These findings suggest that blast energy is transmitted to the CNS via alternative pathways, such as bone conduction. This damage can persist for up to a year after exposure (26). Blast exposure also disrupts thalamocortical circuits and the auditory cortex in the higher parts of the auditory pathway. Evidence suggests that in the acute phase of injury, the long-term functional connectivity between the medial geniculate nucleus of the thalamus and the auditory cortex is disrupted. This disruption is accompanied by reduced synaptic transmission and changes in dendritic spines in the cortex. Such alterations

may cause acute and subacute hearing impairments (27). Furthermore, blast exposure can disrupt the precise tonotopic organization of the primary auditory cortex, contributing to central auditory processing disorders (CAPD) (6). CAPD refers to deficits in the neural processing of auditory information within the central auditory nervous system. These deficits extend from the cochlear nucleus to the auditory cortex. They are not attributable to peripheral hearing loss, cognitive impairment, or language dysfunction. CAPD is characterized by impaired sound localization, auditory discrimination, pattern recognition, temporal processing, and auditory performance in competing or degraded acoustic conditions (28,29). In blast injury, CAPD arises from direct biomechanical damage to the brainstem and cortical auditory structures, secondary neurodegeneration, or combined mechanisms. While auditory frequency maps in control animals are regular and tonotopic, representing the entire frequency spectrum (2–32 kHz), blast-exposed animals show significant irregularities. For example, in animals exposed to a 22 psi blast, these maps become discontinuous, with some narrower frequency ranges being abnormally prominent (6). These structural changes may explain certain central auditory processing deficits, including the inability to comprehend speech in noisy environments and reduced pitch discrimination (30). The exact mechanisms of these changes are currently unknown. Nonetheless, animal studies have reported deterioration of neural pathways in the auditory cortex. Additionally, there is an imbalance of inhibitory and excitatory neurotransmitters, specifically a reduction in GABA and NMDA receptors, which may lead to functional impairments (3,31). Studies indicate that blast injuries can cause bilateral damage to the auditory cortex even when only one side of the head is exposed. These chemical changes occur bilaterally, even when the contralateral ear is protected and surrounding structures remain intact. This suggests that some blast-related damage may be due to mechanical forces directly applied to the brain. However, determining the extent to which traumatic brain injury contributes to these bilateral lesions is difficult. This is due to the numerous bilateral connections in the central auditory pathway (31) . Thus, blast-wave damage involves not only the peripheral system but also complex central structures responsible for precise auditory processing.

Distinguishing Blast Trauma from Conventional Acoustic Trauma

Although blast waves and conventional loud noises both inflict acoustic trauma, their mechanisms differ fundamentally. Blast exposure constitutes a severe form of acoustic insult. Significantly, the presence of the supersonic shock wave creates a unique biomechanical force that causes more severe and widespread damage (5). Comparative studies have shown that blast exposure causes more severe and persistent central auditory processing deficits than non-blast impact noise (32). For instance, animals exposed to blast waves exhibit persistent increases in auditory brainstem response (ABR) thresholds. In contrast, animals exposed to impact noise typically experience only transient changes (25) . This suggests that the shock wave component of the blast is the primary cause of permanent sensorineural damage. It is also a critical factor in the development of tinnitus (33). Notably, damage from blast noise alone may be largely transient. However, combined exposure to blast and noise can cause pathological changes in all stages of the ascending auditory pathway. These changes persist for weeks (32).

Cumulative Effects: Single vs. Repeated Blast Exposures

The frequency of blast exposures plays a crucial role in determining the overall damage to the auditory system and the prognosis for recovery. While a single high-intensity blast causes considerable damage, repeated exposures, even at lower intensities, result in cumulative and often progressive damage (34, 35). As shown in relevant animal studies, three exposures to blast result in more severe and permanent hearing loss compared to a single blast exposure (35). Furthermore, behavioral performance in mice deteriorates after repeated exposures in various neurobehavioral tests. These impairments often worsen months after the last exposure, indicating a progressive neurodegenerative process.Repeated exposures also increase cellular aging markers in the auditory cortex and other brain regions compared to single exposures. This increase may contribute to long-term cognitive and auditory

impairments (34). One study found no significant difference in the degree of degeneration of the cochlear nucleus between single and repeated exposures, highlighting the profound effect of even a single blast event. However, the overall evidence supports a cumulative effect of multiple exposures (7). Importantly, repeated low-level blast exposures can disrupt neurovascular structures and exacerbate neuroinflammation. This process increases the brain's vulnerability to subsequent exposures and chronic neurodegenerative diseases (36).

