# Chapter 67
# Traumatic Brain Injury and Blast Exposures: Auditory and Vestibular Pathology

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**Keywords** Tinnitus • Traumatic brain injury • Tinnitus • Vestibular disorders • Hearing loss

**Abbreviations**

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| mTHB | Mild traumatic brain injury |
| TBI | Traumatic brain injury |

## Introduction

Brain injury has been associated with a variety of neurologic sequelae including the auditory symptoms of hearing loss and tinnitus. Traditionally, we think of brain injury as being secondary to head impact and classify the resultant neurologic damage as mild, moderate, or severe [1]. This classification depends on a variety of factors including length of alteration of consciousness, force of the impact, associated injuries, and neuropathology (such as bleeding). This classification is important since it guides management of the injury and gives health care providers some information about the expected pathologies and best practices for management. There has been a great deal of work done over the years on blunt head injury; however, not all brain injury is secondary to blunt head impact.

The most common etiology of injuries in modern warfare is blast exposure. The use of explosives for terrorism has extended this threat to the civilian world. Such as blunt injury, blast exposure can produce traumatic brain injury. This chapter describes differences between blast injury and blunt head injury from a clinical perspective. We will then consider the audiologic sequelae of blast injury, including tinnitus.

## Pathophysiological Features of Traumatic Brain Injury

A heuristic diagram for understanding the progression of signs and symptoms of traumatic brain injury is shown in Fig. 67.1.

1. The direct injury to the brain is presumed to be the "textbook" neuropathological hallmarks of concussive brain injury, which include subdural hematoma, cerebral contusion, and subarachnoid hematoma.
2. The subdural hematoma can be delayed, emerging later in subacute or chronic stages after injury [1]. Tissue injury responses include, at the cellular level, cellular repair and metabolic pathways and, at the tissue level, wound healing and vascular regulatory responses. Secondary damage includes ischemia and excitotoxic events that reflect imbalances in homeostatic control of both the intracellular and extracellular environments. Plasticity of intact neuronal pathways can also contribute to recovery. The outcomes (functional recovery and permanent functional loss) will obviously depend upon the severity (and location) of the primary trauma and the efficacy of the biological responses to the primary and secondary damage. The signs and symptoms of a patient at any given time will reflect the interplay

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A.R. Møller et al. (eds.), *Textbook of Tinnitus*,
DOI 10.1007/978-1-60761-145-5_67, 

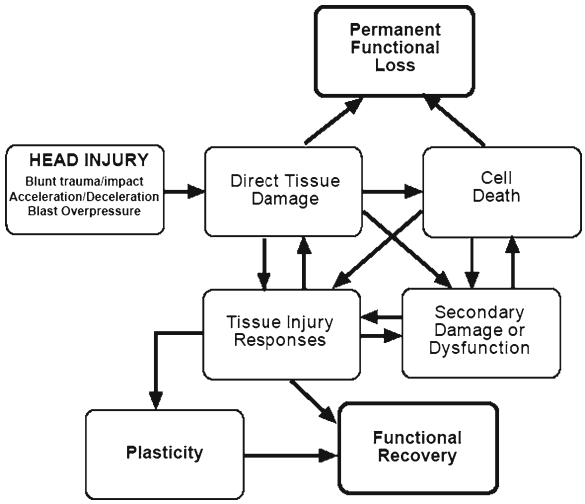


**Fig. 67.1** Schematic representation of the development of neurological and otoneurological signs and symptoms after head injury from blunt and/or blast exposure. Direct damage to intracranial (brain, blood vessels, and meninges) and extracranial tissues triggers sequences of downstream injury and recovery processes that result in an evolving clinical presentation during the acute and subacute post-injury periods. These multiple processes can contribute to tinnitus

between these dynamic mechanisms. One example of this approach is the growing recognition that subarachnoid hemorrhage can contribute to both early and delayed mechanisms of secondary brain injury, including vasospasm, transient ischemia, oxidative stress, excitotoxicity, cortical spreading depression, microcirculatory dysfunction, and delayed thromboembolism [2–4].

## An Introduction to Blast Injury

A shock wave, a blast wind, and an electromagnetic pulse are generated by detonations of explosives. Primary blast injury is defined as the effects of shock wave propagation through tissue. The blast front is a supersonic over-pressure wave, followed immediately by a negative pressure component termed "the under-pressure" [5]. The blast wave produces a positive–negative shift in intracranial pressure that mirrors the incident waveform [6–8]. Unlike primary blunt or acceleration–deceleration brain trauma, low-level blast exposure produces a global compression–decompression of the cranial contents rather than localized brain contusions from impact with the skull. Secondary blast injury is produced by shrapnel or fragments. Tertiary blast injury can produce blunt trauma by impact with objects in the environment. Quaternary blast injury is produced by other detonation products such as heat, electromagnetic pulses, and detonation toxins.

