Introduction

Noise is a common occupational hazard that leads to one of the most common complaints in the adult population seen by the otolaryngologist—noise induced hearing loss (NIHL). The cause and effect relationship between noise exposure and hearing loss has been appreciated for many years. “Boilermaker’s deafness” was a term coined in the 1700s and 1800s to refer to a high frequency hearing loss seen in laborers that could be diagnosed with tuning forks. The increased mechanization seen during the Industrial Revolution was associated with a rise in the incidence of this disorder and today it is estimated that over 9 million American laborers are exposed to potentially hazardous levels of noise throughout their employment. An additional 1 million Americans are affected by non-industrial noise exposure. This means that nearly one third of the 30 million Americans with hearing loss have an impairment caused by noise, making it the most common preventable cause of permanent sensorineural hearing loss (14).

Characteristics of Noise

In everyday language, the term noise is used to refer to an unpleasant or unwanted sound. However, in the context of the medical literature, noise has come to refer to an excessively intense sound capable of producing damage to the inner ear. Noise can be further described by its temporal patterns. Intermittent noise is interrupted with periods of quiet while continuous noise remains constant and fluctuating noise rises and falls over time. Both impact and impulse noises are produced by a sudden intense sound wave but impact noise is caused by a collision while impulse noise is due to an explosion (5).

Noise is typically measured with a sound pressure meter in decibel (dB) units on the A-scale (dBA). This is a scale weighted to place more emphasis on those frequencies to which the human ear is most sensitive while minimizing the effects of the extreme low
and high frequencies. Perhaps a more accurate measure of an individual’s exposure to noise is obtained with a dosimeter. This device, which is similar to that worn by staff in the radiology department, integrates constant and fluctuating noise over time so that total noise exposure may be calculated and risk estimated (13).

**Acoustic Trauma**

Acoustic trauma refers to a sudden permanent hearing loss caused by a single exposure to an intense sound. This is most often caused by an impulse noise, typically in association with an explosion. The sound pressure levels capable of causing acoustic trauma vary between individuals but average around 130-140dB. The degree of hearing impairment seen after acoustic trauma is also variable and may range from a mild to profound SNHL. The mechanism of injury in acoustic trauma is thought to be direct mechanical injury to the sensory cells of the cochlea.

Patients suffering from acoustic trauma tend to present within a short time period following the event. They report a sudden, sometimes painful hearing loss that is often followed by a new onset tinnitus. Otologic examination is often unremarkable but may reveal tympanic membrane disruption or evidence of ossicular damage. Audiogram may show the typical 3-6kHz sensorineural notch that is seen with chronic NIHL but downsloping or flat audiograms that effect a broad range of frequencies are more common. Conductive losses will be seen in cases of tympanic membrane perforation or ossicular discontinuity. Management of acute acoustic trauma injuries most often involves observation with strict noise avoidance. Some improvement can generally be expected in the days immediately following the injury and serial audiograms are performed until hearing levels stabilize. Those patients that present with a complete hearing loss may benefit from middle ear exploration (5,14).

**Chronic NIHL**

Chronic NIHL, in contrast to acoustic trauma, is a disease process that occurs gradually over many years of exposure to less intense noise levels. This type of hearing loss is generally caused by chronic exposure to high intensity continuous noise with superimposed episodic impact or impulse noise. The amount of sound that is capable of producing cochlear damage and subsequent hearing loss is related by so-called “damage risk criteria” which is based upon the equal energy concept. That is to say that it is the total sound energy delivered to the cochlea that is relevant in predicting injury and hearing loss. Both an intense sound presented to the ear for a short period of time and a less intense sound that is presented for a longer time period will produce equal damage to the inner ear. An increase in sound intensity of 3dB is associated with a doubling of sound pressure. Therefore, for each 3dB increase in sound exposure, the time exposed must be cut in half in order to deliver equal sound energy to the ear. Because noise levels are likely to fluctuate throughout the time of exposure, the standard accepted by OSHA is known as the 5dB rule; for every 5dB increase in noise intensity, exposure time must be cut in half. A 90dBA exposure is allowed for 8 hours, a 95dBA exposure is allowed for 4 hours, and so on to a maximum allowable intensity of 115dBA for 15 minutes (1).
Like in acoustic trauma, the hearing loss associated with chronic NIHL is variable between individuals—a subject that will be discussed in more detail later. However, the principal characteristics of chronic, occupational NIHL as specified by the American College of Occupational Medicine Noise and Hearing Conservation Committee include the following:

