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About this Publication

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Executive Summary

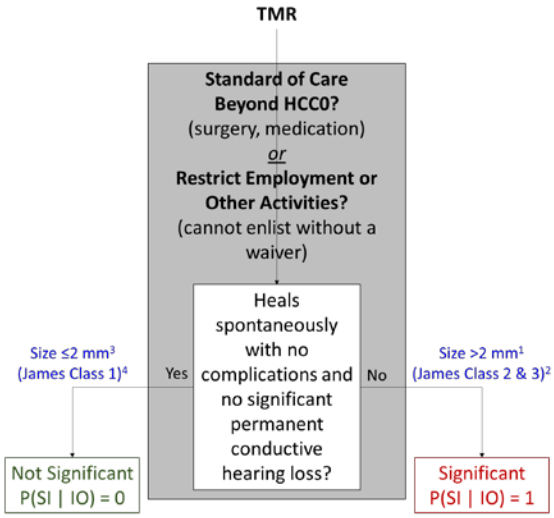
Flashbang grenades employ loud sounds that are intended to temporarily suppress the targeted personnel. The Department of Defense (DoD) uses flashbang grenades in many military missions in which military operators seek to avoid fatalities and permanent injuries (JNLWD 2012, 5, 8). However, there is a risk that flashbang grenades can cause a tympanic membrane rupture (TMR), otherwise known as a burst eardrum.

Flashbang grenades are Non-Lethal Weapons (NLWs). Like all NLWs, they are intended to have reversible effects on personnel and materiel. As part of the DoD development acquisition process, combat developers must compare the capabilities of a novel NLW system to requirements. One particularly important requirement is the NLW system's Risk of [Causing] Significant Injury (RSI), an estimate of the reversibility of the system's effect on the targeted personnel. Per DoD Instruction (DoDI) 3200.19 (DoD 2012), combat developers must quantify a NLW system's RSI, demonstrating that the system's RSI is less than or equal to a numerical threshold. DoDI 3200.19 defines a significant injury as one for which "self-aid, buddy-aid, and combat lifesaver skills" (DoD 2012, 13) are insufficient to treat the injury and/or if the injury results in death or "physical damage that restricts the employment or other activities of the person for the rest of his or her life" (DoD 2012, 14). "Self-aid, buddy-aid, and combat lifesaver skills" are considered to be medical treatment with a Health Care Capability (HCC) index of 0 (HCC0) (DoD 2012, 13).

The purpose of this project was to search the relevant literature to identify attributes of a TMR that can quantitatively, accurately, and precisely predict the significance of the TMR, per the definitions set forth in DoDI 3200.19 (DoD 2012) and consider how these predictive attributes can be estimated during the development acquisition phase of a novel flashbang system. The results of our analysis are illustrated in Figure ES- 1 and are summarized in the following findings and recommendations.

Findings

- Shelley and Chan (2015) created a computational model that estimates the probability that a blast, such as a flashbang detonation, causes a TMR in the three clinical classifications used by James et al. (1982).
- James et al. (1982) classified blast-induced TMRs using subjective criteria, as opposed to quantitative size measurements:
  - James Class 2 and 3 TMRs are described as "large tears, or multiple small holes or tears" and "total disrupture of the drum…," respectively (James et al. 1982, 10).
  - In contrast, James Class 1 TMRs are described as "minor slits, and linear disruption of the drum fibres producing a 'meshlike' effect" (James et al. 1982, 10).



[1] A notable percentage of blast-induced TMRs >2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we err on the side of caution and approximate that TMRs this size caused by flashbang grenades require surgery and are therefore significant, i.e., P(SI | IO) = 1.

[2] The blast-induced TMRs in James Class 2 and 3 are described as "large tears, or multiple small holes or tears" and "total disrupture of the drum…", respectively (James et al. 1982, 10). Therefore, we approximate that James Class 2 and 3 TMRs are >2 mm long.

[3] Zero or almost zero blast-induced TMRs ≤2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we approximate that TMRs this size caused by flashbang grenades do not require surgery to repair the TMR. Furthermore, we approximate a zero likelihood that a spontaneously healed TMR will lead to complications requiring medical treatment beyond HCC0, such as otitis media and cholesteatoma, since this likelihood over all TMRs is already very low at 8% and 7.6%, respectively (Lou, Lou, and Zhang 2012; Kronenberg et al. 1988). Finally, we approximate a zero likelihood that a spontaneously healed TMR will lead to a permanent conductive hearing loss ≥25 dB HL, the bar for significance we found in a previous analysis (King and Cazares 2005), since once a TMR has healed, the conductive hearing loss resolves fully or to a very low level (<10 dB HL) (Taylor and Mueller 2011, 117). Therefore, we approximate these TMRs as not significant, i.e., P(SI | IO) = 0.

[4] The blast-induced TMRs in James Class 1 are described as "minor slits, and linear disruption of the drum fibres producing a 'meshlike' effect" (James et al. 1982, 10). Therefore, we approximate that James Class 1 TMRs are ≤2 mm long.

Figure ES- 1. Classifying a TMR as Significant Based on Its Size

- Other researchers graded blast-induced TMRs using quantitative size measurements and showed that the size of a TMR indicates the likelihood that the TMR will require surgery beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008):
  - A notable percentage of blast-induced TMRs >2 mm long required surgery.
  - In contrast, zero (or almost zero) blast-induced TMRs ≤2 mm long required surgery.
- TMR can lead to complications such as otitis media (infection of the middle ear) or cholesteatoma (torn pieces of the TMR "seeding" in the middle ear) (Kronenberg et al. 1998; Lou, Lou, and Zhang 2012).
  - Severe otitis media requires prescription antibiotics, beyond HCC0 (Waseem 2016).
  - Severe cholesteatoma requires surgery, also beyond HCC0 (Ear Surgery Information Center 2016).

  - Otitis media and cholesteatoma are associated with 8% and 7.6%, respectively, of *all* TMR cases, averaged over both large and small TMRs (Kronenberg et al. 1988; Lou, Lou, and Zhang 2012).

- TMR can lead to conductive hearing loss (Remenschneider et al. 2014; Taylor and Mueller 2011).
- Once a TMR has healed, the conductive hearing loss resolves fully or to a very low level (<10 dB HL) (Taylor and Mueller 2011, 117).
- In a previous analysis, we investigated the significance of permanent hearing loss. We found that a permanent hearing loss ≥25 dB HL was significant, while a permanent hearing loss <25 dB HL was not significant (King and Cazares 2015).
- The literature often uses the term "Permanent Threshold Shift (PTS)" to refer to permanent sensorineural hearing loss due to inner ear injury (Cave, Cornish, and Chandler 2007, 726; Rutka 2003, 3; SCENIHR, 2008, 26), rather than the permanent conductive hearing loss due to middle ear injury such as TMR.
- Permanent conductive hearing loss resulting from blast-induced TMR can be confounded by blast-induced permanent sensorineural hearing loss called "PTS" (Dougherty et al. 2013; Remenschneider et al. 2014). The blast that produced the TMR could have also produced the PTS (Cho et al. 2013).
- A single RSI metric for a flashbang grenade cannot be calculated by simply adding together RSI_{TMR} and RSI_{PTS}, since doing so would double-count the probability that the flashbang grenade caused both TMR and PTS.

Recommendations

Based on our findings, we recommend that:

- Flashbang-induced TMRs should be classified as significant vs. not significant based on the same subjective, size-based, clinical classification scheme used by James et al. (1982). Our rationale is as follows:
  - We approximate that James Class 1 TMRs are ≤2 mm long while James Class 2 and 3 TMRs are >2 mm long. Although the James clinical classifications are subjective and qualitative, we believe they can be mapped to these objective, quantitative size criteria.
  - We approximate that flashbang-induced TMRs >2 mm long (equivalent to James Class 2 and 3) require surgery, beyond HCC0, and are therefore significant. This approximation is based on our finding that a notable percentage of these TMRs require surgery (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008).

- In contrast, we approximate that flashbang-induced TMRs ≤2 mm long (equivalent to James Class 1) are not significant, since
    - We approximate that these small TMRs do not require surgery and can heal spontaneously (HCC0). This approximation is based on our finding that zero or almost zero TMRs ≤2 mm long require surgery to repair the TMR (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008).
        - We approximate that these small TMRs do not lead to cases of otitis media or cholesteatoma that are severe enough to require prescription antibiotics or surgery. That is, we approximate a zero likelihood that a small, spontaneously-healed TMR will lead to otitis media or cholesteatoma, since we found that this likelihood over *all* TMRs, averaged over large and small TMRs, is already very low at 8% and 7.6%, respectively (Kronenberg et al. 1998; Lou, Lou, and Zhang 2012).
            - We approximate that these small TMRs do not lead to a significant permanent conductive hearing loss (≥25 dB HL). This approximation is based on our finding that, once the TMR has healed, hearing loss resolves fully or to a very low level (<10 dB HL) (Taylor and Mueller 2001), below our bar for significance.

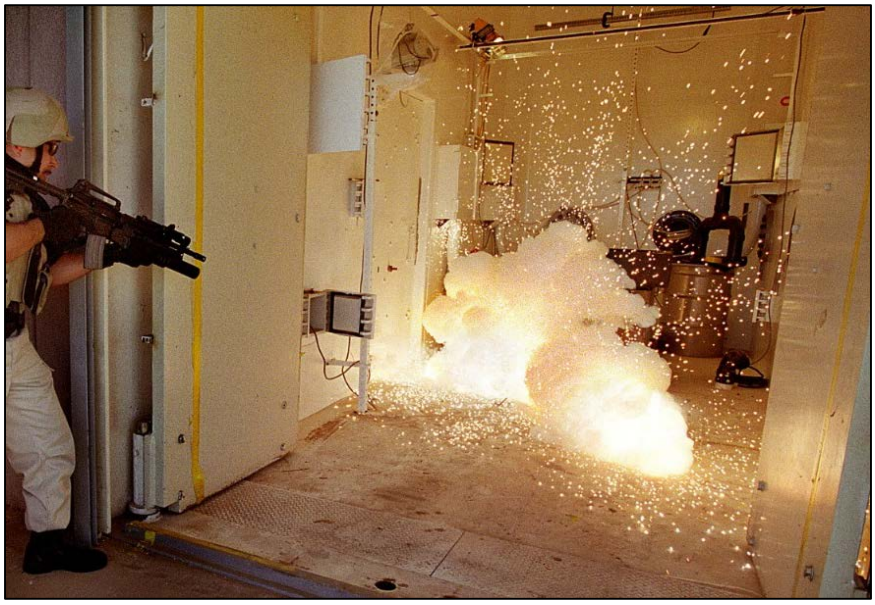
- Based on our recommendations listed above, we conclude that RSI_{TMR} for a flashbang grenade should be estimated as the probability that the flashbang grenade causes a TMR in James Class 2 or 3.
- The Shelley and Chan (2015) computational model should be coded such that it outputs the probability that a blast causes a TMR in James Class 2 or 3, rather than a TMR in James Class 1, 2, or 3, so that it can directly output RSI_{TMR}.
- Combat developers should simultaneously use multiple RSI requirements for a flashbang grenade, one for each potential injury (i.e., one requirement for RSI_{TMR}, another requirement for RSI_{PTS}, and so forth). The flashbang grenade would then have to meet all RSI requirements simultaneously. Each RSI requirement could have a different threshold value.

Contents

1. Introduction

A. Non-Lethal Weapons (NLWs)

The Department of Defense (DoD) defines *Non-Lethal Weapons (NLWs)* as "weapons, devices, and munitions that are explicitly designed and primarily employed to incapacitate targeted personnel or material immediately, while minimizing fatalities, permanent injuries to personnel, and undesired damage to property in the target area or environment" (DoD 2013, 12). Furthermore, "NLWs are intended to have reversible effects on personnel and materiel" (DoD 2013, 12). Some counter-personnel NLWs employ loud sounds that are intended to temporarily suppress the targeted personnel. One example of a sound-based NLW is a flashbang grenade (JNLWD 2012, 5, 8), the focus of this document. Figure 1 shows a photograph of a flashbang detonation.



Source: Singer (2002).

Figure 1. Photograph of a Flashbang Detonation

B. Risk of Significant Injury (RSI)

As part of the DoD technology development acquisition process, combat developers must compare the capabilities of novel NLW systems to requirements, such as the key performance parameters (KPPs) and key system attributes (KSAs) written into capability documents (DoD 2015, B-24). One particularly important KPP/KSA for a counter-personnel NLW system is the system's *Risk of [Causing] Significant Injury (RSI)*, an estimate of the reversibility of the system's

effect on the targeted personnel (DoD 2012, 14). During the development acquisition phase of a novel NLW system, combat developers must quantify the system's RSI (DoD 2012), ideally demonstrating that the system's RSI is less than or equal to a numerical threshold.

DoD Instruction (DoDI) 3200.19 defines a *significant injury* as death, a permanent injury, or an injury requiring medical treatment with a *Health Care Capability (HCC)* index of 1 or higher (HCC1+) (DoD 2012). Furthermore,

- Permanent injury is defined as "physical damage to a person that permanently impairs physiological function and restricts the employment or other activities of that person for the rest of his or her life" (DoD 2012, 14).
- Medical treatment with an HCC index of 1 (HCC1) is defined as "first responder-capability including resuscitation, stabilization, and emergency care" (DoD 2012, 13), while medical treatment with an HCC index of 2 (HCC2) is defined as "forward resuscitative and theater hospitalization capabilities including advanced emergency, surgical, and ancillary services" (DoD 2012, 13).
- In contrast, medical treatment with an HCC index of 0 (HCC0), below the bar for significance, is defined as "limited first-responder capability including self-aid, buddy-aid, and combat lifesaver skills" (DoD 2012, 13).

To summarize, DoD considers an injury to be significant if "self-aid, buddy-aid, and combat lifesaver skills" (DoD 2012, 13) are insufficient to treat the injury and/or if the injury results in death or "physical damage … that… restricts the employment or other activities of the person for the rest of his or her life" (DoD 2012, 14).

A NLW system's RSI is often estimated using a multi-step process (Burgei et al. 2014). First, one estimates *P(injury occurred)*, the probability that a particular injury will occur as a result of the intended use of the NLW system. Then, one estimates *P(injury is significant | injury occurred)*, the probability that the injury is significant, given that it has occurred. Finally, one estimates RSI_{injury} as the product of these two quantities:

$$RSI_{injury} = P(\text{injury occurred}) \times P(\text{injury is significant} \mid \text{injury occurred}),$$

where the *injury* can be any particular injury in question, such as a broken bone, a laceration, and so forth. This project focuses on the second RSI quantity, *P(injury is significant | injury occurred)*. Previous work has estimated this second quantity via a detailed search of the academic and medical literature to identify which physical attributes of a given injury can predict the injury's significance (Hirsch et al. 2015; King and Cazares 2015; Cazares et al. 2016).

It may be possible to bin a given injury into different types, describing each injury type via one or more physical attributes. It may then be possible to approximate $P(injury_{type}\ is\ significant \mid injury_{type}\ occurred)$ for each injury type as either 0 or 1. Approximating this metric as 0 indicates that one cannot consider the given injury type as significant, since the literature suggests that the

injury type does not require HCC1+ medical treatment and does not result in permanent injury that restricts employment or other activities for the rest of one's life. In contrast, approximating this metric as 1 indicates that one can indeed consider the given injury type as significant since the literature indicates that it requires HCC1+ medical treatment and/or results in permanent injury. This process provides a straightforward method for estimating the full RSI_{injury} metric since one must then only estimate that $P(injury_{type}\ occurred)$ for those injury types that are considered significant, effectively disregarding those injury types that are not considered significant.