Functional Deficits and Long-Term Consequences

The substantial physiological damage caused by blast waves leads to a range of severe, often chronic functional impairments. The most commonly reported problems are sensorineural hearing loss, persistent tinnitus, and difficulty understanding speech in noisy environments (15). Deficits in speech perception in noise are a hallmark of blast-induced central auditory processing disorder (CAPD). This disorder can manifest even in individuals with normal or near-normal hearing thresholds. The underlying mechanisms likely involve cochlear synaptopathy and disrupted central auditory pathways (16,28).

Furthermore, neurodegeneration in the brainstem is linked to tinnitus. It may play a more critical role in its etiology than inner ear damage (23). The consequences often extend beyond auditory symptoms. Due to the proximity of the auditory and vestibular systems, blast waves frequently cause vestibular damage, resulting in chronic dizziness and balance deficits (35). Vestibular dysfunction emerges as one of the most debilitating complications in blast-exposed individuals, manifesting as persistent vertigo, postural instability, oscillopsia, and impaired spatial orientation. Studies indicate that up to 40% of individuals with blast-related mild traumatic brain injury experience persistent vestibular symptoms. These can last months to years following the initial exposure. The insidious nature of these symptoms complicates diagnosis and management. They often overlap with post-traumatic stress disorder and cognitive deficits. Therefore, comprehensive vestibular assessment is needed in this population (37). Clinical evaluation of vestibular function in blast-injured patients requires systematic testing beyond standard audiometry. Videonystagmography, vestibular evoked myogenic potentials, and computerized dynamic posturography have proven valuable. They detect subtle vestibular impairments that may not be apparent through bedside examination alone. These objective tests can reveal abnormalities in semicircular canal function, otolith organ integrity, and vestibulospinal reflexes. This is true even when patients maintain normal audiometric thresholds. Early identification of vestibular pathology is crucial. Timely vestibular rehabilitation therapy can substantially improve functional outcomes. It can also reduce fall risk (37). However, the multifactorial nature of dizziness in blast injury necessitates an integrated approach. Dizziness arises from peripheral vestibular damage, central vestibular pathway disruption, and concurrent traumatic brain injury. Therefore, management must address both peripheral and central components. Blast exposure can also induce endolymphatic hydrops (EH), a condition characterized by excessive endolymphatic fluid accumulation in the inner ear. Associated symptoms include variable hearing loss, tinnitus, vertigo attacks, and aural fullness (38). Optical coherence tomography examinations of mice exposed to 196 dB SPL blasts revealed transient swelling of the scala media consistent with EH. Notably, this swelling returned to normal within one da (39). In other studies, exposure to 100 dB SPL noise for two hours was identified as the threshold for EH in rats. This indicates that both explosions and acoustic trauma can effectively induce EH in animal models (40). The relationship between blast exposure, mild traumatic brain injury, and long-term cognitive disorders is well documented. Blast-exposed individuals often exhibit deficits in memory, attention, and executive function. These deficits arise from damage to specific brain regions, such as the hippocampus and cortex (34,41). There is also a significant association with psychiatric disorders, including post-traumatic stress disorder and depression. Repeated blast exposure is now recognized as a significant risk factor for developing chronic traumatic encephalopathy in later life (42). Figure 2 illustrates this complex pathway of injury, mapping the progression from structural damage to clinical symptoms.

