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Otologic Blast Injuries

Michelle Singer

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Otologic Blast Injuries

by

Michelle Singer

A capstone research project submitted to the Graduate Faculty in Audiology in partial fulfillment of the requirements for the degree of Doctor of Audiology, The City University of New York, 2018

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This manuscript has been read and accepted for the
Graduate Faculty in Audiology in satisfaction of the capstone
project requirement for the degree of Au.D.

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Date   John P. Preece, Ph.D. CCC-A
                                                Chair of Examining Committee

_____   _____

Date   John P. Preece, Ph.D. CCC-A
                                                Executive Officer

The City University of New York

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Abstract

The otologic system functions as a highly sensitive pressure transducer. Because of this, the ear is the most commonly affected part of the body in primary blast injury. Frequently encountered symptoms include hearing loss, tinnitus, and tympanic membrane perforations. The ear is repeatedly overlooked during triage and easily forgotten in subjects with multiple injuries after major catastrophic events such as explosions. This systematic review provides an overview of the most common otologic injuries observed after blast exposure in a variety of settings and populations. An analysis of 35 studies and an additional 23 reviews was performed in attempt to uncover patterns of otologic injury after blast exposure. Spontaneous recovery rates and late sequale were examined. The study of otologic blast injury is limited by the vast differences between each blast incident and the inability to predict how physical environments affect blast overpressures. There is an overall lack of data on the long term affects of blast injury on the otologic system. Recommendations for assessment and management of blast-injured patients, both short and long term, are made and future research is discussed.

Acknowledgments

First and foremost, I would like to thank my advisor, Dr. John Preece, for his expert advice, fruitful discussion and continuous encouragement throughout our four years together. Furthermore, I am incredibly grateful to my parents for their unconditional love and enthusiastic support of my academic ambitions. Lastly, I would like to thank my sweet husband, Tzachi, for keeping me grounded through stressful times. I would not be the person I am today without your constant inspiration, reassurance and understanding.

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Introduction

There has been an escalation of violence worldwide among both military and civilian populations (Cohen et al., 2002). With it comes a dramatic increase in the use of explosive devices, often with the intention of causing severe structural damage or the incidence of mass casualties. Explosive weapons can include improvised explosive devices (IEDs), landmines, mortars, bombs and grenades. During recent Operations Enduring Freedom (OEF; 2001-current) and Iraqi Freedom (OIF; 2003-current) there has been an increase in the use of IEDs compared to previous wars (Oleksiak, Smith, Andre, Caughlan, & Steiner, 2012). Blast injuries account for about 75% of combat related casualties (Dougherty et al., 2013). Of those injured, over 60% reported otologic injury, making the ear the most commonly injured part of the body after blast exposure (Cho et al., 2013). In the United States Armed Forces, hearing loss caused by blast exposure has become so common that The Department of Defense Hearing Conservation Program developed a Blast-Related Auditory Injury Database (BRAID). It is "composed of demographic, audiometric, point of injury, and medical outcome data" (Joseph et al., 2016, p. 295). The purpose of BRAID is "... to monitor, assess and investigate blast-related otologic outcomes." As of today, the rate of hearing loss for blast-injured service members is 39% (Joseph et al., 2016).

Historically, blast injuries were seen exclusively during wartime and occasionally in industrial accidents (Sprem, Branica, Dawidowsky, 2001). According to Teter, Newell and Aspinall (1970), the first recorded case of auditory injury from blast exposure was recorded in 1591. At that time, hearing loss was reported in cannoners following battle.

The first recorded incident of complete deafness, due to blast exposure was reported almost 300 years later in a multi-case study by John Orne Green in 1872. His report outlined 10 individual cases of subjects with blast exposure along with their initial subjective reports and otological findings (Green, 1872). Today, IEDs are now the most frequently utilized weapons in terrorist attacks. The frequency of these incidents directed at the civilian population continues to increase across the globe (Mrena, Paakkonen, Back, Pirvola, & Ylikoski, 2004). The study of the effects of blast exposure on the human body is an ongoing interest to medical and rehabilitative professionals, particularly those in otolaryngology and audiology.

Blast injuries can be separated into four categories according to mechanism of injury. Primary blast injuries are those caused directly as a result of the pressure wave. Secondary injuries are caused by projectile objects such as shrapnel or debris propelled by the blast. Tertiary injuries are caused when an individual's body is physically displaced by the blast winds including any subsequent injury caused by impact. Quaternary injuries are other injuries caused indirectly by the blast waves and subsequent blast winds. Quaternary injuries can include, but are not limited to, crush injuries, burns, inhalation of debris and exposure to toxic substances (Tun, Hogan, & Fitzharris, 2009). Table 1 displays how blast injuries are caused and categorized. This review will focus on primary blast injuries.

Table 1. Categorization of blast injuries by mechanism of injury and pattern

Category	Mechanism of Injury	Patterns of Injury on the Body
Primary	Blast pressure wave	Traumatic brain injury (TBI), lungs, tympanic membrane perforation, cochlear and vestibular disturbances
Secondary	Projectile objects	TBI, amputation, fractures, soft tissue injuries
Tertiary	Physical displacement by blast wind	TBI, amputation, fractures
Quaternary	Other	Burns, exposure to toxic substances, crush injuries, exacerbation of chronic illness

Note. Adapted from "Hearing and vestibular dysfunction caused by blast injuries and traumatic brain injuries" by C. Tun, A. Hogan, and K. Fitzharris, 2009, *The Hearing Journal, 62*, p. 24. Copyright 2009 from Wolters Kluwer Health.

Primary blast injuries predominantly occur in hollow organs such as the lungs, gastrointestinal tract and the middle ear cavity (Van Campen, Dennis, Hanlin, King, & Velderman, 1999). The effects of blasts are characterized by their ability to damage both the middle and inner ear. The damage is caused by sudden changes in pressure and volume (Nawaz, Ulhaq, & Khan, 2014; Ziv, Philipsohn, Leventon, & Man, 1973). The ear is the body's most sensitive pressure transducer and the most frequently injured part of the body after blast exposure (Aslier & Aslier, 2016). Exposure to blasts waves can cause instantaneous hearing loss, subsequent tinnitus, dizziness and damage to the middle ear.

Despite injury outcomes varying between individuals (Killion, Monroe, & Drambarean, 2011), there appears to be trends in otologic damage after exposure to

blasts. Additionally, there are subtle differences between the military and civilian populations. Military personnel who are prepared for combat tend to wear more protective gear such as ear plugs, helmets or be inside armored vehicles (Remenschneider et al., 2014). Civilians in the vicinity of bombing attacks tend to be less protected and have a higher potential to sustain severe injury.

Civilian attacks sites are often chosen to maximize casualties and cause chaos. This further delays immediate medical attention so desperately needed by those affected. The lack of appropriate personnel and equipment at local medical facilities exacerbates the problem (Okpala, 2011). Collectively, each barrier encountered increases the time period between insult and initial evaluation which may ultimately affect the prognosis and long term morbidity. Unfortunately, due to their invisible nature, otologic injuries are often overlooked (Chait & Zajtchuk, 1989; Garth, 1995; Okpala, 2011).

A significant proportion of blast injured individuals report persistent hearing loss and tinnitus. There is a need to assess the histology of audiologic symptoms in order to determine hearing-related morbidity. Current literature does not sufficiently provide information about the short and long term effects of blast exposure on hearing (Joseph et al., 2016). In this review, I will cover the most common otologic blast injuries detailing their incidence, prevalence, severity, recovery rates and late sequelae according to a compilation of data from over thirty studies from around the world. Recommendations for initial evaluation, short- and long-term medical management and frequently encountered issues will be discussed.

Additionally, there is a lack of established guidelines for clinical care of subject with blast exposure, especially within the civilian population (Remenschneider et al., 2014). There are a limited number of reviews of the literature detailing the effects of blast exposure on the human ear. Fewer authors have successfully compared findings from studies of otologic blast injuries and made recommendations on how to proceed with evaluation and management of these injuries.

Blasts

A bomb blast wave typically produces a three phase waveform with decreasing amplitude, or force, as it spreads outward from the initial site (Remenschneider et al., 2014). As explosive material abruptly transforms from a solid or liquid to a gaseous state, the first phase begins. A massive increase in volume is generated followed by a massive increase in pressure which produces an irregular shock wave (Nawaz et al., 2014). Subsequently, a positive pressure wave develops applying hundreds of thousands of pounds per square inch. This wave lasts for approximately 5-10 milliseconds. The final phase is a long negative pressure wave that lasts approximately 30 milliseconds before it converts into atmospheric pressure and dissipates into the surrounding environment (Aslier & Aslier, 2016). These rapid changes in pressure are capable of producing barotrauma, which characterizes primary blast injuries (Shah, Ayala, Capra, Fox, & Hoffer, 2014). These phases, in perfect form and unobstructed by objects in the environment, produce what is known as a Friedlander curve (Figure 1).

The middle ear has an active protective mechanism to protect against damaging intense sounds in those without obstructive middle ear or severe inner ear pathology. This mechanism is called the acoustic reflex and it is defined by the contraction of the stapedius muscles. Unfortunately, the acoustic reflex is not triggered quickly enough to protect the cochlea from blast injury to the middle ear. Once a loud noise is introduced into the ear canal, the acoustic reflex has a delayed onset of approximately 5 to 10 milliseconds while blast pressure waves can reach the ear, at times, in under 5 milliseconds (Bruins & Cawood, 1991, Hirsch, 1968).

Figure 1. Simple Friedlander curve depicting blast pressures in an ideal model
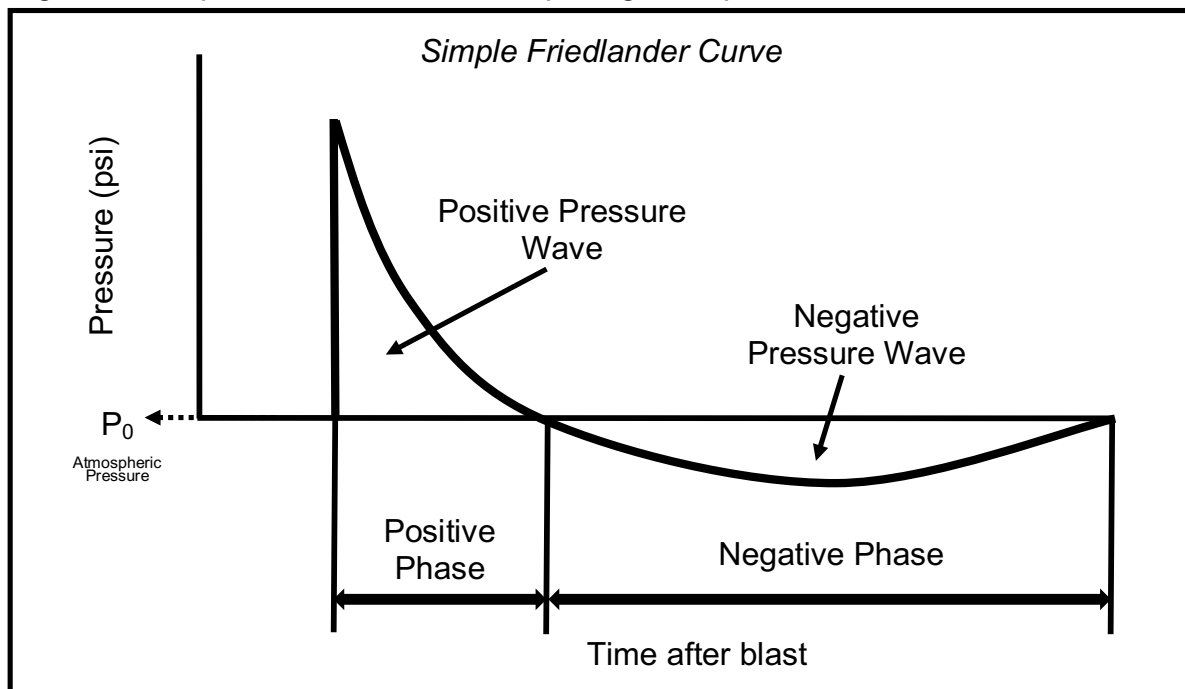
Note. Adapted from "Shock tubes and blast injury modeling" by Y. Ning and Y. Zhou, 2015, *Chinese Journal of Traumatology, 18,* p. 188. CC BY-NC-ND.

Realistically, it is unlikely that individuals in the vicinity of a blast will be exposed to blast pressure waves in the form of a simple Friedlander curve. Different environments have significant influences on the shape and intensity of each phase. For

example, in a confined space, the initial shock wave will be modified by multiple reflections from surrounding surfaces. The heating of gases within the space will give rise to a gradual increase in pressure (Patterson & Hamernik, 1997). Researchers agree that of all phases of a blast, the positive pressure wave is the most dangerous and is the direct cause of most blast injuries (Ziv et al, 1973).

The shape and severity of the blast wave more closely resembles a complex Friedlander curve depicted in Figure 2 and is dependent on multiple factors (Kerr & Byrne, 1975):

- Rise time, or speed in which the pressure wave builds
- Height of the peak pressure wave
- Duration of the positive wave
- Site of the blast

Many of these factors are influenced by the speed in which pressure can escape. Blasts occurring in confined spaces produce a higher peak pressure and a longer positive pressure wave. Moreover, reflected waves can strike the subject again. The transformation of blast waves caused by the confined space can generate a more powerful blast and thus, more severe injuries compared to blasts occurring in an open field (Nageris, Attias, & Shemesh, 2008; Okpala, 2011).

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Figure 2. An example of a complex Friedlander curve

Complex Friedlander Curve
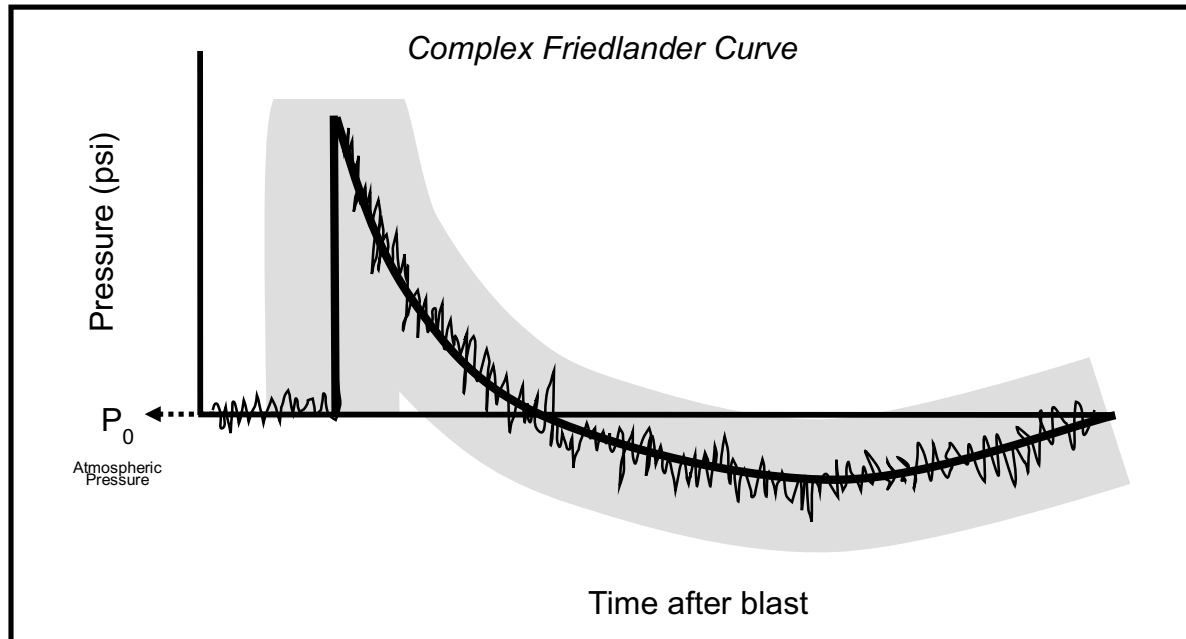
Note. The grey area represents potential variability in the Friedlander curve once influenced by environmental factors. Adapted from "Shock tubes and blast injury modeling" by Y. Ning and Y. Zhou, 2015, *Chinese journal of traumatology, 18,* p. 188. CC BY-NC-ND.