For example, consider the case in which an injury is binned into two types: one injury type is characterized by a physical attribute that is less than a threshold T, while the other injury type is characterized by that same attribute greater than or equal to T. The two injury types are mutually exclusive and collectively exhaustive. Furthermore, the first injury type is considered to be not significant, while the second injury type is considered to be significant. Then RSI_{injury} can be calculated as follows:

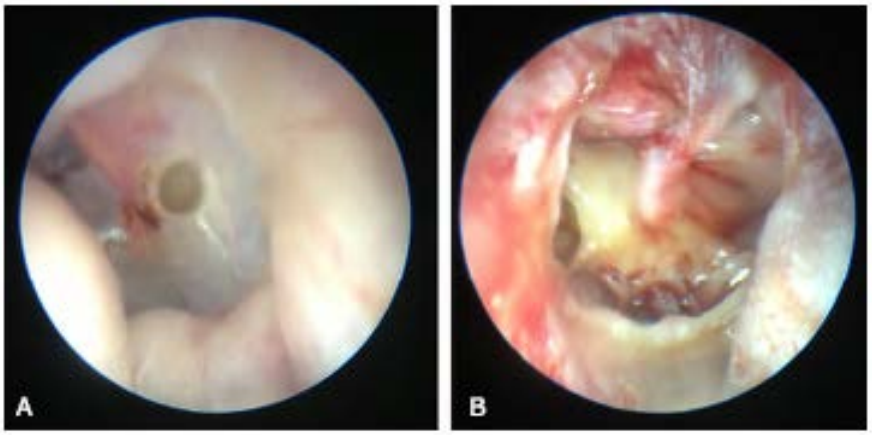
$$\begin{aligned} RSI_{Injury} &= [P(Injury_{attribute<T}\ occurred) \times P(Injury_{attribute<T}\ is\ significant \mid Injury_{attribute<T}\ occurred)] \\ &\quad + [P(Injury_{attribute\geq T}\ occurred) \times P(Injury_{attribute\geq T}\ is\ significant \mid Injury_{attribute\geq T}\ occurred)] \\ &= [P(Injury_{attribute<T}\ occurred) \times 0] + [P(Injury_{attribute\geq T}\ occurred) \times 1] \\ &= P(Injury_{attribute\geq T}\ occurred). \end{aligned}$$

C. Permanent Threshold Shift (PTS)

There is a risk that flashbang grenades can lead to an injury called *Permanent Threshold Shift (PTS)*, an irreversible hearing loss caused by exposure to intense sound (as opposed to the reversible *Temporary Threshold Shift (TTS)* that also results from intense sound exposure) (Farlex Partner Medical Dictionary 2012c). Such injury falls under codes H90.3, H90.41, H90.92, and H90.5 of the International Classification of Diseases, 10th edition, Clinical Modification (ICD-10-CM) (WHO 2016a, 380). Computational models such as Auditory 3.0 (now updated to Auditory 4.0) have been developed and validated to estimate $P(PTS_{\geq threshold}\ occurred)$, the probability that an intense impulse sound, such as those emanating from flashbang grenades, will cause a PTS greater than a user-selected hearing loss (HL) threshold level (Chan and Ho 2008; Comeaux, Dayton, and Parker 2010; Chan, Ho, and Ryan 2016). Auditory 4.0 can be used to estimate a flashbang grenade's RSI, without the need for animal or human experimentation. To use the model for this purpose, though, combat developers need guidance on what hearing loss threshold level to select when estimating a flashbang grenade's RSI_{PTS} (i.e., what threshold level can bin the PTS into significant vs. not significant types). Therefore, in the first phase of this project, the Institute for Defense Analyses (IDA) performed a search of the relevant literature to determine what hearing loss threshold level should be selected when estimating a flashbang grenade's RSI_{PTS} (King and Cazares 2015). The threshold level of 25 dB HL was selected.

D. Tympanic Membrane Rupture (TMR)

Flashbang grenades could also lead to *tympanic membrane rupture (TMR)* or *tympanic membrane perforation*, a disruption of the epithelium that separates the ear canal from the middle ear (Farlex Partner Medical Dictionary 2012d, 2012f). For this project, we use the term *TMR* to refer to both tympanic membrane rupture and tympanic membrane perforation. These injuries fall under the ICD-10-CM codes H72.00–H72.93 (WHO 2016a, 373–374). The tympanic membrane is colloquially referred to as the *eardrum* (Farlex Partner Medical Dictionary 2012a). Figure 2 shows photographs of two TMRs. The photograph on the left (A) shows a small TMR covering <25% of the eardrum area. In contrast, the photograph on the right (B) shows a large TMR covering >50% of the eardrum area, with associated secondary burns to the external ear canal.



Source: Remenschneider (2014, 1828, Figure 1).

Figure 2. Photographs of (A) a Small TMR and (B) a Large TMR

A computational model was recently developed to estimate $P(TMR_{type}\ occurred)$, the probability that an intense sound emanating from a flashbang grenade will cause a mild, moderate, or major TMR, where the terms "mild," "moderate," and "major" are based on a subjective, qualitative determination of the size of the TMR (Shelley and Chan 2015). Plans are in place for validating this model with experimental data (Shelley and Chan 2015).

However, no analysis has yet been done within the NLW community to estimate the second quantity of the RSI equation, $P(TMR_{type}\ is\ significant \mid TMR_{type}\ occurred)$, the probability that a mild, moderate, or major TMR is significant, given that it has occurred. Furthermore, no analysis has yet been done to confirm that the size of the TMR is the appropriate attribute for approximating $P(TMR_{type}\ is\ significant \mid TMR_{type}\ occurred)$ as either 0 or 1. Such analyses are needed to estimate a flashbang grenade's RSI_{TMR} in a cost-effective manner.

E. Objective

The purpose of this phase of the project was to search the relevant literature to

- Identify physical attributes of a TMR that can quantitatively, accurately, and precisely approximate the significance of a TMR type as either 0 or 1, per the definitions set forth in DoDI 3200.19, and
- Consider how those predictive attributes can be estimated during the development acquisition phase for a novel flashbang system.

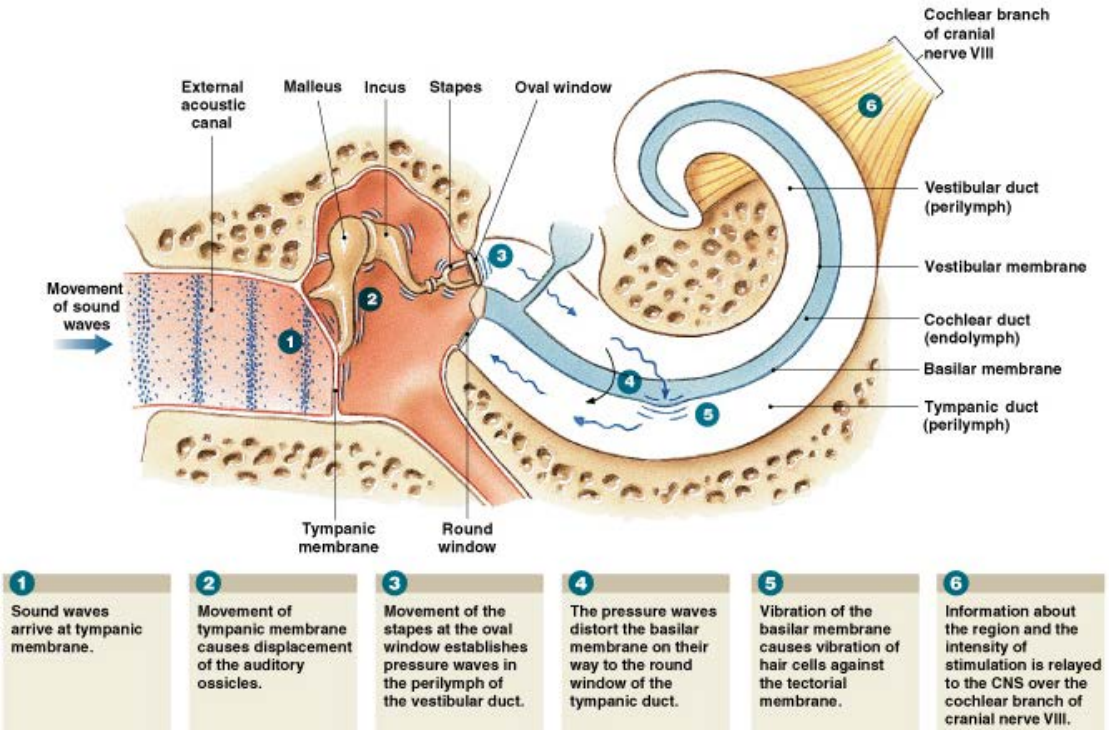
F. Overview

In this document, we first review the anatomy of the ear and the different injury mechanisms resulting in TMR vs. PTS. Then, we review the state of the art on computational models used to estimate $P(TMR_{type}\ occurs)$, the probability that a TMR with a particular attribute (i.e., of a particular size) occurs (the first metric needed to calculate RSI_{TMR}). Next, we present findings from our search of the literature for data on TMR and propose recommendations for TMR attributes that can be used to estimate $P(TMR_{type}\ is\ significant \mid TMR_{type}\ occurred)$, the probability that a TMR with a particular attribute is significant, given that it has occurred (the second of the two metrics needed to calculate RSI_{TMR}). We carefully list the approximations that we must make to bin this second metric as either 0 or 1. We then reconcile the attributes used to predict the first vs. second metrics and discuss how one can use the existing computational model along with our proposed predictive attributes to calculate RSI_{TMR}. Finally, we conclude with a discussion on the interrelation between RSI_{TMR} vs. RSI_{PTS}, the risk of causing a significant TMR vs. the risk of causing a significant PTS. We briefly suggest that animal studies could be done to quantify the statistical dependence between these two metrics, such that the two metrics can be numerically consolidated into a single *RSI* metric. In the absence of such studies, though, we propose methods to separately quantify and assess both RSI_{TMR} and RSI_{PTS} during the development acquisition phase of a novel flashbang system.

2. Anatomy and Injury Mechanisms of the Ear

A. Ear Anatomy

The ear is composed of three major sections: the outer ear, middle ear, and inner ear. Figure 3 describes the anatomical structure of the ear. This section provides an overview of each compartment's structure and function, based upon the work of Guyton and Hall (1996), combined with an overview of injury mechanisms.



Source: van der Willigen (2008, 4).

Figure 3. The Human Ear

1. Outer Ear

The exterior, cartilaginous structure of the ear, called the pinna, serves to collect sound and channel the acoustic energy into the external auditory canal (i.e., the ear canal). At the end of the ear canal lies the eardrum, or tympanic membrane.

2. Middle Ear

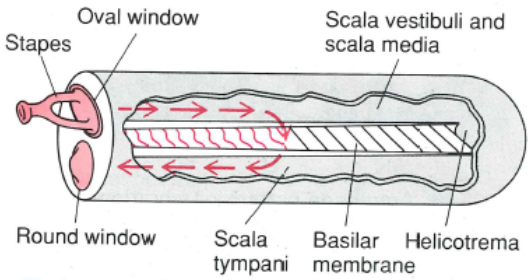
The middle ear consists of the tympanic membrane and ossicular system. Referred to as the conduction system, the middle ear conducts airborne acoustic energy from the outer ear to the inner ear for sensing.

The tympanic membrane is cone shaped. This structure acts like a drum head that transduces incoming acoustic waves into mechanical motion. The membrane consists of two regions: an upper region called the pars flaccida, and a lower region called the pars tensa. The lower region is thicker and maintains tension for conducting vibrations to the ossicular system. The pars tensa is the region of the tympanic membrane that is most commonly associated with TMR (Kronenberg, Ben-Shoshan, and Wolf 1993; Kronenberg et al. 1988; Persaud et al. 2003).

Viewed from the exterior, the pars tensa possesses a linear indentation. This indentation is formed by the attachment of the ossicular system on the interior surface. The ossicular system consists of three small bones. The first bone is the hammer (or malleus), whose handle attaches to the tympanic membrane. The hammer's other end attaches to the anvil (or incus), which attaches to the stirrup (or stapes). The three bones are held together with a network of ligaments that allow them to articulate and conduct the mechanical vibrations of the tympanic membrane to the sensory element of the ear: the cochlea. The ossicular system provides *impedance matching*, the efficient coupling of the inbound air waves with the mechanical movement of the fluid inside the cochlea. The bones conduct pressure from the tympanic membrane (which is approximately 55 mm^2 in area, roughly 8 mm in diameter) to the cochlear oval window (which is only approximately 3.2 mm^2 in area, roughly 2 mm in diameter), leading to a $55 \div 3.2 = 17$-fold increase in pressure at the cochlea. This pressure increase is necessary given the higher impedance (i.e., resistance to flow) of the fluid within the cochlea. Without this phenomenon, the acoustic energy would reflect and fail to couple efficiently into the cochlea. The lever displacement ratio of the bones provides additional pressure amplification. The ossicular system's impedance matching is optimized for frequencies between 300 and 3,000 Hz. Without the middle ear conduction system, acoustic conduction is far less efficient, resulting in hearing loss.

3. Inner Ear

Resembling a snail shell, the cochlea is a system of three fluid-filled tubes that are coiled up into a spiral structure. The three tubes (scala vestibuli, scala media, and scala tympani) lie side by side. Perhaps the most straightforward way to understand the function of the cochlea is to envision the tubes uncoiled into a straight line, as shown in Figure 4. The ossicular system of the middle ear pushes against the oval window, which, in turn, pushes fluid within the scala vestibuli, which pushes fluid within the neighboring scala media and, finally, the scala tympani. As fluid movement is conducted between these three compartments, a sensory element called the basilar membrane picks up the vibrations.

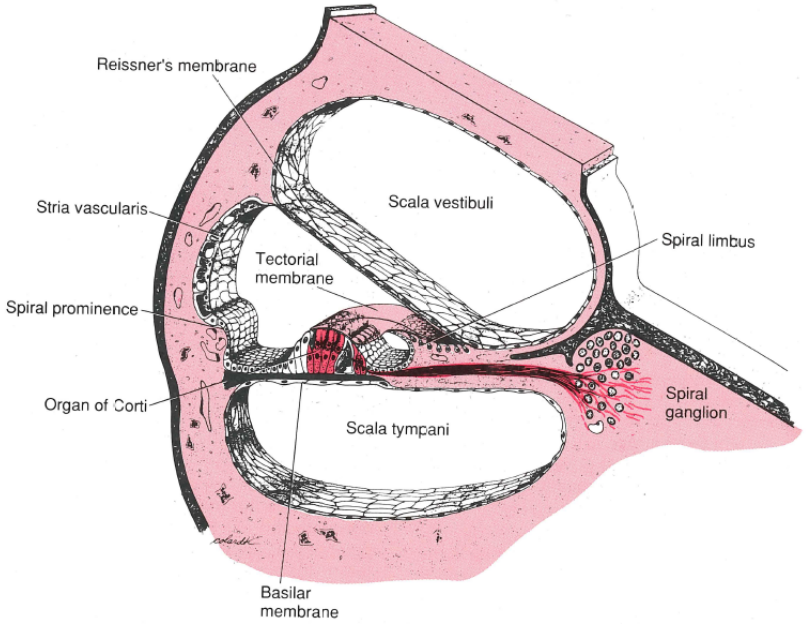


Source: Guyton and Hall (1996, 665).

Figure 4. Simplified Depiction of the Three Cochlear Chambers

The basilar membrane is composed of stiff, reed-like fibers. The fibers' stiffness and length vary along the length of the cochlea. This variation in stiffness and length allows different fibers to resonate at different frequencies. The fibers are arranged such that those that resonate at high frequencies are located close to the front of the cochlear oval window while those that resonate at low frequencies are located at the far end of the cochlea.

Along the basilar membrane lies the Organ of Corti, as shown in Figure 5. The Organ of Corti acts as the final sensory transduction element, converting vibrations into a sensory neural signal. When a local region of the basilar membrane vibrates at the proper frequency, the sensory hair cells within the corresponding section of the Organ of Corti are displaced. This displacement causes the hair cells to depolarize, creating a neural signal and the perception of sound within higher orders of the central auditory system.



Source: Guyton and Hall (1996, 665).

Figure 5. Cross Section of the Cochlea, Illustrating the Three Cochlear Chambers, the Basilar Membrane, and the Organ of Corti

The inner ear also consists of the vestibular system, which consists of three fluid-filled tubes located in different, orthogonal planes. Hair cells within these tubes detect inertial changes in the head's orientation, providing sensory perceptive input for balance and orientation.

B. Mechanisms of Ear Injury

Hearing loss can be attributed to degraded function in any step of the sensory pathway, ranging from reduced outer and middle ear conduction, sensory neural damage within the cochlea, and higher order loss of function within the central nervous auditory system. The literature reports injury and recovery statistics from a variety of injury mechanisms ranging from moderate (slap injuries, scuba diving) to severe (puncture wounds, bomb blasts) (Orji and Agu 2008; Lou, Lou, and Zhang 2012). Since this project considers NLW systems, specifically flashbang grenades, pertinent injury mechanisms are confined to blast-induced primary acoustic injuries within the middle ear (conductive injuries) and inner ear (sensorineural injuries).