1. It is always sensorineural.
2. It is nearly always bilateral and symmetric.
3. It will only rarely produce a profound loss.
4. It will not progress once noise exposure is stopped.
5. The rate of hearing loss decreases as the threshold increases.
6. The 4kHz frequency is the most severely effected and the higher frequencies (3-6kHz) are more affected than the lower frequencies (500Hz-2kHz).
7. Maximum losses typically occur after 10-15 years of chronic exposure.
8. Continuous noise is more damaging than intermittent noise (5,6).

The majority of chronic NIHL is due to occupational or industrial exposure. It is important to remember, however, that in today’s noisy society even people with quiet jobs may suffer from NIHL. Such non-occupational NIHL is also called socioacusis. Sources of non-occupational noise include gunfire, loud music—via concerts or headphones, open vehicles such as motorcyclies, snowmobiles or tractors, and power tools to name just a few. This hearing loss also demonstrates the characteristics listed above. One caveat to these features would be the individual who had significant noise exposure secondary to rifle shooting. In this case, an asymmetrical loss, with the ear nearest the gun barrel (the left ear in a right handed shooter) demonstrating slightly worse hearing, would be expected (5).

The development of chronic NIHL progresses through two phases. The first stage is characterized by a temporary threshold shift (TTS). This is brief hearing loss that occurs after noise exposure and completely resolves after a period of rest. This can be thought of as auditory fatigue and most studies indicate that it is associated with no sensory cell damage or minimal, reversible cell changes. After repeated exposure to noises intense enough to produce TTS, eventually a permanent threshold shift (PTS) will occur. This is the second stage of chronic NIHL and is an irreversible increase in hearing thresholds. At this point, there has been irreversible hair cell damage (5,6).

Patients suffering from chronic NIHL commonly present at the urging of family members or friends who are frustrated by the patients hearing loss. Upon further questioning, patients report difficulty not so much with hearing speech as with understanding speech. This difficulty is primarily noticed in environments with significant background noise. High frequency hearing loss is characterized by a loss of consonant discrimination. Consonant sounds such as f, s, t, d, sh, and k are all high frequency sounds (3-6kHz) and although they are not responsible for the acoustic power of speech, they are very important to the intelligibility of speech. Otoscopic examination will most often be normal and the audiogram will likely demonstrate the characteristics as listed above (5,14).
Many studies have been done looking for an effective medicinal treatment for NIHL caused by either acute trauma or chronic exposure. Dextran-40, carbogen, nicotinic acid, vitamins A, B1, E, and ephedrine are just a few agents that have not proven themselves beneficial (11). A report published in 1998 treated patients with sudden SNHL, acoustic trauma or NIHL whose hearing loss had failed to improve after a short trial of medical therapy with hyperbaric oxygen therapy (HBO). This study found that if the onset of the hearing loss was 2-6 weeks prior to HBO therapy, 1/3 of patients showed a marked hearing improvement—more than 20dB in at least three frequencies. Another 1/3 of patients had a moderate hearing gain—10-20dB while 13% had no improvement. If the onset was between 6 weeks and 3 months prior to therapy, 13% had marked gain, 25% had moderate gain and 62% had no improvement. If the hearing loss had been present for more than 3 months, HBO offered no benefit in terms of hearing improvement. From this data, the conclusion was made that a new hearing loss diagnosed within 3 months may improve with HBO treatment (10). Obviously, further studies are required to support or refute these findings.