How the blast wave ultimately affects the ear is determined by:

- Device specifications including the type and amount of explosive material (Okpala, 2011)

- Distance the individual is from the blast (Remenschneider et al., 2014; Yetiser & Ustun, 1993)

- Positioning of the individual in relation to the blast (Patterson & Hamernik, 1997)

- The physical environment (Hirsch, 1968)

- Presence of obstructing objects and the proximity of surrounding structures (Walsh, Pracy, Huggon, & Gleeson, 1995)

Most would assume, that the larger the amount of explosive material, the larger the blast wave will be. Another reasonable assumption, is that the closer an individual is to the center of the blast, the more severe their injuries will be. However, Kerr and Byrne (1975) argue that distance does not always indicate severity of injury due to the inherent reflections of pressure waves. They detailed otologic findings of those inside the building when a 5-lb bomb was detonated in Abercorn restaurant in Belfast, Ireland (March, 1971). They found that some people close to the blast escaped without otologic injury while others further away had severe injuries including sensorineural hearing loss and tympanic membrane perforations. Therefore, due to the vast irregularity of the initial shock wave caused by objects in the environment, distance no longer becomes a key indicator of presence or severity of injury for those in the vicinity of blasts occurring in confined spaces.

Physical environment heavily determines the effects of the wave on nearby subjects. Walls, doors, windows, hallways, vehicles and furniture can all produce reflected waves. Van Campen et al. (1999) discussed the the connection between damage to buildings and the human ear. They stated that is it presumed that more structural damage means more severe otologic injury is to be expected. In their findings, they spectate that buildings may absorb some energy produced by blasts thus shielding subjects within its walls.

The positioning of the subject in relation to the blast can directly affect the injuries sustained (Hirsch, 1968). Darley and Kellman (2010) outlined the effect of the vector at which the blast wave hits the ear canal. They asserted that blasts occurring directly in

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front or behind the ear have less intensification of pressure waves within the ear canal and middle ear system than those striking the ears from the sides due to presence of the pinna.

Blasts are occurring more frequently and in more settings outside of war. The locations in which blasts occur have an enormous effect on the intensity and duration of pressure waves which, in turn, partially determine the incidence and severity of injury of subjects in the surrounding area. Blasts occurring indoors are theorized to be more destructive to the otologic system as reflections from the environment strike a second time. No one pressure wave is alike due to the subtitle differences in type of explosive used and the location in which it is detonated. These factors substantially influence the overall destruction and injury caused by the blast.

Methods

Literature searches utilizing EBSCO Complete, PubMed and Google Scholar (1950–December 2017) were performed. Preference was placed on more recent literature, published after 2000. Search terms included relevant terminology such as blast injury, blast, explosion, bombing, ear, otologic, audiologic, hearing loss, threshold shift, spontaneous recovery, tinnitus and tympanic membrane perforation. Articles with topics pertinent to primary blast injury of the ears were selected through a search of their titles or abstracts. After an article was selected and initially reviewed, a search of its references was performed to identify additional sources. Thirty-five studies and an

additional 23 review articles have been selected from more than 200 screened based on their historical, pathophysiological, and clinical significance.

Studies and review articles were then analyzed for findings related to otologic blast injuries. Relevant data was extracted and, when suitable, compared to related studies. Author recommendations for evaluation, management and follow up were collected and summarized in this review in order to create a comprehensive and straightforward inventory for the use of medical professionals. Findings may be of particular interest to those working in emergency medicine, otolaryngology and audiology.

Hearing Loss

Loss of hearing sensitivity, to some degree, is a common complaint after exposure to blasts. Hearing loss can be conductive, sensorineural or a combination of the two which is known as mixed hearing loss. Conductive hearing loss may occur when there is injury to the tympanic membrane or middle ear structures, including the ossicular chain or supportive muscles. Damage to any of these structures limits their ability to conduct sounds pressure into the cochlea. Tympanic membrane perforations are the most common cause of conductive hearing loss in those exposed to blasts (Cave, Cornish, & Chandler, 2007).

Sensorineural hearing loss occurs when there is damage to the cochlea. During a blast, intense pressure hits the tympanic membrane, is conducted through the

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ossicular chain into the cochlea through the oval window. The oval window is forced inward creating a hydraulic pressure on the perilymph in the scala vestibuli. The pressure then passes to the endolymph of the cochlear duct onto the basilar membrane exposing the Organ of Corti to trauma (Ziv et al., 1973). The basilar membrane, inner and outer hair cells, and associated cilia within the Organ of Corti are most susceptible to acoustic trauma. These intense blast waves can tear the inner and outer hair cells from their support cells which ruptures the reticular lamina. When the reticular lamina is ruptured, perilymph and endolymph mix creating a toxic environment causing death of the hair cells (Shah et al., 2014). While research is currently being conducted, there is no effective treatment that can regenerate hair cells in humans. In most cases with inner ear damage resulting in hearing loss, the damage is considered permanent. However, it is possible that sensorineural hearing loss is transient and recovers over a short period of time. This phenomenon is called a temporary threshold shift (TTS). TTS will be discussed in further detail later in this review.

In 1953, IV Congress of the International Audiological Society accepted a classification system defining blast trauma, as Jagade et al. (2008) described, as injuries:

"… associated with a single exposure to an explosion where stimulation duration is greater than 1.5 m./sec and where middle ear damage is not uncommon, while noise induced hearing loss occurs as a result of long term exposure to elevated sound intensities" (p. 324).

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Garth (1995) further discerned that peak overpressures from blasts are often tens of thousands of Pa while impulse noises are usually less than 2000 Pa and that blast waves involve movement of substantial volumes of combustible material and air whereas impulse noise often only contains low frequency mechanical clatter. It is crucial to be able to distinguish one from the other when analyzing post-blast injuries.

The percentage of subjects exposed to blasts and reported subjective hearing loss at their initial evaluation in this review can be found in Table 2. Data ranged from 24.3% to 100% of subjects reporting subjective hearing loss after blast exposure. The largest percentage was reported by Persaud, Hajioff, Wareing, and Chevretton (2003). The authors assessed otologic injuries incurred after the Soho Nail Bombing (April, 1999), in which an explosive device packed with long nails exploded inside of a public house in London. All 17 subjects reported subjective hearing loss at their initial evaluations which occurred within six days of the incident. Alternatively, Pahor (1981) had the lowest reported percentage of subjective hearing loss when examining a similar occurrence. In this study, he examined subjects who were in the vicinity of two separate explosions in crowded public houses in Birmingham (November, 1974). At initial evaluations, only 27 of the 111 subjects (24.3%) reported difficulty hearing.

More recently and with the largest sample size, Jagade et al. (2008) found upon initial evaluation that 48 of their 132 subjects (36.4%) who survived an explosion in Mumbai, India (August, 2003) reported subjective hearing loss. Despite initial medical interviews taking place within 24 hours of the incident, Jagade and colleagues (2008) found an incidence of subjective hearing loss toward the lower end of our range and

closer to that of Pahor (1981). All other authors reporting incidence of subjective hearing loss fell between the 24.3% and 100% with the average of the studies in this review being approximately 45%.

Table 2. Reports of subjective hearing loss at initial evaluation

Authors (Year)	Total No. of Subjects	Subjects Reporting Hearing Loss	
		No.	%
Aslier & Aslier (2014)	25	10	40
de Regloix et al. (2017)	41	25	61
Jagade et al. (2008)	132	48	36.4
Mrena et al. (2004)	29	16	55.2
Nawaz et al. (2014)	30	21	70
Pahor (1981)	111	27	24.3
Persaud et al. (2003)	17	17	100
Tungsinmunkong et al. (2007)	33	24	72.7

After the initial interview and reports of subjective hearing loss, many researchers performed subjective audiometric testing to determine if hearing loss was truly present. Table 3 shows the percentage of subjects exposed to blasts that were found to have abnormal audiograms, to any degree. Similar to subjective reports of hearing loss, there are mixed findings. The percentage of abnormal audiograms range from 18% on the lower end to 100%. Cohen et al. (2002) found that 100% of their 17 subjects who were on or near a bus where an IED was detonated by a suicide bomber (October, 1994) had abnormal audiometric findings. Their initial evaluations were conducted one to three days after the initial incident. On the other hand, Qureshi, Awan, Hassan, Aftab, and Ali

(2017), found only 18 of the 100 subjects (18%) evaluated after blast exposure over a

two-and-a-half-year period at the Aga Khan University Hospital in Karachi, Pakistan, to

have abnormal audiometric findings. Both of these studies had relatively small to

medium sample sizes. The largest sample of audiometric evaluations after blast

exposure in this review was reported by Cave et al. (2007) in reports of otologic findings

in American military personnel following return from deployment in the Middle East.

They found that of 258 soldiers with blast injuries, about 165 (64%) revealed abnormal

audiometric findings. The findings of Cave and colleagues (2007), came close to the

average calculated incidence of audiometric abnormalities of 60%. It should be noted

that asymmetrical hearing loss is twice as common in those with blast exposure than

those without (Joseph, Shaw, Clouser, MacGregor, & Galarneau, 2017).

Table 3. Hearing loss at initial audiological evaluation

Authors (Year)	Total No. of Subjects	Subjects With Hearing Loss*	
		No.	%
Breeze et al. (2011)	57	30	52.6
Cave et al. (2007)	258	165	64
Cohen et al. (2002)	17	17	100
de Regloix et al. (2017)	41	25	61
Jagade et al. (2008)	42	36	85.7
Mrena et al. (2004)	29	21	75.7
Qureshi et al. (2017)	100	18	18
Tun et al. (2009)	50	26	52
Tungsinmunkong et al. (2007)	33	27	81.8
Van Campen et al. (1999)	83	59	71.1
Walsh et al. (1995)	12	9	75

*any type and degree of hearing loss in one or both ears

The literature denotes that hearing loss rates were nearly double when exposure occurred indoors as opposed to outdoors (Ziv et al., 1973). Reportedly, not only were there more incidences of hearing loss for those with blast exposure occurring indoors, but these subjects also tended to have more severe hearing losses (Kerr & Byrne, 1975). Within this review, Breeze et al. (2011) disclosed the location of their subjects at the time of incident in British military personnel serving in Iraq and Afghanistan between 2006 and 2009. They distinguished between those inside a vehicle at the time of incident versus those who were out "in the open" (p. 15). Of 16 subjects who had abnormal audiograms, the majority, 12 (75%), were in a vehicle while only 3 (18.8%) were out "in the open." The location of 1 subject (6.3%) at the time of blast was unknown.

Van Campen et al. (1999) also reported the location of their subjects in relation to incidence of hearing loss in his sample of subjects exposed to the Oklahoma City Bombing (April, 1995). Of their total 83 subjects, 53 (63.9%) had some degree of hearing loss. In this study, Van Campen et al. (1999) defined abnormal as any threshold over 22.5 dB HL citing their "age-corrected thresholds were not in 5-dB increments… [therefore] ""normal"" threshold was defined as <22.5 dB HL" (p 233). Of those 53 subjects with abnormal audiograms at initial evaluation, 48 (90.6%) were either within the federal building where the bomb detonated, inside of a building within the vicinity or in a vehicle when the blast occurred. Researchers in this review examined the incidence of hearing loss in regards to location of their subjects. Their research supported the

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findings of Ziv et al. (1973) that explosions occurring in confined spaces produced an overall higher incidence of hearing loss compared to those occurring in open areas.

The classification of hearing loss, by type at initial evaluation, can be seen in Table 4. de Regloix et al. (2017) reviewed the cases of 41 subjects who were referred to one of two military hospitals in Paris for injuries secondary to blast exposure between 2002 and 2014. They found mixed hearing loss to be the most commonly revealed type of hearing loss in their sample. Of their 41 subjects, or 82 ears examined separately, over half (54.9%) had mixed hearing loss compared to the 26.5% with purely sensorineural and 8.8% with purely conductive losses. Cohen et al. (2002) also found mixed hearing loss to be predominant with 61.8% of their subjects having mixed type hearing loss at initial evaluation.

Table 4. Hearing loss at initial evaluation by type

Authors (Year)	Total No. of Ears Evaluated	Type of Hearing Loss (%)			
		SNHL	MHL	CHL	Normal
Cohen et al. (2002)	34	9 (26.5)	21 (61.8)	3 (8.8)	1 (2.9)
de Regloix et al. (2017)	82	24 (29.3)	45 (54.9)	12 (14.6)	1 (1.2)
Remenschneider et al. (2014)	158	29 (18.4)	20 (12.7)	34 (21.5)	75 (47.5)
Ziv et al. (1973)	154	44 (28.6)	11 (7.2)	8 (5.2)	91 (59.9)

Note. SNHL = sensorineural hearing loss, MHL = mixed hearing loss, CHL = conductive hearing loss

Alternatively, Ziv and colleagues (1973) found the highest rate of normal audiometric findings in this review. They evaluated subjects who were in one of two

incidences: a destroyer hit by missiles (October, 1967) or an accidental detonation of a truck loaded with explosives (January 1970). In their sample of 77 subjects, or 154 ears, almost 60% were determined to be within the normal limits of hearing. Remenschneider et al. (2014) also found a high incidence (47.5%) of normal hearing in their study of those subjected to the blasts of the Boston Marathon Bombings. It is plausible the vast variability between the incidents, including location, size of explosive and individual susceptibility, may account for differences in findings of the affected ears.

Configurations

Researchers have been exploring the concept of a characteristic audiometric configuration associated with blast-exposure. Noise-induced hearing loss, or hearing loss associated with repeated or prolonged exposure to hazardous noise, typically manifests as a decrease in hearing thresholds centered around 4000 Hz (Fausti, Wilmington, Gallun, Myers, & Henry, 2009, p. 799), also called a noise notch. One of the aims of this review of the literature is to answer the question: does blast exposure also produce its own identifiable pattern?

In this review, a multitude of audiometric configurations were identified in blast-exposed subjects after complete audiometric evaluation. The most common configurations include:

- Dip
- Trough shaped

- Sloping (high frequency) loss

- Rising (low frequency) loss

- Flat loss

Teter et al. (1970) defined a dip as "hearing loss by which air conduction is 15 dB greater at a given frequency than at the octave frequency on both sides of it" (p. 1123) (Figure 3a-c). In this review, dips in hearing sensitivity were reported at 1000 Hz, 4000 Hz and 6000 Hz. A dip can occur at one or more frequencies. However, if there are more than three consecutive frequencies with recovery of hearing sensitivity on both sides, the configuration is then called trough-shaped (Figure 3d). Sloping losses tend to start at or above 2000 Hz and become more severe as the octaves increase with no recovery in hearing (Figure 3e). Rising losses are typically seen as decreased hearing sensitivity at or below 1000 Hz rising to normal hearing above 1000 Hz (Figure 3f). Flat losses are a generally even loss of hearing sensitivity across all octaves tested (Figure 3g).

Teter et al. (1970) examined a total of 81 ears in an undisclosed number of subjects exposed to blasts. About 71% of ears presented with dip configurations. Of the total 81 ears, 29 (36%) had dips at 1000 Hz, 17 (20%) had dips at 4000 Hz and 12 (15%) had dips at 1000 Hz and 4000 Hz. Additionally, 23 (28%) of ears presented with sloping losses.

