Synopsis of Causation

Blast Injury of the Ear

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Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

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1. Definition

1.1. The term **acute acoustic trauma** is often used to encompass injury to the ear due to **blast** or to **impulse noise**.

1.2. **Blast injury of the ear** (*Syn.* blast overpressure injury to the ear) The classification of blast injury of the ear is inconsistent in the literature, but the term encompasses the various effects of explosive forces on the <u>auditory</u> system and the following categorisation is frequently employed:

   1.2.1. **Explosive blast injury of the ear** This term refers to otological trauma due to the detonation of explosives.

   1.2.2. **Non-explosive blast injury to the ear** Here, otological trauma is caused by a blow to the ear that seals the <u>external auditory meatus</u>, causing a shock wave to be transmitted along the <u>auditory canal</u>.

1.3. **Ear injury due to impulse noise** Impulse noise is also inconsistently defined, and there is no single uniformly accepted method for its measurement, but in general it is taken to mean single or slowly repeated very loud sounds which are not associated with blast. One recommendation is that an impulse noise may be defined as a single-pulse sound or a burst of sound with a duration of between 0.001 and 1 second.[1] Impulse noise is typified by a sound, typically equal to or greater than 140dB, which rapidly rises to a sharp peak and then quickly fades. However, impulses which occur in very rapid succession (such as with some jack hammers), would be regarded as chronic noise rather than impulse noise and in some cases the distinction is difficult to make.

1.4. **Noise induced hearing loss** (NIHL) in contrast, develops over a period of several years as a result of exposure to continuous or intermittent loud noise. Damage from chronic exposure to high ambient sound levels is cumulative, and the onset of persistent hearing loss is usually gradual. The Synopsis *Sensorineural Hearing Loss* deals with chronic noise exposure in more detail and the topic is not considered further in this Synopsis.

1.5. **Barotrauma** Otological injury due to pressure change is generally referred to as barotrauma. These cases are usually distinguished from those attributable to explosive blast or impulse noise. In barotrauma, damage to the ear is caused by such events as a change in atmospheric pressure, due to diving, travel in an aircraft, etc., or by a rapid change in ear pressure in a normal atmosphere, which may occur in sneezing, heavy lifting, etc. These cases are not considered further in this Synopsis.

2. Clinical features

2.1. **Explosive blast injury** As an air-filled organ the ear is most susceptible to blast injury, and exposure to the detonation of explosives may result in severe auditory and vestibular disturbance.

2.1.1. **The external ear** (the pinna and external auditory canal) is unlikely to be damaged by the shock wave, but flying debris may cause secondary missile injuries, and amputation of the ear lobe may occur in close-proximity blast.[2]

2.1.2. **The tympanic membrane** is stretched and displaced by blast, and may be perforated in one or more places, sometimes with the formation of everted or inverted flaps. Perforations may be punched out, ragged, linear or multiple and occasionally the tympanic membrane is destroyed completely.[15,3] It may however remain intact. In one report of terrorist bombing in Northern Ireland, 45% of those who died had rupture of the tympanic membrane.[4] Discharge or bleeding from the ears may or may not be present. Small tissue fragments from the outer surface of the membrane can be propelled into the inner ear where in some 12% of cases they may form destructive masses of debris known as cholesteatoma.[5]

2.1.3. Perforation of the tympanic membrane and disruption of the ossicles may give some degree of protection to the cochlea as damaging force is not transmitted to the inner ear. Paradoxically therefore less cochlear damage may sometimes occur after larger blasts.[6,6]

2.1.4. **Lateralisation** Usually, both ears are affected, but the ear closest to the source of the explosion is likely to be more severely affected. Lateralisation of otological damage is however made more complex by the reflection of blast by surrounding structures, and in one incident where a nail bomb was detonated inside a closed building, unilateral tympanic membrane perforation occurred in two patients in the ear furthest from the blast. This was attributed to reflection from a nearby wall.[7]

2.1.5. **Pain** Acute pain in the ear is common, following exposure to blast. This is usually temporary but pain or discomfort may last up to several weeks.

2.1.6. **Ossicular damage** There may be damage to the ossicles in the middle ear, and dislocation of the incudo-stapedial joint is the commonest injury in this location. Where there is total functional disruption of the ossicular chain the loss of air conduction usually exceeds 50dB.[8]

2.1.7. **Damage to the inner ear** Any exposure to blast that has damaged the tympanic membrane or ossicles is likely to have damaged the sensory epithelium of the inner ear also. The injury is mainly of a mechanical type, affecting the basilar membrane of the inner ear. Detachment of the sensory cells from the basilar membrane is induced by excessive displacement of the basilar membrane.