Although NIHL is not amendable to medical or surgical therapy, it is entirely preventable. To address the increasing concern over occupational NIHL, many industries have adopted hearing conservation programs (HCPs). An effective HCP has five components: 1.) assessment of noise levels, 2.) engineering controls, 3.) administrative controls, 4.) use of personal hearing protectors, and 5.) serial audiograms. Hazardous noise levels can be identified with sound pressure meters or individual dosimeters as mentioned previously. In order to obtain the most accurate measurement of noise, sound surveys are performed that measure noise levels over long periods of time. Once dangerous noise levels have been identified, various control measures are taken to minimize exposure. Engineering controls involve changes in the technology or equipment used in industry. Examples of this would include replacing riveting with welding, applying mufflers to pneumatic drills, or redesigning machinery to enclose noisy gear wheels. Administrative controls include limiting time of exposure to noise, providing a less noisy work environment, and educating workers about the prevention of NIHL (1). When engineering and administrative controls fail to reduce noise to an acceptable level, personal hearing protective devices (PHPD) are vital to prevent NIHL. Insert earplugs, earmuffs and canal caps are the three main types of PHPDs. Earplugs fit directly into the EAC and may be sized, custom-made or moldable. While earplugs are often felt to be less cumbersome than other devices, their effectiveness in attenuating sound depends on an adequate seal within the EAC and proper fit is essential. Earmuffs encompass both ears with rubber or plastic cups connected by a headband. Again, the effectiveness of these devices depends on an adequate seal and a snug fit. Canal caps seal the external meatus with a soft rubber or plastic cap that is held in place with a headband. They do not require sizing or custom fitting like earplugs and are less bulky than earmuffs, but still must be worn tightly to assure an adequate seal. PHPDs, in general, are more effective in attenuating sounds above 1kHz but the absolute reduction of sound reaching the inner ear is highly variable. Earmuffs with a tight seal are capable of reducing sound levels by about 45dB in the high frequencies while earplugs average about 30dB of attenuation. Worn together, they provide a maximum of approximately
50dB of sound reduction. Obviously no PHPD will be effective unless the worker consistently wears it. Therefore, the most important aspect of choosing a PHPD is worker comfort and confidence in using the device (1,7,13). Finally a HCP involves screening audiometry to allow early identification of individuals with worsening hearing and to assess the efficacy of the program. Changes of 10dB or greater at any frequency or an average change of 10dB or more at all frequencies may warrant a referral to an otolaryngologist for further evaluation and a reassessment of the HCP. This annual exam serves not only to identify hearing loss but also to provide a opportunity for counseling on the importance of hearing conservation and assuring proper fit and compliance with PHPDs (1).

**Physiology, Pathophysiology and Histopathology**

Although the histopathologic correlate to chronic NIHL is injury to the cells of the inner ear, the pathogenesis involves interactions between all three divisions of the auditory system—the external, middle and inner ears. The importance of the external ear centers on the resonant characteristics of the external auditory canal (EAC). Tubes that are open at one end have an inherent resonant frequency that is determined primarily by the length of the tube. The average human EAC is 25mm in length, using this value in the formula: resonant frequency = speed of sound/4 x EAC length, means the average resonant frequency of the human ear is 3200Hz. Additionally, the configuration of the EAC can serve to amplify mid-frequency sounds by as much as 20dB. The clinical importance of these characteristics is twofold. Studies have shown that the most severe hearing loss is demonstrated ½-1 octave higher than the offending noise. The broadband noise seen in industry is converted by the fundamental resonance of the EAC to a 3Hz noise. This leads to the characteristic 4Hz notch seen on the audiogram in noise-exposed individuals. Secondly, as mentioned previously, significant variability exists in different individuals response to similar noise exposure. One explanation for this variability could be differences in EAC configuration and inherent resonance (5,8).