In practice, audiologists and researchers are encouraged to take a detailed audiological history of their subject. Occupational noise exposure and acoustic trauma

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can become confounding variables. Prior damage can potentially appear audiometrically, and interfere with the accurate effects of the blast. Because of this, Bruins and Cawood (1991) analyzed these two populations separately. Sixty-one percent of ears with abnormal audiograms had high frequency sensorineural hearing loss. Only 6 of 41 ears (14.6%) had low frequency losses; all of which were conductive in nature and resolved with the closure of present tympanic membrane perforations. The most astounding discovery was that, when examining subjects with previous occupational noise exposure or acoustic trauma, 6 of 7 ears (85.7%) of which had dips at 6000 Hz at initial evaluation had a recovery of hearing by 6 months after the initial incident. This finding denotes that the 6000 Hz dip may be directly associated with blast exposure and stresses the importance of routinely testing inter-octaves (3000 Hz and 6000 Hz).

Perez, Gatt and Cohen (2000) examined 143 subjects sent to the Department of Otolaryngology in a city hospital after blast exposure. Of the 200 abnormal audiograms that were obtained from this sample, 93 (46%) had a sloping loss and 25 (12%) had flat losses. Furthermore, 82 (41%) had dips at varying frequencies; most notably 38 (19%) were dips at 6000 Hz which corroborates Bruins and Cawood's (1991) findings of a potential blast-induced audiometric configuration. Perez et al. (2000) further pointed out that subjects with sloping losses tended to be significantly older than those with dips configurations. As significant as medical history, age should be taken into account when controlling for confounding variables. Presbycusis, or age-related hearing loss, may manifest on the audiogram in conjunction with damage incurred from blasts.

Many authors whose research has centered around blast-exposed subjects, both within the military and civilian populations, have analyzed the incidence of distinguishing audiometric patterns that appear as a result of blast exposure. It should be noted that while the mechanism of injury for noise-induced hearing loss and blast-induced hearing loss are similar, as detailed earlier, they are not entirely the same. It has been observed, through many decades of research, that blast-exposure does not always produce this stereotypical high frequency noise notch. This further supports the differences in mechanism of injury (Patterson & Hamernik, 1997; Ritenour et al., 2008). Instead, it produces a assortment of configurations that Perez et al. (2000) and colleagues say are determined by a variety of factors unique to each blast as well as each individual subject's anatomical variations and medical history. They state that the configuration of hearing loss is never random and can, in fact, give researchers more detailed insight as to how and where the damage occurred in the inner ear. For example, Perez et al. (2000) theorized that sloping configurations indicated that the inner ear was more affected by the pressure wave while the presence of a dip signified that the inner was more affected by the impulse noise created by the blast.

There is agreement among researchers in this review that exposure to blasts most frequently affects the higher frequency range (2000 Hz to 8000 Hz) and is sensorineural in nature (Garth, 1995; Nageris et al., 2008; Shah et al., 2014), regardless of pattern. While low frequency losses were less common, had a tendency to be conductive in nature, and resolved with the spontaneous recovery or surgical repair of middle ear injury (Bruins & Cawood, 1991). Alternatively, Singh and Ahluwalia (1968) found predominately flat configurations in their study of 79 otologic blast injuries from

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Figure 3. The most common audiometric configurations after blast exposure
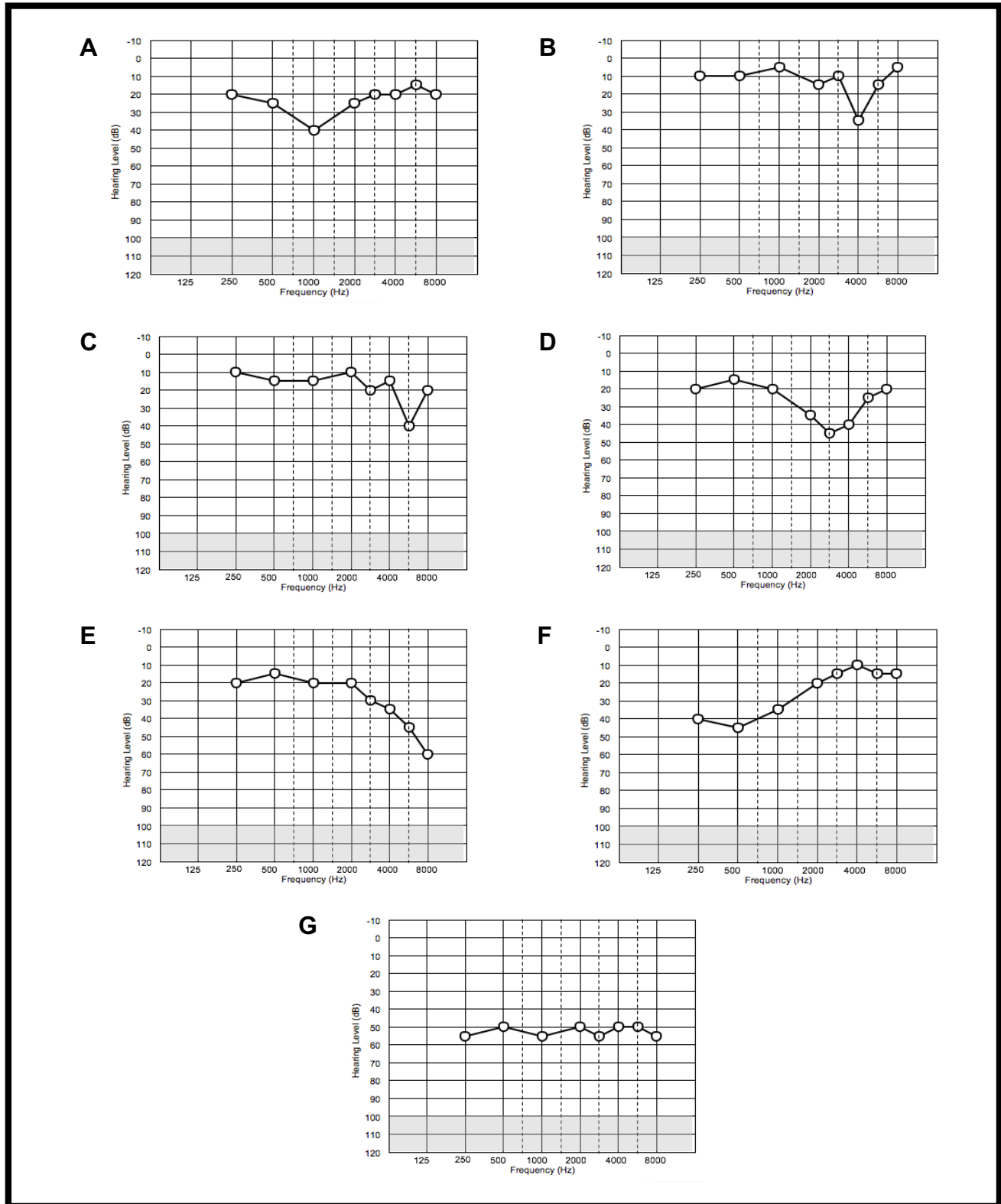

Figure 3a-g. Example audiograms displaying the most common configurations after blast exposure in this review. 3a. 1000 Hz dip. 3b. 4000 Hz dip. 3c. 6000 Hz dip. 3d. Trough shaped.

the Indo-Pakistan conflict in 1965. Ziv et al. (1973) noted a high preponderance of 4000

Hz dip configurations followed by flat and sloping losses. Sloping losses and dip

configurations were the most frequently noted in this review with special focus on dips

occurring at 6000 Hz. Currently, there is no one definite audiometric configuration that

can be linked exclusively to blast-exposure (Jagade et al., 2008; Perez et al., 2000).

More research is needed in order to identify a configuration linked directly to blast

pressure waves. Because of the distinctive features of each blast and the multitude of

factors that affect the intensity and vector in which a pressure wave enters the ear

canal, it can be difficult to compare findings from one incident to another. A distinctive

pattern of how hearing loss, as a primary blast injury, presents audiometrically, would

help aid in differential diagnosis between blast-induced hearing loss and other

etiologies.

Tinnitus

Tinnitus is defined as the perception of sound in the absence of external stimuli.

The sound perceived does not have a definitive quality but can be drastically different

from individual to individual. Some recurrent descriptive words include buzzing, hissing,

static or ringing in one or both ears. Similar to hearing loss, tinnitus can be transient or

permanent. Tinnitus can also be present intermittently or constantly. The majority of

tinnitus cases do not cause disruption to an individual's activities of daily living.

However, for those unable to habituate, severe or bothersome tinnitus can be

detrimental to their psychological health and overall quality of life. While there may be

multiple etiologies responsible for tinnitus, researchers have yet to determine a definite

cause for most cases. There is no current consensus between interdisciplinary professionals on the most effective method to manage severe cases of bothersome persistent tinnitus. While hearing aids or generated noise can help mask or alleviate the perception of tinnitus, there is no cure to tinnitus at this time. Tinnitus can trigger distress and has been strongly correlated with depression, anxiety, irritability and difficulty sleeping. Tinnitus can negatively influence cognitive functioning and, in extreme cases, has been linked to suicide (Erlandsson & Tyler, 2000).

Tinnitus is the number one service-connected disability for United States veterans, followed by hearing loss as a close second (Folmer, McMillan, Austin, & Henry, 2011). Veterans Association Medical Centers do not have standardized clinical protocols for tinnitus management. Additionally, de Régloix et al. (2017) found that there is a significantly higher incidence of tinnitus in service members with post traumatic stress disorder (PTSD) which is rampantly prevalent in the military population. Fagelson (2007) found that PTSD is strongly associated blast-induced tinnitus reporting that 34% of the 300 veterans of the United States Armed Forces in his study whom were experiencing tinnitus also carry a diagnosis of PTSD. Sounds triggering episodes of PTSD can often intensify the perception of tinnitus supporting Fagelson's (2007) findings that those who reported qualities such as severity, suddenness of onset, and "sound-triggered exacerbation" of tinnitus were more common among subjects with both tinnitus and PTSD compared to those with solely tinnitus. Andersson, Baguley, McKenna, and McFerran (2005) further expanded that psychological disorders, in general, when comorbid with tinnitus, have the potential to intensify each other.

Almost all individuals experience tinnitus immediately after blast exposure (Kerr & Byrne, 1975). The acoustic characteristics and duration of tinnitus vary case by case. They found that most subject's tinnitus was described as high frequency and hissing in nature. If unilateral, tinnitus is usually perceived in the ear most exposed to the blast which can either be by direct pressure waves or those deflected by objects in the environment (Bruins & Cawood, 1991). If present in both ears, it is usually perceived as worse in the ear with more exposure relative to the contralateral ear. Overall, Yetiser and Ustun (1993) found, during their research with a military population in Turkey, that the subjective severity of tinnitus cannot be associated to the severity of hearing loss, if hearing loss is present.

Reports of tinnitus at initial evaluations in this review can be found in Table 5. The findings range from 8 of 100 subjects (8%) reporting tinnitus after exposure to bomb blast incidents in South Asia to 100% of subjects exposed to bomb blasts in Israel, the United Kingdom and Pakistan over multi-year inclusion periods (Nageris et al., 2008; Nawaz et al., 2014; Qureshi et al., 2017; Walsh et al., 1995). The two authors with the largest samples produced similar findings. Miller, McGahey and Law (2002) retrospectively evaluated 138 subjects exposed to blast when a 440-lb car bomb detonated on a busy street corner in Omagh, Ireland (August, 1998). Sixty-four of these subjects (46.4%) reported tinnitus at their initial evaluation anywhere from one day to ten months after the incident. It should be noted that due to the potential for spontaneous recovery of tinnitus within the maximum ten-month time period, this finding should be interpreted with caution. Cave et al. (2007) produced similar findings as 127 of his 258 subjects (49.2%) reported tinnitus at their initial evaluations. The average

incidence of tinnitus of all relevant studies in this review was found to be slightly higher at 57.2%.

Table 5. Reports of tinnitus at initial evaluation

Authors (Year)	Total No. of Subjects	Subjects Reporting Tinnitus	
		No.	%
Aslier & Aslier (2014)	25	21	84
Cave et al. (2007)	258	127	49.2
Cohen et al. (2002)	17	15	88.2
de Regloix et al. (2017)	41	36	87.8
Jagade et al. (2008)	52	20	38.5
Miller et al. (2002)	138	64	46.4
Mrena et al. (2004)	29	19	65.5
Nageris et al. (2008)	73	73	100
Nawaz et al. (2014)	30	30	100
Pahor (1981)	111	26	23.4
Persaud et al. (2003)	17	15	88
Qureshi et al. (2017)	100	8	8
Remenschneider et al. (2014)	44	30	68.2
Shah et al. (2014)	110	89	81
Tun et al. (2009)	50	44	88
Tungsinmunkong et al. (2007)	33	22	66.7
Van Campen et al. (1999)	83	48	57.8
Walsh et al. (1995)	12	12	100

Tympanic Membrane Perforations

Pressure up to 5 pounds per square inch (psi) (35 kPa) can rupture the tympanic membrane and pressures up to 15 psi (105 kPa) will rupture the tympanic membrane in 50% of cases (Abbas, Arshad & Ghani, 2014; Branica, Dawidowsky, Sprem, & McKinnon, 2008; Chait & Zajtchuk, 1989; Garth, 1995; Hirsch, 1968; Okpala, 2011). Additionally, peak pressures of 130 dB SPL or higher can cause tympanic membrane perforations (Reichenbach, 2016). Most blasts produce pressures greater than those needed to rupture the tympanic membrane. Consequently, perforations are the most common ear injury sustained by those exposed. Newman et al. (2015) reports that tympanic membrane perforations are the most common significant combat related ear injury as well. In the past, tympanic membrane perforations were thought to be indicators of other internal blast injuries (Darley & Kellman, 2010; de Régloix et al., 2017). While it is now known that tympanic membrane perforations can occur without injury to any other organ, blast injury to any other organ is almost always associated with tympanic membrane perforations (Ziv et al., 1973).

Numerous researchers agree that tympanic membrane perforations are usually caused by the positive phase of the pressure waves (Abbas et al., 2014; Chait & Zajtchuk, 1989; Shah et al., 2014; Yetiser & Ustun, 1993; Ziv et al., 1973). Yetiser and Ustun (1993) frequently found tympanic membrane epithelium in the middle ear space and portions of the tympanic membrane folding in on itself toward the middle ear. It is, however, possible that some may be caused by the sucking of the negative pressure wave (Kerr & Byrne, 1975) when the edges of the perforation are pulled peripherally

towards the outer ear. Earlier research by Perlman (1941) theorized that if the negative pressure following the initial positive overpressure of a blast wave is substantial in size and prolonged, a tympanic membrane that was not perforated by the positive pressure may be done so by the subsequent negative pressure.

The size and severity of the perforations can be dependent on multiple factors. One is the force of the blast meaning the larger the blast, the larger the perforation (de Régloix et al., 2017). The closer an individual is to the location of the blast, the stronger the pressure waves are upon impact with the tympanic membrane. It is thought that distance was directly related to the size of the perforation. Examination of injuries after the SCUD missile explosion revealed more bilateral tympanic membrane perforations within 30 feet of the blasts relative to those further away (Patow, Bartels & Dodd, 1994). Alternatively, Remenschneider et al. (2014) found that distance had little effect on the the presence of perforations. After the Boston Marathon Bombing (2013), those as far as 30 feet from the blasts did not have perforations but some as close as 5 feet were unaffected as well. These contradictory findings exemplify the tremendous effects environment can have on severity of injury. Multiple studies found that younger people are less susceptible to perforations than the elderly (Darley & Kellman, 2010; Hirsch, 1968; Walsh et al., 1995, Yetiser and Ustun, 1993). Additionally, past otologic insult affects the resistance of the tympanic membrane to perforations. Scarring lessens resistance while chronic pathologic conditions can thicken the tympanic membrane making it more resistant to perforation (Hirsch, 1968).