2.1.8. Even without injury to the tympanic membrane and ossicles, most victims will suffer marked hearing loss ("temporary threshold shift") following a blast, often accompanied by tinnitus. Usually, a proportion of this hearing loss resolves quickly, but as many as 30-55% of cases experience some permanent loss, which may be sensorineural, conductive, or mixed. Persistent tinnitus is not unusual and may have important

functional and psychological consequences.[14,19,2]

2.1.9. **Vestibular and balance function** Reports of blast-related disequilibrium are inconsistent. In some incidents of blast the number of victims complaining of dizziness and vertigo were small, and the symptoms regressed quickly. An investigation of victims of the Oklahoma City bombing survivors however found that 72% of survivors had some persisting vertigo, dizziness or imbalance.[9]

2.2. **Non-explosive blast injury** The effects of this not uncommon injury differ from explosive blast injury only in that they are usually much less severe. Short term hearing loss, vertigo and tinnitus are usual but ossicular damage is rare.[10,11] Limited trauma to the inner ear is the usual consequence of this type of injury. Where the injury is associated with water sports, contamination of the middle ear frequently occurs, resulting in post-traumatic suppurative otitis media.

2.3. **Injury due to impulse noise** The commonest effects of exposure to impulse noise are sensorineural hearing loss and tinnitus.

2.3.1. There is experimental evidence to suggest that the cochlear damage which occurs in response to impulse noise comprises both metabolic and mechanical components. Furthermore, a conditioning effect appears to exist, whereby prior noise exposures may diminish the severity of any permanent threshold shift from subsequent high intensity impulse noise exposures. However the mechanism of this effect is not clear, although it is thought that such a conditioning response may be attributable to an adaptive alteration in cochlear mechanics.[12]

2.3.2. In addition, experimental work has demonstrated that recovery of hearing following exposure to impulse noise is non-linear. Recovery begins immediately after exposure to impulse noise but stops after 30-60 minutes. Hearing loss then increases once more until 5 to 12 hours have elapsed, whereupon hearing recovery resumes and eventually reaches some stable level. It was thought that this triphasic process reflected mechanical damage to the cochlea, and histological studies have confirmed this relationship.[21]

2.3.3. It appears from early research that the cochlear damage resulting from exposure to impulse noise relates not only to the loudness of the noise but also to its duration and waveform,[13] and that there is probably a critical level of impulse noise above which cochlear damage occurs that encompasses all these factors.

3. Aetiology

3.1. **Explosive blast injury** Primary explosive blast injury may occur in military and civilian detonations and from the firing of weapon systems. In blast, an almost instantaneous wave of overpressure travels at a velocity greater than that of sound, followed by a region of gas flow. This positive-pressure phase lasts only a few milliseconds and is followed by a longer negative-pressure phase, which may last for tens or hundreds of milliseconds. A blast occurring outdoors unmodified by nearby structures produces a characteristic pressure-time tracing known as a Friedlander wave.

3.1.1. In practice, the waveform of blasts is often very complex as the blast wave is modified by reflections and rarefactions, and the heating of gases within a restricted space may result in a more sustained period of positive pressure.

3.1.2. The orientation of the auditory canal to the blast wave has a significant effect on the characteristics of the wave as it travels within the canal, and it has been shown experimentally that there is a lower peak overpressure at the drum when the wave incidence is at an angle to the canal. The pressure distribution of sound waves within the canal has been shown experimentally to vary considerably on reaching the surface of the tympanic membrane and it is probable that a blast wave possesses similar characteristics. Furthermore, due to uneven stress distribution and normal biological variability, apparently similar explosions may result in otological injuries of widely varying severity.[14,15,16] For example, it is clear from experimental work that in addition to the peak sound pressure level, the rate of pressure increase and the characteristics of the pressure waveform are important variables in producing rupture of the tympanic membrane.[16]

3.1.3. Blast overpressure is usually distinguished from **impulse noise**. The essential difference lies in the shape, magnitude and velocity of the pressure impulse. Although the distinction is arbitrary, the following criteria are helpful:[17]

- Blast waves involve the movement of considerable volumes of combustion products and air

- In blast, peak overpressures often reach tens of kilopascals (kPa) while in impulse noise they are usually less than 2 kPa

- Impulse noise often contains low-frequency mechanical clatter

- The victim of an explosive blast will often give a history of being lifted up or bowled over

3.1.4. Both ears are usually affected by exposure to blast, but the lateralisation of injury depends on the angle of the ear canals to the pressure wave, as discussed above, and the manner in which the blast is modified by reflections from surrounding surfaces. These factors have a significant effect on the severity of the damage sustained by the ear.[18] Unconfined free-field blasts are likely to cause significantly less injury than those occurring within an enclosed space, such as a military vehicle or the inside of a building.

