Noise Induced Hearing Loss

**DEFINITION**

- The term noise-induced hearing loss refers to reduction in auditory acuity associated with noise exposure.
- May be **TTS** (Temporary Threshold Shift) or **PTS** (Permanent Threshold Shift).
- **TTS** can be from hours to days.
- **PTS** can be because of repeated attacks of TTS or single loud noise exposure.
- The term 'acoustic trauma' has, however, been utilized to describe the situation where a single exposure to an intense sound leads to an immediate hearing loss.
- In PTA a notch at **3-6 kHz**, and recovery at **8 kHz**. But absence of a notch doesn’t rule out NIHL.

**PATHOLOGY**

- TTS usually **metabolic cochlear pathology** whereas PTS mainly cause of **structural**.

**METABOLIC PATHOLOGY**:

1. **Increased** inhibitory neurotransmitter release i.e. **Glutamine** which affects transduction function of Cochlea.
2. **Decreased blood supply to cochlea** noted on exposure to loud sounds whereas increased on moderate sound exposure
3. Glucocorticoid receptor affection
4. Outer Hair cell Plasma membrane fluidity loss
5. Oxidative Stress

**STRUCTURAL PATHOLOGY**:

1. **Depolymerization of actin filaments** in stereocilia may be a substrate of TTS.
2. Additionally, changes to nonsensory elements of the cochlea, such as **swelling of the stria vascularis, afferent nerve endings** and of supporting cells have been noted.

**APOPTOSIS AND NECROSIS**

- There is evidence that both apoptosis (programmed cell death) and necrosis plays role in noise-induced hearing loss.
- The progression of OHC death well after the cessation of noise has implicated apoptotic mechanisms.
- **Apoptotic Changes** → 5 min following loud noise
- **Necrotic Changes** → 30 min following loud noise
- There is a role of **Caspases** in cochlear hair cell pathology
- **High frequency hair cells die** rapidly after noise injury, but that low frequency hair cells may survive but without auditory function.
**PREDISPOSING FACTORS**

- Genetic Basis – *Ahl gene* (also plays role in Age related hearing loss)
- Relation between NIHL and Age related hearing loss
- The suggestion is that as individuals with noise-induced hearing loss age, the effect of ageing upon thresholds affected by noise-induced hearing loss is slowed, but for adjacent frequencies is accelerated.
- **Smoking, Diabetes, Cardiovascular disease**, greater extent of TTS in patients with Blue eye colour.
- **Recreational drug use** causes greater risk of NIHL and tinnitus.
- Industrial solvent exposure

**DIAGNOSIS**:

- Historically, the diagnosis of noise-induced hearing loss has included separating the effects of ageing from the effects of noise. This process has been accomplished by reference to one or more of the many standardized reference tables detailing hearing thresholds with age for typical screened and unscreened populations.
- Assumptive Diagnosis based on Age component, Noise exposure and idiopathic degenerative component.
- The clinician's task is to separate and calculate the relative contribution (if any) from the three sources.
- **HISTORY →** Usually Male in early middle age *(women mostly present with acoustic shock)*, Hearing difficulty in presence of background noise, Lack of clarity rather than a lack of volume.
- Difficult telephone conversations
- Slowly progressive for many years.
- Tinnitus commonly accompanies NIHL.
- **Hyperacusis** *(hypersensitive to particular frequencies of sound)*
- History of sufficient noise exposure to cause NIHL
- Tinnitus usually present in TTS.

**Table 238b.2** Summary of symptoms found in acoustic shock.

<table>
<thead>
<tr>
<th>Common</th>
<th>Less frequent</th>
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<tbody>
<tr>
<td>Otalgia</td>
<td>Neck pain</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>Shoulder pain</td>
</tr>
<tr>
<td>Hyperacusis</td>
<td>Panic attacks</td>
</tr>
<tr>
<td>Dizziness</td>
<td></td>
</tr>
<tr>
<td>Headaches</td>
<td></td>
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<tr>
<td>Sleep disturbance</td>
<td></td>
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<tr>
<td>Poor concentration</td>
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</tbody>
</table>
Acoustic Shock ➔ Pain, tinnitus, balance disturbance and phobic symptoms following exposure to sudden unexpected noise, usually seen in call centre workers.

**EXAMINATION**

- Typically, otological examination will be normal.

**INVESTIGATIONS**

- PTA showing notch at 4 or 6 kHz, with some recovery at 8 kHz. However, the notch is often absent but significant audiometric loss at frequencies below 2 kHz is extremely uncommon.
- So if low frequency loss present suspect some other pathology.
- PTA ➔ Impedance Audiometry ➔ In those individuals in whom a significant nonorganic component (feigned thresholds) is suspected, cortically evoked reflex audiometry may be required.
- In cases with significant asymmetry, magnetic resonance imaging (MRI) may be required to exclude a vestibular schwannoma.

**DIAGNOSIS AND REPORT WRITING**

- Diagnosis is simple in an individual with a clear and prolonged history of unprotected exposure to excessive noise, no evidence of any other otological pathology and an audiogram showing good preservation of mid and low frequencies but a significant high-tone hearing loss with classical notching at 4 – 6 kHz.

**MANAGEMENT**:

- Three broad areas: Psychological and practical (both nonspecific) and sensory (specific).

**Nonspecific management**

- Improve acoustic environment (decrease background noise, face to face communication, lip reading classes)

**Specific management**

- Hearing aids.
- Avoid Noise exposure.
- Ear plugs or ear muffs can be used
- Employers are under a statutory duty under the Health and Safety Act 1974 to minimize risks to employees.
- The employer is required to identify those areas where hearing protection use is required.
- Regular hearing tests should be offered to employees where and when a potential risk is recognized.
- Electronic equipments to cancel out background noises.
- Hearing aids (binaural) help in low intensity losses.
- Optimization of acoustic environment.