Generally, ears facing the blast have larger or more severe perforations (Bruins & Cawood, 1991; Kerr & Byrne, 1975). However, when obstacles such as walls or doors are present in an environment, ears closer to those surfaces tend to have larger perforations as pressure waves are reflected (Chait, Casler, & Zajtchuk, 1989; Persaud et al., 2003).

Categorization

Of the nine studies in this review, which not only categorized tympanic membrane perforations by size but also provided their detailed categorization criteria, the two most commonly found are displayed in Figure 4. While three authors (Helling, 2004; Patow et al., 1994; Remenschneider et al., 2014) chose to utilize a standardized grading system created by Griffin (1979), others established their own guidelines for the purpose of their individual studies. Despite similar descriptions, there are major discrepancies between most grading scales in this review.

The grading system standardized by Griffin (1979), based on percentage affected, is as follows and can be visualized in Figure 4:
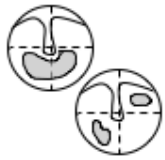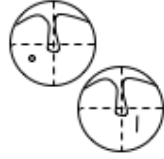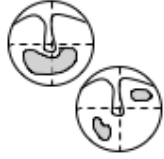
- Grade I – less than 25%

- Grade II – 25 to 50%

- Grade III – 50 to 75%

- Grade IV – greater than 75%

Alas, another three studies –two of which share similar authorship- (Kronenberg, Ben-Shoshan, & Wolf, 1993; Ritenour et al., 2008; Wolf, Kronenberg, Ben-Shoshan, & Roth, 1991) utilized a similar, but ultimately different, grading scale which can be visually compared in Figure 4:

- Grade I – a pinpoint or linear tear less than 2 millimeters

- Grade II – less than 25%

- Grade III – 25 to 50%

- Grade IV – greater than 50% or subtotal

Figure 4. Differences in tympanic membrane perforations grading scales

Studies Utilizing Grading Scale	Grade I	Grade II	Grade III	Grade IV
Helling (2004) Patow et al. (1994) Remenschneider et al. (2014)	Less than 25% or with one quadrant affected	25% to 50% or with two quadrants affected	50% to 75% or with three quadrants affected	Greater than 75% or subtotal with four quadrants affected
Kronenberg, Ben-Shoshan, and Wolf (1993) Ritenour et al. (2008) Wolf, Kronenberg, Ben-Shoshan, and Roth (1991)	Pinpoint or linear tear up to 2mm	Less than 25% affected	25% to 50% affected	Greater than 50% affected or subtotal

Note. Adapted from "Tympanic membrane perforation in survivors of a SCUD missile explosion" by C.A. Patow, J. Bartels, and K.T. Dodd, 1994, Otolaryngology and Head & Neck Surgery, 110(2), *p. 216.*

More inconsistencies exist between the two aforementioned grading scales with others in this review. Several additional studies disregarded these systems altogether

and chose a more simplistic method. They categorized the perforation sizes as small, medium, or large based on subjective otoscopic findings. Many authors omitted their operational definitions of these descriptors. Consequently, comparison of non-corroborating grade scales would yield deceptive findings. The inconsistencies noted throughout reports of tympanic membrane severity prove to be problematic when analyzing initial findings and predicting the long term prognosis.

The percentage of subjects who had tympanic membrane perforations of any size at initial evaluation and the percentage of those who had bilateral perforations can be found in Table 6. In virtually all of the studies examined in this review, unilateral perforations were much more common than bilateral perforations. As seen in Table 6, some authors did not provide the amount of subjects with bilateral perforations.

When examining solely the presence of tympanic membrane perforations within relevant studies in this review, Cohen et al. (2002), once again, reported the highest rate of tympanic membrane perforations as all 17 subjects (100%) exposed to blasts inside of a bus has tympanic membrane perforations at initial evaluation. The average percentage of subjects with any perforations of the relevant studies in this review is 35.6%. It is plausible that Cohen and colleagues' (2002) findings are significantly higher than the average because, as discussed earlier, this incident occurred in a confined space. Peak pressures may have been higher, the positive pressure wave may have lingered for a longer time and reflected waves may have struck subjects causing more severe injuries as compared to if the same explosive device would have detonated in an open area (Nageris et al., 2008; Okpala, 2011). Abbas et al. (2014) provided the largest

sample size when examining all potential subjects with blast injuries who arrived at the ENT Department of the Combined Military Hospital in Peshawar, Pakistan. Of the 393 potential subjects Abbas vetted for further investigation secondary to spontaneous recovery and concurrent inner ear symptomology, 74 (18.8%) presented with tympanic membrane perforations. The lowest rates of tympanic membrane perforation in this review were described by Aslier and Aslier (2014) in their chart review of 25 law enforcement officers with blast trauma who were initially treated at Silopi State Hospital in Turkey. Additionally, Tun et al. (2009) assessed 50 blast exposed soldiers injured in the Iraq war who later presented to the Boston Veterans Affairs Medical Center's poly-trauma outpatient clinic in 2008. Incidence of tympanic membrane perforations in these studies were only 3 of the total 25 subjects (12%) and only 4 of the total 50 subjects (8%) respectively. Location of these subjects, whether inside or outside, as well as their physical orientation to the blasts were not reported.

While unilateral perforations were a significantly more common finding, the presence of bilateral perforations at initial evaluation were also examined. Persaud et al. (2003) found the highest rates of bilateral tympanic membrane perforations after the Soho Nail Bomb in London with 7 of his 17 subjects (41.2%) having bilateral perforations. The lowest rates were found by Abbas et al. (2014) who reported only 21 of their 393 subjects (5.3%) to have bilateral perforations. The average percentage of subjects with bilateral tympanic membrane perforations of the studies disclosing this information is 13%. This calculated average most closely associated to the findings of Miller et al. (2002) whose evaluation of subjects exposed to a large-caliber car bomb detonated on a street corner in Ireland and those of Remenschneider et al. (2014) of

whom examined subjects affected by the Boston Marathon bombings. It should be

noted that both of these incidents occurred in crowded roadside locations.

Table 6. Tympanic membrane perforations at initial evaluation

Authors (Year)	Total No. of Subjects	No. of Subjects with Perforations		No. of Subjects with Bilateral Perforations	
		No.	%	No.	%
Abbas et al. (2014)	393	74	18.8	21	5.3
Aslier & Aslier (2014)	25	3	12		
Bruins & Cawood (1991)	20	8	40	7	35
Cave et al. (2007)	258	82	31.8	20	7.8
Cohen et al. (2002)	17	17	100	10	5.8
de Regloix et al. (2017)	41	21	51.2	15	36.6
Jagade et al. (2008)	52	40	76.9	15	28.8
Katz et al. (1989)	29	22	75.9		
Miller et al. (2002)	138	105	76.1	19	13.8
Mrena et al. (2004)	29	8	27.6	1	3.4
Nageris et al. (2008)	73	27	37	14	19.2
Nawaz et al. (2014)	30	11	36.7		
Pahor (1981)	111	20	18	9	8.1
Patow et al. (1994)	172	62	36	28	16.3
Persaud et al. (2003)	17	14	82.4	7	41.2
Raju (2015)	13	3	23.1		
Remenschneider et al. (2014)	94	48	51.1	19	14.9
Shah et al. (2014)	110	18	16.4	9	8.1
Tun et al. (2009)	50	4	8		
Tungsinmunkong et al. (2007)	33	22	66.7	9	27.3
Walsh et al. (1995)	12	3	25	1	8.3

When it comes to management or treatment of tympanic membrane perforations, medical professionals are presented with three options: immediate tympanoplasty, patching the affected area, or delaying either and allowing time for spontaneous closure (Wolf, Kronenberg, Ben-Shoshan, & Roth, 1991).

Role of Tympanic Membrane Perforations on Inner Ear Damage

Previously, it was thought perforations to the tympanic membrane served to protect the inner ear against damage. It was theorized that when the conducting system of the middle ear was interrupted, less energy would be transferred to the cochlea. A series of authors produced four studies that examined the presence of inner ear pathology including hearing loss, tinnitus, and vestibular manifestations in subjects with tympanic membrane perforations.

Wolf et al. (1991) was the earliest of the four in this review to detail inner ear subjective reports and audiometric findings in 147 subjects who suffered from blast injury during military service and sustained tympanic membrane perforations in one or both ears. They found that 139 ears (66%) with tympanic membrane ruptures had a sensorineural component of hearing loss. Furthermore, 79 of these 147 subjects (53.9%) reported tinnitus in one or both ears. Only 9 of these subjects (6%) reported vestibular symptoms supporting the overall low prevalence rates continuously reported throughout this review. Ritenour et al. (2008) encountered a similar figure when retrospectively examining the medical charts of US service members injured in combat

explosions in Afghanistan and Iraq over a three-year period. All subjects had tympanic membrane perforations of varying magnitudes noted. They found 37 of their 65 subjects (56.9%) reported subjective hearing loss at their initial evaluations. Additionally, 21 of their subjects (32.3%) reported tinnitus and 3 (4.6%) reported vestibular symptoms.

Abbas et al. (2014) retrospectively reviewed the records of 53 blast injured subjects with 74 tympanic membrane perforations in Pakistan and found that 100% of ears with perforations were accompanied by a report of subjective hearing loss and 67 (90.5%) by a report of tinnitus. Most recently, Pusz and Robitschek (2017) conducted a prospective analysis of military personnel with tympanic membrane perforations from primary blast injury over a two-year period at The United States Army Landsthul Regional Medical Center (LRMC) in Germany. The authors found that of the 59 ears examined, 29 (49%) had sensorineural hearing loss and 50 ears (84.8%) presented with tinnitus.

Both Wolf et al. (1991) and Ritenour et al. (2008) found relatively low rates of vestibular complaints. Alternatively, Pusz and Robitschek (2017) reported subjective vestibular symptomology in a significantly higher number of their subjects. There appears to be no clear pattern for vestibular disturbances after blast exposure in this review.

Examination of this particular data presents with limitations. While Wolf et al. (1991) and Ritenour et al. (2008) reported their findings per subject, Pusz and Robitschek (2017) and Abbas et al. (2014) did so by ear despite the lack of reports

localizing potential vestibular pathology by use of standardized testing. Comparison can prove difficult as each author chose to report their findings differently. There is limited agreement in findings between these groups of researchers in regards to the amount of inner ear damage incurred with tympanic membrane perforations after blast exposure. However, the significant presence of inner ear damage in this review does refute the theory that tympanic membrane perforations serve a protective role against inner ear injury.

Symptoms of inner ear damage such as hearing loss, tinnitus and vestibular pathologies did not diminish with the presence of a tympanic membrane perforation. Continually, research has shown no statistical difference in the presence of inner ear damage in relation to the tympanic membrane perforations. The data from this review of four studies as well as investigations from other reviews of the literature produce no evidence of protective quality of tympanic membrane perforations on the inner ear (Aslier & Aslier, 2016; de Régloix, 2017; Kerr & Byrne, 1975; Yetiser & Ustun, 1993; Ziv et al., 1973). Shah and colleagues (2014), after assessment of blast and non-blast injured service members returning from Middle East deployment, found no difference in hearing thresholds with and without perforations with the exception of one particular within subject comparison. Interestingly enough, Shah and colleagues (2014) discovered a difference in 6000 Hz thresholds in those with unilateral perforations compared to their contralateral ear.

Animal models such as one presented by Cho and colleagues (2013) further contested the dated theory that tympanic membrane perforations protect the inner ear

from damage. Cho et al. (2013) exposed mice to fabricated pressure waves in a blast chamber. Compared to their control mice who had surgically-placed tympanic membrane perforations before exposure, the thresholds shifts were larger in those whose tympanic membranes were perforated by the blast winds. These findings indicate cochlear damage had occurred and further supports the theory that tympanic membrane perforations had no influence on blast-induced hearing loss outcomes. In humans, Hirsch (1968) stated that, after reviewing the literature regarding the effects of blast overpressures on the ear, while middle ear damage is associated with inner ear damage, the absence of middle ear damage does necessarily not indicate the absence of inner ear damage.

Role of Tympanic Membrane Perforations on Conductive Hearing Loss

It is widely known that tympanic membrane perforations can and usually do produce some degree of hearing loss. While no correlation between the size of tympanic membrane perforations and magnitude of a sensorineural component has been found in recent studies, Persaud et al. (2002) emphasized the strong and direct correlation between the size of the perforation and magnitude of a conductive component.

Mehta, Rosowski, Voss, O'Neil and Merchant (2006) explored this concept further when they analyzed 62 perforated tympanic membranes in a total of 56 patients. They utilized pure tone audiometry explicitly using supra-aural headphones citing their previous study in which it was revealed that tympanic membrane perforations can affect

the sound pressure in the ear canal that is generated by insert earphones. At the conclusion of that study, results revealed hearing measured with insert earphones were significantly worse than the subject's actual loss (Voss et al., 2000). Mehta and colleagues (2006) also assessed the size of the middle ear space with tympanometry, hypothesizing that ears with smaller middle ear spaces would produce larger conductive losses. Their results corroborated Persaud et al. (2002) as they also found a significant increase in air-bone gaps with increasing perforation size at all frequencies measured (250 to 8000 Hz) as well as a significant inverse correlation between middle ear size and present air-bone gaps. Consistent with the findings in this review, the largest air-bone gaps were present at the lower frequencies and diminished with increasing frequency.

Data has shown that the pars tensa is the most susceptible to blast injury as no injury to the pars flaccida alone has ever been reported (Chait et al., 1989) and that perforations most commonly occur in the anterioinferior quadrant of the tympanic membrane (Persaud et al., 2003; Yetiser & Ustun, 1993). In 2018, Sood, Pal and Kumar examined 100 patients with tympanic membrane perforations from various incidents with aim of determining the effect of location of perforations on magnitude of conductive hearing loss incurred. While they observed that maximum hearing loss was found in subjects with central perforations and perforations in the posterior quadrant produced a higher average hearing loss than those in the anterior quadrants, ultimately, none of the findings had statistical significance. Results from this study and that of Mehta et al. (2006) found that, contrary to popular belief, that location of a perforation is not a determinate for severity of conductive hearing loss.

Vestibular Injuries

Several authors stated that vestibular symptoms were most likely attributed to secondary or tertiary blast injuries rather than a symptom of primary blast injury. Thereby insinuating that most vestibular manifestations were central findings secondary to head trauma (Chait & Zajtchuk, 1989; de Régloix et al., 2017; Garth, 1995; Kerr & Byrne, 1975; Okpala, 2011). Currently, there are a number of research centers investigating the effects of blast exposure on the inner ear's balance system. Vestibular complaints can encompass anything from dizziness and true vertigo to double vision and various sensations such as drifting or falling. Benign paroxysmal positional vertigo (BPPV) is the most commonly reported vestibular complaint after blast exposure. Subjects with BPPV typically report provoked vertigo with changes in movement such as laying down, getting up or changes in head positions (Tun et al., 2009).

Darley & Kellman (2010) reported that most cases of vestibular dysfunction brought on by blast exposure were transient and recovery spontaneously overtime. As many authors did not divulge their vestibular findings post-evaluation, initial subjective reports are listed in Table 7. There appears to be a discrepancy in the prevalence of vestibular symptoms reported post blast exposure as 44% of Oklahoma City bombing (1995) victims (Van Campen et al., 1999) reported persistent dizziness, vertigo and balance issues whereas findings from incidents such as the SoHo Nail Bombing in London (1991), the London Bridge bombing (1992) and a series of bombing attacks in bazaars in Peshawar, Pakistan (Nawaz et al., 2004; Persaud et al., 2003; Walsh et al.,

1995) found no reports (0%) of vestibular symptoms. While this great variability may also be attributed to differences in pressure waves manipulated by the surrounding environment, further research and publication of blast-induced vestibular findings after formal evaluation is of particular interest to the field of audiology and those studying blast injuries in general.

