THE EFFECT OF SONIC BOOM EXPOSURE TO THE GUINEA PIG COCHLEA

By

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ABSTRACT

Thirty guinea pigs with normal hearing were used. Six were controls and eight each were exposed to 1,000 sonic booms at approximately 130 dB of 2, 4.76 and 125 msec N-wave pulse duration, respectively. The cochleae were dissected, stained with osmium tetroxide, and the organ of Corti removed for histological examination. It was observed that hair cell damage occurred in the apical turn of the cochleae of the exposed guinea pigs while the other turns were unaffected. Damage occurred in the same place with all pulse signatures tested.

INTRODUCTION

The imminent appearance of supersonic commercial aircraft makes the threat of frequent exposure to sonic booms a reality. It is thus very urgent that we evaluate the effect on ear tissues of these high intensity pulses under controlled conditions. The ability to fashion sonic booms of different spectral content and wave configuration may permit us to seek out that pulse which is least harmful to auditory structures. With this information, aircraft design could be modified to be less detrimental to hair cells (Howes, 1967; Koegler, 1967.)

Research on sonic boom involving human subjects has largely taken for granted that no hair cell damage would occur in any turns of the cochlea (Nixon, 1969; Hubbard and Mayes, 1967; Kryter, 1969.) This is probably due to the benign loudness levels in assessing apical hair cell damage by standard audiometric procedures.

Therefore, we decided to subject guinea pigs to controlled sonic booms in order to objectively evaluate damage to the auditory mechanism. The cochleae of the animals could be removed and studied microanatomically for hair cell damage and thus demonstrate positively any effects of sonic boom trauma.

METHODS

From 30 guinea pigs, six were selected randomly as controls. These were tested for Preyer reflex and sacrificed. The 24 remaining were divided into three groups, tested for Preyer reflex, exposed to one of three types of sonic booms, retested for Preyer reflex, and then sacrificed. The cochleae were removed and prepared for histological examination.

Spectral analyses were also carried out of the sonic booms for estimations of spectral power density.

Preyer Reflex Testing

A GR tone burst generator Type 1396A was used as an electronic switch. A Wavetek Model 111 was used as the tone oscillator and was set for sine wave output. A HP 651A oscillator was used to time the tone burst generator to deliver a burst of 250 msec duration at an interval of 1/sec. A GR octave band noise analyzer Type 1558AP was used as a band pass filter to eliminate unwanted frequencies produced by the sudden onset of the tone. The output was amplified by a McIntosh 40 amplifier and delivered to a B&K Artificial Mouth Type 4216. (An Electro Voice 16W speaker was used for frequencies below 500 Hz.) A B&K 1/2" microphone was placed at 0 degrees incidence to the Artificial Mouth right above the guinea pig's ear which was also at 0 degrees incidence to the Artificial Mouth. The microphone input was put into a B&K microphone amplifier Type 2603 which permitted intensity monitoring. A sound intense enough to cause a pinna jerk was produced and attenuated stepwise until no movement of the pinna could be observed. The intensity of this sound at the guinea pig's ear was recorded as the Preyer reflex threshold.

Sonic Boom Generation

The guinea pig was put into a circular hardware cloth cage and pushed into a plane wave tube ahead of a 9' fiber glass wedge. The tube was 20' long and one foot in diameter. (For the 125 msec pulse, the wedge was not used and the tube was extended by addition of a 25 cu. ft. resonator box. The guinea pig's cage was suspended in the box.) A B&K 1/2" microphone was mounted over the guinea pig's ear at the same 0 degrees incidence as the ear to the Electro Voice Model 30W speaker mounted at the end of the tube. The microphone led out of the tube to a B&K microphone amplifier Type 2603. The output from this amplifier went to a Tektronix Type 545B oscilloscope for display. To produce the pulse, the Wavetek Model 111 oscillator was set to deliver N-waves and fed into the GR tone burst generator Type 1396A. Initially, the tone burst generator was set for 10 pulses per second. The fundamental frequency of the N-wave was either 210 Hz, 500 Hz, or 8Hz. The output of the tone burst generator went to a GR decade attenuator Type 1450TBR. This was set initially for 50 dB attenuation. The output from the attenuator went to the McIntosh 40 amplifier after a junction to the Tektronix Type 545B oscilloscope for display. The gain of the McIntosh was set to deliver 80 dB at the vicinity of the guinea pig microphone. The meter dial on the B&K microphone amplifier was set to RMS slow. Thus, 80 dB was measured. The output from the tone burst generator was then set to 1 pulse per sec. and the decade attenuator then set to 0 dB attenuation. Thus, the sound pressure level at the guinea pig's ear was theoretically 130 dB. Figures 1-3 demonstrate the shape of the N-wave from the Wavetek oscillator at the top and the wave configuration at the guinea pig's ear on the bottom. A total of 1,000 bursts were delivered to the guinea pig. These were counted with a Beckman Universal Eput and Timer Model 7350A. The guinea pig was then removed from the tube.