In our experience, most journal articles in the literature use the term "PTS" to refer to a permanent hearing loss caused by *sensorineural* injuries to the inner ear, such as trauma to the sensory hair cells in the Organ of Corti (e.g., Cave, Cornish, and Chandler 2007, 726; SCENIHR, 2008, 26; Rutka 2013, 3). Therefore, in this and our previous document (King and Cazares 2015), we also use the term "PTS" to refer to permanent sensorineural hearing loss only, ICD-10-CM codes H90.3, H90.41, H90.42, and H90.5 (WHO 2016a, 380). However, permanent hearing loss can also occur due to *conductive* injuries to the *middle* ear, such as TMR (Remenschneider et al. 2014). Therefore, in this document, we investigate the permanent conductive hearing loss resulting from TMR, ICD-10-CM codes H90.0, H90.1, and H90.2 (WHO 2016a, 380). We contrast this condition with the permanent sensorineural hearing loss referred to as "PTS" in our previous document (King and Cazares 2015).

1. Middle Ear Injury (Conductive Injury)

The tympanic membrane is a compliant structure that distends when exposed to changes in air pressure. The membrane is highly sensitive to pressure changes by necessity since the typical fluctuation in air pressure for normal conversation is about 0.02 Pa, which is 60 dB of sound pressure level (SPL)[1] (van der Willigen 2007, 17). The ear possesses a large dynamic range, capable of operating over many orders of magnitude in intensity, with sensory pain reported for sounds at or above about 20 Pa (1,000 times louder than conversation) and ultimately TMR for pressures at or above about 34.4 kPa (1.8 million times louder than conversation) (Hirsch 1966). Parker et al. (2012) report a flashbang grenade system that provides peak pressures of less than 2 psi (13.7 kPa, which is 176 dB) at a distance 6 ft. from the device, well below Hirsch's (1966)

[1] SPL = $20 \times \log_{10}$(Sound Pressure/0.00002 Pa), where 0.00002 Pa is considered to be the hearing threshold—the lowest sound pressure that the average, young, healthy human can detect (ANSI 2013).

threshold for TMR (34.4 kPa, which is 184 dB). Table 1 lists the sound pressures and SPLs of different exposures.

Table 1. Sound Pressures and SPLs of Different Exposures

Sound	Sound Pressure (Pa)	SPL (dB)
Hearing threshold	0.00002	0
Conversation	0.02	60
Pain threshold	20	120
Flashbang at 6 ft.	13,700	176
TMR rupture	35,000	184

Note that all of the dynamic pressure intensities in Table 1 are small compared to static standard atmospheric air pressure of 101 kPa (Oxtoby, Nachtrieb, and Freeman 1994). While TMRs may occur at approximately 35 kPa, this magnitude is only about one-third the size of the static atmospheric pressure. This phenomenon explains the common occurrence of TMRs with conditions that present sizeable pressure changes across the tympanic membrane (e.g., scuba diving, ear infections, slaps to ear, and blast overpressure).

For blast-induced TMR, the literature reports a variety of factors that can influence the likelihood of a TMR, such as the subject's age (Kerr and Byrne 1975), the orientation of the subject's ear to the blast wave (James et al. 1982), and the blast dynamics (James et al. 1982). Blast dynamics prove important to this framework since blast dynamics predict the size of the TMR (James et al. 1982), which, in turn, predicts the likelihood of spontaneous healing of the TMR (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008; Lou, Lou, and Zhang 2012). We discuss this phenomenon to greater extent later in Section 4.A.1.

Spontaneous healing/recovery of a TMR tends to occur within about 6 months of injury onset (Kerr and Byrne 1975; Persaud et al. 2003). When spontaneous healing does not occur within 3 to 6 months, surgical intervention (tympanoplasty) is the standard of care (Kerr and Byrne 1975; Garth 1995). These factors are discussed in greater detail in Section 4.A.1.

Conductive hearing loss is associated with TMR. Taylor and Mueller state in their audiometry textbook that, "The amount of hearing loss caused by a perforated TM [tympanic membrane] varies by both size of the perforation and the opening… With large perforations, it's common to see a conductive hearing loss of 30 to 40 dB" (2011, 116). This conductive hearing loss tends to be temporary. "Once the perforation heals, hearing is usually recovered fully (maybe with a slight 5- to 10-dB drop due to scarring)" (Taylor and Mueller 2011, 116–117). This recovery occurs with the healing/repair of the tympanic membrane within 3 to 6 months (Persaud et al. 2003). We discuss these factors in greater detail in Section 4.B.

2. Inner Ear Injury (Sensorineural injury)

When intense acoustic energy is coupled to the cochlea, three forms of hearing problems may result: tinnitus, TTS, and PTS.

Tinnitus is the perception of sound (e.g., ringing, whistling, hissing, roaring, or booming) in the absence of an external stimulus (Farlex Partner Medical Dictionary 2012e). The ICD-10-CM codes tinnitus as H93.11, H93.12, H93.13, and H93.19 (WHO 2016a, 382–383). Tinnitus is the most common symptom associated with blast injury, reported in about 19% of blast cases (Dougherty et al. 2013) and 50% of all blast-induced TMR cases (Ritenour et al. 2008). The damage mechanism associated with tinnitus is still speculative but is perhaps due to temporary changes in membrane permeability at the Organ of Corti (Garth 1995). The "ear ringing" associated with acute exposure to intense sound tends to dissipate within hours after exposure (Garth 1995). Due to its often temporary effect, this mechanism of injury was not considered further in our project.

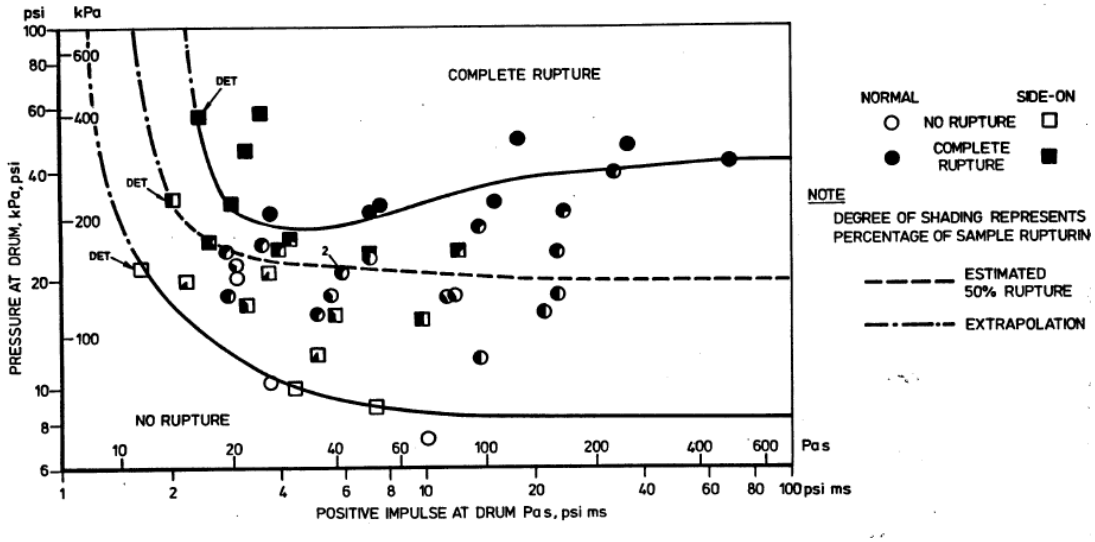
TTS represents sensorineural hearing loss that recovers over days (Nordman, Bohne, and Harding 2000). Because TTS is, by definition, temporary, this condition is beyond the scope of our analysis. In contrast, our analysis does touch upon PTS, the *permanent* sensorineural hearing loss caused by damage to the inner ear, as we discuss later in Sections 5.B and 6. The mechanisms of cochlear damage and the physiological differences that lead to TTS vs. PTS remains a topic of debate in the literature but may result from membrane buckling and dislodgment of hair cells within the Organ of Corti (Nordman, Bohne, and Harding 2000). For blast exposure cases, reports are common of high levels of PTS (>40 dB HL) at high frequencies (4,000, 6,000, and 8,000 Hz) due to sensorineural damage in the cochlea (Persaud et al. 2003). We discuss PTS in greater detail in Sections 5.B and 6.

3. Predictive Attributes of TMR Occurrence

As discussed in Section 2, the tympanic membrane is a compliant structure that distends when exposed to changes in air pressure and ultimately fails with sufficiently high pressure. Assessing the membrane's susceptibility to rupture due to a blast is beyond the scope of this project. In fact, the Joint Non-Lethal Weapons Directorate (JNLWD) commissioned earlier studies that investigated this behavior (Shelley and Chan 2015). However, in this section, we briefly revisit the state of the art on this subject and discuss methods to estimate $P(TMR_{type}\ occurred)$, the probability that a blast will cause a TMR type characterized by a particular physical attribute (i.e., size). In a later section, we will discuss how one can use this information along with estimates of $P(TMR_{type}\ is\ significant \mid TMR_{type}\ occurred)$ to quantify RSI_{TMR}, the probability that the blast will cause a significant TMR.

Abundant literature is available on blast-induced TMR. Hirsch (1966) showed that the fast dynamic pressure waves commonly associated with explosions begin to rupture tympanic membranes at peak pressures of about 5 psi (34.4 kPa, which is 184 dB). This SPL has often been considered a minimum injury threshold for TMR. However, above this threshold remains significant variability in pressure sensitivity of TMR. Confounding factors include individual anatomy (pinna and ear canal geometry, membrane thickness, compliance, size), occlusions (ear wax, ear plugs), orientation (ears normal or side-on with respect to the blast), and so forth (James et al. 1982). Perhaps the most comprehensive and cited work on blast-induced TMR is the report by James et al. (1982), who examined 395 human cadaver ears under controlled blast conditions. Results showed that the notable attributes of a blast that can induce TMR are its peak pressure and the duration of its positive pressure phase.

The James et al. findings establish the realm of conditions that can produce TMR. Figure 6 summarizes the influence of pressure dynamics, plotting peak pressure (Pa) at the eardrum vs. the duration of exposure (s), a quantity that James et al. refer to as *impulse* (Pa•s). The shaded markers indicate the percentage of samples (ears) that exhibited a TMR post-blast, ranging from 0% of samples ruptured (no shading) to 100% of samples ruptured (full shading). The figure presents three isodamage curves. The bottom curve marks the "no rupture" boundary (below which no ears exhibited TMR), the middle curve marks the "50% rupture" boundary (50% of ears exhibited TMR), and the top curve marks the "complete rupture" boundary (above which 100% of ears exhibited TMR). The area between the top and bottom curves marks the envelope of conditions where TMR may result. Note that the pressures reported in this figure are those recorded at the eardrum, which may be many times larger than the pressure recorded outside the ear due to amplification of the pressure wave by the ear canal.



Source: James et al. (1982, 40, Figure 13).

Figure 6. TMR Rupture Statistics Given the Impulse at the Eardrum and Peak Pressure at the Eardrum

For large explosions facing 90 degrees normal to the ear (the optimal conditions for inducing TMR), James et al. estimate that 56 kPa is the minimum threshold peak pressure (at the eardrum) needed to rupture the eardrum, which translates to about 15 kPa outside the ear. Interestingly, the 15 kPa value reported by James et al. (1982) is about 50% lower than the standard value of 34.4 kPa reported by Hirsch (1966). However, Hirsch (1966) cites other studies that report minimum injury-causing peak pressure values that also dip as low as 15 kPa. Conditions that rupture 100% of samples begin at about 220 kPa, which translates to about 51 kPa outside the ear.

Regrettably, James et al. (1982) do *not* report the statistical likelihood of TMRs of particular sizes, which is unfortunate since other literature indicates that the size of the membrane tear is indicative of the likelihood of spontaneous healing, discussed later in more detail in Section 4.A.1. Fortunately, though, the James et al. report does include the data needed to construct a computational model that estimates the statistical likelihood of three different TMR sizes (Shelley and Chan 2015). The reported data detail the conditions and observations for all 395 human cadaver ear samples, including (1) orientation, (2) peak pressure, (3) impulse, and, most importantly, (4) clinical classification of the TMR size. James et al. classified each eardrum post-blast via an ordinal score of 0, 1, 2, or 3 (1982, 10):

- 0 = “No rupture.”
- 1 = “Mild rupture. This included minor slits, and linear disruption of the drum fibres producing a ‘meshlike’ effect.”

- 2 = “Moderate damage. Large tears or multiple small holes or tears.”
- 3 = “Major damage. Total disrupture of the drum with large flaps of drum; some were inverted into the ear and some everted.”

Shelley and Chan (2015) used these data to develop a computational model that estimates $P(TMR_{type}\ occurred)$, the probability that a given blast will cause a TMR type with a particular tear size. The tear size is binned based on the James clinical classifications. For example, $P(TMR_{JamesClass1\text{-}3})$ is the probability that a blast will cause a TMR with any size of tear (James clinical classification of 1, 2, or 3), while $P(TMR_{JamesClass2\text{-}3})$ is the probability that a blast will cause a TMR with only a moderate or major tear size (James clinical classification of 2 or 3).

4. Predictive Attributes of TMR Significance

In the previous section, we discussed methods to estimate $P(TMR_{type}\ occurred)$, the probability that a blast will cause a TMR type with a particular physical attribute (e.g., a TMR size with a James clinical classification of 1, 2, or 3). In this section, we now discuss methods to estimate $P(TMR_{type}\ is\ significant \mid TMR_{type}\ occurred)$, the probability that a TMR type with a particular attribute (i.e., size) is significant, given that it has occurred. Both of these metrics must be estimated to quantify a blast's RSI_{TMR}. Furthermore, the attributes used to estimate the first quantity must be numerically reconciled with the attributes used to estimate the second quantity.

A. HCC Level Required to Treat TMR

DoDI 3200.19 specifies that one way in which an injury can be considered significant is if HCC0 medical treatment, defined as "self-aid, buddy-aid, and combat lifesaver skills" (DoD 2012, 13), is insufficient to treat the injury. In this section, we propose quantitative attributes of a TMR that can predict the HCC index of the medical treatment required to treat the TMR. We also consider the HCC index of medical treatment required to treat any complications of the TMR.

1. Spontaneous Healing

The size of the TMR indicates the likelihood that the membrane will heal spontaneously, without medical treatment (HCC0). This subsection proposes quantitative thresholds to bin the TMRs according to size. We explain how TMR size predicts whether the TMR will heal with HCC0 vs. HCC1+ medical treatment and how our quantitative size bins relate to the "1, 2, 3" clinical classifications used by James et al. (1982).

For cases involving blast-induced TMR, the literature reports spontaneous healing rates of about 80% (Kerr and Byrne 1975; Persaud et al. 2003). However this bulk statistic masks a major determinant of spontaneous healing—the TMR size. The literature indicates that larger TMRs produce worse spontaneous healing outcomes (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008; Lou, Lou, and Zhang 2012). Ritenour et al. (2008) examined 97 blast-inducted TMRs exhibited by 65 patients in a military setting. They assigned an ordinal score of I to IV to each TMR, post-blast, based on an objective, quantitative measurement of the size of the TMR:

- I = a pinpoint or linear tear ≤2 mm long,
- II = a linear tear >2 mm long up to a rupture of <25% of the eardrum area,
- III = a rupture of 25%–50% of the eardrum area, and
- IV = a rupture of >50% of the eardrum area.

Ritenour et al. (2008) documented outcomes that ranged from 100% likelihood of spontaneous healing for their small Grade I TMRs to only an 11% likelihood of spontaneous healing for their large Grade IV TMRs. Table 2 summarizes the Ritenour et al. findings and conveys two key messages:

- Larger perforations result in worse spontaneous healing outcomes (second to last column).
- Surgical repair/intervention (HCC1+) is prescribed as the standard of care when spontaneous healing does not result (last column).

Table 2. Spontaneous Healing Outcomes for TMRs of Different Sizes

Grade	No. of TMs Perforated	Perforation (%)	No. with Outcome	Healed (%)	Surgery (%)
I	5	5 ± 0	3	100	0
II	28	17 ± 6	18	83	17
III	19	42 ± 8	15	33	67
IV	32	81 ± 1	27	11	89
Total (all TMs)	98	47 ± 30	74	46	54

Source: Ritenour et al. (2008, S176, Table 3).