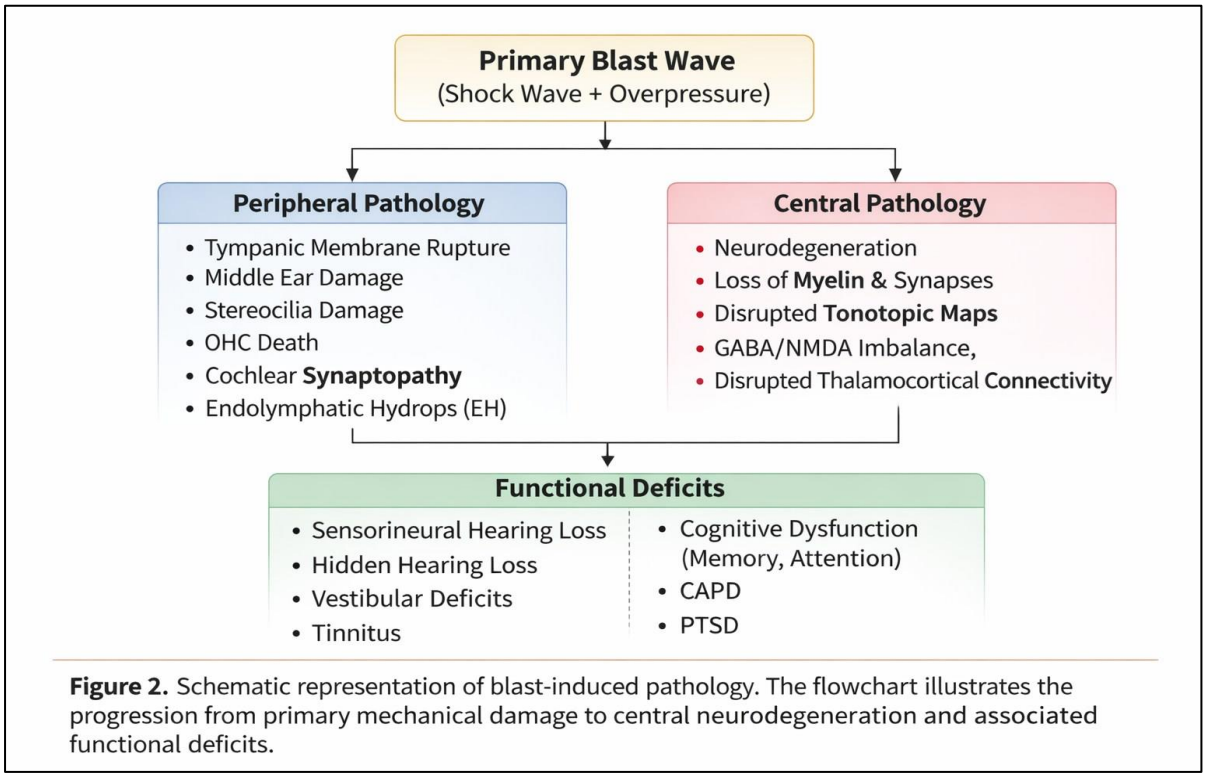

Fig 2. Schematic representation of blast-induced pathology.

The flowchart illustrates the progression from primary mechanical damage to central neurodegeneration and associated functional deficits.

Finally, Table 2 provides a consolidated overview of the key studies examined in this review, detailing their experimental models and clinical implications.

Table 2. Summary of studies on blast-related auditory damage and management.

Study	Subject	Exposure	Tests / Methods Used	Findings
Cho et al. 2013	Mouse	Blast wave (14 psi)	ABR, Histology (Hair cell count)	Higher vulnerability of OHCs compared to IHCs. ABR Wave I reduction indicating synaptic damage.
Hickman et al. 2018	Chinchilla	Blast overpressure	ABR, Synaptic ribbon count	Synaptic ribbon loss (cochlear synaptopathy) identified as primary cause of hidden hearing loss.
Grant et al. 2024	Human (Veterans)	Combat blast	Pure Tone Audiometry, Speech-in-Noise	Evidence of functional hearing deficits/CAPD in veterans despite normal audiometric thresholds.
Mao et al. 2021	Mouse	Single vs. Triple blast	ABR, DPOAE, Vestibular test	Cumulative effect observed; triple exposures caused severe vestibular/auditory damage vs. single blast.
Kim et al. 2018	Mouse	Blast wave	OCT Imaging, ABR	Prevention of secondary synaptopathy and endolymphatic hydrops achievable via osmotic stabilization.
Jiang et al. 2021	Chinchilla	Blast + Helmet	ABR, Biomechanical Modeling	Helmets reduced peripheral injury but failed to block bone-conducted transmission to the brain.
Masri et al. 2023	Rat	Blast wave	Electrophysiology, Immunohistochemistry	Correlation found between cortical parvalbumin neuron loss and auditory processing deficits.
Saunders et al. 2018	Human (Veterans)	Rehabilitation	Hearing aid fitting, FM system trial	Significant improvement in speech understanding in noise using FM systems.

Discussion

Diagnostics, Prevention, and Rehabilitation

Early and accurate diagnosis of blast-related hearing injuries is crucial yet challenging. Standard pure-tone audiometry often fails to reveal the full extent of the pathology. Therefore, employing a complete set of non-invasive electrophysiological tests is essential. This comprehensive diagnostic battery, utilizing various electrophysiological and behavioral tools, improves the identification of blast-induced auditory deficits.

Otoacoustic Emissions (OAEs): OAEs are effective for assessing OHC function and detecting peripheral cochlear damage after blast exposure (43). Research has demonstrated that longer blast wave exposure results in greater damage. It also leads to a lower likelihood of recovery (35). While noise exposure primarily leads to elevated high-frequency thresholds by affecting OHCs, blast exposure causes broader damage. It can disrupt the integrity of both OHCs and IHCs. It can even damage IHC synaptic connections. This potentially reduces the overall number of auditory nerve fibers (32 ,16).