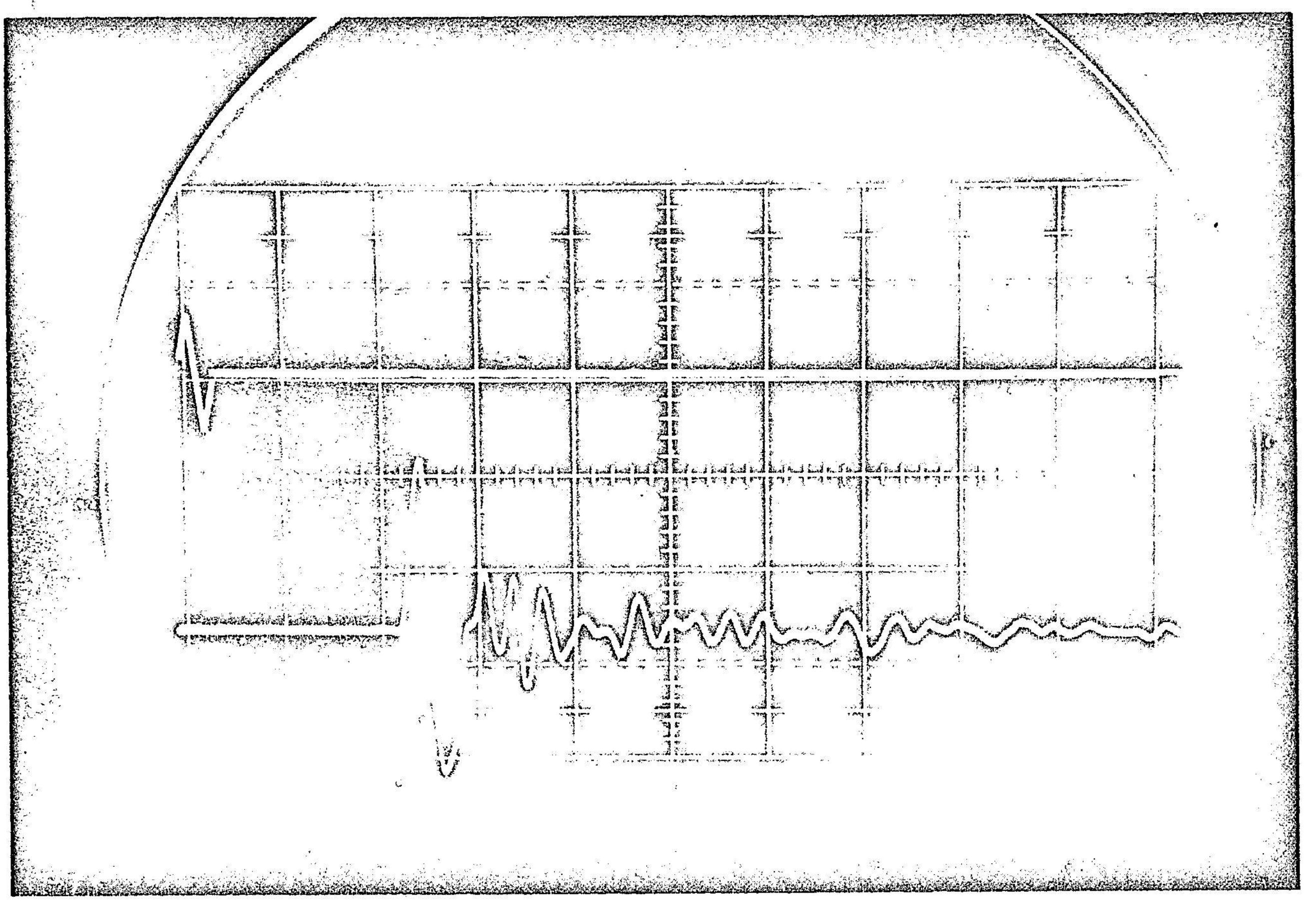


Figure 1. 2 msec duration N-wave