The contribution of the middle ear to the response to noise is the action of the acoustic reflex. The middle ear structures involved in this reflex are the tensor tympani muscle, which is attached to the head of the malleus and the stapedius muscle, which is attached to the head of the stapes. Two cranial nerves—the trigeminal (V) and facial (VII)—participate in the reflex. Stimulation of the reflex by a sudden intense sound causes muscle contraction. The action of the tensor tympani is to tense the tympanic membrane (TM) by pulling the malleus medially while the stapedius pulls the stapes perpendicular to its axis on the oval window. The combined action of these muscles is to stiffen the middle ear structures thereby reducing the sound energy reaching the inner ear. This system is most effective in attenuating low frequency sounds (<2kHz). Human and animal studies have shown that malfunction of the acoustic reflex is related to more temporary and permanent hearing threshold shifts. Specifically, patients with Bell’s palsy developed more TTS on the side of the facial paralysis when exposed to moderate noise. Additionally, differences in reflex latency, threshold, strength of muscle contraction and resistance to adaptation have been found and may help to explain inter-individual differences in NIHL (5,8,9).
The injurious action of noise is believed to affect not only the sensory cells of the inner ear, but also the supporting cells, nervous structures and blood vessels. The outer hair cells (OHC) are more vulnerable to noise injury than the inner hair cells (IHC). This is likely secondary to several characteristics including the location of the OHC, which is close to the point of maximal basilar membrane displacement, the direct shearing forces on the stereocilia of the OHC against the tectorial membrane, and the relative lack of supporting cells around the OHC. Early noise induced injury involves alterations in hair cell membranes which eventually lead to a failure in the regulation of intracellular ionic composition. A chain of events is set off that involves cell swelling or herniation, increased number of lysosomes and changes in essentially all cellular organelles. The hair cell cilia may become floppy, disordered, splayed, fractured or fused. Some of these changes seen in the cilia are reversible—this may be seen clinically as a TTS. However, at some point the cell is unable to recover from these injuries and degenerates—causing a PTS. With prolonged noise exposure, supporting cells and IHC undergo similar changes and eventual loss. After IHC loss, retrograde degeneration of cochlear nerve fibers may also be seen. Noise exposure has also been found to cause changes in the vascular system of the cochlea. Reductions in the number of capillaries, evidence of vessel occlusion, and alterations of RBC packing density have all been demonstrated in noise damaged ears. Although all of these pathologic changes have been well documented in both animal and human studies, a clear relationship between the degree of hearing loss and cochlear pathology has not been documented. Histologic study of ears that demonstrate identical audiograms may reveal markedly different pathology. Not only does this, once again, point to differences in susceptibility to NIHL, but it also has implications regarding differences in successful rehabilitation of hearing loss (5,8).

Susceptibility and Interactions

As has been mentioned several times, individual susceptibility to NIHL is highly variable. Several large studies have been done which have shown that, on average, 5% of individuals with long-term exposure to noise levels of 80dBA will have significant hearing loss. This risk increases to 5-15% with 85dBA noise and 15-25% with 90dBA noise (1). These averages are useful in terms of counseling patients on the risks of noise exposure, but we do not have a good understanding why, within a population exposed to the same noise intensity for the same time period, some individuals will have a significant reduction in hearing thresholds and others will not. Studies have evaluated the relationship between such things as gender, race, eye-color, other medical conditions or smoking history and susceptibility to noise but have not been able to demonstrate a connection (9). Attempts have also been made to predict the likelihood of a PTS based upon the degree of TTS after a noise exposure. Again, no such association has been proven (5).

One thing that we do know is that chronic noise exposure can interact with other factors to produce a hearing loss that may differ from that expected if each factor were delivered separately. Aging and noise exposure are the two most common causes of hearing loss. Since we know that chronic NIHL occurs over years of exposure, many
patients will experience some degree of age induced hearing loss (AIHL) in addition to NIHL. The interaction between AIHL and NIHL follows the idea of decibel additivity up to a cumulative loss of approximately 40dB. This concept predicts that the normal progression of hearing loss associated with aging will occur in subjects with NIHL from an early age. Similarly, subjects with pre-existent AIHL will experience the same degree of NIHL with any given noise exposure as will those without AIHL. Once the total loss exceeds 40dB then one must factor in a “compression term” which takes into account that both AIHL and NIHL occur via the same mechanism—hair cell loss. Such that, when a number of hair cells have been previously damaged by one factor, there exists less chance for the other factor to cause further damage and the total loss will not simply be the sum of the two individual losses. In this case, total loss can be calculated with the following formula:

\[
\text{Total HL} = \text{NIHL} + \text{AIHL} - \frac{(\text{NIHL})(\text{AIHL})}{120}
\]