Table 7. Reports of vestibular symptoms at initial evaluation

Authors (Year)	Total No. of Subjects	Subjects Reporting Vestibular Symptoms	
		No.	%
Bruins & Cawood (1991)	20	2	10
Cave et al. (2007)	258	38	14.7
Cohen et al. (2002)	17	7	41.2
de Regloix et al. (2017)	41	8	19.5
Mrena et al. (2004)	29	4	13.8
Nawaz et al. (2014)	30	0	0
Pahor (1981)	111	2	1.8
Persaud et al. (2003)	17	0	0
Remenschneider et al. (2014)	44	0	0
Tun et al. (2009)	50	10	20
Tungsinmunkong et al. (2007)	33	1	3
Van Campen et al. (1999)	83	36	44
Walsh et al. (1995)	12	0	0

Vestibular symptoms can have a delayed onset, appearing months to years after the initial incident. Similarly, one author reported no incidence of vestibular symptoms at the initial evaluation of 44 subjects at the Boston Marathon bombing (2013). However,

at the 6 month follow up evaluation, 8 of the original 44 subjects (14.2%) reported vestibular symptoms with onsets weeks to months after the incident (Remenschneider et al., 2014).

Other Injuries

Damage to the outer ear injuries or injuries to the pinna are usually the result of secondary, tertiary and quandary blast injuries such as projectile fragments and burns They are typically not life threatening. Damage to any part of the face, including the ears, can have long standing psychological and social implications (Reichenbach, 2016).

Difficulty hearing without the presence of hearing loss especially in the presence of background noise is a common complaint of subjects with blast exposure. Blast exposure may affect auditory processing and while audiometrically, a subject may not show any abnormalities, they may present to an audiologist or ENT reporting auditory difficulties (Tun et al., 2009). A considerable population of blast-trauma patients with central auditory system injury may have been misdiagnosed due to the lack of assessment criteria for this population (Fausti et al., 2009).

The literature has shown that blast exposure, regardless of the presence of hearing loss, affects localization abilities. Kubli, Brungart, and Northern (2017) found, within a population of blast exposed military personnel, localization accuracy in a simple situation such as one-on-one conversation was not impaired compared to the control

group. Subtle negative effects of blast-exposure were observed on localization accuracy when the subjects were tasked with a more complex listening situation, such as multiple talkers. The results of their study are consistent with the common subjective report of difficulty with speech in background noise and suggest blast-exposure may cause difficulty localizing in complex acoustic environments. Damage to the outer ear and pinna can further complicate localization abilities (Reichenbach, 2016). These finding are most crucial to the military population where localizing threats within one's environment and communicating with fellow soldiers in the presence of competing noise are vital for survival.

Although extremely rare, evaluation for perilymph fistula should be considered in subjects with fluctuating hearing loss and persistent vertigo. (Aslier & Aslier, 2017; Okpala, 2011; Pusz & Robitschek, 2017)

Cholesteatoma

Cholesteatoma are abnormal and rare benign skin growths that can develop in the middle ear. They can be congenital, forming as a result of recurrent middle ear infection, or be brought upon by traumatic implantation of skin cells into the middle ear thereby being labeled "acquired" (Wolf, Megirov, & Kronenberg, 1999). Often times cholesteatoma develop as a cyst that sheds layers of old skin. The dead skin accumulates, the lesion grows and can erode and destroy important structures of the middle ear, temporal bone and base of the skull…" (Darley & Kellman, 2010, p. 148) and are caused by a traumatic event as opposed to those occurring secondary to

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genetic predisposition. Cholesteatoma occur when squamous epithelium debris is driven into the middle ear through traumatic events such as a tympanic membrane perforation (Chait et al., 1989; Darley & Kellman, 2010). Overtime and undetected, cholesteatoma can affect hearing, balance, and facial nerve function.

A study examining 110 Vietnam war veterans with tympanic membrane perforations caused by blast exposure reported a 12% incidence of squamous epithelium in the middle ear (Seaman & Newell, 1971). Kronenberg, Ben-Shoshan, Modan, and Leventon (1988) reported a 7.6% incidence of cholesteatoma in a 210 blast injured ears. Sridhara, Rivera, and Littlefield (2013) found the development of cholesteatoma in 3 of their 34 subjects (9%) who underwent tympanoplasty to repair blast-induced tympanic membrane perforations at Walter Reed Army Medical Center and National Naval Medical Center with one neurologist in particular. Keller et al. (2017) reported that cholesteatoma complicated 15 of their 352 tympanoplasty procedures (4.3%). They also noted a significant relationship between increasing size of perforation and risk of cholesteatoma development. Darley and Kellman identified this same relationship in 2010. In different study with similar authorship, Song, Sridhara, and Littlefield (2017) reported that that same neurologist discovered a total of 8 cholesteatoma (11.7%) in while evaluating 68 ears pre- and post-tympanoplasty over a 5-year period.

Cholesteatoma have delayed onset as they take time to develop and can usually be detected 10-48 months after injury (Darley & Kellman, 2010; Wolf et al., 1991). Undetected, cholesteatoma can cause both sensorineural and conductive hearing loss,

vestibular dysfunction, cranial nerve palsy and central nervous system complications (Darley & Kellman, 2010) as the erosive lesion expands. It can be directly attributed to the severity of the initial tympanic membrane perforation and subsequent lack of spontaneous recovery or appropriate medical intervention.

Ossicular Damage

Ossicular damage is rarely found in blast exposure individuals (Hirsch, 1968). The incidence of ossicular damage can be directly associate to the intensity of the blast pressure as larger blast pressures produce a higher incidence of ossicular damage (Darley & Kellman, 2010). The incus, or more specifically, the incudostapedial joint is most susceptible to this trauma because of its anatomical position compared to the other ossicles in the ear. Trauma affecting the long process of incus can result in conductive hearing loss (Aldosari & Thomassin, 2017; Okpala, 2011).

Sridhara et al. (2013), in the same study analyzing various clinical findings and their effect on the success of tympanoplasty, found ossicular abnormalities in 6 of their 34 subjects (18%). Keller et al. (2017) performed ossicular chain reconstruction in 32 of their 352 blast-exposed ears (9.1%). Song et al. (2017) reported that the same neurologist discovered 5 ossicular disruptions (7.4%) in 68 ears pre- and post-tympanoplasty over a 5-year period. While rare, clinicians must be aware of the audiometric presentations associated with ossicular damage.

Late Sequelae

Due to the limited number of studies reflecting long term follow up of individuals with blast exposure, there is limited data about the prevalence and long term effects of late sequelae stemming from the original insult and management of injuries. The data that exists directs attention to four detrimental conditions which can, in the best case scenario, effect long term mental health and quality of life and in the worst cases, be lethal.

Hearing loss in those exposed to blasts can be transient or permanent. Hearing is said to recover in 30% to 55% of those who report hearing loss upon initial evaluation (Fisher, 2008). Research suggests that hearing thresholds are generally stable by 1-year post injury (Fisher, 2008; Raju, 2015; Van Campen et al., 1999). Any residual hearing loss at that time is most likely permanent. Permanent hearing loss, regardless of cause, has an adverse effect on communication abilities. When an individual has difficulty communicating and does not seek treatment or alternative solutions, such as amplification, they may socially isolate themselves.

Similar to the loss of hearing sensitivity, tinnitus can be transient or permanent. Persistent bothersome tinnitus can affect a subject's mental health and overall quality of life. Tinnitus can constantly diverge attention away from events in the environment and towards the sounds perceived. Bothersome tinnitus can prevent individuals from communicating when the perceived sound is louder than speech and more commonly, inhibit normal sleep patterns.

When tympanic membrane perforations do not heal properly or are not surgically closed after failure to spontaneously recover, an individual is at a higher risk for infection (Chait & Zajtchuk, 1989; Shah et al., 2014).

Testing

In an ideal world, initial and subsequent otologic and audiometric examinations of blast-exposed subjects would include a variety of subjective and objective testing. Also included should be in-depth interviews about the details of the event and a collection of an extensive medical history. In many of the articles analyzed in this paper and in most real-world situations, this is not the case. Physicians, first responders and audiologists in this review utilized at least two or more of the following assessment tools:

- Medical history, including a detailed otological history
- Otoscopic examination (Raju, 2015)
- Tuning fork tests (Weber and Rinne)
- Pure tone audiometry (air and bone conduction) (Kerr & Byrne, 1975)
- Speech audiometry (speech recognition thresholds [SRT] and word recognition score [WRS]) (Kerr & Byrne, 1975)
- Tympanometry (Reichenbach, 2016)
- Otoacoustic emissions (OAEs) (Reichenbach, 2016)
- Auditory brainstem response (ABR) thresholds assessment testing (Raju, 2015)

- In cases of vestibular assessment, audiologists utilized (Mills & Jones, 2012):

  o Ocular motor testing

  o Sinusoidal harmonic acceleration

  o Velocity step test

  o Visual vestibular interaction

  o Electrocochleography (ECoG)

  o Vestibular evoked myogenic potentials (VEMP)

  o Bithermal calorics

When initial assessment occurred in theater for military personnel or outside of an official medical facility, screening procedures were employed and reported intermittently.

Spontaneous Recovery

Hearing

Immediately after a blast, most individuals report profound hearing loss. Some report that they do not realize their hearing has been affected until they see people talking but hear little to no sound (Chait & Zajtchuk, 1989; Garth, 1995; Kerr & Byrne, 1975; Shah et al., 2014). In a combat setting, the instantaneous loss of hearing can compromise communication and environmental awareness putting the individual in imminent danger (Chait & Zajtchuk, 1989). This sudden loss of hearing is usually the

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result of a temporary threshold shift (TTS) which can resolve in the matter of hours to days. Researchers theorize that TTSs, similar to the insult causing permanent sensorineural hearing loss, occurs from structural changes within the cochlea caused by excessive displacement of the basilar membrane. These mechanical injuries also challenge the integrity of the tight cell junctions of the reticular lamina and may affect membrane permeability (Okpala, 2011). The mixing of perilymph and endolymph thereby creating a toxic environment interfering with physiological events in the cochlea similar to that producing sensorineural hearing loss. When the damage to the reticular lamina is less severe, the breaches are spontaneously repaired, the TTS resolves and hearing is restored. (Garth, 1995; Patterson & Hamernik, 1997; Persaud et al., 2003). Patterson and Hamernik (1997) report that auditory changes can become worse immediately after blast exposure before a recovery period ending in a stable but permanent hearing loss. The level of hearing restoration and the time period in which the resolution takes place varies from individual to individual.

The rates of spontaneous resolution of hearing loss in those exposed to blasts retrieved from the authors in this review can be found in Table 8. Limited data is currently available on the spontaneous recovery of hearing after blast exposure. Cohen et al. (2002) re-evaluated their patients 6 months after the initial incident and found that of 33 ears with abnormal audiometric findings at initial evaluation, 6 (18.2%) had complete recovery of hearing in that all depressed thresholds returned to normal and 11 (33.1%) had partial recovery, or significant and stable improvement at at least one frequency tested. Tungsinmunkong, Chongkolwatana, Piyawongvisal, Atipas, and Namchareonchaisuk (2007) reported their audiometric re-evaluation 3 months after the

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initial incident and revealed spontaneous resolution of hearing loss in 8 of the original 27 ears with abnormal audiometric findings at initial evaluation. Lastly, Walsh et al. (1995) discovered 7 of 12 subjects (58.3) with abnormal audiometric findings at initial evaluation had spontaneous recovery in hearing when they performed re-evaluations from 6 to 12 months after the initial incident.

Table 8. Spontaneous resolution rates of hearing loss

Authors (Year)	No. with Abnormal Audiograms Initial Evaluation	Time of Assessment Post-Injury	Spontaneous Resolution of Hearing Loss*	
			No.	%
Cohen et al. (2002)	33 ears	6 months	6	18.2
Partial Resolution**			11	33.1
Tungsinmunkong et al. (2007)	27 ears	3 months	8	29.6
Walsh et al. (1995)	12 subjects	6-12 months	7	58.3
*Resolution to normal hearing levels at all frequencies tested				
**Significant and stable Improvement in thresholds at one or more frequencies				

Many authors whose work was examined in this review found limited evidence of recovery in hearing thresholds after 6 months (Branica et al., 2008; Raju, G., 2015; Yetiser & Ustun, 1993). Others have stated, more conservatively, that hearing thresholds are generally stable by 1 year (Fisher, 2008; Van Campen et al., 1999) and therefore recommend frequent audiometric monitoring of blast-exposed patients with hearing loss within the first year after the initial incident and annually thereafter unless a

subjective change is noticed. Additionally, it was reported that even with recovery in sensorineural hearing loss, many subjects still reported persistent hyperacusis and difficulty in noise (Remenschneider et al., 2014).

The conductive component of a hearing loss generally recovers after closure of the tympanic membrane perforations either with spontaneous recovery or surgical intervention (Persaud et al., 2003). It reported that individuals who required surgical closure of their perforations generally had worse air conduction thresholds than those whose perforations spontaneously recovered (Remenschneider et al., 2014). Largely, the more severe the blast-induced hearing loss, the more likely there will be persistent hearing loss regardless of the amount of recovery overtime (Persaud et al., 2003).

Kerr & Byrne (1975), Garth (1995), Shah et al. (2014), and Chait and Zajtchuk (1989) detailed the immediate, typically severe but transient in nature, hearing loss many reportedly experience after blast exposure. Of the majority of subjects who suffered this acute deficit, Argyros (1997) stated that hearing resolved quickly when subjects were allowed to recover in a quiet environment. Furthermore, he reported that remaining in noisy environments has deleterious effects on residual hearing. These conclusions originate from a study in 1976 by Melinek, Naggan, and Altman when they examined a sample of 433 soldiers in Israel with hearing loss, tinnitus or both. They divided this group into two. One subgroup was sent on to work in a quieter non-combat unit where the other sub-group remained in combat units where they would incur further occupational noise exposure. For those who were transferred to the quieter work environment, 30.8% revealed an improvement in hearing while 4% showed

deterioration. Those who stayed in a noisy environment only 8.7% revealed improvement in hearing while 30.4% showed deterioration. Therefore, allowing for the recovery of hearing in a quieter environment can prove advantageous by preventing further insult to the auditory system.

Tinnitus

Spontaneous recovery of tinnitus often times parallels that of hearing loss as it tends to disappear as hearing recovers however, this is not always the case (Chait & Zajtchuk, 1989; Fisher, 2008; Wolf et al., 1991). Some may experience the perception of spontaneous recovery when they are able to habituate to their tinnitus. The rates of spontaneous resolution of tinnitus can be found in Table 9. In a study of 20 subjects injured when 800 kg of high explosives detonated in Peterborough, United Kingdom (March, 1989), Bruins & Cawood (1991) found that of the 40 ears that subjects reported to perceive tinnitus, 25 (62.5%) had spontaneous resolution of the perception of tinnitus by 1 year after the event and further stated that as their hearing improved, the tinnitus diminished; first becoming intermittent before completely disappearing from perception. While relevant, the findings of Bruins & Cawood (1991) could not effectively be compared to those of other authors in this review listed in Table 9 as all others were organized by subject as opposed to ear. It can be argued that Bruins & Cawood (1991) described their findings in a more valid approach as tinnitus can, and often is, unilateral, especially in cases of trauma. However, spontaneous rates of resolution of tinnitus was found more commonly reported by subject. The findings in this review ranged from spontaneous resolution in only 5 of 73 civilian and military subjects (6.8%) exposed to

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explosive terrorist attacks in Israel over a two-year period (2002-2004) at 6-month

follow-ups (Nageris et al., 2008) all the way to 100% spontaneous resolution rates at 3-

month follow up evaluations of subjects at both the reports on the Soho Nail Bomb in

London (Persaud et al., 2003) and those from incidence of terrorist bombings over a 5-

month period from Yala Provincial Hospital, Thailand. The average rate of spontaneous

resolution of the perception of tinnitus in their review fell centrally at 54.8%. Limitation to

comparison lies in the timing of follow up assessment of spontaneous resolution of

symptoms therefore findings should be interpreted with caution.