This latter point has been stated by other researchers (Kerr and Byrne 1975; Xydakis et al. 2005; Remenschneider et al. 2014). Kerr and Byrne (1975) recommended surgical repair if the TMR fails to close within 6 months. Remenschneider et al. (2014, 1831) recommended that, "small perforations (<50% [of the eardrum area]) should be observed for spontaneous closure, whereas larger perforations (sub-total) will likely require tympanoplasty [surgical repair of the tympanic membrane]." Xydakis et al. (2005) recommended surgical intervention for TMR, including removing debris and TM fragments from the middle ear and repositioning the torn edges of the TM, all under general anesthesia. Note, though, that Xydakis et al. (2005) do not comment on the difference in care needed for TMRs that do vs. do not heal spontaneously. However, due to their reference to "large tears" and "middle ear debris" (Xydakis et al. 2005, 504), we assume that they were referring to large TMRs, the same type of TMRs as in the Ritenour Grade IV. The guidance from Kerr and Byrne (1975), Xydakis et al. (2005), Ritenour et al. (2008), and Remenschneider et al. (2014) anchors a major conclusion of our analysis: a TMR that does not spontaneously heal is a significant injury because the standard treatment for such a TMR is surgical intervention (HCC1+).

Figure 7 presents the Ritenour et al. findings in scatter plot form, plotting the percent of TMRs requiring surgery vs. the size of the TMR. The horizontal error bars represent ±1 standard deviation around the mean TMR area, quantified as the percentage of the total eardrum area. This figure suggests a method for estimating the significance of a TMR by associating its size to its need for surgical intervention (HCC1+). Ideally, we may wish to know the TMR size that leads to

a 50% likelihood of requiring surgical intervention. All TMRs this size or greater could then be considered significant.

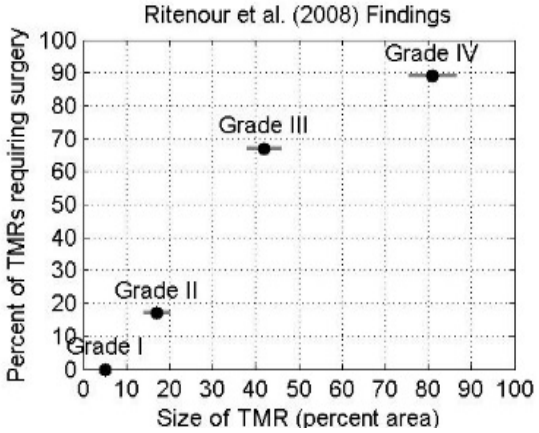


Figure 7. Percent of TMRs Requiring Surgery vs. the Size of the TMR

At first glance, one may be tempted to fit the data in Figure 7 to a statistical model to estimate this 50% likelihood cut-off threshold. However, such an exercise would require prior information about what the functional form of the model should be: linear, quadratic, or so forth. In the absence of this prior information, we instead focus on the few data points that Ritenour et al. collected empirically, in reverse order of grade:

- Grade IV TMRs were large, >50% of the eardrum area, with a mean size 81% ± 11% of the eardrum area. Almost all (89%) of these TMRs required surgical intervention. Therefore, we err on the side of caution and approximate that TMRs >50% of the eardrum area caused by flashbang grenades require surgery and can therefore be considered significant.
- Grade III TMRs were 25%–50% of the eardrum area, with a mean size of 42% ± 8% of the eardrum area. A large percentage (67%) of these TMRs required surgery. Therefore, we also err on the side of caution here and approximate that TMRs caused by flashbang grenades that are 25%–50% of the eardrum area also require surgery and can therefore also be considered significant.
- Grade II TMRs were longer than 2 mm but <25% of the eardrum area, with a mean size of 17% ± 6% of the eardrum area. A notable percentage (17%) of these TMRs required surgical intervention. (Note that it is coincidence that the mean size and the percentage requiring surgery were both 17%.) We believe these TMRs represent a “grey area” of significance since while a notable percentage (17%) of them required surgery, this percentage is still rather low (i.e., less than 50%). However, we choose to err on the side of caution here again and approximate that Grade II TMRs caused by flashbang grenades also require surgery and can also be considered significant.

- Grade I TMRs were small, ≤2 mm long, with a mean size of only 5% ± 0% of the eardrum area. None (0%) of these TMRs required surgical intervention. We therefore conclude that TMRs ≤2 mm long caused by flashbang grenades do not require surgery.

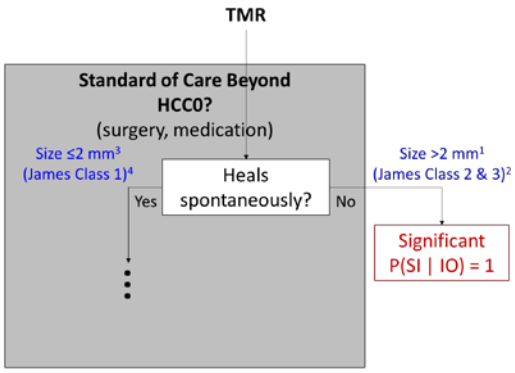
We compared the results of the Ritenour et al. study to those of other research groups. In an earlier study, Kronenberg, Ben-Shoshan, and Wolf (1993) used the same Grades I–IV to classify blast-induced TMRs in a military setting. They found that a similar percentage of the TMRs in each grade required surgery: 8% in Grade I, 16% in Grade II, 45% in Grade III, and 80% in Grade IV. Remenschneider et al. (2014) used a slightly different scheme to classify blast-induced TMRs, this time in a civilian setting: the 2013 Boston Marathon bombings. Their size-based classifications had less resolution at the small end of the scale than did the Ritenour/Kronenberg grades, effectively lumping both the Ritenour/Kronenberg Grades I and II into one class: TMRs <25% of the eardrum area. Therefore, it is difficult to compare their spontaneous healing rates for small TMRs to the rates published by Ritenour et al (2008) and Kronenberg, Ben-Shoshan, and Wolf (1993). They did show a high rate of persistent perforation (i.e., a low rate of spontaneous healing) for large TMRs: 12 (57%) of 21 TMRs that were 26–50% of the eardrum area (Ritenour/Kronenberg Grade III) did not heal spontaneously. Similarly, 16 (73%) of 22 TMRs that were >50% of the eardrum area (Ritenour/Kronenberg Grade IV) did not heal spontaneously (Remenschneider et al. 2014, 1827).

Thus, as shown in Figure 8, we approximate that medium and large TMRs caused by flashbang grenades with tears >2 mm long (Ritenour/Kronenberg Grades II, III, and IV) can be readily binned as significant since we err on the side of caution and approximate that they require surgical intervention (HCC1+):

$$P(TMR_{size>2mm} \text{ is significant} \mid TMR_{size>2mm} \text{ occurred}) = 1.$$

In contrast, at this point in our analysis, those TMRs ≤2 mm long cannot be readily binned as not significant, even though we approximate a zero likelihood that they will require surgical invention. It may be that HCC1+ medical treatment is needed to treat any downstream complications resulting from these small TMRs, rather than the TMR itself. Or, it may be that these small TMRs "restrict the employment or other activities of [a] person for the rest of his or her life," to use language from DoDI 3200.19 (DoD 2012, 14), even though HCC1+ medical treatment is not needed to treat the TMR or its complications. Therefore, these small TMRs must be analyzed further to determine whether they are or are not significant. Accordingly, the left arm of the decision tree in Figure 8 is left unfinished, for now:

$$P(TMR_{size\leq 2mm} \text{ is significant} \mid TMR_{size\leq 2mm} \text{ occurred}) = \text{to be decided in later sections.}$$



[1] A notable percentage of blast-induced TMRs >2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we err on the side of caution and approximate that TMRs this size caused by flashbang grenades require surgery and are therefore significant, i.e., P(SI | IO) = 1.

[2] The blast-induced TMRs in James Class 2 and 3 are described as "large tears, or multiple small holes or tears" and "total disrupture of the drum…", respectively (James et al. 1982, 10). Therefore, we approximate that James Class 2 and 3 TMRs are >2 mm long.

[3] Zero or almost zero blast-induced TMRs ≤2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we approximate that TMRs this size caused by flashbang grenades do not require surgery. Further analysis is needed to determine if they are or are not significant, however.

[4] The blast-induced TMRs in James Class 1 are described as "minor slits, and linear disruption of the drum fibres producing a 'meshlike' effect" (James et al. 1982, 10). Therefore, we approximate that James Class 1 TMRs are ≤2 mm long.

Figure 8. Beginning to Classify a TMR as Significant:
Classifying the TMR as Significant Based on Its Likelihood of Spontaneously Healing

In Section 3, we discussed a computational model that estimates the probability that a TMR of a certain size will occur, based on the data from James et al. (1982). Unfortunately, the James et al. (1982) vs. Kronenberg, Ben-Shoshan, and Wolf (1993)/Ritenour et al. (2008) data sets use different schemes to assess the size of the TMR. While Kronenberg, Ben-Shoshan, and Wolf and Ritenour et al. bin their TMRs into grades that are based on quantitative measurements of the sizes of the tears, James et al. provide only a qualitative description of the sizes of the TMRs in each of their three classes. Furthermore, while Kronenberg, Ben-Shoshan, and Wolf and Ritenour et al. provide quantitative statistics on the percentage of TMRs in each of their four grades that required surgical intervention, James et al. merely state, "Looking at the degree of rupture clinically, one has the impression that type (1) ruptures would heal spontaneously but that type (3) ruptures would require surgical repair" (1982, 10). Since the James et al. TMRs were induced in cadaver ears, there was no way to test their hypothesis, since cadavers do not heal. Furthermore, James et al. offered no hypothesis for their type (2) ruptures.

As will be discussed in Section 5, the James et al. (1982) and Kronenberg, Ben-Shoshan, and Wolf (1993)/Ritenour et al. (2008) findings are needed to estimate RSI_{TMR}. We must therefore reconcile the two different size attributes, as shown in Table 3. To accomplish this goal, we approximate:

- The boundary between James Class 1 vs. James Class 2 is equivalent to the boundary between Ritenour/Kronenberg Grade I vs. Ritenour/Kronenberg Grade II due to the fact that the blast-induced TMRs in James Class 1 are described as "minor slits and linear

disruption of the drum fibres producing a 'meshlike' effect" (James et al. 1982, 10). Therefore, we approximate that James Class 1 TMRs are ≤2 mm long, which is the maximum size of the Ritenour/Kronenberg Grade I TMRs.

- The boundary between James Class 2 vs. James Class 3 is equivalent to the boundary between Ritenour/Kronenberg Grade III vs. Ritenour/Kronenberg Grade IV due to the fact that the blast-induced TMRs in James Class 3 are described as "total disrupture of the drum…" (James et al. 1982, 10). Therefore, we approximate that James Class 3 TMRs are >50% of the eardrum area, the minimum size of the Ritenour/Kronenberg Grade IV TMRs.

Table 3. Reconciling the James vs. Ritenour/Kronenberg Schemes for Evaluating the Size of a TMR

James Clinical Classification	Ritenour/Kronenberg Grade (Percent of Eardrum Area)
1: Minor (small tears or slits)	I: ≤2 mm long (5 ± 0)
2: Moderate (large tears or multiple small tears)	II: >2 mm long to <25% area (17 ± 6) III: 25% - 50% area (42 ± 8)
3: Major (complete tear)	IV: >50% area (81 ± 11)

We have already concluded that TMRs ≤2 mm long (i.e., Ritenour/Kronenberg Grades II, III, and IV) are significant since we erred on the side of caution and approximated that those TMRs caused by flashbang grenades require surgical intervention (HCC1+). Therefore, we now also conclude that TMRs in James Class 2 and 3 are significant, since James Class 2 and 3 correspond to Ritenour/Kronenberg Grades II, III, and IV. Thus,

$$P(TMR_{JamesClass2\text{-}3} \text{ is significant} \mid TMRJames_{Class2\text{-}3} \text{ occurred}) = 1.$$

In contrast, we must further analyze the TMRs in James Class 1 to determine whether they are or are not significant:

$$P(TMR_{JamesClass1} \text{ is significant} \mid TMR_{JamesClass1} \text{ occurred}) = \text{to be decided in later sections.}$$

2. Complications

A TMR can lead to additional medical complications. The complications may require HCC1+ medical treatment above and beyond the treatment required to repair the TMR itself. We factor the two main complications into our analysis: otitis media and cholesteatoma.

a. Otitis media

Otitis media is an inflammation of the middle ear (Farlex Partner Medical Dictionary 2012b). Otitis media is more often associated with the *cause* of TMR rather than the *result* of TMR. However, the following discussion is confined to acute otitis media *resulting* from TMR. This

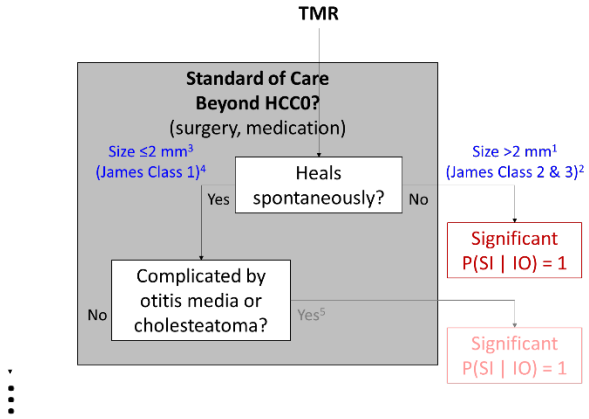
complication falls under ICD-10-CM codes H65.00–H65.07 and possibly H65.191–H65.199 (WHO 2016a, 365–366).

Pertinent otitis media pathologies following TMR arise from middle ear infection. The tympanic membrane serves as a physical barrier, protecting the middle ear from bacterial growth and infection. Following TMR, the middle ear becomes exposed to the natural environment, which makes the middle ear more susceptible to infection. Infection following TMR occurs at a low rate. According to Lou, Lou, and Zhang (2012), middle ear infections were associated with only 42 (8%) of 504 TMR cases. Note that these statistics pertain to blast-related and non-blast-related TMR cases. In their study, scarce data are available for blast-specific cases only. Also note that infection should not be confused with ear discharge (otorrhea), which is a common non-threatening side effect associated with TMR wound healing (McGraw-Hill Concise Dictionary of Modern Medicine 2002). In fact, Lou, Lou, and Zhang (2012) suggest that the presence of a "wet ear" may even facilitate healing.

Recommended care for acute otitis media may include antibiotic administration if the symptoms are severe (fever, and so forth) (Waseem 2016). We did not find information regarding how often a TMR leads to a case of otitis media that is severe enough to require antibiotics. In the absence of this information, we err on the side of caution and simply approximate that TMRs that lead to otitis media require antibiotics. In the United States, acquiring a prescription for antibiotics requires a visit to a health care professional (HCC1+), which leads to another approximation in our analysis: a TMR that results in otitis media is a significant injury because prescription antibiotics (HCC1+) are needed. The question now remains, how often do TMRs lead to otitis media?

In the previous section, we concluded that any TMR that fails to heal spontaneously (i.e., one >2 mm long, equivalent to James Class 2 or 3) must be considered a significant injury since surgical intervention (HCC1+) is the standard of care for treating the TMR. Therefore, we do not need to consider these TMRs further. They are already considered significant. Instead, we only need to consider the likelihood of otitis media for TMRs that heal spontaneously (i.e., those $\leq$2 mm long, equivalent to James Class 1), as shown in Figure 9.

Unfortunately, the literature lacks the resolution to isolate these sets of cases. However, we assume that the likelihood of otitis media resulting from a TMR that heals spontaneously is less than Lou, Lou, and Zhang's (2012) reported 8% likelihood of otitis media resulting from *all* TMRs. Therefore, we approximate that we can disregard the possibility of otitis media in spontaneously healed TMRs. That is, its likelihood is approximately zero, with an uncertainty that is similar to the uncertainties of other quantities estimated to determine RSI_{TMR}. As a result, the otitis media arm of the decision tree in Figure 9 is greyed out. This approximation is backed up by the fact that in our review of the literature, we have not seen any reports of infection after spontaneous healing.



[1] A notable percentage of blast-induced TMRs >2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we err on the side of caution and approximate that TMRs this size caused by flashbang grenades require surgery and are therefore significant, i.e., P(SI | IO) = 1.

[2] The blast-induced TMRs in James Class 2 and 3 are described as "large tears, or multiple small holes or tears" and "total disrupture of the drum…", respectively (James et al. 1982, 10). Therefore, we approximate that James Class 2 and 3 TMRs are >2 mm long.

[3] Zero or almost zero blast-induced TMRs ≤2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we approximate that TMRs this size caused by flashbang grenades do not require surgery.

[4] The blast-induced TMRs in James Class 1 are described as "minor slits, and linear disruption of the drum fibres producing a 'meshlike' effect" (James et al. 1982, 10). Therefore, we approximate that James Class 1 TMRs are ≤2 mm long.