Auditory brainstem response (ABRs): The ABR is a key tool for assessing the health of the auditory nerve and brainstem pathways. A reduction in the amplitude of Wave I is considered an important marker of cochlear synaptopathy (16). Furthermore, ABRs reflect functional deficits in the central auditory nerve and brainstem (32). Changes in ABRs over time are indicative of secondary biochemical effects in the central auditory system (44).

Auditory Event-Related Potentials (ERPs): Higher-level tests such as the P3 ERP can measure the speed and differentiation of neural processing. This indicates cortical processing deficits (45).

Behavioral Tests: For central auditory processing damage, specialized behavioral tests are necessary. Tools such as the Gaps-in-Noise test are considered appropriate for these subjects. This test is sensitive to temporal processing deficits (45). This comprehensive diagnostic toolkit improves the identification and understanding of complex lesions caused by blast-induced trauma.

Prevention and Mitigation

Given the severity of blast-induced injuries, effective prevention strategies are critical:

Personal Protective Equipment: Devices such as earplugs, noise-canceling headsets, and helmets are the primary preventive measures. In animal studies, combining earplugs and helmets has been shown to reduce the severity of blast-induced hearing damage by approximately 20 dB SPL (46). However, these devices cannot completely block blast wave transmission through bone conduction. Consequently, damage to the central auditory system may still occur despite protection (47).

Pharmacological Interventions: Drug research is a promising area for mitigation. Antioxidant therapies, including N-acetylcysteine and HPN-07, have demonstrated efficacy in animal models. By reducing oxidative stress, these compounds limit cochlear damage. They also prevent permanent hearing loss (2,16). Additionally, emerging drugs have shown significant potential in protecting hair cells and auditory neurons. These include the multifunctional redox regulator HK-2 and the GLP-1 receptor agonist liraglutide (48,49).

Rehabilitation Strategies

For people with blast-induced hearing impairment, a multidisciplinary rehabilitation strategy is necessary. This improves auditory processing and quality of life.

Assistive Technology: Some individuals have difficulty understanding speech in noisy environments despite normal audiograms. For these patients, frequency modulation (FM) systems and remote microphone technology are very effective. Findings demonstrate that FM systems significantly improve speech understanding in noise and help alleviate cognitive problems among blast-exposed veterans (50). Additionally, environmental modifications are crucial. Strategies such as acoustic insulation and reducing reverberation in living and workspaces are prioritized. This improves the signal-to-noise ratio. It facilitates better speech understanding for patients struggling with central auditory processing deficits (30, 50, 51).

Therapeutic and Medical Management: In cases of acute sensorineural hearing loss after a blast, prompt administration of corticosteroids remains the mainstay of treatment. These can be given either orally or by intratympanic injection (51). Given the high prevalence of concomitant

balance and cognitive problems, a comprehensive rehabilitation program should be developed by a multidisciplinary team. This approach must address balance issues, tinnitus, and any associated psychological or cognitive disorders (52). Table 3 systematically compares key findings from animal experimental studies and human clinical investigations, highlighting both convergent evidence and translational considerations.

Table 3. Comparison of Key Findings: Animal Models vs Human Clinical Studies.

Pathological Feature	Animal Model Evidence (n=27)	Human Clinical Evidence (n=8)
Peripheral Damage (Cochlea & Hair Cells)	Direct stereocilia damage, OHC loss, stereocilia bundle disruption. Dose-dependent injury with blast intensity. Recovery limited even with low-intensity exposures.	Sensorineural hearing loss documented in 40-60% of blast-exposed veterans. Permanent threshold shifts at high frequencies most common.
Cochlear Synaptopathy	Ribbon synapse loss between IHCs and auditory nerve fibers. Preferential loss of low-SR, high-threshold fibers. Occurs even with normal audiograms.	Suspected based on speech-in-noise difficulties and normal audiometry. Direct histological confirmation not available in humans.
Central Auditory Pathology	Neurodegeneration in cochlear nucleus, inferior colliculus, auditory cortex. Loss of myelin and synapses. Disrupted tonotopic maps. GABA/NMDA imbalance.	CAPD documented in 20-30% of blast-exposed individuals. ABR and ERP abnormalities suggest central involvement. Functional deficits exceed peripheral damage.
Vestibular Dysfunction	Direct labyrinthine damage. Disrupted otolith function. Balance deficits correlate with blast intensity.	Persistent vertigo and imbalance in 25-40% of cases. Abnormal VNG and VEMP findings. Often comorbid with auditory deficits.
Temporal Progression	Acute damage (0-24h): mechanical trauma, cell death. Subacute (days-weeks): neuroinflammation, secondary degeneration. Chronic (months): persistent functional deficits.	Limited longitudinal data. Some recovery in first 3-6 months. Many symptoms persist beyond 1 year. Repeated exposures worsen outcomes.
Hidden Hearing Loss	Documented extensively. Normal thresholds but reduced amplitude of ABR wave I. Impaired temporal processing. Speech-in-noise deficits.	Suspected in many cases with normal audiograms but subjective hearing difficulties. Difficult to diagnose with standard clinical tools.
Translational Considerations	Controlled blast parameters. Isolated auditory injury. Species differences in anatomy and physiology. Limited behavioral assessment.	Complex polytrauma. Psychological comorbidities (PTSD). Individual variability. Real-world exposure conditions.