## Clinical Contrasts: Neurologic Aspects of Mild Blast Trauma Vs. Mild Blunt Head Trauma

A clinical picture is now emerging from a series of studies conducted with active duty military personnel who sustained a mild traumatic brain injury (mTBI) as a consequence of pure blunt head injury or pure blast head injury. These studies have been presented in detail in other publications, but will be summarized here [9, 10]. The mTBI was defined by the Department of Defense Policy for Mild Traumatic Brain Injury (October 2007) criteria as the presence of a documented head trauma or blast exposure event followed by a change in mental status, which could include nausea, dizziness/balance problems, temporary headache, sensitivity to noise or light, tinnitus, vomiting, fatigue, insomnia/sleep disturbances, drowsiness, blurred vision, memory problems, or poor concentration. One study examined males with purely blunt head injury during service in Iraq (34 individuals) or with purely blast injury during service in Iraq (21 individuals) within 9 months of injury. The clinical characteristics varied markedly between the blunt and blast-exposed patients. Specifically, the blast mTBI group had a much higher prevalence of clinically significant hearing loss (43% vs. 7% of the blunt head injury group) and cognitive impairment (90% vs. 17% of the blunt head injury group). Rotational chair balance test results also suggested a different pattern of functional impairment in the two groups with more unilateral, peripheral vestibular symptoms in the blunt group than the blast group [9, 10]. A second study used dynamic posturography to assess postural control after mTBI. The 33 blunt head injury patients and 39 blast injury patients in this study all received mTBI in Iraq and entered the study within 9 months after injury. There was a significant difference in the sensory organization test results of a portion of the blunt patients as compared to all the blast patients. The group mean scores of the motor

control test of the patients with blast injuries were markedly worse than the group mean score of the patients who had suffered blunt injuries [9, 10]. In summary, our laboratory results demonstrate that the head injury and resultant sequelae seen after blast injuries are markedly different than those seen after blunt head injury. The implication of these findings is that we cannot utilize our decades of knowledge on blunt head injury to predict the pathologies or discern the best management practices in individuals with blast exposure.

## Auditory Pathology after Blast Exposure

The rate of hearing loss, documented by pure-tone audiometry, increases slightly as a function of time from injury to presentation in individuals with blast exposure and resultant head trauma [9]. Tinnitus was noted initially by from 33% and in 43% of those seen later than 1 month after their most recent blast exposure. However, the occurrence of tinnitus is greater than the occurrence of hearing loss in both groups. Nearly 70% of individuals with documented mild traumatic brain injury report tinnitus in the first 72 h after the blast. This number decreases over time, but the rate of tinnitus exceeds the rate of hearing loss at all time points.

There are many factors that might account for the tinnitus seen after blast injury in our mild traumatic brain injury population. Of course, in many individuals, the tinnitus occurs along with the hearing loss, and the postulated etiology would be from primary damage to the ear. However, as stated earlier, tinnitus is more common than hearing loss, and many individuals who have been exposed to a blast wave have normal pure-tone hearing tests but show abnormalities in hearing noise and in central auditory processing. Most of these individuals complain of tinnitus despite their aforementioned normal audiograms. In this regard, it is critical to note that the subjective tinnitus can be produced by mechanisms that range from localized disturbances in the peripheral auditory system and central nervous system to systemic metabolic disturbances [11, 12]. Particularly, germane to blast TBI is the association of tinnitus with stroke and cerebral hemorrhage [11, 13–16]. Somatic tinnitus can accompany acceleration–deceleration injuries, such as whiplash, in the absence of hearing loss [17]. Blast injury is also associated with a higher than expected rate of posttraumatic Ménière's disease. Thus, it is quite possible that the central and peripheral sequelae of blast injury produce tinnitus independent of direct ear damage. These factors may contribute to a higher than expected rate of tinnitus and suggest the need for more comprehensive diagnostic tests and a broader range of therapeutic approaches. At the same time, this may allow us to intervene specifically in the primary etiology of the tinnitus and/or more effectively manage the tinnitus after it develops.

## Conclusions

Ultimately, the tinnitus seen after blast exposure and brain injury is likely multi-factorial and a product of end organ damage, brain injury, and/or a pathology that develops over time. Several factors remain unclear. We have very little data documenting the rate of tinnitus in those with blast exposure who do not have resultant mild traumatic brain injury. Given the rate of blast exposure in current operational settings, this is a very important piece of information. Also, while we have candidate pathologies to account for the tinnitus seen in blast-exposed individuals with mTBI, we still have a great deal of work to do in this area. More targeted and specific tinnitus tests need to be done on this population. We are obligated to better characterize the disorder, so that we can help develop diagnostic and management strategies to initially treat and, in the future, prevent tinnitus associated with blast exposure.

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