[5] TMR can lead to otitis media or cholesteatoma (Lou, Lou, and Zhang 2012; Ear Surgery Information Center 2015). Severe otitis media requires prescription antibiotics, beyond HCC0 (Waseem 2016). Severe cholesteatoma requires surgery, also beyond HCC0 (Ear Surgery Information Center 2015). Otitis media and cholesteatoma are associated with 8% and 7.6%, respectively, of all TMR cases (Lou, Lou, and Zhang 2012; Kronenberg et al. 1988). However, we assume that the likelihood of otitis media or cholesteatoma resulting from TMRs that heal spontaneously is even less than the already low values of 8% and 7.6% reported for all TMRs, and is approximately zero. Therefore, we disregard the possibility of this branch of the decision tree. Further analysis is still needed to determine if these TMRs are or are not significant, however.

Figure 9. Continuing to Classify a TMR as Significant: Classifying a Spontaneously Healed TMR as Significant Based on its Likelihood of Otitis Media or Cholesteatoma Complications

a. Cholesteatoma

Cholesteatoma is another potential complication of a TMR (Kronenberg et al. 1988). Following a TMR, epithelial (outer skin) cellular matter from the membrane may dislodge and "seed" inside the middle ear. This cellular matter may proliferate, forming cholesteatomas, which manifest as isolated "pearls" or more invasive growths into the mastoid bone beside the middle ear. This complication falls under ICD-10-CM code H71.10–H71.13 (WHO 2016a, 372–373).

Medical intervention is often indicated for cholesteatoma since the growths may disrupt middle ear function. In more severe cases, these growths may exert pressure on local facial nerve fibers and present facial weakness or paralysis (Ear Surgery Information Center 2016). Isolated pearls can be surgically removed through the ear canal, while mastoid growths require more invasive surgery that removes the wall separating the middle ear and the mastoid bone. This phenomenon is thought to occur more readily from a blast-related TMR (Kronenberg et al. 1988). Based on this information, we approximate that the standard of care for cholesteatoma is surgical resection (HCC1+). Thus, we make the following approximation: a TMR that results in

cholesteatoma is a significant injury because of the standard of care for cholesteatoma is surgical intervention (HCC1+). Our question is now: how often do TMRs lead to cholesteatoma?

Kronenberg et al. (1988) investigated blast-induced TMR cases in military settings, finding a cholesteatoma incidence in only 16 (7.6%) of 210 cases. They report that the likelihood of cholesteatoma is associated with the size of the TMR. Only 1 (2%) of 51 Grade I TMRs (i.e., those ≤2 mm long) in their study led to cholesteatoma. In contrast, 3 (20%) of 15 Grade IV TMRs (i.e., those >50% of the eardrum area) led to cholesteatoma. Kronenberg et al. found this trend to be statistically significant (P = 0.001) but noted that other researchers offered contradictory findings. They also report elevated hearing loss associated with cholesteatoma, but their article lacks adequate data to quantify this association.

Once again, though, we point out that we only need to investigate the likelihood of cholesteatoma for TMRs that have spontaneously healed (i.e., those ≤2 mm long, equivalent to James Class 1) since TMRs that do *not* spontaneously heal (i.e., those >2 mm long, equivalent to James Class 2 and 3) are already considered significant. We assume that the likelihood of cholesteatoma in spontaneously healed TMRs is even lower than Kronenberg et al.'s (1998) reported 7.6% likelihood in *all* TMRs. Therefore, as with otitis media, we assume that we can disregard the possibility of cholesteatoma in spontaneously healed TMRs. That is, we approximate its likelihood as zero, with an uncertainty that is within the uncertainties of other quantities estimated to determine RSI_{TMR}. As such, we grey out the corresponding arm in the decision flowchart of Figure 9.

Note, though, that Kronenberg et al. state the following: "In two cases, cholesteatoma developed after the tympanic membrane had healed. These perforations apparently closed, leaving trapped epithelium in the middle ear space which later developed into cholesteatoma. Seaman [1971] reported a 12% chance that epithelium may be implanted behind a healed tympanic membrane. These findings reinforce the need for long-term follow-up in cases of blast injury, even if the tympanic membrane is closed" (1988, 129). Remember, though, that for this project, we need only consider the likelihood of cholesteatoma in TMRs that have spontaneously healed, which, based on the previous section of this document, we define as those <2 mm long. Kronenberg et al. (1988) found that only 1 (2%) of the 51 TMRs in this grade led to cholesteatoma. These results indicate that cholesteatoma is extremely rare for those TMRs in the upper, left arm of Figure 9, giving further weight to our decision to grey out the lower, right arm in the figure.

B. Restrictions to Life Caused by TMR

In the previous section, we identified quantitative attributes of a TMR (i.e., its size) that can predict the HCC index of the medical treatment required to treat the TMR and its complications, and, thus, that can predict the significance of the TMR. DoDI 3200.19 also specifies a second way in which an injury, such as a TMR, can be considered significant: the injury results in death or "physical damage … that … restricts the employment or other activities of the person for the rest

of his or her life" (DoD 2012, 14). Therefore, in this section, we propose quantitative attributes of a TMR that can predict the effect of a TMR on a person's employment or other life activities.

For this project, we approximate that failure to meet fitness for duty criteria in the U.S. military is an adequate surrogate for "restrictions on employment." This approximation is due to the fact that flashbang grenades can potentially cause harm not only to the personnel targeted with the flashbang detonation, but also to the U.S. operators who employ the flashbang grenades. Furthermore, based on guidance from the JNLWD, we do not differentiate between the significance of injuries to U.S. operators vs. the personnel targeted by U.S. operators.

DoDI 1332.18 states that "a Service member will be considered unfit [for duty] when the evidence establishes that the member, due to disability, is unable to reasonably perform duties of his or her office, grade, rank, or rating, including those during a remaining period of Reserve obligation" (DoD 2014, 27). The thought among U.S. military medical officers in the field is that a current TMR renders a soldier "unfit for duty" (Xydakis et al. 2005, 504). Furthermore, per DoDI 6130.03, the pre-enlistment medical standards for the U.S. military stipulate that new recruits cannot have a current TMR or "history of surgery to correct a [TMR] during the preceding 180 days" (DoD 2010, 14).

Service members who are considered to be "unfit for duty" are referred to the Integrated Disability Evaluation System (IDES) to initiate the Military Disability Process (Military Disability Made Easy 2013). The Veterans Affairs Schedule for Rating Disabilities (VASRD) rates a TMR as a 0% disability if there are no other complications (DVA 2003). Similarly, Xydakis et al. observed that "many soldiers can function well despite the perforation, provided that they have no other incapacitating injuries from the blast" (Xydakis et al. 2005, 504). However, if the TMR results in permanent conductive hearing loss, the VASRD rates that hearing loss separately (DVA 1999).

For this analysis, we equate a non-zero disability from the VASRD as a proxy for "restrictions on employment," per the language in DoDI 3200.19 (DoD 2012). Based on this argument, then, TMR in the absence of other complications (including permanent conductive hearing loss) should *not* be considered significant (unless, of course, it has already been classified as significant due to its need for HCC1+ treatment). However, hearing loss associated with a TMR may be separately rated as a non-zero disability and therefore could potentially be considered significant. We discuss this possibility below.

In the first phase of this project, we recommended hearing loss thresholds to bin PTS as significant vs. not significant (King and Cazares 2015). That analysis focused on permanent *sensorineural* hearing loss due to inner ear damage, ICD-10-CM codes H90.3–H90.5 (WHO 2016a, 380). That type of hearing loss differs from the permanent *conductive* hearing loss caused by middle ear damage, such as a TMR, coded as H90.0–H90.02 in the ICD-10-CM (WHO 2016a, 380). A patient may experience difficulties hearing different frequencies with sensorineural vs. conductive hearing loss, with sensorineural hearing loss often affecting high frequencies (OSHA

2013, 11). In our previous report (King and Cazares 2015), we recommended that JNLWD explore the possibility of developing computational models that can estimate sensorineural hearing loss at individual pure-tone frequencies. In the absence of such models, though, we focused on the mean sensorineural hearing loss averaged over all pure-tone frequencies, the metric used by the VASRD when rating a veteran's hearing disability. Therefore, we do the same for this analysis, and focus on the mean conductive hearing loss averaged over all pure-tone frequencies.

Based on this decision, we see no reason why the mean threshold for significance developed for sensorineural hearing loss in our previous report should not also be applied to *conductive* hearing loss caused by TMR. Therefore, we borrow our previous recommendations for sensorineural hearing loss (King and Cazares 2015): Combat developers should select 26 dB HL as the significance threshold for permanent *conductive* hearing loss. Per the definitions listed in DoDI 3200.19 (DoD 2012), a permanent hearing loss ≥26 dB HL is significant since such a level could restrict a person's employment or other activities for the rest of his or her life and may even require the use of hearing aids, which require HCC1+ medical care. In contrast, a permanent hearing loss ≤25 dB HL should *not* be considered significant, since the World Health Organization (WHO) categorizes this condition as "no impairment" (WHO 2016b).[2]

In footnote 14 of King and Cazares (2015, 30–31), we explained that the Auditory 4.0 computational model has been programmed to estimate $P(PTS_{\geq 25dBHL}\ occurs)$ but not $P(PTS_{\geq 26dBHL}\ occurs)$. We commented that the ramifications of using a significance threshold of 25 dB HL vs. 26 dB HL is likely to be negligible compared to the uncertainty in estimating the other quantities used to calculate RSI_{PTS}. Therefore, we now adjust our previous recommendations to state that a permanent conductive hearing loss ≥25 dB HL should be considered significant, with <25 dB HL considered *not* significant. That is,

$$P(PermanentHearingLoss_{\geq 25dBHL}\ is\ significant \mid HearingLoss_{\geq 25dBHL}\ occurred) = 1,$$

and

$$P(PermanentHearingLoss_{<25dBHL}\ is\ significant \mid PermanentHearingLoss_{<25dBHL}\ occurred) = 0.$$

Our question now becomes, what is the likelihood that a TMR will result in a permanent conductive hearing loss that is ≥25 dB HL? As discussed previously in Section 4.A.1, we have already approximated that TMRs >2 mm long (equivalent to James Class 2 and 3) are significant, since a notable percentage require HCC1+ medical treatment (i.e., surgical repair). Therefore, we do not have to consider these TMRs any further. Instead, we must only consider if the remaining TMRs (those ≤2 mm long, equivalent to James Class 1) will or will not lead to a significant permanent conductive hearing loss. Unfortunately, many challenges are associated with determining the level of permanent conductive hearing loss resulting from a blast-induced TMR.

[2] It is our understanding that audiometric test results are rounded to the nearest integer, such that clinicians quantify hearing loss as 25 dB HL or 26 dB HL but not in between.

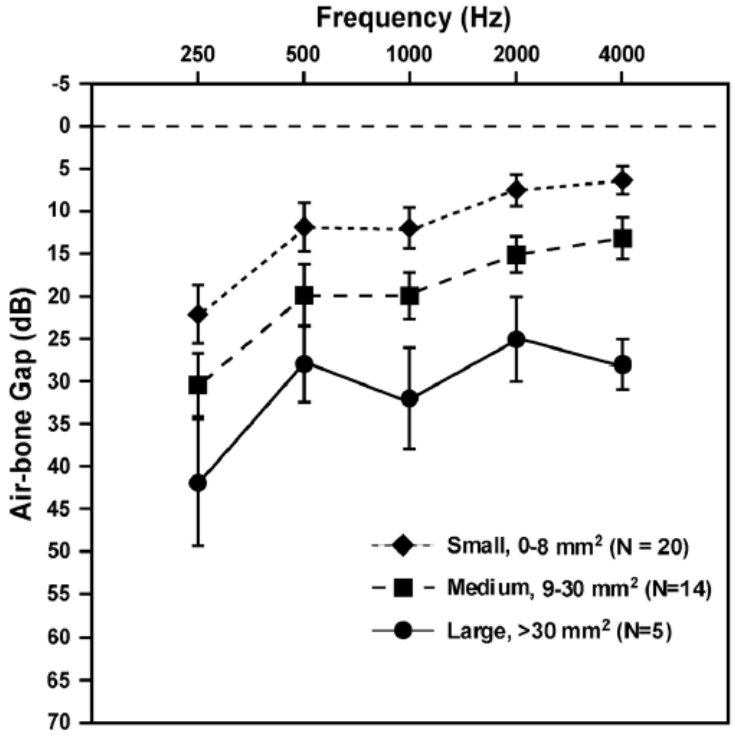
First, many studies concern bombing victims in which there is an absence of pre-injury audiograms to establish baseline hearing performance. Any hearing loss noted after the blast may have already been in place before the blast. Some studies involving blast-related ear injuries among military personnel do include baseline data (e.g., Cave, Cornish, and Chandler 2007; Ritenour et al. 2008; Breeze et al. 2011; Dougherty et al. 2013), but, again, it is difficult to find a causative link between TMR and permanent hearing loss, and, admittedly, researchers state that existing datasets are often incomplete (Cave, Cornish, and Chandler 2007; Ritenour et al. 2008; Breeze et al. 2011; Shah et al. 2014).

To complicate matters further, reports in the literature often refer to hearing loss that occurs while the TMR is still in the process of healing (e.g., Mrena et al. 2004; Mehta et al. 2006; Shah et al. 2014; Park et al. 2015) rather than the permanent hearing loss that remains after the TMR has already healed or has already been repaired via surgical intervention. Hearing loss of this nature can be thought of as a *temporary* hearing loss and, therefore, is beyond the scope of this analysis. These datasets can still be useful, though, if we assume that the permanent hearing loss that remains *after* the TMR has healed is no greater than the temporary hearing loss that occurs *before* the TMR has healed.

For example, Mehta et al. (2006) investigated 62 TMRs from 56 patients[3], binning the TMRs as small, medium, and large. Small TMRs were those ≤8 mm^2 in area. TMRs ≤2 mm long (equivalent to James Class 1) fall into this category. In contrast, medium and large TMRs were those 9–30 mm^2 and >30 mm^2 in area, respectively. Mehta et al. administered audiometric tests to quantify the patients' air-bone gap (ABG), a measure of his or her level of conductive, rather than sensorineural, hearing loss.[4] The ABG was measured at five pure tone frequencies: 250, 500, 1,000, 2,000, and 4,000 Hz. Figure 10 summarizes their results. Note that the ABG increases downwards on the vertical axis, as is the convention in audiometric tests. Error bars represent ±1 standard errors around the means. Analysis of this figure shows that when averaging over all five frequencies, small TMRs led to an average ABG (i.e., an average conductive hearing loss) of only approximately 12 dB HL, while medium and large TMRs exhibited larger ABGs of approximately 20 and 31 dB HL, respectively.

[3] Mehta et al. (2006) do not report the cause of the TMRs in these 56 patients. We speculate that few, if any, of these TMRs were caused by blast, due to the fact that the research took place at the Massachusetts Eye and Ear Infirmary, a civilian hospital. Therefore, we caution that the characteristics of the TMRs on which these results are based may not extrapolate perfectly to the TMRs caused by flashbang grenades.

[4] The pure-tone air conduction hearing test is used to determine the softest sound a person can hear at least 50% of the time over a range of different frequencies (Kutz 2016). The patient sits in a booth and listens through earphones for pure tone pulses and indicates at what intensity each pulse is heard. Bone conduction hearing tests are performed in a similar manner. However, instead of earphones, a bone conduction vibrator is placed behind the ear on the mastoid bone. The sound circumvents the outer and middle ear and travels directly through the skull to the inner ear (Meier 2016). If the air and bone conduction thresholds differ (i.e. if the air conduction threshold is ≥10 dB higher than the bone conduction threshold), then a conductive hearing loss is indicated and the difference in threshold is referred to as the air-bone gap (ABG) (Kutz 2016).



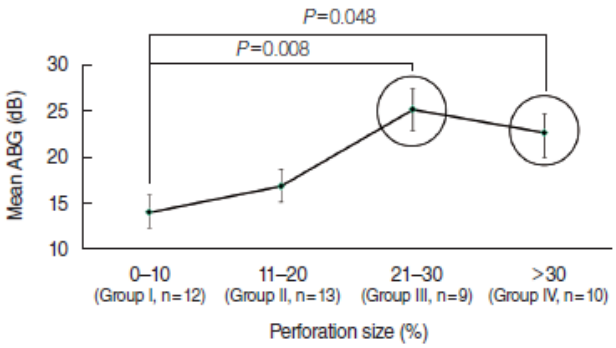
Source: Mehta et al. (2006, 13, Figure 4).