3.1.5. **Nature of the explosive** In an explosion, the conversion of solid or liquid material into a gaseous form is accompanied by the release of large amounts of energy. The characteristics of this process differ according to the explosive involved, and agents such as gunpowder release energy slowly, while high-energy explosives ('high explosives') such as trinitrotoluene (TNT) produce a very rapid release of energy.[19]

3.2. **Non-explosive blast injury** Non-explosive blast injury is due to the sudden increase of air pressure within the external auditory canal which strikes the tympanic membrane. In one series of 91 cases of non-explosive blast injury the authors note that 60 were caused by a slap or a fist, 13 occurred during sporting activities (mainly ball games), and 18 during swimming or water sports. In all cases there was acute perforation of the tympanic membrane.[20]

3.3. **Injury due to impulse noise** Peak overpressures from extremely loud acoustic waves are generally less than 0.3kPa, whereas it has been estimated that 8-56 kPa is required to rupture the human tympanic membrane. Nevertheless impulse noise may cause acoustic damage, and tinnitus, pain and hearing loss are not uncommon following exposure to extremely loud noise. Examples of sources of impulse noise include commercial blasting, sonic boom, muzzle blast and backfire from an internal combustion engine.[21] A single impulse, for example a gunshot or firecracker, may only result in a temporary impairment of hearing (temporary threshold shift, TTS) or may be sufficient to produce a permanent audiometric change (permanent threshold shift, PTS) in a sensitive ear. However predicting susceptibility to otological damage from such an exposure is not yet possible.

3.3.1. Risk criteria for impulse noise are not yet internationally established, and the situation is further complicated by the effect of superadded chronic exposure to steady state noise. It has been demonstrated that when impact noise is superimposed on continuous noise, the injurious potential is synergistically enhanced.[22]

4. Prognosis

4.1. Hearing loss and tinnitus usually follow exposure to blast or impulse noise. This may resolve quickly but may in some cases persist, with a proportion of victims experiencing permanent sensorineural, conductive or mixed hearing loss and tinnitus. It is estimated that permanent loss may occur in as many as 50% of victims of explosive blast and 20% of cases of non-explosive blast. Although high-frequency sensorineural loss is common, and inversely related to the individual's distance from an explosive blast, it rarely causes objective symptoms. While a 4kHz dip in audiometric threshold is common in noise-induced hearing loss, this does not appear to be a characteristic of patients exposed to acute acoustic trauma.[7] Tinnitus often resolves as hearing recovers, but it may persist in some cases.[23]

4.2.   In about 50-80% of cases of acute acoustic trauma, perforations of the tympanic membrane will heal spontaneously.[15,3,24,25] Large and central perforations have the least tendency to heal and usually require surgical intervention. In one series of 147 patients (210 affected ears) the rate of spontaneous healing of tympanic membrane perforations was evaluated in relation to time after blast injury and the size and location of perforations. Spontaneous healing was seen in 155 ears (74%); 131 (62%) healed within the first 3 months and 145 ears (69%) healed within 10 months. 32 patients were treated by immediate patching of the perforated drum and of these 84% healed.[26] Surgical intervention is usually undertaken when the tympanic membrane remains unhealed for 10-12 months. Some 3% of cases of explosive blast exposure develop chronic otitis media, which may pursue a prolonged and refractory course

4.3. Attempts have been made to reduce the degree of permanent hearing loss after blast injury by the use of corticosteroids, low molecular weight dextran, magnesium, vasodilators and other therapeutic agents, and by the use of hyperbaric oxygen but evidence of their effectiveness is limited.

4.4. It is generally accepted that, providing there has been no further exposure to harmful noise, further deterioration in hearing is unlikely to be attributable to the initial injury a year after an episode of acute acoustic trauma,[27] and any residual loss is considered permanent. If however there is continued exposure to noise following such an event it is likely to be accompanied by a progressive deterioration in audiometric findings.[27]

5. Summary

5.1. Blast injury to the ear (acute acoustic trauma) may be caused by explosive or non-explosive forces, or by impulsive sound. The resulting injuries may include perforation of the tympanic membrane, damage to middle ear structures and injury to the inner ear.

5.2. Short term hearing loss and tinnitus are the commonest effects. A proportion of cases suffer permanent hearing loss which may be sensorineural, conductive or mixed.

5.3. No further deterioration is likely once a year has elapsed since an episode of acute acoustic trauma.

6. Related Synopses

Conductive Hearing Loss

Otitis Externa

Otosclerosis

Sensorineural Hearing Loss

Vertigo

7.   Glossary

auditory	Pertaining to the hearing mechanisms of the body. (External) auditory canal: the passage leading to the ear drum.
basilar membrane	A thin layer of tissue in the cochlea which supports the organ of Corti, the assembly of cells that transmit sound vibrations to the nerve fibres.
cochlea	The inner ear.
conductive deafness	Hearing impairment caused by interference with sound transmission through the external canal and middle ear.
external auditory meatus	Outer opening of the auditory canal.
incudo-stapedial joint	The joint between two of the *ossicles* (q.v.) of the middle ear; the incus and the stapes.
kilopascal (kPa)	A unit of pressure.
mixed hearing loss	Hearing loss which involves a combination of conductive and sensorineural factors (*qv*).
ossicles, ossicular chain	A chain of three small bones which traverse the middle ear and transmit vibration from the tympanic membrane to the inner ear.
overpressure	A transient air pressure greater than the surrounding atmospheric pressure (average 101.325 kilopascals).
sensorineural deafness	Hearing impairment due to damage to or disorders of the auditory nerve.
tinnitus	A sensation of high-pitched whistling or hissing in the ears.
tympanic membrane	The ear drum.
vestibular	Relating to the vestibular apparatus; the organ of balance.

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