Simultaneous exposure to noise and ototoxic medications may have an amplifying affect on hearing loss, producing more threshold elevation than with either factor alone. This effect has been definitively demonstrated in noise- exposed animals given aminoglycoside antibiotics. The chemotherapy drug cisplatin was not found to cause hearing loss alone, but in animals given the drug and exposed to noise the hearing was worse than in animals exposed to noise alone. Although the diuretic furosemide is potentially ototoxic when given intravenously to patients with altered renal function, oral administration in people with normal kidneys has not been associated with hearing loss. It has also not been shown to worsen NIHL. The literature on the combined effect of salicylates and noise is contradictory. Some studies have demonstrated a potentiating effect, while others have not. Two separate studies done in the mid 1980’s found that in noise exposed individuals, if higher doses of aspirin (1.9 gr/day) were taken, their TTS was of greater magnitude and slower to recover. Therefore, it seems reasonable to counsel patients with significant noise exposure to avoid high dose aspirin therapy (5,9).

Simultaneous exposure to hazardous noise and certain chemical pollutants may have an additive effect on hearing loss. Toluene, carbon monoxide and carbon disulphide in combination with noise are known to cause a more severe high frequency hearing loss than noise alone. Other agents such as lead, mercury, xylene and trimethyltin are suspected to either worsen NIHL or alter susceptibility to NIHL (9,12).

It is not unusual for significant vibration to accompany noisy environments. Although vibration alone is known not to cause hearing loss, it is not known if vibration has any influence on NIHL. Animal studies have found more severe hearing loss and hair cell loss in animals exposed to both vibration and noise compared to those exposed to noise alone. In humans, vibration causes a larger TTS after a noise exposure, however, it is not clear if this can be translated to larger PTS also (5,12).

**Impairment, Handicap and Disability**
Hearing impairment, handicap and disability are terms that are frequently, though incorrectly, used synonymously. The correct definitions as set forth by the American Academy of Otolaryngology Committee on Hearing and Equilibrium in 1979 are as follows. Hearing impairment refers to “a change for the worse in either structure or function, outside the range of normal.” A hearing handicap is “the disadvantage imposed by an impairment sufficient to affect a person’s efficiency in the activities of daily living.” And, a hearing disability is “an actual or presumed inability to remain employed at full wages.” The otolaryngologist is often called to evaluate patients with hearing loss and should therefore be familiar with the appropriate use of these terms, particularly in those cases that may involve some compensation for the loss (2).

Although several methods exist to calculate hearing handicap, the most commonly accepted formula is the AAO-1979 rule. In this formula, pure tone audiometry is used to establish hearing thresholds at 500Hz, 1, 2, and 3kHz for each ear and the average monaural thresholds are calculated using these values. Then, using the assumptions that hearing handicap begins when PTA thresholds exceed 25dB and increases by 1.5% for each decibel loss above 25dB, the monaural percent impairment is calculated: \( MI = 1.5(PTA-25) \). Finally, the hearing handicap is calculated by applying a 5:1 weight favoring the better hearing ear: \( HH = \left\{5(MI_b) + (MI_w)\right\}/6 \). The following is an example of the calculation of hearing handicap:

……………… SEE NEXT PAGE …………………
1. Calculate monaural PTA.

<table>
<thead>
<tr>
<th>PTA thresholds</th>
<th>500Hz</th>
<th>1kHz</th>
<th>2kHz</th>
<th>3kHz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right ear</td>
<td>25</td>
<td>30</td>
<td>35</td>
<td>45</td>
</tr>
<tr>
<td>Left ear</td>
<td>35</td>
<td>40</td>
<td>55</td>
<td>70</td>
</tr>
</tbody>
</table>

Right ear: \[
\frac{25 + 30 + 35 + 45}{4} = \frac{135}{4} = 33.8 \text{dB}
\]

Left ear: \[
\frac{35 + 40 + 55 + 70}{4} = \frac{200}{4} = 50 \text{dB}
\]

2. Calculate monaural impairment.

<table>
<thead>
<tr>
<th></th>
<th>Right ear: [33.8 \text{dB} - 25 \text{dB} = 8.8 \times 1.5% = 13.2%]</th>
<th>Left ear: [50 \text{dB} - 25 \text{dB} = 25 \times 1.5% = 37.5%]</th>
</tr>
</thead>
</table>

3. Calculate hearing handicap.

\[
\text{HH} = \left[ \frac{5(13.2\%) + (37.5\%)}{6} \right] = 66 + \frac{37.5}{6} = 103.5/6 = 17\% \quad (5,6).
\]

The establishment of a hearing disability is an “administrative” decision. It is an estimate of the individual’s present and future ability to earn wages based, in part, on the hearing handicap. Any compensation that an individual will receive is dependent on the determination of a disability (2).