Figure 10. The Level of Conductive Hearing Loss, as Measured by the ABG, Increases with the Size of the TMR in Patients with Large Middle Ear Volumes

Park et al. (2015) showed similar results. Figure 11 reproduces their summary figure. Error bars represents ±2 standard errors around the means. Of the 42 patients in their study[5], 12 patients exhibited very small TMRs (<10% of the eardrum area) and an additional 13 patients exhibited TMRs that were only slightly larger (11–20 % of the eardrum area). We believe both sets of TMRs would likely have diameters ≤2 mm (equivalent to James Class 1). These two sets of patients exhibited an average ABG across all pure tone frequencies (i.e., an average conductive hearing loss) of approximately 14 dB HL and 16 dB HL.[6] The remaining patients exhibited larger TMRs with larger hearing losses (approximately 25 dB HL for TMRs that were 21–30% of the eardrum area and approximately 23 dB HL for TMRs that were ≥30% of the eardrum area).

[5] Park et al. (2015) do not report the cause of the TMR in these 42 patients. We speculate that few, if any, of their TMRs were caused by blast, since they enrolled their patients from the set of adult patients who underwent surgical repair of the TMR at the Seoul National University Hospital between 2010 and 2012. Therefore, we caution that the characteristics of the TMRs on which their results are based may not extrapolate perfectly to the TMRs caused by flashbang grenades.

[66] Park et al. (2015) measured the ABG at the same five frequencies as Mehta et al. (2006): 250, 500, 1,000, 2,000, and 4,000 Hz.



Source: Park et al. (2015, 94).

Figure 11. The Level of Conductive Hearing Loss, as Measured by the ABG, Increases with the Size of the TMR in a Pooled Set of Patients with Both Small and Large Middle Ear Volumes

Both Mehta et al. and Park et al. also investigated other middle ear attributes that can predict the level of conductive hearing loss in patients with TMR. Both research groups found that, contrary to popular belief, the location of the TMR did not affect the level of conductive hearing loss (Mehta et al. 2006, 14; Park et al. 2015, 95). Mehta et al. explain, "We speculate that the common clinical belief of the importance of location may have resulted from attempts to explain variations in hearing loss with similar sized perforations that were, in fact, caused by inter-ear differences in middle ear volumes" (Mehta et al. 2006, 7). Both Mehta et al. and Park et al. found that "in addition to size of a perforation, one must consider the middle-ear volume in estimating perforation-induced hearing loss. Larger middle-ear volumes are associated with smaller ABGs" (Mehta et al. 2006, 7). In particular, Mehta et al. found that the average ABGs over all pure-tone frequencies in ears with small middle ear volume (≤4.3 ml) were 10–20 dB HL larger than in ears with large volumes (>4.3 ml) (2006, 4). Park et al. showed a similar trend (2015, 94).

Unfortunately, neither Mehta et al. (2006) nor Park et al. (2015) publish data focusing specifically on the conductive hearing loss experienced by patients with small TMRs (the TMRs of interest to us in this section) and small middle ear volumes (the worst case scenario). Park et al.'s figure (reproduced here in Figure 11) appears to pool data from patients with both small and large middle ear volumes. Mehta et al.'s figure (reproduced here in Figure 10) was based on only those ears with a large middle ear volume (>4.3 ml), the best case scenario. They do not show their corresponding data for ears with a small middle ear volume (≤4.3 ml), the worst case scenario, likely due to the fact that the sample size was low. They merely state that the same trend remains: at each pure-tone frequency, average binaural level (ABL) increases with TMR size. It is possible that this unseen data may show that even small TMRs (area ≤8 mm^2, equivalent to James Class 1) have an average ABG over all pure tone frequencies that is ≥25 dB HL, surpassing our bar for significance. Mehta et al. (2006) propose use of their previously-published, physics-based model (Voss et al. 2001) to predict a patient's ABG at each pure-tone frequency based on the patient's TMR diameter and middle ear volume.

However, we note that both Mehta et al. (2006) and Park et al. (2015) presented data on the conductive hearing loss experienced by patients *before* the TMR had healed or had been surgically repaired. As Taylor and Mueller (2011) point out in their audiometry textbook, "Once the perforation heals, hearing is usually recovered fully (maybe with a slight 5- to 10-dB drop due to scarring)" (Taylor and Mueller 2011, 116–117). Therefore, even if small TMRs (equivalent to James Class 1) with small middle ear volumes (the worst case scenario) do indeed lead to significant conductive hearing loss *before* they heal, we assume that the conductive hearing loss will no longer be significant *after* they heal (which, as we found in the previous section, will occur spontaneously in all or almost all of these cases). As such, we disregard the possibility that a spontaneously-healed, blast-induced TMR with no otitis media or cholesteatoma (i.e., ≤2 mm long, equivalent to James Class 1) will result in a *permanent* hearing loss that is significant (i.e., ≥25 dB HL). That is, we approximate that the likelihood of this possibility is zero, with an uncertainty that is within the uncertainties of other quantities estimated to determine RSI_{TMR}. Therefore, the corresponding arm of the decision tree in Figure 12 is greyed out.

We now restate two of our main approximations:

- A spontaneously healed TMR (i.e., ≤2mm long, equivalent to James Class 1) will not lead to otitis media or cholesteatoma.
- A spontaneously-healed TMR with no otitis media or cholesteatoma (i.e., ≤2 mm long, equivalent to James Class 1) will not lead to a significant, permanent, conductive hearing loss (i.e., ≥25 dB HL).

Factoring in these approximations allows us to consolidate our decision tree from Figure 12 to that shown in Figure 13. All greyed-out branches of the decision tree have been eliminated since they represented likelihoods that we approximated as zero. Only one branch remains: the likelihood that the TMR will spontaneously heal, which simplifies our analysis to only one predictive attribute: size.

We can now write the following expressions:

$$P(TMR_{size>2mm} \text{ is significant} \mid TMR_{size>2mm} \text{ occurred}) = 1,$$

and

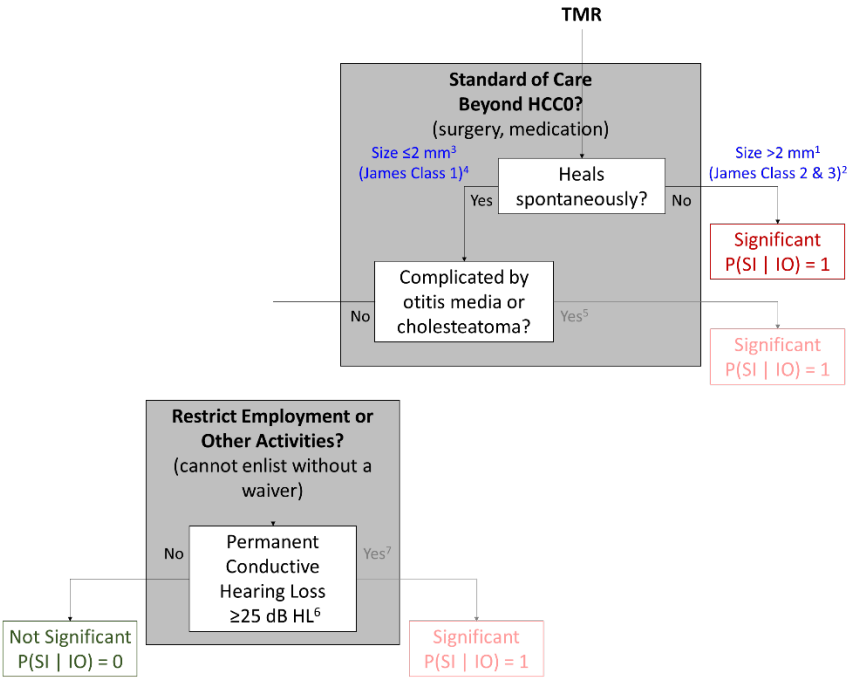
$$P(TMR_{size\leq 2mm} \text{ is significant} \mid TMR_{size\leq 2mm} \text{ occurred}) = 0,$$

which, as we have already discussed, is equivalent to

$$P(TMR_{JamesClass2\text{-}3} \text{ is significant} \mid TMR_{JamesClass2\text{-}3} \text{ occurred}) = 1,$$

and

$$P(TMR_{JamesClass1} \text{ is significant} \mid TMR_{JamesClass1} \text{ occurred}) = 0.$$



[1] A notable percentage of blast-induced TMRs >2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we err on the side of caution and approximate that TMRs this size caused by flashbang grenades require surgery and are therefore significant, i.e., P(SI | IO) = 1.

[2] The blast-induced TMRs in James Class 2 and 3 are described as "large tears, or multiple small holes or tears" and "total disrupture of the drum…", respectively (James et al. 1982, 10). Therefore, we approximate that James Class 2 and 3 TMRs are >2 mm long.

[3] Zero or almost zero blast-induced TMRs ≤2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we approximate that TMRs this size caused by flashbang grenades do not require surgery.

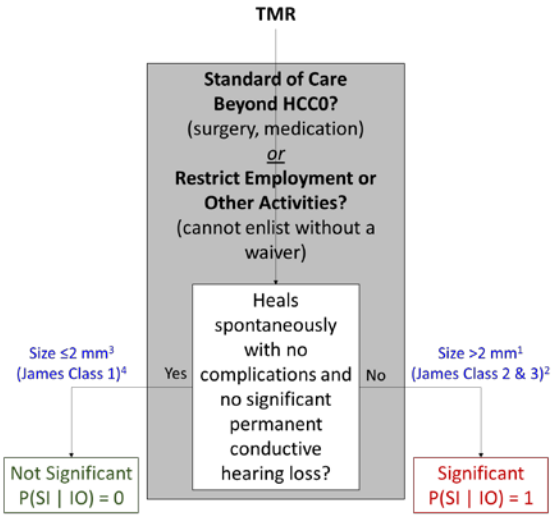
[4] The blast-induced TMRs in James Class 1 are described as "minor slits, and linear disruption of the drum fibres producing a 'meshlike' effect" (James et al. 1982, 10). Therefore, we approximate that James Class 1 TMRs are ≤2 mm long.

[5] TMR can lead to otitis media or cholesteatoma (Lou, Lou, and Zhang 2012; Ear Surgery Information Center 2015). Severe otitis media requires prescription antibiotics, beyond HCC0 (Waseem 2016). Severe cholesteatoma requires surgery, also beyond HCC0 (Ear Surgery Information Center 2015). Otitis media and cholesteatoma are associated with 8% and 7.6%, respectively, of all TMR cases (Lou, Lou, and Zhang 2012; Kronenberg et al. 1988). However, we assume that the likelihood of otitis media or cholesteatoma resulting from TMRs that heal spontaneously is even less than the already low values of 8% and 7.6% reported for all TMRs, and is approximately zero. Therefore, we disregard the possibility of this branch of the decision tree.

[6] We previously approximated that a permanent sensorineural hearing loss ≥25 dB HL was significant (King and Cazares 2015). We assume an equivalent bar for significance applies to permanent conductive hearing loss resulting from TMR.

[7] Once a TMR has healed, the conductive hearing loss resolves fully or to a very low level (<10 dB HL) (Taylor and Mueller 2011, 117). Therefore, we assume that after the TMR has healed spontaneously with no complications, the permanent conductive hearing loss remains below our ≥25 dB HL bar for significance. Therefore, we disregard the possibility of this branch of the decision tree. As a result, we approximate these TMRs as not significant, i.e., P(SI | IO) = 0.

Figure 12. Continuing to Classify a TMR as Significant: Classifying a Spontaneously Healed TMR with No Otitis Media or Cholesteatoma as Significant Based on Its Likelihood of Causing a Significant Permanent Conductive Hearing Loss



[1] A notable percentage of blast-induced TMRs >2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we err on the side of caution and approximate that TMRs this size caused by flashbang grenades require surgery and are therefore significant, i.e., P(SI | IO) = 1.

[2] The blast-induced TMRs in James Class 2 and 3 are described as "large tears, or multiple small holes or tears" and "total disrupture of the drum…", respectively (James et al. 1982, 10). Therefore, we approximate that James Class 2 and 3 TMRs are >2 mm long.

[3] Zero or almost zero blast-induced TMRs ≤2 mm long require surgery, beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008). Therefore, we approximate that TMRs this size caused by flashbang grenades do not require surgery to repair the TMR. Furthermore, we approximate a zero likelihood that a spontaneously healed TMR will lead to complications requiring medical treatment beyond HCC0, such as otitis media and cholesteatoma, since this likelihood over all TMRs is already very low at 8% and 7.6%, respectively (Lou, Lou, and Zhang 2012; Kronenberg et al. 1988). Finally, we approximate a zero likelihood that a spontaneously healed TMR will lead to a permanent conductive hearing loss ≥25 dB HL, the bar for significance we found in a previous analysis (King and Cazares 2005), since once a TMR has healed, the conductive hearing loss resolves fully or to a very low level (<10 dB HL) (Taylor and Mueller 2011, 117). Therefore, we approximate these TMRs as not significant, i.e., P(SI | IO) = 0.

[4] The blast-induced TMRs in James Class 1 are described as "minor slits, and linear disruption of the drum fibres producing a 'meshlike' effect" (James et al. 1982, 10). Therefore, we approximate that James Class 1 TMRs are ≤2 mm long.

Figure 13. Classifying a TMR as Significant Based on its Size

5. Estimating RSI for TMR and PTS

In this section, we explain how RSI_{TMR} can be calculated, based on the results of the previous two sections. We also explain how RSI_{PTS} can be calculated in a similar manner, based on the results of our previous work (King and Cazares 2015).

A. TMR

RSI_{TMR} is the likelihood (or "risk") that a significant TMR will occur. In Section 3, we reviewed the computational model that estimates $P(TMR_{type}\ occurred)$, where the TMR type is binned according to the James et al. (1982) clinical classifications: James Class 1, 2, and 3. This quantity is the first quantity needed to estimate RSI_{TMR}. Furthermore, in Section 4, we reviewed the literature on TMR and proposed our rationale for approximating $P(TMR_{type}\ is\ significant \mid TMR_{type}\ occurred)$ as either 0 or 1. This quantity is the second quantity needed to estimate RSI_{TMR}.

We initially binned the TMR type based on quantitative size measurements: ≤2 mm vs. >2 mm in length. We proposed arguments for approximating that these two bins represented TMRs that were not significant vs. significant, respectively. In addition, we then proposed our rationale for reconciling these quantitative size measurements to the James clinical classifications: James Class 1 is equivalent to ≤2 mm long (not significant) while James Classes 2 and 3 are equivalent to >2 mm long (significant).

With this reconciliation complete, we can now calculate RSI_{TMR} as the product of the two quantities, $P(TMR_{type}\ occurred)$ and $P(TMR_{type}\ is\ significant \mid TMR_{type}\ occurred)$:

$$\begin{aligned} RSI_{TMR} &= P(TMR_{JamesClass1}\ occurred) \times P(TMR_{JamesClass1}\ is\ significant \mid TMR_{JamesClass1}\ occurred)] \\ &\quad + [P(TMR_{JamesClass2\text{-}3}\ occurred) \times P(TMR_{JamesClass2\text{-}3}\ is\ significant \mid TMR_{JamesClass2\text{-}3}\ occurred)] \\ &= [P(TMR_{JamesClass1}\ occurred) \times 0] + [P(TMR_{JamesClass2\text{-}3}\ occurred) \times 1] \\ &= P(TMR_{JamesClass2\text{-}3}\ occurred). \end{aligned}$$

Shelley and Chan (2015) used the James et al. (1982) data to develop a computational model that estimates the likelihood that a blast will cause a TMR in James Class 1, 2, or 3. However, we are not concerned with the TMRs in James Class 1 since we approximate that these heal spontaneously and, based on our other assumptions and approximations, are not considered significant in our framework. We believe that it is feasible to code the Shelley and Chan model to estimate the likelihood of TMR in only James Class 2 or 3, such that this model could then directly output RSI_{TMR}.

