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REPORT NO. 686

RECOVERY FROM IMPULSE NOISE INDUCED ACOUSTIC TRAUMA

by

Lt Colonel J. L. Fletcher, MSC and Captain A. B. Cairns, MC

22 November 1966

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UNITED STATES ARMY Medical Research and Development Command

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RECOVERY FROM IMPULSE NOISE INDUCED ACOUSTIC TRAUMA

by

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ABSTRACT

RECOVERY FROM IMPULSE NOISE INDUCED ACOUSTIC TRAUMA

OBJECTIVE

To follow recovery from impulse noise induced acoustic trauma in order to determine amount and rate of recovery.

METHOD

Military personnel traumatized on firing ranges were given serial audiograms in order to determine initial loss and recovery from such loss.

RESULTS

Recovery was essentially complete at the speech frequencies (500-2,000 Hz) within two weeks. Some recovery was still occurring at higher frequencies (up to 8,000 Hz) as long as six months post exposure. Low frequency losses appeared to be sensori-neural rather than conductive in nature.

CONCLUSIONS

Two weeks is sufficient time to allow for recovery from speech frequency TTS. A longer time must be allowed for complete recovery from TTS at higher frequencies. The human ear can achieve almost complete recovery from TTS's as great as 85 dB at 4,000 Hz.

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RECOVERY FROM IMPULSE NOISE INDUCED ACOUSTIC TRAUMA

INTRODUCTION

There is a considerable body of literature regarding noise induced hearing loss. In nearly all cases, the losses described are occasioned by long term exposure to noise, usually steady state noise. Obviously, in such cases it is difficult, if not impossible to point to any one exposure, relate hearing loss to that particular exposure and plot recovery from the exposure. For many reasons, medical, legal, and humanitarian, it would be of value to know more about the rate, amount, and frequency characteristics of recovery from acoustic insult.

In the present report, attention is restricted to impulse noise induced acoustic trauma in order that we can be assured of the time and nature of the trauma. Impulse noise induced trauma is of particular interest, additionally, because almost 50% of the aural rehabilitation cases seen at the Audiology and Speech Clinic, Walter Reed Army Hospital (WRAH), are ~aceable to impulse noise exposure.

Specifically, then, this study will deal with impulse noise induced acoustic trauma, resulting from exposure on one day (or less), on a firing range. Recovery from loss will be followed over time.

METHOD

All data were secured from young, male soldiers, stationed at Fort Knox, Ky. Educational sessions were held prior to data collection with physicians staffing the dispensaries at Fort Knox, Ky. The purpose of these sessions was to assure that cases would be identified as soon as possible after exposure. The physicians were briefed on symptoms suggestive of acoustic trauma, i.e., usually complaints of severe bilateral hearing loss with sudden onset on a range, or persistent tinnitus following range firing. We tried, where at all possible to get cases on the day of exposure (Day 0), but to increase the number of cases available we also secured data on persons exposed the day prior to their being seen.

When a person was referred for suspected acoustic trauma, a noise exposure history was taken to determine the type of exposure suffered, when it occurred, and in what period of time (i.e., how many rounds fired or how long on the range). Past noise exposure and past medical history was also considered in the evaluation of the patients' suitability for inclusion in the study. The next step in processing entailed a thorough otological examination, including pure tone air and bone conduction thresholds, and intelligibility testing.

Air and bone audiograms, as well as intelligibility testing, were done in the ENT Clinic in an IAC Model #1200 Booth with either a Beltone 15C or a Beltone 15A Audiometer. Subsequently, on the same day, pure tone audiograms were determined using a Grason-Stadler Model E-800 Békésy Audiometer in the Army Medical Research Laboratory Anechoic Chamber.

RESULTS

Time and frequency characteristics of recovery from impulse noise induced acoustic trauma are shown in Figure 1. Note the rapid recovery from Day 0 to Day 1 at 500, 1,000, and 2,000 cps and the less rapid recovery at higher frequencies. By the end of two weeks,

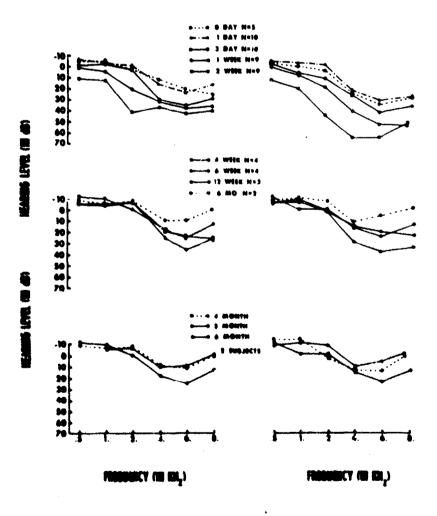


Figure 1

post-exposure recovery was essentially complete for frequencies from 500 to 2,000 cps. However, examination clearly shows continuing recovery for frequencies higher than 2,000 cps up to the maximum six months covered in this study. Even then, some residual loss can be seen at 4,000 and 6,000 cps, perhaps a little at 8,000 cps. In view of the magnitude of initial losses (some were down as much as 85 dB at 4,000 cps); the recovery observed is remarkable.

DISCUSSION

Clearly, for legal purposes, six months is a minimum waiting period before attempting to determine permanent hearing loss. However, recovery at the speech frequencies (and also those used in determining compensation in hearing loss cases) from 500 to 2,000 cps is essentially complete in about two weeks.

There has been some speculation in the past regarding the low frequency losses noted in acoustic trauma with some holding that they were conductive in nature. The belief was that they could be due to a de-coupling of the ossicular chain analogous to a sprain, resulting in a temporary conductive type loss. Data from our study support the thesis that the low frequency loss is a sensori-neural, not conductive, loss. Specifically, we believe that since the bone conduction curves closely approximate the air conduction curves on the air and bone audiograms done in the ENT Clinic, the loss most likely is sensori-neural. There was no apparent correlation between the PB score and the degree of hearing loss; however, the SRT generally followed the average loss in the speech frequencies. Some individuals experienced a drop in intelligibility and some did not. (Standard CID lists-W22 were used and presented by live voice). It is interesting to note that often symptoms of tinnitus and stuffiness in the ear persisted beyond complete recovery in the speech range. Also, even in patients with 100% PB scores, as well as in exposed patients with normal audiograms, a complaint of decreased intelligibility (as well as ear stuffiness and tinnitus) was noted. Physical examinations of the ears in these patients were essentially normal. Occasionally, a little canal hyperemia was noted, especially in the vascular strip area, but nothing more. An associated complaint found in almost all subjects was that of a stopped-up feeling in the ear (or ears) much like that noted in rapid altitude change.

It is obvious that fairly large initial threshold shifts can be incurred from impulse noise exposure and still be followed by almost complete recovery. TTS's as large as 35 dB from 500-2,000 cps were observed with cosentially complete recovery. At the higher frequencies, shifts of magnitudes as great as 75 - 85 dB were observed with good recovery most of the time. Valid pre-exposure audiograms were not available for all Ss in this study. Recovery at low frequencies which approached -10 dB HL could reasonably be assumed to be close to pre-exposure levels at 500, 1,000, and 2,000 cps. However, at 4, 6, and 8,000 cps recovery to 10-15 dB HL could not be so interpreted since they might have been below that level before trauma, and therefore, had not recovered.

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