Table 9. Spontaneous resolution rates of tinnitus

Authors (Year)	No. of Subjects Reporting Tinnitus at Initial Evaluation	Time of Assessment Post-Injury	Spontaneous Resolution of Tinnitus	
			No.	%
Cohen et al. (2002)	15	6 months	9	60
Nageris et al. (2008)	73	1 year	5	6.8
Persaud et al. (2003)	15	3 months	15	100
Tungsinmunkong et al. (2007)	22	3 months	22	100
Van Campen et al. (1999)	83	1 year	63	76

Alternatively, Garth (1995) found that, largely, tinnitus did not improve from the

time of initial evaluation to follow up evaluations months to years later. Nageris et al.

(2008) reported a spontaneous resolution of tinnitus in only 5 of 73 victims of explosive

attacks (6.8%) one year after the initial incident. Even more so, the perception of tinnitus can potentially deteriorate, progressively getting worse as time goes on (de Régloix et al., 2017).

Tympanic Membrane Perforations

Rates of spontaneous closure of tympanic membrane perforations are not found to be related to gender or side of injury. However, there is significant correlation between spontaneous closure and (Al-Juboori, 2014):

- Initial size of the perforation

- Severity of hearing loss

- Age

- Time post injury

The rate of spontaneous closure of tympanic membrane perforations was found to be directly related to size (Aslier & Aslier, 2016; Ritenour et al., 2008). It is suggested that the size of the perforation be used as criterion for surgical intervention, if immediate treatment is the preferred course of action (Chait & Zajtchuk, 1989). The larger the initial perforation, the less likely spontaneous closure will occur. Both de Régloix et al. (2017) and Ritenour et al. (2008) examined large groups of blast-exposed subjects and found that no spontaneous closure occurred in perforations greater than 80% of the surface area of the tympanic membrane. Chait and Zajtchuk (1989) indicated that tympanic membranes heal at an average rate of 10% per month. Due to the variable definitions

and categorization of size described earlier in this review, an analysis of tympanic membrane perforations by size was not included.

In regards to age, tympanic membrane perforations in younger subjects tended to heal faster as "they have a higher protein turnover" (Al-Juboori, 2014, p. 2) than that of older subjects. Furthermore, Ritenour et al. (2004) indicated that perforations to the inferior portion of the tympanic membrane had the highest rates of spontaneous recovery rate while those located in the central region had the lowest rates. All of these factors should be taken into account when making management decisions for each individual subject. Chait & Zajtchuk (1989) offer a far less conservative approach than most stating that if no healing has occurred within 10 to 15 days of the incident, further healing is improbable, and medical intervention options should be explored. The lower the odds of spontaneous closure (i.e. older subjects, subjects with large perforations, or subjects whose perforations have not closed after a considerable amount of time) the more medical professionals should consider more aggressive options for closure.

The rates of spontaneous recovery of tympanic membrane perforations can be found in Table 10. In this review, the largest sample was that outlined in studies by Kronenberg et al. (1993) and Wolf et al. (1991) when analyzing 147 subjects with blast injury over a 19-year period. They found that after 3 months, 131 of 210 ears (62.4%) with perforations had spontaneous closure. By 10 months that 141 (69%) had closed spontaneously and that by one year after the initial incident 151 (71.9%) of tympanic membrane perforations caused by blast exposure had spontaneously recovered without surgical intervention. Another relatively large study by Miller et al. (2002) found

significantly lower rates of spontaneous recovery when at 3 months after the incident they found only 29 of 124 (23.4%) tympanic membrane perforations had spontaneously closed and by one year, only 47 of the original 124 perforations had spontaneously recovered.

Several authors found spontaneous recovery rates above 80% while Miller et al. (2002) had the lowest spontaneous closure rates in this review. Al Juboori (2014) reported the highest rate at 82.3% when 51 of 62 perforations healed spontaneously by 3 months in his evaluation of 60 subjects with traumatic tympanic membrane perforations at Al-Fallujah Teaching Hospital in Iraq. While not all attributable to blasts, Al Juboori (2014) highlights the insignificance of etiology in regards to analysis of spontaneous recovery and attributes the failure of perforations to spontaneously recover to loss of tissue and secondary infections. The average rate of spontaneous recovery of tympanic membrane perforations at 3 month follow up evaluations in this review was 54.7%. Of those reporting at 6 months, the figure dropped to 47.8%. By one year after the incident, the average spontaneous recovery rate was, of those who reported it, was 60.2%.

A notable pattern in recovery of hearing loss is connected to the closure of tympanic membrane perforations which subsequently can affect the closure of air-bone gaps. The initial presentation in those with tympanic membrane perforations is commonly a mixed hearing loss. Closure of the tympanic membrane perforation, either spontaneously or with surgical intervention, is typically followed by at least partial recovery of the conductive component (Fausti et al., 2009).

Table 10. Spontaneous recovery rates of tympanic membrane perforations

Authors (Year)	Total No. of Perforations	Time of Assessment Post-Injury	Spontaneous Recovery of TM Perforations	
			No.	%
Abbas et al. (2014)	74	3 months	39	52.7
Al Juboori (2014)	62	3 months	51	82.3
Bruins & Cawood (1991)	15	1 year	12	80
Cohen et al. (2002)	27	6 months	15	55.6
de Regloix et al. (2017)	36	3 months	27	75
Jagade et al. (2008)	52	6 weeks	30	46.2
Kronenberg et al. (1993); Wolf et al. (1991)	210	3 months	131	62.4
		10 months	145	69
		1 year	151	71.9
Miller et al. (2002)	124	3 months	29	23.4
		1 year	47	37.9
Nawaz et al. (2014)	11	1 month	8	72
Pahor (1981)	25	Unknown	20	80
Persaud et al. (2003)	9	3-6 months	7	77.8
Remenschneider et al. (2014)	50	6 months	19	38
Ritenour et al. (2008)	74	Unknown	34	46
Tungsinmunkong et al. (2007)	31	2 months	23	74.2
Walsh et al. (1995)	4	6 months	2	50
Note. TM = tympanic membrane				

Limitations

In depth analysis of the data should be interpreted with caution. No study or review is without its limitations. In fact, many studies encountered similar limitations

therefore it is important to keep in mind how such factors influence the outcomes and findings. While compiling data for this review, the most restrictive consideration was the difficulty encountered by compare findings from one blast event to another. This is due to each individual events having a set of unique characteristics that affect the outcome, particularly the qualitative and quantitative findings of blast injuries. As discussed previously, blast characteristics (amount and material of explosive), location (in a confined space or open field) and objects present in the environment can play an immense role in determining otologic damage. Furthermore, individual factors such as age, previous otological history, TBI or cognitive deficits and the utilization of hearing protection can also affect the outcome. Because of this, it can be problematic comparing one event to another.

Additionally, within all research, there is a variability in the ways in which authors choose to analyze their data. Differences in inclusion criteria, categorization, recruitment and terminology used can all differ from study to study (Mills & Jones, 2012; Van Campen et al., 1999). An example of this are the grading systems employed for tympanic membrane perforation size. Information depicted in Figure 4 further allows one to visual the differences between multiple studies in this review thereby exhibiting why comparison of spontaneous recovery findings would be problematic. Furthermore, between study variability for categorizing severity of hearing loss was found to be just as, if not more, problematic for comparison. Ultimately, these two factors were not chosen as a variable in this review due to these vast inconsistencies.

The range of time between incident and initial evaluation was drastically large in this sample creating further complications in regards to comparison (Perez, Gatt, & Cohen, 2000). While it is ideal to assess the subjects immediately after exposure to blasts, it is not realistic. This review found initial evaluations performed anywhere from a couple of hours to 5 months after the incident of exposure with most performed within about 2 weeks. When long periods of time pass without evaluation, researchers may not obtain the true symptomology and immediate effect of the blast on the ear and auditory system. Perez et al. (2000) suggested that the timing of the initial audiological evaluation can actually determine the audiometric configuration of the hearing loss. Most symptoms tend to be transient in nature and spontaneously recover over time therefore early assessment of otologic injuries yields more accurate findings. Studies that reported their initial findings on subject's weeks, months or years after the initial incident, risk making inaccurate statements about the true effects of the blast.

A small number of researchers or medical professionals performing initial evaluations performed an oral history intake on each subject including questions about prior medical history. It is imperative to consider previous noise exposure, otologic symptoms and occupation when analyzing subject symptoms after blast exposure, especially within the military population. Unfortunately, subjective report and objective findings may not always correlate due to either poor reporting, cognitive functioning or malingering (Raju, 2015). Raju (2015) provided data from a study encompassing this concept when he and his team audiologically assess 13 subjects working in an explosive manufacturing unit in Andhra Pradesh, India who were exposed to blast trauma. The authors noted that none of the subjects had occupational noise exposure

and all wore hearing protection while working. At initial evaluation using pure tone audiometry, 10 of the 13 subjects (77%) showed significant sensorineural hearing loss. Two to 3 years after the initial incident, per protocol at this facility, they were referred for disability evaluation which would ultimately grant financial compensation, if approved. At this point in time, 12 of the 13 subjects (92%) indicated significant sensorineural hearing loss which did not clinically correlate. After re-evaluation using objective testing such as acoustic reflex, OAEs and ABR testing, none of the subjects were found to have significant hearing loss. This study and cases like it outline the necessity of employing objective measures of hearing sensitivity when evaluating for compensation.

Most studies lacked long term follow up. Otologic injuries incurred from blast exposure were rarely reassessed after 1 year. Because of this, little is known about the late sequelae, morbidity and long term quality of life of affected subjects (Nageris et al., 2008). Clinicians and future researchers should follow up with their subjects, as medically indicated, until symptoms resolve or stabilize. Annual audiometric evaluations should be recommended to monitor changes in hearing and other otologic symptomology with a late onset. de Régloix et al. (2017) found worsening in the perception of tinnitus in some of their subjects as time went on. Furthermore, the addition of presbycusis to auditory trauma caused by blast exposure could become detrimental to a subject's social and communicative abilities and overall quality of life.

The extreme variation in vestibular findings can similarly be explained by the variation of each event however there is limited understanding of how vestibular function is affected by blast exposure. This can make assessment and identification of

vestibular symptoms as primary blast injuries rather than secondary, tertiary or so forth, challenging. Numerous authors have studied the vestibular symptomology of subjects with blast exposure but many report that they are unable to separate the mechanism of injury including those with blunt head trauma, blast head trauma, or unknown causes. They proactively state that blast-exposed subjects may have additionally experienced secondary blunt head trauma at the time of the incident. Some authors have presented exceedingly limited data. These factors have undoubtedly made assessment of the vestibular system post- blast injury problematic. Moreover, of those who have published data, results are not corroborative and there is extensive variation from study to study (Mills & Jones, 2012).

Recommendations

Because exposure to blasts frequently results in poly-trauma, life threatening injuries should be ruled out or managed first. Once stabilized, the ears should be assessed (Garth, 1995). Unless there is extensive outer ear or pinna injuries, damage to the auditory system including the middle and inner ear are virtually invisible in the absence of subject report. Furthermore, many subjects who are unable to report symptoms or those with confounding cognitive factors, such as TBI, may be overlooked or misdiagnosed. Because of the frequently poly-traumatic nature of blast exposure, it is important to differentiate the symptomology of psychological pathologies versus auditory damage as they can easily be misdiagnosed.

Simple screening procedures should be utilized by first responders and other medical professionals who are the first to come in contact with blast-exposed subjects (Chait & Zajtchuk, 1989). Any suspicion of injury should warrant a referral to an otologist or otolaryngologist. All subjects in proximity to the blast should be screened for otologic injuries regardless of their distance from blast or absence of other injuries (Okpala, 2011). Examining the audiological and vestibular systems as soon as possible can lead to better outcomes and morbidity.

Initial evaluations should include a subject interview with complete medical history, including a detailed otologic history, and recollection of the event, if possible. With this, audiologists should consider the subject's occupation and prior noise exposure. Researchers should always take into account, especially when assessing military populations, that "there is an accumulative effect on hearing when multiple acoustical insults are sustained by an individual" (Hirsch, 1968, p. 156; Remenschneider et al., 2014). Subject's should be assessed for proximity and head orientation in relation to the blast along with the presence of any otologic symptoms such as bleeding from the ears, otalgia, the sensation of pressure, tinnitus, pre-existing hearing loss and the status of the tympanic membrane (Chait & Zajtchuk, 1989). Audiometry should be performed as soon as possible (Okpala, 2011). For military personnel, a complete audiological evaluation should be performed immediately after evacuation and stabilization of life threatening injuries. As it is now protocol for the United States Armed Forces, these post-incident audiometric findings should be compared to pre-deployment audiograms to assess the extent of auditory injury whenever possible. Audiometric examinations should include: the aforementioned

detailed history, otoscopic examination to report on the status of the external ear canal and tympanic membrane, immittance measures including tympanometry and acoustic reflex measures to assess the functional capacity of the eardrum and middle ear, pure tone audiometry with air conduction thresholds from 250 Hz to 8000 Hz (including 3000 Hz and 6000 Hz inter-octaves) and bone conduction thresholds from 250 Hz to 4000 Hz. All thresholds should be appropriately masked when indicated. At times where audiological equipment is not readily available, otoscopic examinations and tuning fork tests, such as Weber and Rinne, should be employed. For subjects reporting difficulty hearing without the presence of abnormal audiometric findings, clinicians should consider the appropriate referral for examination of central auditory processing skills. In their early stages, signs of auditory dysfunction may not appear in typical audiometric evaluations. When appropriate, audiologists should consider the adoption of OAEs into protocol for blast-exposed subjects as researchers have found OAEs to be more sensitive to noise trauma than standard audiograms (Nageris et al., 2008).

In cases where legal liability or compensation are involved, or any instance where a medical professional is concerned about malingering, objective testing should be employed. Audiologists may utilize OAE and ABR testing to corroborate subjective findings (Raju, 2015). Additionally, a Stenger test may be utilized for those who are suspected of malingering a unilateral hearing loss.

The appearance of vestibular symptomology after blast exposure has proven to be mixed in this review as well as many others. These findings ultimately indicated that all reports of vestibular dysfunction as a result of primary, or even secondary, blast

injury should not be overlooked. The appropriate recommendations for full vestibular evaluation should be made promptly in order to properly accommodate these subjects with suitable interventions. Referrals to vestibular rehabilitation may be warranted.

In regards to tinnitus, the subjective severity tends to dictate clinical recommendations for treatment and management. At times, subjects may report the presence of tinnitus but that their tinnitus does not bother them. It is also possible that subjects with long-standing tinnitus have habituated and therefore audiologists may note its presence and provide no further recommendations at that time. Nevertheless, it is imperative that subjects reporting bothersome tinnitus be advised about and referred to appropriate management options. While currently, there is no definitive treatment for the perception of tinnitus, there are a variety of management options that should irrefutably be attempted.

Subjects with concurrent hearing loss that is aid-able should utilize hearing aids. For the last 70 years, hearing aids have been found to partially or even completely mask tinnitus (Tunkel et al., 2014) when utilized in conjunction with suitable counseling. Searchfield, Kaur and Martin (2010) compared two groups of 29 subjects with tinnitus each for the effect of hearing aids as a treatment option. One group chose to utilize hearing aids while the other chose to forgo hearing aids, both group received the same counseling thereby controlling for that potential variable. He found that the group who wore hearing aids during this showed significant improvement on subjective outcome measures compared to those who did not utilize hearing aids.