B. PTS

RSI_{PTS} is the likelihood (or "risk") that a significant PTS will occur. Note that we consider "PTS" to refer to permanent sensorineural hearing loss due to damage to the cochlea, rather than permanent conductive hearing loss caused by TMR. We can also calculate RSI_{PTS} in a similar fashion as RSI_{TMR}. As we summarized in Section 4.B, the Auditory 4.0 computational model estimates $P(PTS_{\geq threshold}\ occurred)$ (Chan and Ho 2008; Chan, Ho, and Ryan 2016), where PTS is binned according to whether or not it is greater than or equal to some threshold. In our previous report (King and Cazares 2015), we reviewed the literature on PTS and proposed our rationale for estimating $P(PTS_{\geq threshold}\ is\ significant \mid PTS_{\geq threshold}\ occurred)$, where the PTS can be binned into two classes: <25 dB HL (not significant) vs. ≥25 dB HL (significant). We can combine the two quantities into the following expression:

$$\begin{aligned} RSI_{PTS} &= [P(PTS_{<25dBHL}\ occurred) \times P(PTS_{<25dBHL}\ is\ significant \mid PTS_{<25dBHL}\ occurred)] \\ &\quad + [P(PTS_{\geq 25dBHL}\ occurred) \times P(PTS_{\geq 25dBHL}\ is\ significant \mid PTS_{\geq 25dBHL}\ occurred)] \\ &= [P(PTS_{<25dBHL}\ occurred) \times 0] + [P(PTS_{\geq 25dBHL}\ occurred) \times 1] \\ &= P(PTS_{\geq 25dBHL}\ occurred). \end{aligned}$$

The current version of the Auditory 4.0 model can already estimate the likelihood of a PTS ≥25 dB HL, directly outputting RSI_{PTS}.

Challenges remain, however. We note that Auditory 4.0 assumes no TMR (Shelley and Chan 2015). Therefore, the RSI_{PTS} values estimated via Auditory 4.0 will not take any concomitant TMR into consideration. Similarly, the Shelley and Chan (2015) model does not consider hearing loss at all. Therefore, the RSI_{TMR} values estimated via the Shelley and Chan model will not take any concomitant permanent hearing loss into account. In the following section, we review the literature regarding the interdependency of TMR and PTS and propose suggestions for dealing with this interdependency during the development acquisition phase of a novel flashbang grenade system.

6. TMR vs. PTS

For simplicity, combat developers may be tempted to write capability documents that specify only a single RSI requirement. As a result, they would then need to formulate and calculate a single RSI metric that encapsulates the risk of causing *all* possible significant injuries. Unfortunately, calculating a single, all-encompassing RSI metric is difficult for flashbang grenades. One reason for this difficulty is that one cannot simply sum RSI_{TMR} and RSI_{PTS} due to the fact that, for blast stimuli like flashbang detonations, RSI_{TMR} and RSI_{PTS} are not independent quantities. Instead, both quantities are dependent on the same phenomenon: the blast. Simply summing together RSI_{TMR} and RSI_{PTS} would double-count the risk that a blast like a flashbang detonation will cause both TMR *and* PTS. Thus, a correction factor is needed to prevent such double-counting. The difficulty lies in estimating the value of that correction factor. In this section, we review the literature regarding the dependence between TMR and PTS. We then propose two methods for dealing with this challenge during the development acquisition phase of a novel flashbang grenade.

The ear is particularly sensitive to blast injuries. Both conductive and sensorineural injuries can result (Remenschneider et al. 2014). The conductive hearing loss resulting from blast-induced TMR is confounded with blast-induced sensorineural hearing loss. Dougherty et al. (2013) suggests that blast-induced TMR can be an indicator of hearing loss 1 year after the blast. However, they believe that the hearing loss is more a result of the blast exposure affecting inner ear structures. That is, the same blast that caused the TMR may have also caused sensorineural hearing loss (Cho et al. 2013). In Ritenour et al.'s (2008) study of spontaneous healing rates of blast-related TMR, they cited a previous study (Chait, Casler, and Zajtchuk 1989) which reported that 30% of patients exhibited high-frequency hearing loss >30 dB measured at 4,000 and 8,000 Hz 1 year after blast exposure. Upon further investigation, though, we found that the cited article was a literature review and that this result (Chait, Casler, Zajtchuk 1989, 9) was taken from Kerr and Byrne (1975, 560) which reported on *sensorineural* hearing loss following a 1972 restaurant bombing in Belfast, Ireland.

Some researchers suggest that TMR can be thought of as a protective measure that reduces sound energy transmission and, thus, damage to the inner ear (Koike et al. 2003; Helling et al. 2004). However, Kerr and Byrne (1975) state that they saw no evidence of TMR as a protective mechanism against sensorineural hearing loss. In their study of 24 patients involved in the 1972 Belfast restaurant bombing, all who suffered "serious bilateral [hearing] damage" (≥40 dB (HL) in the "speech frequencies") also had TMR (Kerr and Byrne 1975, 560). When examining the Kerr and Byrne (1975) data, Shah et al. (2014) theorize that because the explosion happened in a confined space (a restaurant), the "pattern of injury may be different than in open spaces" (Shah et al. 2014, 273). Shah et al. believe that TMR does not offer protection from hearing loss and,

although they could not link TMR to degree of hearing loss, state that blast-exposed individuals' "neurotologic symptoms may be indicative of SNHL [sensorineural hearing loss]" (2014, 276).

Based on these studies, we conclude that it is difficult to determine how much a blast-induced TMR contributes (either positively or negatively) to the PTS (i.e., the permanent sensorineural hearing loss) that was also caused by the blast. Therefore, further information is needed to combine RSI_{TMR} and RSI_{PTS} into one all-encompassing RSI metric. Combat developers can address this challenge in at least two different ways:

- Perform a series of animal experiments to elucidate how much a blast-induced TMR is associated with a blast-induced PTS. Results of this study could be used to estimate the correction factor needed to prevent double-counting the risk that a flashbang grenade causes both TMR *and* PTS. However, animal experimentation is often constrained by ethics, cost, and logistics.
- Include multiple RSI requirements into the capability documents of a novel flashbang grenade system—one requirement for RSI_{TMR}, another for RSI_{PTS}, and perhaps even other requirements for the other injuries that could potentially be caused by flashbang grenades (e.g., skin burns, blast injuries to the lung, and so forth). The flashbang system would have to meet all requirements simultaneously. Note that different thresholds could be placed on each requirement. For example, one requirement may stipulate that RSI_{PTS} must be ≤15%, meaning that the flashbang grenade must cause a PTS ≥25 dB HL in ≤15% of the target population. Simultaneously, another requirement may stipulate that RSI_{TMR} must be ≤75%, meaning that the flashbang grenade must cause a James Class 2 or 3 TMR in ≤75% of the target population. Combat developers could then track the progress of their flashbang grenade system against each requirement in parallel.

7. Conclusions

We have reviewed the literature to craft a method to estimate RSI_{TMR} during the development acquisition phase of a novel flashbang grenade system. We first reviewed an existing computational model that estimates that a blast will induce a TMR. We then reviewed the literature and proposed our rationale for approximating that moderate and major TMRs are significant while minor TMRs are not. Next, we wrote down expressions for calculating RSI_{TMR} based on the output of the computational model and our significance approximations. Finally, we commented on the confounds between TMR (and its resulting, albeit small, permanent conductive hearing loss) vs. PTS (otherwise known as permanent sensorineural hearing loss). We propose methods for simultaneously assessing a flashbang grenade's RSI_{TMR} and RSI_{PTS}. Below we list our major findings and recommendations.

A. Findings

- Shelley and Chan (2015) created a computational model that estimates the probability that a blast, such as a flashbang detonation, causes a TMR in the three clinical classifications used by James et al. (1982).
- James et al. (1982) classified blast-induced TMRs using subjective criteria, as opposed to quantitative size measurements:
  - James Class 2 and 3 TMRs are described as "large tears, or multiple small holes or tears" and "total disrupture of the drum…", respectively (James et al. 1982, 10).
  - In contrast, James Class 1 TMRs are described as "minor slits, and linear disruption of the drum fibres producing a 'meshlike' effect" (James et al. 1982, 10).
- Other researchers graded blast-induced TMRs using quantitative size measurements and showed that the size of a TMR indicates the likelihood that the TMR will require surgery beyond HCC0 (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008):
  - A notable percentage of blast-induced TMRs >2 mm long required surgery.
  - In contrast, zero (or almost zero) blast-induced TMRs ≤2 mm long required surgery.
- TMR can lead to complications such as otitis media (infection of the middle ear) or cholesteatoma (torn pieces of the TMR "seeding" in the middle ear) (Kronenberg et al. 1998; Lou, Lou, and Zhang 2012).
  - Severe otitis media requires prescription antibiotics, beyond HCC0 (Waseem 2016).

  - Severe cholesteatoma requires surgery, also beyond HCC0 (Ear Surgery Information Center 2016).
  - Otitis media and cholesteatoma are associated with 8% and 7.6%, respectively, of *all* TMR cases, averaged over both large and small TMRs (Kronenberg et al. 1988; Lou, Lou, and Zhang 2012).
- TMR can lead to conductive hearing loss (Taylor and Mueller 2011; Remenschneider et al. 2014).
- Once a TMR has healed, the conductive hearing loss resolves fully or to a very low level (<10 dB HL) (Taylor and Mueller 2011, 117).
- In a previous analysis, we investigated the significance of permanent hearing loss. We found that a permanent hearing loss ≥25 dB HL was significant, while a permanent hearing loss <25 dB HL was not significant (King and Cazares 2015).
- The literature often uses the term "PTS" to refer to permanent sensorineural hearing loss due to inner ear injury (Cave, Cornish, and Chandler 2007, 726; Rutka 2003, 3; SCENIHR, 2008, 26), rather than the permanent conductive hearing loss due to middle ear injury, such as a TMR.
- Permanent conductive hearing loss resulting from blast-induced TMR can be confounded by blast-induced permanent sensorineural hearing loss called "PTS" (Dougherty et al. 2013; Remenschneider et al. 2014). The blast that produced the TMR could have also produced the PTS (Cho et al. 2013).
- A single RSI metric for a flashbang grenade cannot be calculated by simply adding together RSI_{TMR} and RSI_{PTS}, since doing so would double-count the probability that the flashbang grenade caused both TMR and PTS.

B. Recommendations

Based on our findings, we recommend that:

- Flashbang-induced TMRs should be classified as significant vs. not significant based on the same subjective, size-based, clinical classification scheme used by James et al. (1982). Our rationale is as follows:
  - We approximate that James Class 1 TMRs are ≤2 mm long while James Class 2 and 3 TMRs are >2 mm long. Although the James clinical classifications are subjective and qualitative, we believe they can be mapped to these objective, quantitative size criteria.
  - We approximate that flashbang-induced TMRs >2 mm long (equivalent to James Class 2 and 3) require surgery, beyond HCC0, and are therefore significant. This

approximation is based on our finding that a notable percentage of these TMRs require surgery (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008).

- In contrast, we approximate that flashbang-induced TMRs ≤2 mm long (equivalent to James Class 1) are not significant, since
    - We approximate that these small TMRs do not require surgery and can heal spontaneously (HCC0). This approximation is based on our finding that zero or almost zero TMRs ≤2mm long require surgery to repair the TMR (Kronenberg, Ben-Shoshan, and Wolf 1993; Ritenour et al. 2008).
        - We approximate that these small TMRs do not lead to cases of otitis media or cholesteatoma that are severe enough to require prescription antibiotics or surgery. That is, we approximate a zero likelihood that a small, spontaneously-healed TMR will lead to otitis media or cholesteatoma, since we found that this likelihood over *all* TMRs, averaged over large and small TMRs, is already very low at 8% and 7.6%, respectively (Kronenberg et al. 1998; Lou, Lou, and Zhang 2012).
            - We approximate that these small TMRs do not lead to a significant permanent conductive hearing loss (≥25 dB HL). This approximation is based on our finding that, once the TMR has healed, hearing loss resolves fully or to a very low level (<10 dB HL) (Taylor and Mueller 2001), below our bar for significance.

- Based on our recommendations listed above, we conclude that RSI_{TMR} for a flashbang grenade should be estimated as the probability that the flashbang grenade causes a TMR in James Class 2 or 3.
- The Shelley and Chan (2015) computational model should be coded such that it outputs the probability that a blast causes a TMR in James Class 2 or 3, rather than a TMR in James Class 1, 2, or 3, so that it can directly output RSI_{TMR}.
- Combat developers should simultaneously use multiple RSI requirements for a flashbang grenade, one for each potential injury (i.e., one requirement for RSI_{TMR}, another requirement for RSI_{PTS}, and so forth). The flashbang grenade would then have to meet all RSI requirements simultaneously. Each RSI requirement could have a different threshold value.

Appendix A.
Illustrations

Figures

Tables

Appendix B. References

ANSI. 2013. *American National Standard Acoustical Terminology*. ANSI S1-1-2013. New York City, NY: Acoustical Society of America, October 14.

Breeze, J., H. Cooper, C. R. Pearson, S. Henney, and A. Reid. 2011. "Ear Injuries Sustained by British Service Personnel Subjected to Blast Trauma." *The Journal of Laryngology & Otology* 125 (01) (January): 13–17. http://dx.doi.org/10.1017/S0022215110002215.

Burgei, Wesley, Shannon Foley, Jennifer Preston, Ashley Raba, James Simonds, Thomas Dayton, Michael Jirjis, et al. 2014. *Implementation Guidance Document: General Risk of Significant Injury Equation.* JNLW14-067. Quantico, VA: Joint Non-Lethal Weapons Directorate. http://www.dtic.mil/cgi-bin/GetTRDoc?AD=ADA606576.

Cave, Kara, Elizabeth M. Cornish, and David W. Chandler. 2007. "Blast Injury of the Ear: Clinical Update from the Global War on Terror." *Military Medicine* 172 (7) (July): 726–730. http://www.ncbi.nlm.nih.gov/pubmed/17691685.

Cazares, Shelley, Allison King, Leon Hirsch, Jenny R. Holzer, and Michael Finnin. 2016. "Predicting the Significance of Injuries Potentially Caused by Non-Lethal Weapons." Presented at the *NDIA Human Systems Conference*, Springfield VA, February 9–10.

Chait, Robert H., John Casler, and Joan T. Zajtchuk. 1989. "Blast Injury to the Ear: Historical Perspective." Supplement. *Annals of Otology, Rhinology, and Laryngology* 98 (May): 9–12. http://www.ncbi.nlm.nih.gov/pubmed/2497701.

Chan, Philemon, and Kevin Ho. 2008. *Impulse Noise Injury Model.* J0910-07-330RI. San Diego, CA: L3 Communications Applied Technologies, September.

Chan, Philemon, Kevin Ho, and Allen F. Ryan. 2016. "Impulse Noise Injury Model." *Military Medicine* 181 (5): 59 – 69. http://www.ncbi.nlm.nih.gov/pubmed/27168554.

Cho, Sung-II, Simon. S. Gao, Anping Xia, Rosalie Wang, Felipe T. Salles, Patrick D. Raphael, Homer Abaya, et al. "Mechanisms of Hearing Loss after Blast Injury to the Ear." *PLoS ONE* 8 (7): e67618. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3698122.

Comeaux, James A., Thomas Dayton, and James E. Parker. 2010. *V&V Report for the Capability of Auditory M&S Version 3.0 to Support Impulse Sound Weapon Use.* San Antonio, TX: General Dynamics, June 30.

DoD. 2010. *Medical Standards for Appointment, Enlistment, or Induction in the Military Services.* Department of Defense Instruction (DoDI) 6130.03. Washington, DC: USD(P&R), April 28. Incorporating Change 1, September 13, 2011. http://dtic.mil/whs/directives/corres/pdf/613003p.pdf.

DoD. 2012. *Non-Lethal Weapons (NLW) Human Effects Characterization.* Department of Defense Instruction (DoDI) 3200.19. Washington, DC: USD(AT&L), May 17. http://www.dtic.mil/whs/directives/corres/pdf/320019p.pdf.

DoD. 2013. *DoD Executive Agent for Non-Lethal Weapons (NLW), and NLW Policy.* Department of Defense Directive (DoDD) 3000.03E. Washington, DC: USD(AT&L), April 25. http://www.dtic.mil/whs/directives/corres/pdf/300003p.pdf.

DoD. 2014. *Disability Evaluation System (DES).* Department of Defense Instruction (DoDI) 1332.18. Washington, DC: USD(P&R), August 5. http://www.dtic.mil/whs/directives/corres/pdf/133218p.pdf.

DoD. 2015. *Manual for the Operation of the Joint Capabilities Integration and Development System (JCIDS).* Washington DC: Chairman of the Joint Chiefs of Staff, February 12. https://dap.dau.mil/policy/Documents/2015/JCIDS_Manual_with_errata_through_20151218.pdf.

Dougherty, Amber L., Andrew J. MacGregor, Peggy P. Han, Erik Viirre, Kevin J. Heltemes, and Michael R. Galarneau. 2013. "Blast-Related Ear Injuries among U.S. Military Personnel." *Journal of Rehabilitation Research & Development* 50 (6) (November 6): 893–904. http://www.rehab.research.va.gov/jour/2013/506/dougherty506.html.