**Legislation and Compensation**

The introduction of legislation for noise regulation came in 1969 with the Walsh-Healey Public Contracts Act. This established a maximum noise exposure of 90dB over an eight-hour workday with increases of 5dB of exposure requiring a decrease in duration by half. The stipulation was made that when these levels were exceeded, employers must use control measures to limit exposure. In 1970, with the establishment of the Occupational Safety and Health Act, these requirements were applied to all employees involved in interstate commerce. Also in 1970, the Clean Air Act created the Office of Noise Abatement and Control within the EPA, which was designed to identify sources of noise and its effect on public health and welfare. Bulletin #334: Guidelines to the Department of Labor’s Occupational Noise Standards was published in 1971. In addition to restating the maximal allowable exposure levels (90dB for 8hr), this standard issued a requirement that employers provide personal protective equipment to those employees whose exposure exceeded these levels. Secondarily, industries in which noise exceeded the maximal levels were required to establish hearing conservation programs that included periodic screening audiograms and noise surveys. The Noise Control Act of 1972 served to establish noise emission standards for construction, transportation equipment, motors, engines and electrical equipment. Lastly, the Hearing Conservation Amendment, Final Rule was set forth by the Department of Labor in 1983. This lowered the noise exposure that necessitated implementation of a hearing conservation program to 85dB over eight hours instead of the previously stated 90dB. Employers were required to
provide personal hearing protectors to those workers exposed to 90dB or greater noise levels (3,15).

During and immediately following the Industrial Revolution, the majority of responsibility for personal safety in the workplace rested upon individual employees. Employers limited their liability by establishing three common-law defenses. The fellow servant rule stated that the employer was not liable if an action of one worker caused injury to another. The assumption of risk policy stated that when a worker accepted wages they assumed the risks of the job. And, the limited responsibility policy stated that the employer could fire or transfer any worker who was negligent in their job. Thus, prior to 1948, compensation for occupational NIHL was essentially unheard of. However, between 1948 and 1959, several court decisions served to establish occupational hearing loss as a compensable injury. The original concept behind worker’s compensation was to provide payment to cover lost wages and medical expenses accrued by a worker as a result of an injury sustained on the job. Employers gave up their common-law defenses, employees gave up their right to sue, and limits were set on the amounts of liability. In the United States, the federal government oversees three worker’s compensation programs and each of the 50 states has such a program that covers occupational NIHL. The state programs are similar but may differ in terms of statute of limitations, waiting periods, exposure levels, use of hearing protectors or prior hearing loss. Hearing handicap is most often the criterion upon which compensation awards are based. Although other methods to calculate this are available, 32 of the 50 states use the AAO-1979 rule as outlined above (3). A study published in 1982 found that average compensation for hearing loss in private industry was $3,000 per claim, while federal employees received $8,000 per claim (15). In 1992, the average maximum payment for total bilateral hearing loss was $45,000 but in the majority of cases the claimant does not receive this maximum. Instead, the percentage handicap is multiplied by the state maximum to determine an individual award (4).

The otolaryngologist’s role in cases of NIHL that may involve compensation centers on accurate diagnosis and succinct reporting of findings. Patients must undergo a complete otologic history and examination to rule out other potential sources of hearing loss. A complete audiogram is obtained and compared to previous studies if possible. Then, taking all of this data into account, the otolaryngologist must provide clear diagnostic conclusions in his or her report or testimony. In workers compensation cases the standard of “reasonable medical certainty” is utilized. This means that if a diagnosis is determined to be “more probable than not” the source of the disability, causation may be assigned and compensation awarded (6).
Bibliography