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Audiologists, when programming hearing aids for subjects with bothersome tinnitus, should apply a prescriptive formula that maximizes audibility while monitoring maximum output levels. An attempt should be made to place emphasis on amplifying low-frequency sounds. The overall fitting objective should be to correct the hearing loss and by doing so, connecting the subject to his or her environment. Introducing additional low-frequency and intensity stimuli may help divert attention away from the tinnitus (Searchfield, 2015). Additionally, McNeill, Tavora-Vieira, Alnafjan, Searchfield, and Welch (2012) found that effective masking of tinnitus was more likely in subjects with good low frequency hearing and with those whose identifiable pitch of their tinnitus was within the frequency range of the hearing aids.

In 2015, researchers compared the use of a hearing aid to a noise generator for effectiveness in treatment of blast-induced chronic tinnitus. They did so by assessing subject satisfaction of over a two-year period in 974 veterans of the Iran-Iraq War (1980-1988) with blast injuries and attributable chronic tinnitus. Approximately 84% of subjects preferred using a hearing aid alone while only 2.7% preferred the noise generator alone. They concluded that hearing aids were the most effective, long-term, treatment for blast-induced chronic tinnitus (Jalilvand, Pourbakht, & Haghani, 2015). For those suffering from bothersome tinnitus who do not have accompanied hearing loss, these ear-level noise generators may be an appropriate recommendation as they were still effective at reducing the perception of tinnitus in some subjects.

When tinnitus is accompanied by any degree of psychological disorders such as anxiety, depression or PTSD, referrals to appropriate mental health professionals must

be made. Common therapeutic strategies for tinnitus include cognitive behavioral therapy (CBT) and tinnitus retraining therapy (TRT). Subsequent psychological and psychopharmacological therapies may be warranted (Tunkel et al., 2014). Currently, developers and clinicians alike are working on the ability to deliver these vital psychological services remotely to those who cannot easily access medical facilities due to physical impairment or distance.

There are differing views on how, when, and even if to appropriately treat tympanic membrane perforations. Abbas et al. (2014), Darley and Kellman (2010) suggest waiting up to 3 months to allow time for spontaneous closure of all perforations, regardless of size, before pursuing any definite management. Immediate surgical intervention should only be the precautionary removal of foreign materials and debris (Okpala, 2011). This conservative "watch and wait" approach is popular for small to medium sized perforations that encompass less than than 75% of the tympanic membrane as most perforations of this size close within this by 3-month period. The larger the tympanic membrane perforation, the less likely a successful spontaneous closure (Persaud et al., 2003). Water precautions should be taken during this time as to prevent any foreign substances or bacteria into the middle ear (Chait & Zajtchuk, 1989). Furthermore, if there is a medical need for antibiotics in the ear, physicians should be cautious to avoid the use of ototoxic or vestibulotoxic antibiotics, when possible (Reichenbach, 2016) to prevent further injury.

If a perforation has not closed by the end of this waiting period, the option of surgical closure via tympanoplasty should be explored (Kerr & Byrne, 1975). Sprem et

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al. (2001) refuted that this conservative approach has any detriment on hearing sensitivity when they found that the number of days elapsed between the initial incident and tympanoplasty had no statistical difference in post-operative air-bone gaps. Once a tympanic membrane perforation has successfully been closed either through spontaneous recovery or surgical intervention, subjects should be re-evaluated and monitored for late sequelae, such as secondary infections, persistent hearing loss or cholesteatoma, twice a year for the subsequent 2 years (Darley & Kellman, 2010).

Because of the potential dangers of late sequelae, there is urgent need for long term follow up that is not currently standard practice in related fields. Most authors did not report on follow up with their subjects past one year. Many of which ending their follow up with the resolution or stabilization of subjective symptoms such as hearing loss or tinnitus, the spontaneous recovery or surgical closure of tympanic membrane perforations or, inopportunely, in absence of complaint upon initial evaluation. Follow up, for many studies, ended in as little as 3 months. From this review of the literature, one now knows that symptoms may have delayed onsets, severity is relative to the individual, and the occurrence of late sequelae can be deadly if not closely monitored.

While it has been proven that tympanic membrane perforations can occur without blast injury to any other organ, the presence of a perforation should indicate further exploration of other air-filled organs in the body as a precaution. It is possible to have middle ear damage, such as a perforated tympanic membrane or ossicular damage, without inner ear damage. Alternatively, it is possible to have inner ear damage such as hearing loss, tinnitus or vestibular symptoms without damage to the middle ear. This is

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because damage to the inner ear occurs at pressure levels above those which are needed to injure the middle ear (Patterson & Hamernik, 1997).

Individuals in combat settings or at high-risk for blast exposure should utilize ear and hearing protection. de Régloix et al. (2017) examined of both military personnel and civilians referred to two military training hospitals in France, between May 2002 and October 2014, for blast injuries to the ear. They found that those wearing hearing protection at the time of blast exposure did not have tympanic membrane perforations and had less severe hearing loss than those who did not wear any protection. While simple ear plugs are effective in reducing the incidence of tympanic membrane perforations, they can be impractical in combat situations where survival is dependent on discreet communication (Ritenour et al., 2008) and situational awareness becomes hindered by their linear attenuation. Further advancements in technology call for digital hearing protection that not only attenuates loud sounds from the auditory system and blast pressure waves from entering the ear canal but also does not disrupt communication and localization abilities vital for survival for combat soldiers in theater.

As many factors can influence the nature of behavioral responses during subjective testing, objective testing such as an ABR, ASSR or ARTs should be utilized to corroborate subjective findings (Reichenbach, 2016) or when obtaining standard audiometry is not feasible due to behavioral, physical or cognitive impairments.

While still being trialed, little evidence supports the use of steroids, vasodilators or vitamin supplements to aid in recovery of sensorineural hearing loss caused by blast exposure (Patterson & Hamernik, 1997).

Future Research

Unfortunately, the population needed to produce more research on the effect of blast exposure cannot be created or controlled for as violence and physical harm are prerequisites for such studies. Researchers must recruit significant populations of military personnel or civilians who have fallen victim to violent attacks in order to gather further data and advance understanding of how blast pressure waves affect the otologic system.

There is a demand for research that follows the full pathophysiological course of otologic blast injuries from initial insult through many years of follow up in furtherance of understanding their long term effects. There is a lack of research exposing these long term effects of blast exposure on the ear and auditory system. Reasons for this can include subject loss to follow up, medical professionals under-emphasizing the importance of regular evaluations to identify late sequelae and all around shortage of evidence of necessity of long term management of blast-exposed subjects. Stigma will ultimately play a role in many subject's decisions on long term management of persistent otologic symptoms such as hearing loss and tinnitus.

The studies examined in this review varied in sample size from 258 subjects (Cave et al., 2007) to 12 subjects (Walsh et al., 1995) with an average of approximately 93. Future research needs to utilize larger populations of subjects in order to allow their findings to be as generalizable as possible given the variability possessed by this type of data. Despite each blast having its own unique characteristics that play a major role in the manifestation of otologic symptoms, the more incidents that can be amalgamated, the closer the world gets to understanding the effect of blasts and leads to better identification, treatment and management.

For the majority of cases, the perception of tinnitus is subjective to the subject experiencing it. There is a demand for objective testing for tinnitus not only in those with blast exposure but in the general population. Tinnitus affects millions of people in the United States and around the globe. Recent epidemiologic studies reveal a prevalence in the range from 8% to 25.3% of the US population and similar surveys conducted in other countries produced comparable findings in the range of 4.6% to 30% of their populations (Bhatt, Harrison, Lin, & Bhattacharyya, 2014). Objective testing could potentially help identify the typically unknown etiology of this common phenomenon that leaves many medical professionals looking for answers. Blast-exposed individuals are frequently military personnel and tinnitus, being the number one service connected disability with the largest financial payout, is given service connection and compensation based solely on subjective complaint. Current research is examining the recording of neural activity to identify tinnitus in those who report severe symptomology (Dohrmann, Elbert, Schlee, & Weisz, 2007). For these reasons, reputable and evidenced based objective testing for tinnitus is becoming increasingly desirable.

Currently, there is a limited understanding of vestibular function following blast exposure. With reported vestibular symptoms ranging from 0% to 44% in this review alone, further research is needed to establish standardized test batteries for vestibular testing of subject presenting with or reporting vestibular symptoms. These assessments should be performed at any point from immediately to years after an incident of blast exposure on subjects with reported vestibular dysfunction. Research is being conducted regarding specific vestibular pathologies that could be linked to blast exposure at this time. Furthermore, intervention and management protocols are being examined (Tun et al., 2009). Due such limited research, little is known about the spontaneous recovery rates of vestibular symptomology (Cohen et al., 2002). Mills and Jones (2012) emphasized the need for "improved rehabilitative approaches" (p. 32) to resolve vestibular dysfunction caused by blast exposure. More effective rehabilitation can further improve long term quality of life.

To avoid confounding variables within the human population, animal models are being utilized by researchers such as Cho et al., (2013) and Newman et al. (2015) in order to examine the effects of simulated blast pressure waves, similar to those experienced by human subjects, on the otologic system. Cho et al.'s (2013) utilization of mice in a fabricated blast chamber found a 100% rate of tympanic membrane perforation and even more interestingly, a 100% spontaneous recovery rate of these perforations. Furthermore, they performed ABR testing and DPOAEs on all mice. They found threshold shifts that were later able to be correlated with blast intensities. Newman and his team have recently developed a low-cost blast generator to replicate

studies such as Cho et al.'s (2013) on animal models, overcoming fiscal and space limitations, thus paving the way for more opportunities to study the effects of blast exposure on the otologic system.

Future advances could include the delivery of crucial audiological and psychological counseling through tele-health outlets such clinician-driven, internet-delivered treatment for tinnitus outlined by Andersson (2015), or the development of technologically-driven hearing protection for our military personnel with fast-acting active noise-canceling hearing protection that provides the life-dependent balance between communication and situational awareness. Additionally, future research should include the advancements in pharmacological hearing loss prevention agents as Campbell et al. (2007) explores the use of d-methionine for prevention of noise- and drug-induced hearing loss. Lastly, ability to restore function to an already damaged auditory system is being closely examined as more research is being performed with avian, amphibian and aquatic animal models for their ability to regenerate hair cells.

Conclusion

Blasts can have a multitude of effects on the ear and auditory system. The most common manifestations are hearing loss, tinnitus and tympanic membrane rupture. While rare, vestibular symptoms, disruption in the ossicular chain and perilymph fistulas can occur. Assessment, management and treatment of these subjects should always take on a multidisciplinary approach (Chandler, 2006). Physicians and audiologists, alike, are urged to conduct thorough initial examinations and obtain detailed medical

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histories when assessing subjects exposed to blasts. There is limited data on the long term effects of blast exposure on the auditory system but research indicates that long term follow up is needed to prevent late sequelae, such as the development of cholesteatoma or other infections. Permanent hearing loss and tinnitus that goes unmanaged create psychological implications and social isolation which negatively impacts a subject's quality of life.

Unfortunately, we cannot predict the otologic damage caused by blasts because we cannot calculate the blast overpressures that will be produced in any given incident. Not only must one consider the complex wave that is created by the reflections and attenuation of pressure waves from the objects in the environment but also the augmentation of the subjects and their individual anatomical differences including medical histories (Garth, 1995). It is hard to quantify the vulnerability the human ear as, so far, we cannot eqaute overpressure levels to severity of damage. For example, a condition as simple and common as the overproduction of cerumen and the consequential "plug" impaction can create, has been found to have a protective effect against blast injuries (Chandler, 2006; Darley & Kellman, 2010). This small entity can completely alter the effect of the blast overpressures on a subject.

Because of all the factors influencing the intensity of the blast, the incidence and prevalence ranges were quite extensive. Despite the detailed accounts of the blast-exposed subjects and the environment in which the incident occurred, comparison between two or more events remains complicated and can yield vastly different otologic findings. More research is needed in order to better understand how the environment

and individual anatomical variances affect the severity of blast injuries to the ear and audiologic system. Researchers should continue to monitor subjects even after surgical correction and subjective improvement of symptoms as subject report is seldom correlated to true objective findings. Audiologists regularly encounter cases epitomizing this concept. Bruins and Cawood (1991) observed a poor correlation between subjective hearing loss and objective audiometric findings when many of their subjects stated they felt their hearing was normal despite previous acoustic trauma. Alternatively, many of their subjects reported subjective hearing loss in the absence of abnormal audiometric findings. This concept can be exemplified by examining the differences between Table 2 outlining subjective complaints of hearing loss and Table 3 which details the objective finding of abnormal audiograms after audiometric evaluation.

With the ever-changing geopolitical climate of our world and continued growth in the use of explosive devices for violence in warfare and against civilians, further research is needed to determine the most effective protocols for assessment, management, treatment and long term follow up of blast exposed subjects.

References

Abbas, S., Arshad, M., & Ghani, S. (2014). Tympanic membrane perforations secondary to blast trauma-an experience of 74 affected ears. *Isra Medical Journal*, *6*(4), 267-269.

Aldosari, B., & Thomassin, J. M. (2017). Audiological results of endoscopic surgical repair of the long process of incus. *World Journal of Otorhinolaryngology-Head and Neck Surgery*, *3*(3), 148-152.

Al-Juboori, A. N. (2014). Evaluation of spontaneous healing of traumatic tympanic membrane perforation. *General Medicine: Open Access*, 1-3.

Andersson, G. (2015). Clinician-supported internet-delivered psychological treatment of tinnitus. *American Journal of Audiology*, *24*(3), 299-301.

Andersson, G., Baguley, D. M., McKenna, L., & McFerran, D. (2005). Tinnitus: A multidisciplinary approach. London: Whurr.

Argyros, G. J. (1997). Management of primary blast injury. *Toxicology*, *121*(1), 105-115.

Aslıer, M., & Aslıer, N. G. Y. (2017). Analysis of otologic injuries due to blast trauma by handmade explosives. *Turkish Archives of Otorhinolaryngology*, *55*(2), 64-68.

Bhatt, J. M., Lin, H. W., & Bhattacharyya, N. (2016). Prevalence, severity, exposures, and treatment patterns of tinnitus in the United States. *JAMA Otolaryngology– Head & Neck Surgery*, *142*(10), 959-965.

Branica, S., Dawidowsky, K., Sprem, N., & McKinnon, B. (2008). Otologic blast trauma: experience from Croatian War. *Explosion and Blast-Related Injuries*, 353-369.

Breeze, J., Cooper, H., Pearson, C., Henney, S., & Reid, A. (2011). Ear injuries sustained by British service personnel subjected to blast trauma. *Journal of Laryngology & Otology*, *125*(1), 13-17.

Bruins, W.R., & Cawood, R.H. (1991). Blast injuries of the ear as a result of the Peterborough lorry explosion: 22 March 1989. *J Laryngol Otol, 105*, 890-895.

Campbell, K. C., Meech, R. P., Klemens, J. J., Gerberi, M. T., Dyrstad, S. S., Larsen, D. L., ... & Hughes, L. F. (2007). Prevention of noise-and drug-induced hearing loss with D-methionine. *Hearing research*, *226*(1-2), 92-103.

Cave, K. M., Cornish, E. M., & Chandler, D. W. (2007). Blast injury of the ear: clinical update from the global war on terror. *Military Medicine*, *172*(7), 726-730.

Chait, R.H., & Zajtchuk, J.T. (1989). Treatment of blast injury to the ear. *Ann Otol Rhinol Laryngol Suppl, 140*, 13–16.

Chait, R.H., Casler, J., & Zajtchuk, J.T. (1989). Blast injury of the ear: historical perspective. *Ann Otol Rhinol Laryngol, 98*, 9-12.