DVA. 1999. "4.85 – Evaluation of Hearing Impairment." *38 CFR Book C, Schedule for Rating Disabilities.* Washington, DC: DVA. http://www.benefits.va.gov/warms/docs/regs/38CFR/BOOKC/PART4/S4_85.doc.

DVA. 2003. "4.87 – Schedule of Ratings – Ear." *38 CFR Book C, Schedule for Rating Disabilities.* Washington, DC: DVA. http://www.benefits.va.gov/warms/docs/regs/38CFR/BOOKC/PART4/S4_87.doc.

Ear Surgery Information Center. 2016. *Cholesteatoma: About Surgery*. http://www.earsurgery.org/conditions/cholesteatoma/cholesteatoma-about-the-surgery/.

Farlex Partner Medical Dictionary. 2012a. "Eardrum." http://medical-dictionary.thefreedictionary.com/eardrum.

Farlex Partner Medical Dictionary. 2012b. "Otitis Media." http://medical-dictionary.thefreedictionary.com/otitis+media.

Farlex Partner Medical Dictionary. 2012c. "Permanent Threshold Shift." http://medical-dictionary.thefreedictionary.com/Permanent+Threshold+Shift.

Farlex Partner Medical Dictionary. 2012d. "Rupture of the Tympanic Membrane." http://medical-dictionary.thefreedictionary.com/rupture+of+the+tympanic+membrane.

Farlex Partner Medical Dictionary. 2012e. "Tinnitus." http://medical-dictionary.thefreedictionary.com/tinnitus.

Farlex Partner Medical Dictionary. 2012f. "Tympanic Membrane Perforation." http://medical-dictionary.thefreedictionary.com/Tympanic+membrane+perforation.

Garth, R. J. N. 1995. "Blast Injury of the Ear: An Overview and Guide to Management." *Injury* 26 (6) (July): 363–366. http://www.ncbi.nlm.nih.gov/pubmed/7558254.

Guyton, Arthur C., and John E. Hall. 1996. "The Sense of Hearing." In *Textbook of Medical Physiology*, 9th ed., 663–673. Philadelphia, PA: W.B. Saunders and Company.

Guyton, Arthur C., and John E. Hall. 2006. "The Sense of Hearing." In *Textbook of Medical Physiology*, 11th ed., 651–662. Philadelphia, PA: Elsevier, Inc.

Helling, Eric Robert. 2004. “Otologic Blast Injuries Due to the Kenya Embassy Bombing.” *Military Medicine* 169 (11) (November): 872–876. http://www.ncbi.nlm.nih.gov/pubmed/15605933.

Hirsch, Frederic G. 1966. *Effects of Overpressure on the Ear – A Review.* Technical Progress Report. Albuquerque, NM: Lovelace Foundation for Medical Education and Research, November. http://www.dtic.mil/dtic/tr/fulltext/u2/653129.pdf.

Hirsch, Leon R., Jenny Holzer, Michael Finnin, and Shelley Cazares. 2015. *Significance of Retinal Lesions Potentially Caused by Dazzling Lasers*. IDA Document D-5691. Alexandria, VA: Institute for Defense Analyses, December.

James, D. J., V. C. Pickett, K. J. Burdett, and A. Cheesman. 1982. *The Response of the Human Ear to Blast. Part 1: The Effect on the Ear Drum of a “Short” Duration, “Fast” Rising, Pressure Wave*. Joint AWRE/CDE Report No. 0-4/82. Aldermaston, UK: Atomic Weapons Research Establishment.

JNLWD. 2012. *Non-Lethal Weapons (NLW) Reference Book.* Quantico, VA: Joint Non-Lethal Weapons Directorate. http://www.dtic.mil/dtic/tr/fulltext/u2/a565971.pdf.

Kerr, A. G, and J. E. T. Byrne. 1975. “Surgery of Violence. IV. Blast Injuries of the Ear.” *British Medical Journal* 1 (5957) (March 8): 559–561. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1672656/.

King, Allison, and Shelley Cazares. 2015. *Significance of Permanent Threshold Shift Potentially Caused by Sound-Based Non-Lethal Weapons*. IDA Document D-5692. Alexandria, VA: Institute for Defense Analyses, December.

Koike, Takuji, Hiroshi Wada, Fumiaki Ito, Kazuyoshi Takayama, and Toshimitsu Kobayashi. 2003. “High Speed Video Observation of Tympanic Membrane Rupture in Guinea Pigs.” *JMSE International Journal, Series C* 46 (4): 1434–1440. https://www.jstage.jst.go.jp/article/jsmec/46/4/46_4_1434/_article.

Kronenberg, Jona, Josef Ben-Shoshan, and Michael Wolf. 1993. “Perforated Tympanic Membrane Injury after Blast.” *American Journal of Otology* 14 (1) (January): 92–94. http://www.ncbi.nlm.nih.gov/pubmed/8424486.

Kronenberg, Jona, Josef Ben-Shoshan, Micheala Modan, and George Leventon. 1988. “Blast Injury and Cholesteatoma.” *American Journal of Otology* 9 (2) (March): 127–130. http://www.ncbi.nlm.nih.gov/pubmed/3407744.

Kutz, Joe Walter. 2016. “Audiology Pure-Tone Testing.” *Medscape*. Last updated April 7. http://emedicine.medscape.com/article/1822962-overview.

Lou, Zeng-Cai, Zi-Han Lou, and Qing-Ping Zhang. 2012. “Traumatic Tympanic Mebrane Perforations: A Study of Etiology and Factors Affecting Outcome.” *American Journal of Otolaryngology* 33 (5) (September–October): 549–555. http://www.ncbi.nlm.nih.gov/pubmed/22365389.

McGraw-Hill. 2002. “Otorrhea.” *McGraw-Hill Concise Dictionary of Modern Medicine*. http://medical-dictionary.thefreedictionary.com/otorrhea.

Mehta, Ritvik P., John J. Rosowski, Susan E. Voss, Ellen O'Neil, and Saumil N. Merchant. 2006. "Determinants of Hearing Loss in Perforations of the Tympanic Membrane." *Otology & Neurotology* 27 (2) (February): 136–143. http://www.ncbi.nlm.nih.gov/pubmed/16436981.

Meier, Glen R. 2016. "Hearing Tests." *Audiology Awareness Campaign*. http://www.audiologyawareness.com/hearinfo_heartest.asp.

Military Disability Made Easy. 2013. "The Integrated Disability Evaluation System." http://www.militarydisabilitymadeeasy.com/integrateddisabilityevaluationsystem.html.

Mrena, Roderik, Rauno Pääkkönen, Leif Bäck, Ulla Pirvola, and Jukka Ylikoski. 2004. "Otologic Consequences of Blast Exposure: A Finnish Case Study of a Shopping Mall Bomb Explosion." *Acta Oto-Laryngologica* 124 (8) (October): 946–952. http://www.ncbi.nlm.nih.gov/pubmed/15513532.

Nordman, Amy S., Barbara A. Bohne, and Gary W. Harding. 2000. "Histopathological Differences between Temporary and Permanent Threshold Shift." *Hearing Research* 139 (1–2) (January): 13–30. http://www.ncbi.nlm.nih.gov/pubmed/10601709.

Orji, F. T., and C. C. Agu. 2008. "Determinants of Spontaneous Healing in Traumatic Perforations of the Tympanic Membrane." *Clinical Otolaryngology* 33 (5) (October): 420–426. http://www.ncbi.nlm.nih.gov/pubmed/18983374.

OSHA. 2013. "Noise." *OSHA Technical Manual.* Occupational Health and Safety Administration, United States Department of Labor, August 15. https://www.osha.gov/dts/osta/otm/new_noise/index.pdf.

Oxtoby, David W., Norman H. Nachtrieb, and Wade A. Freeman. 1994. *Chemistry: Science of Change.* Philadelphia, PA: Saunders College Publishing.

Park, Hanaro, Seung No Hong, Hyo Sang Kim, Jae Joon Han, Juyong Chung, Myung-Whan Seo, Seung-Ha Oh, Sun-O Chang, and Jun Ho Lee. 2015. "Determinants of Conductive Hearing Loss in Tympanic Membrane Perforation." *Clinical and Experimental Otorhinolaryngology* 8 (2) (June): 92–96. http://www.ncbi.nlm.nih.gov/pubmed/26045905.

Parker, James E., Thomas Dayton, Eric Nelson, James Stringham, and Allen J. DeNeve. 2012. *FY11 Analysis of Improved Flashbang Grenade, Engineering and Manufacturing Development Phase 5 (EMD-5) Test Devices for Blast, Flash, and Noise Injury.* AFRL-RH-FS-TR-2011-0007. Fort Sam Houston, TX: Air Force Research Laboratory, 711th Human Performance Wing, Human Effectiveness Directorate, Bioeffects Division, Human Effects Center of Excellence, January.

Persaud, R., D. Hajioff, M. Wareing, and E. Cheveretton. 2003. "Otological Trauma Resulting from the Soho Nail Bomb in London, April 1999." C*linical Otolaryngology* 28 (3) (June): 203–206. http://www.ncbi.nlm.nih.gov/pubmed/12755756.

Remenschneider, Aron K., Sarah Lookabaugh, Avner Aliphas, Jacob. R. Brodsky, Anand K. Devaiah, Walid Dagher, Kenneth M. Grundfast, et al. 2014. "Otologic Outcomes after Blast Injury: The Boston Marathon Experience." *Otology & Neurotology* 35 (10) (December): 1825–1834. http://www.ncbi.nlm.nih.gov/pubmed/25393974.

Ritenour, Amber E., Aaron Wickley, Joshua S. Ritenour, Brian R. Kriete, Lorne H. Blackbourne, John B. Holcomb, and Charles E. Wade. 2008. "Tympanic Membrane Perforation and Hearing Loss from Blast Overpressure in Operation Enduring Freedom and Operation Iraqi Freedom Wounded." Supplement. *The Journal of Trauma, Infection, and Critical Care* 64 (2) (February): S174–S178. http://www.ncbi.nlm.nih.gov/pubmed/18376162.

Rutka, John. 2013. *Hearing Loss and Tinnitus.* Discussion Paper. Ontario, Canada: The Workplace Safety and Insurance Appeals Tribunal, July. http://www.wsiat.on.ca/english/mlo/hearing_loss.htm.

SCENIHR. 2008. *Potential Health Risks of Exposure to Noise from Personal Music Players and Mobile Phones Including a Music Playing Function*. Preliminary Report. Brussels, Belgium: European Commission, Directorate-General for Health and Consumers. http://ec.europa.eu/health/archive/ph_risk/committees/04_scenihr/docs/scenihr_o_017.pdf.

Seaman, Richard W., and Robert C. Newell. 1971. "Another Etiology of Middle Ear Cholesteatoma." *Archives of Otolaryngology* 94 (5) (November 1): 440–442. http://archotol.jamanetwork.com/article.aspx?articleid=603866.

Shah, Anil, Marco Ayala, Gregory Capra, David Fox, and Michael Hoffer. 2014. "Otologic Assessment of Blast and Nonblast Injury in Returning Middle East-Deployed Service Members." *The Laryngoscope* 124 (1) (January): 272–277. http://www.ncbi.nlm.nih.gov/pubmed/23686673.

Shelley, David, and Philemon Chan. 2015. *Eardrum Rupture Phase-1 Final Report.* J0258-15-680. San Diego, CA: L3 Applied Technologies, August 11.

Singer, Neal. 2002. "Reusable Explosive Device to Help Law Enforcement." News Release. Albuquerque, NM: Sandia National Laboratories, April 3. http://www.sandia.gov/media/NewsRel/NR2002/flash.htm.

Taylor, Brian, and H. Gustav Mueller. 2011. *Fitting and Dispensing Hearing Aids.* San Diego, CA: Plural Publishing, Inc.

van der Willigen, Rob. 2007. *Auditory Perception.* Nijmegen, Netherlands: Radboud University. http://www.mbfys.ru.nl/~robvdw/DGCN22/COLLEGES/2010/P13/AudPerc_2008_P4_V13.ppt.

van der Willigen, Rob. 2008. *Auditory Perception.* Nijmegen, Netherlands: Radboud University. http://www.mbfys.ru.nl/~robvdw/DGCN22/COLLEGES/2010/P4/AudPerc_2008_P4_Anatomy_Physiology_V2.ppt.

Voss, Susan E., John J. Rosowski, Saumil N. Merchant, and William T. Peake. 2001. "Middle-ear Function with Tympanic-membrane Perforations. II. A Simple Model." *Journal of the Acoustic Society of America* 110 (3): 1445 – 1452. http://www.ncbi.nlm.nih.gov/pubmed/11572355

Waseem. 2016. "Otitis Media – Management." *Medscape*. Last updated March 15. http://emedicine.medscape.com/article/994656-overview.

WHO. 2016a. *International Classification of Diseases, 10th edition, Clinical Modification*. Atlanta, GA: National Center for Health Statistics, Center for Disease Control and Prevention. Last updated April 29. http://www.cdc.gov/nchs/icd/icd10cm.htm#icd2016.

WHO. 2016b. “Prevention of Blindness and Deafness: Grades of Hearing Impairment.” http://www.who.int/pbd/deafness/hearing_impairment_grades/en/.

Xydakis, Michael S., Michael D. Fravell, Katherine E. Nassar, and John D. Casler. 2005. “Analysis of Battlefield Head and Neck Injuries in Iran and Afghanistan.” *Otolaryngology-Head and Neck Surgery* 133 (4) (October): 497–504. http://www.ncbi.nlm.nih.gov/pubmed/16213918.

Appendix C. Abbreviations

ABG	air-bone gap
ABL	average binaural level
ANSI	American National Standards Institute
CM	Clinical Modification
CFR	Code of Federal Regulations
dB	decibel
DoD	Department of Defense
DoDD	Department of Defense Directive
DoDI	Department of Defense Instruction
DVA	U.S. Department of Veterans Affairs
HCC	health care capability
HCC0	HCC Index 0
HCC1	HCC Index 1
HCC2	HCC Index 2
HL	hearing loss
ICD	International Classification of Diseases
IDA	Institute for Defense Analyses
IDES	Integrated Disability Evaluation System
JNLWD	Joint Non-Lethal Weapons Directorate
JSME	Japan Society of Mechanical Engineers
KPP	key performance parameter
KSA	key system attribute
NLW	Non-Lethal Weapon
Pa	Pascals
PTS	Permanent Threshold Shift
RSI	Risk of Significant Injury
SCENIHR	Scientific Committee on Emerging and Newly Identified Health Risks
SNHL	sensorineural hearing loss
SPL	sound pressure level
TMR	tympanic membrane rupture
TTS	Temporary Threshold Shift
USD(AT&L)	Under Secretary of Defense for Acquisition, Technology and Logistics
USD(P&R)	Under Secretary of Defense for Personnel and Readiness
VASRD	Veterans Affairs Schedule for Rating Disabilities
WHO	World Health Organization

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14. ABSTRACT

Flashbang grenades are Non-Lethal Weapons (NLWs) that employ loud sounds intended to temporarily suppress the targeted personnel while minimizing permanent injuries. However, there is a risk that these loud sounds can lead to Tympanic Membrane Rupture (TMR), a burst eardrum. Per Department of Defense Instruction 3200.19, an injury caused by a NLW, such as a TMR, is considered significant if "self-aid, buddy-aid, and combat lifesaver skills" (p. 13) are insufficient to treat the injury and/or if the injury results in death or "physical damage that restricts the employment or other activities of the person for the rest of his or her life" (p. 14). A novel NLW system's Risk of [Causing] Significant Injury (RSI) must be quantified during acquisition. The objective of this project was to search the relevant literature to identify attributes of a TMR that can quantitatively, accurately, and precisely predict the significance of the TMR and then determine how those predictive attributes could be used to quantify a novel flashbang grenade's RSI during acquisition. We recommend that NLW developers define a TMR as "significant" if the tear spans more than 2 mm of the membrane diameter and "not significant" if the tear spans 2 mm or less. We also recommend that an existing computational model be reconstructed such that it outputs the probability that a blast causes a moderate or major TMR (i.e., one spanning more than 2 mm), rather than simply any TMR. This quantity could then be used as an estimate of a novel flashbang grenade's risk of causing a significant TMR.

15. SUBJECT TERMS

Non-Lethal Weapon, NLW, Less Lethal Weapon, Less Than Lethal Weapon, Flashbang, Blast, Tympanic Membrane, Tympanic Membrane Rupture, TMR, Tympanic Membrane Perforation, TMP, eardrum, otitis media, cholesteatoma, hearing loss, Permanent Threshold Shift, PTS, Risk of Significant Injury, RSI, Health Care Capability, HCC

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