This table synthesizes key findings from 27 animal experimental studies and 8 human clinical studies included in this review. OHC, outer hair cell; IHC, inner hair cell; SR, spontaneous rate; CAPD, central auditory processing disorder; ABR, auditory brainstem response; ERP, event-related potential; VNG, videonystagmography; VEMP, vestibular evoked myogenic potential; PTSD, post-traumatic stress disorder.

Conclusion

The evidence in this review is derived predominantly from animal experimental studies (75%) and human clinical investigations (22%). While animal models have provided mechanistic insights into synaptopathy and neurodegeneration pathways, important limitations must be acknowledged. Animal studies use controlled blast parameters. In contrast, human injuries occur in complex environments involving polytrauma and psychological comorbidities. Species differences in cochlear anatomy and auditory pathway organization further limit direct extrapolation. Nonetheless, convergent findings from both animal and human studies support core mechanisms. These include ribbon synapse loss and central processing deficits. Clinical recommendations presented here are based primarily on available human evidence. Animal data providing a mechanistic context. Evidence suggests that blast waves cause damage far beyond the peripheral

hearing system. This damage extends from the middle ear's mechanical structures to the complex neural circuits of the auditory cortex. Studies demonstrate that the resulting pathology extends beyond peripheral hearing loss to include significant central nervous system damage. Key features include cochlear synaptopathy, brainstem neurodegeneration, and cortical synaptic dysfunction. Collectively, these injuries lead to debilitating deficits. These include tinnitus and impaired speech processing in noise (26,27). Moreover, the cumulative effect of this damage is concerning. Repeated exposure, even at sub-threshold intensities, can cause progressive functional decline. It also leads to poorer recovery outcomes (34,35). Standard pure-tone audiometry is insufficient to assess the full severity of blast-related injuries. It often fails to detect hidden hearing loss caused by synaptopathy and CAPD. Consequently, a comprehensive diagnostic suite is required. This should include objective, noninvasive tools such as ABRs and ERPs. These accurately assess the integrity of the central auditory pathway. Additionally, given the high comorbidity of hearing impairment with vestibular, cognitive, and psychiatric deficits.

Therefore, multidisciplinary rehabilitation strategies addressing the individual's holistic health are essential. Despite existing research, significant gaps remain in the scientific literature. While animal models are valuable, longitudinal, prospective, and large-scale studies in human populations are recommended. These are necessary to examine damage progression over time. They also help identify risk factors for long-term functional decline. Also, the precise mechanisms of blast shock wave transmission to central nervous structures remain an active area of investigation. Finally, the optimal therapeutic window for pharmacological interventions post-blast is not yet clearly defined.

Future studies should focus on several key areas. Clarifying these complex mechanisms is essential for developing better protection strategies. It also improves the quality of life for at-risk individuals. Priority must be given to developing sensitive diagnostic tools for the early detection of central hearing damage. Research into the efficacy of otoprotective drugs is also critical. These include antioxidants and novel redox modulators, is also critical. Furthermore, validating targeted auditory rehabilitation paradigms specifically designed for blast-induced CAPD is essential. Beyond conventional amplifiers, these strategies should also include remote microphone systems and structured auditory training. Elucidating the complex pathophysiology of this damage will eventually enable better protection, diagnosis, and treatment. This will improve the long-term quality of life for at-risk individuals.

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List of Abbreviations

ABR – Auditory Brainstem Response
CAPD – Central Auditory Processing Disorder
dB SPL – Decibel Sound Pressure Level
EH – Endolymphatic Hydrops
ERPs – Event-Related Potentials
FM – Frequency Modulation
GABA – Gamma-Aminobutyric Acid
IHCs – Inner Hair Cells
NMDA – N-Methyl-D-Aspartate
OAEs – Otoacoustic Emissions
OHCs – Outer Hair Cells
Pa – Pascal
psi– Pounds per Square Inch

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