Chandler, D. (2006). Blast-related ear injury in current U.S. military operations: role of audiology on the interdisciplinary team. *ASHA Leader*, *11*(9), 8-29.

Cho, S., Gao, S. S., Xia, A., Wang, R., Salles, F. T., Raphael, P. D., & ... Oghalai, J. S. (2013). Mechanisms of hearing loss after blast injury to the ear. *Plos One*, *8*(7), e67618.

Choi, C. H. (2012). Mechanisms and treatment of blast induced hearing loss. *Korean Journal of Audiology*, *16*(3), 103-107.

Cohen, J. T., Ziv, G., Bloom, J., Zikk, D., Rapoport, Y., & Himmelfarb, M. Z. (2002). Blast injury of the ear in a confined space explosion: auditory and vestibular evaluation. *Imagj-Ramat Gan*, *4*(7), 559-562.

Darley, D. S., & Kellman, R. M. (2010). Otologic considerations of blast injury. *Disaster Medicine and Public Health Preparedness*, *4*(2), 145-152.

de Régloix Ballivet, S., Crambert, A., Maurin, O., Lisan, Q., Marty, S., & Pons, Y. (2017). Blast injury of the ear by massive explosion: a review of 41 cases. *Journal of the Royal Army Medical Corps*, *163*(5), 333-338.

Dohrmann, K., Elbert, T., Schlee, W., & Weisz, N. (2007). Tuning the tinnitus percept by modification of synchronous brain activity. *Restorative Neurology and Neuroscience, 25*(3-4), 371-378.

Dougherty, A. L., MacGregor, A. J., Han, P. P., Viirre, E., Heltemes, K. J., & Galarneau, M. R. (2013). Blast-related ear injuries among U.S. military personnel. *Journal of Rehabilitation Research & Development*, *50*(6), 893-904.

Erlandsson, S., & Tyler, R. S. (2000). Psychological profiles of tinnitus subjects. *Tinnitus Handbook*, 25-27.

Fagelson, M. A. (2007). The association between tinnitus and posttraumatic stress disorder. *American Journal of Audiology*, *16*(2), 107-117.

Fausti, S. A., Wilmington, D. J., Gallun, F. J., Myers, P. J., & Henry, J. A. (2009). Auditory and vestibular dysfunction associated with blast-related traumatic brain injury. *Journal of Rehabilitation Research & Development*, *46*(6), 797-809.

Fisher, T., (2008). Blast injury of the ear. *Synopsis of causation, Ministry of Defense, Edinburgh*.

Folmer, R. L., McMillan, G. P., Austin, D. F., & Henry, J. A. (2011). Audiometric thresholds and prevalence of tinnitus among male veterans in the United States:

Data from the National Health and Nutrition Examination Survey, 1999-

2006. *Journal of Rehabilitation Research & Development*, *48*(5), 503-515.

Garth, R. J. N. (1995). Blast injury of the ear: an overview and guide to

management. *Injury*, *26*(6), 363-366.

Green, J.O. (1872) Cases of injury to the ear from external violence. *Transactions of

The American Otological Society*, 5, 88-98.

Griffin, W. L. (1979). A retrospective study of traumatic tympanic membrane

perforations in a clinical practice. *The Laryngoscope*, *89*(2), 261-282.

Helling, E. R. (2004). Otologic blast injuries due to the Kenya embassy

bombing. *Military Medicine*, *169*(11), 872-876.

Hirsch, F.G. (1968). Effects of overpressure on the ear: a review. *Ann NY Acad Sci,*

152(1), 147-162.

Jagade, M. V., Patil, R. A., Suhail, I. S., Kelkar, P., Nemane, S., Mahendru, J., ... &

Kewle, P. (2008). Bomb blast injury: effect on middle and inner ear. *Indian*

Journal of Otolaryngology and Head & Neck Surgery, *60*(4), 324-330.

Jalilvand, H., Pourbakht, A., & Haghani, H. (2015). Hearing aid or tinnitus masker: which one is the best treatment for blast-induced tinnitus? The results of a long-term study on 974 patients. *Audiology and Neurotology, 20*(3), 195-201.

Joseph, A. R., Horton, J. L., Clouser, M. C., MacGregor, A. J., Louie, M., & Galarneau, M. R. (2016). Development of a comprehensive Blast-Related Auditory Injury Database (BRAID). *Journal of Rehabilitation Research & Development, 53*(3), 295-306.

Joseph, A. R., Shaw, J. L., Clouser, M. C., MacGregor, A. J., & Galarneau, M. R. (2017). Impact of Blast Injury on Hearing in a Screened Male Military Population. *American Journal of Epidemiology. 187*(1), 7-15.

Katz, E., Ofek, B., Adler, J., Abramowitz, H. B., & Krausz, M. M. (1989). Primary blast injury after a bomb explosion in a civilian bus. *Annals of Surgery, 209*(4), 484-488.

Keller, M., Sload, R., Wilson, J., Greene, H., Han, P., & Wise, S. (2017). Tympanoplasty following blast injury. *Otolaryngology–Head and Neck Surgery, 157*(6), 1025-1033.

Kerr, A.G. & Byrne, J.E.T. (1975). Concussive effects of bomb blast on the ear. *Journal of Laryngology and Otology, 89*, 131-143.

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Killion, M. C., Monroe, T., & Drambarean, V. (2011). Better protection from blasts without sacrificing situational awareness. *International Journal of Audiology*, *50*, 38-45

Kronenberg, J., Ben-Shoshan, J., Modan, M., & Leventon, G. (1988). Blast injury and cholesteatoma. *Otology & Neurotology*, *9*(2), 127-130.

Kronenberg, J., Ben-Shoshan, J., & Wolf, M. (1993). Perforated tympanic membranes after blast injury. *Am J Otol, 14*(1), 92-94.

Kubli, L. R., Brungart, D., & Northern, J. (2017). Effect of Blast Injury on Auditory Localization in Military Service Members. *Ear and Hearing*.

McNeill, C., Tavora-Vieira, D., Alnafjan, F., Searchfield, G. D., & Welch, D. (2012). Tinnitus pitch, masking, and the effectiveness of hearing aids for tinnitus therapy. *International Journal of Audiology*, *51*(12), 914-919.

Mehta, R. P., Rosowski, J. J., Voss, S. E., O'Neil, E., & Merchant, S. N. (2006). Determinants of hearing loss in perforations of the tympanic membrane. *Otology & Neurotology, 27*(2), 136-143.

Melinek, M., Naggan, L. and Altman, M. (1976) Acute acoustic trauma, a clinical investigation and prognosis in 433 symptomatic soldiers. *Isr. J. Med. Sci. 12*, 562-569.

Miller, I.S.M., McGahey, D., & Law, K. (2002). The otologic consequences of the Omagh bomb disaster. *Otolaryngol Head Neck Surg, 126*(2), 127-128

Mills, K. N., & Jones, S. M. (2012). Current perspectives on blast injury and inner ear function. *SIG 6 Perspectives on Hearing and Hearing Disorders: Research and Diagnostics*, *16*(1), 26-34.

Mrena, R., Paakkonen, R., Back, L., Pirvola, U., & Ylikoski, J. (2004). Otologic consequences of blast exposure: A Finnish case study of a shopping mall bomb explosion. *Acta Otolaryngol, 124(8),* 946–952.

Nageris, B.I., Attias, J., & Shemesh, R. (2008). Otologic and audiologic lesions due to blast injury. *J Basic Clin Physiol Pharmacol., 19*(3-4), 185-191.

Nawaz, G., Ulhaq, N., & Khan, A. R. (2014). Bomb blast injuries to the ear: The Peshawar Experience. *Cell*, *22(4)*, 193-196.

Newman, A. J., Hayes, S. H., Rao, A. S., Allman, B. L., Manohar, S., Ding, D., & ... Salvi, R. (2015). Low-cost blast wave generator for studies of hearing loss and brain injury: blast wave effects in closed spaces. *Journal of Neuroscience Methods*, *24,* 282-292.

Ning, Y. L., & Zhou, Y. G. (2015). Shock tubes and blast injury modeling. *Chinese Journal of Traumatology*, *18*(4), 187-193.

Okpala, N. (2011). Management of blast ear injuries in mass casualty environments. *Military Medicine*, *176*(11), 1306-1310.

Oleksiak, M., Smith, B. M., Andre, J. S., Caughlan, C. M., & Steiner, M. (2012). Audiological issues and hearing loss among veterans with mild traumatic brain injury. *Journal of Rehabilitation Research & Development*, *49*(7), 995-1003.

Pahor, A.L. (1981). The ENT problems following the Birmingham bombings. *J Laryngol Otol, 95*(4), 399-406.

Patterson, J.H., & Hamernik, R.P. (1997). Blast overpressure induced structural and functional changes in the auditory system. *Toxicology, 121*, 29-40.

Patow, C. A., Bartels, J., & Dodd, K. T. (1994). Tympanic membrane perforation in survivors of a SCUD missile explosion. *Otolaryngology and Head & Neck Surgery*, *110*(2), 211-221.

Perez, R., Gatt, N., & Cohen, D. (2000). Audiometric configurations following exposure to explosions. *Archives of Otolaryngology and Head & Neck Surgery*, *126*(10), 1249-1252.

Perez, R., Gatt, N., & Cohen, D. (2007). Long-term follow-up of sensorineural hearing loss in subjects exposed to explosions. *Mediterr J Otol, 3*, 1-5.

Perlman, H. B. (1941). Acoustic trauma in man: Clinical and experimental

studies. *Archives of Otolaryngology*, *34*(3), 429-452.

Persaud, R., Hajioff, D., Wareing, M., & Chevretton, E. (2003). Otological trauma

resulting from the SoHo nail bomb in London, April 1999. *Clin Otolaryngol, 28*,

203-206.

Pusz, M. D., & Robitschek, J. (2017). Traumatic hearing loss in the context of blast-

related tympanic membrane perforation. *Military Medicine*, *182*(1), 1645-1648.

Qureshi, T. A., Awan, M. S., Hassan, N. H., Aftab, A. H., & Ali, S. A. (2017). Effects of

bomb blast injury on the ears: The Aga Khan University Hospital experience.

Journal of the Pakistan Medical Association, *67*(9), 1313-1317.

Raju, G. (2015). Disability evaluation in acoustic blast trauma. *Indian Journal of

Occupational and Environmental Medicine*, *19*(3), 138-140.

Reichenbach, T. (2016). Hearing damage through blast. *Blast Injury Science and

Engineering: A Guide for Clinicians and Researchers*, 307-314.

Remenschneider, A.K., Lookabaugh, S., Aliphas, A., Brodsky, J.R., Devaiah, A.K.,

Dagher, W., Grundfast, K.M., Heman-Ackah, S.E., Rubin, S., Sillman, J., Tsai,

A.C., Vecchiotti, M., Kujawa, S.G., Lee, D.J., Quesnel, A.M. (2014). Otologic

outcomes after blast injury: The Boston Marathon experience. *Otol Neurotol, 35*(10), 1825–1834.

Ritenour, A. E., Wickley, A., Ritenour, J. S., Kriete, B. R., Blackbourne, L. H., Holcomb, J. B., & Wade, C. E. (2008). Tympanic membrane perforation and hearing loss from blast overpressure in Operation Enduring Freedom and Operation Iraqi Freedom wounded. *Journal of Trauma and Acute Care Surgery*, *64*(2), 174-178.

Seaman, R., & Newell, R. (1971). Another etiology of middle ear cholesteatoma. *Archives of Otolaryngology, 94*(5), 440-442.

Searchfield, G. D. (2015). Hearing aids for tinnitus. *Tinnitus: Clinical and Research Perspectives*, 197-212.

Searchfield, G. D., Kaur, M., & Martin, W. H. (2010). Hearing aids as an adjunct to counseling: Tinnitus patients who choose amplification do better than those that don't. *International Journal of Audiology*, *49*(8), 574-579.

Shah, A., Ayala, M., Capra, G., Fox, D., & Hoffer, M. (2014). Otologic assessment of blast and non-blast injury in returning Middle East-deployed service members. *The Laryngoscope*, *124*(1), 272-277.

Singh, D., & Ahluwalia, K. J. S. (1968). Blast injuries of the ear. *The Journal of Laryngology & Otology*, *82*(11), 1017-1028.

Song, S. A., Sridhara, S. K., & Littlefield, P. D. (2017). Tympanoplasty outcomes for blast-induced perforations from Iraq and Afghanistan: 2007-2012. *Otolaryngology–Head and Neck Surgery*, *156*(2), 353-359.

Sood, A. S., Pal, P., & Kumar, A. (2018). Tympanic membrane perforation: correlation of hearing loss with its site and size. *International Journal of Otorhinolaryngology and Head and Neck Surgery*, *4*(2), 1-6.

Sprem, N., Branica, S., & Dawidowsky, K. (2001). Tympanoplasty after war blast lesions of the eardrum: retrospective study. *Croat Med J*, *42*, 642-645.

Sridhara, S. K., Rivera, A., & Littlefield, P. (2013). Tympanoplasty for blast-induced perforations: the Walter Reed experience. *Otolaryngology--Head and Neck Surgery*, *148*(1), 103-107.

Teter, D. L., Newell, R. C., Colonel, L. T., & Aspinalln, K. B. (1970). Audiometric configurations associated with blast trauma. *The Laryngoscope*, *80*(7), 1122-1132.

Tun, C., Hogan, A., & Fitzharris, K. (2009). Hearing and vestibular dysfunction caused by blast injuries and traumatic brain injuries. *Hearing Journal*, *62*(11), 24-26.

Tungsinmunkong, S., Chongkolwatana, C., Piyawongvisal, W., Atipas, S., & Namchareonchaisuk, S. (2007). Blast injury of the ears: the experience from Yala Hospital, Southern Thailand. *Medical Journal of the Medical Association of Thailand*, *90*(12), 2662.

Tunkel, D. E., Bauer, C. A., Sun, G. H., Rosenfeld, R. M., Chandrasekhar, S. S., Cunningham, E. R., ... & Henry, J. A. (2014). Clinical practice guideline: Tinnitus. *Otolaryngology-Head and Neck Surgery*, *151*(2), 1-40.

Van Campen, L.E., Dennis, J.M., Hanlin, R.C., King, S.B., & Velderman, A.M. (1999). One-year audiologic monitoring of individuals exposed to the 1995 Oklahoma City bombing. *J Am Acad Audiol, 10*(5), 231–247.

Voss, S. E., Rosowski, J.J., Merchant, S.N., Thornton, A,R., Shera, C.A., Peake, W.T. (2000). Middle-ear pathology can affect the ear-canal sound pressure generated by audiologic earphones. *Ear Hear, 21*, 265–274.

Walsh, R. M., Pracy, J. P., Huggon, A. M., & Gleeson, M. J. (1995). Bomb blast injuries to the ear: The London Bridge incident series. *Emergency Medicine Journal*, *12*(3), 194-198.

Wolf, M., Kronenberg, J., Ben-Shoshan, J., & Roth, Y. (1991). Blast injury of the ear. *Military Medicine, 156*, 651-653.

Wolf, M., Megirov, L., & Kronenberg, J. (1999). Multifocal cholesteatoma of the external auditory canal following blast injury. *Annals of Otology, Rhinology & Laryngology*, *108*(3), 269-270.

Yetiser, S., & Ustun, T. (1993). Concussive blast-type aural trauma, eardrum perforations, and their effects on hearing levels: an update on military experience in Izmir, Turkey. *Military Medicine*, *158*(12), 803-806.

Ziv, M., Philipsohn, N. C., Leventon, G., & Man, A. (1973). Blast injury of the ear: treatment and evaluation. *Military Medicine*, *138*(12), 811-813.