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No one would argue the point that modern technology has introduced rapid growth and advancement throughout the world within the last few decades. While achievements are countless, one detrimental adversity which has been, until recently, an unknown consequence, is the effect of noise on man. Specifically, noise has introduced irreversible hair cell damage to the cochlea reducing man's potential for normal hearing sensitivity. Noise induced hearing loss (NIHL) may be caused by either long term exposure above the damage risk criteria, or instantaneous exposure. In any event, hair cell damage to the cochlea is the end result. Depending on man's exposure to noise, either temporary threshold shifts (TTS) or permanent threshold shifts (PTS) will ensue. The intent of this paper is to present current research in the area of noise exposure as it affects physiological changes within the auditory system. In addition, concomitant changes that face man due to a reduction in hearing sensitivity are discussed. Finally, current research by the present author in the area of noise exposure, ototoxicity and the additive effects on high risk infants are presented.

While intense noise exposure in excess of 160 dB SPL may produce structural damage to the middle ear system such as rupturing of the tympanic membrane or fracturing of the ossicular chain, the primary damaging mechanism seems to be metabolic stress on the stimulated sensory hair cells. Most vulnerable to acoustic trauma is the region of the organ of corti approximately eight to ten millimeters from the basilar end of the cochlea, an area which corresponds to the 4000 Hz sensitivity region. This frequency region will produce the largest threshold shifts regardless of the stimulus frequency, and is most probably due to the auditory system which acts as a built-in filter whose band pass somewhat limits the frequency of sounds which are hazardous to the organ of corti. A contributing factor to this filter system is the contraction of the middle ear muscles which attenuate transmission of sound in the lower frequencies. The acoustic middle ear contraction magnitude increases with increased SPL up to about 30 dB above the reflex threshold, thereby altering the ear's sensitivity due to the efficiency of the middle ear. Other factors related to the maximum threshold shift seen at the 4000 Hz region may be attributed to the physiological and anatomical differences of the basilar membrane, the direction and pattern of the travelling wave within the cochlea, and the resonance characteristics of the external auditory canal.

Physiologically, two basic alterations occur to the auditory system due to noise exposure and are reflected in the degree of hearing sensitivity loss and its method of measurement. These are adaptation and fatigue. Adaptation deals with low intensity stimulation between 20 and 90 dB SPL and is measured as a change in the perception or threshold while the stimulus is present. Adaptation is basically a neural phenomenon and results in a decreased rate of neural firing or action potential. Adaptation occurs in a normal, healthy ear and produces no permanent damage to the cochlea. Fatigue, on the other hand, deals with high intensity stimulation, usually greater than 80 dB SPL and is measured approximately 3 minutes after the cessation of the stimulus. The temporary threshold shift measured is due to fatigue, basically a cochlear phenomenon. Physiological changes which result from fatigue include alterations in both the cochlear and action potentials, a substantial reduction in the vascular supply thereby diminishing oxygen to the auditory system, distortion of the basilar membrane and organ of corti; all resulting in tissue damage to the sensory receptor hair cells in the cochlea.

The most common index of auditory fatigue is the temporary threshold shift and is measured by determining the ear's threshold, exposing the ear to fatigue, measuring the exposed threshold and the differences between them; that is, the pre and post thresholds are considered the degree of threshold shift. It is presumed that anytime you have induced a TTS of more than 40 dB, you have induced actual damage. As long as the TTS has not exceeded this dB value, recovery time all tends to meet at approximately 1000 minutes or 16 hours.

The consequence of reduction in hearing will not only reduce the ear's auditory sensitivity to tonal stimulus, but will also reduce the systems ability to discriminate speech, man's obvious means of communication. Because consonant phonemes are high frequency, low intensity in nature and provide meaningfulness to speech, a concomitant reduction in hearing sensitivity will reduce the high frequency resolution capabilities of the auditory system while introducing a masking effect by the stronger low frequency vowel phonemes. Taking this into consideration, as well as the low frequency noise in our environment, it is no wonder the major complaint of individuals with sensori-neural hearing losses is that, while speech is usually loud enough to hear, the discrimination of the speech signal is unintelligible.

Recently, I have done some extensive work in the area of noise measurement as it relates to potential hearing loss in premature infants who have spent, in some cases, the first three to six months of their lives in incubators. The results of my investigation are consistent with other research in the literature. Using B & K equipment and measuring noise intensity on dBA and linear settings, we found that the frequency distribution of incubator noise produced peak levels between 31.5 and 250 Hz with approximately 90% of all sound level energy below 500 Hz. Depending upon the incubator measured, mean intensity values ranged between 62 and 67 dBA and 70 to 77 unweighted. Interestingly, and consistent with Dr. Mencher's work at the Izaak Walton Killam Children's Hospital in Halifax, the most intense noise measured was attributed to the closing of the incubator door, introducing impact measurements as much as 115 dB.

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According to well established damage-risk criteria, the maximum sound intensity an adult may safely tolerate is 80 dBA regardless of the duration. Thus, according to our measurements, incubator noise should not be considered a potential risk; however, a number of factors must be taken into consideration before accepting this conclusion. First, damage-risk criteria are established on adult subjects and based on intermittent noise during an eight hour per day exposure. Research has revealed that continuous noise is more damaging than intermittent. In addition, animal evidence has shown that hair cell damage following noise exposure with associated ototoxic antibiotics appear to affect the auditory system more than just additively. It should be noted that the large numbers of premature infants concomitantly receive antibiotics such as gentamycin, which may have ototoxic effects on the auditory system. While conclusive evidence has not been formulated, longitudinal studies are in order to determine any potential hazards that may exist.

This paper was presented at the Annual Meeting of the CAA, Halifax, Nova Scotia, November 1978.

## CORRECTION

In "The Ford Auditorium" (October 1978 issue, page 16), "Lewis M. Dimenco" was named as design architect when Lewis M. Dickens was in fact responsible. We apologise to Mr. Dickens and Mr. Dimenco.