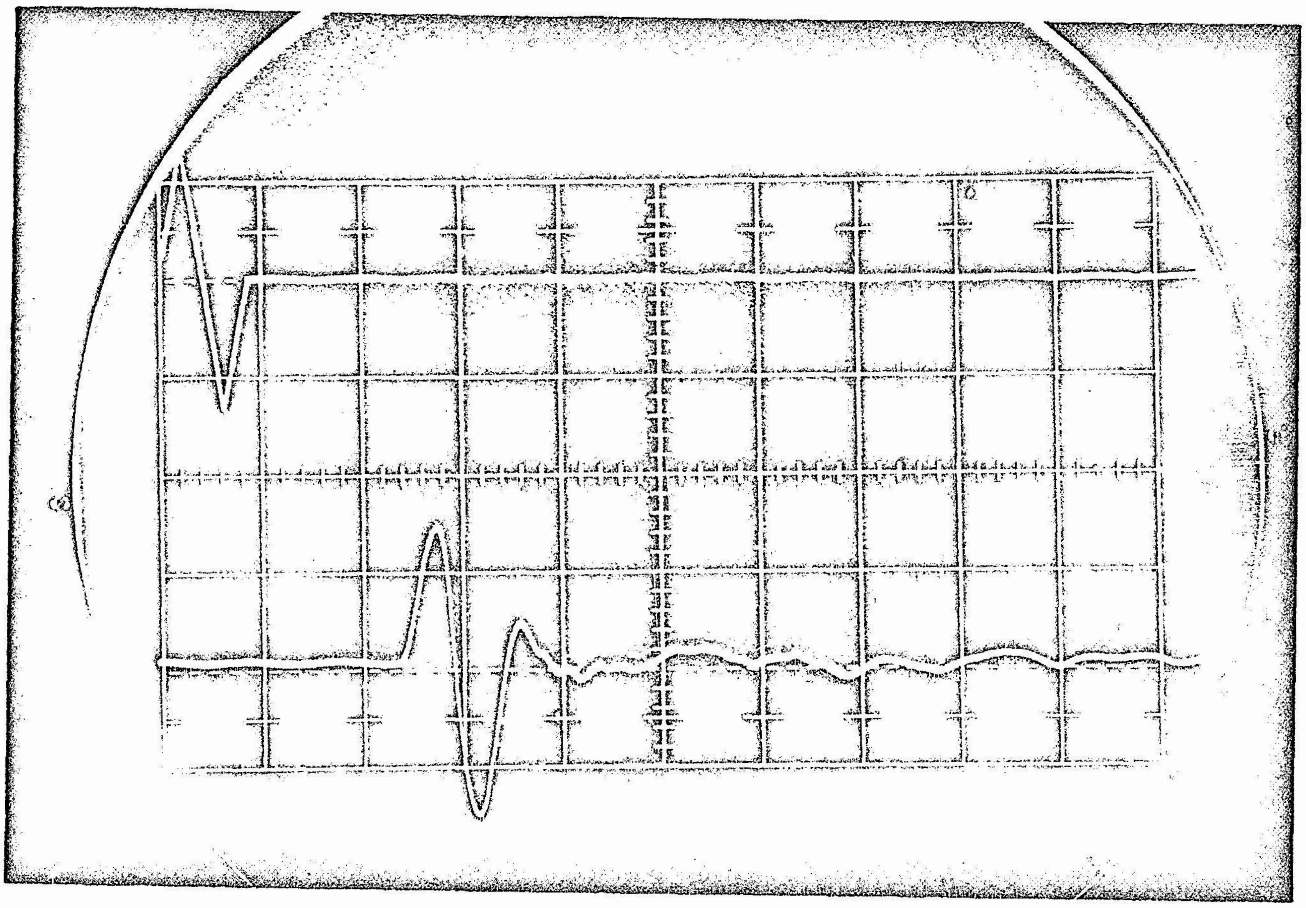


Figure 2. 4.76 msec duration N-wave

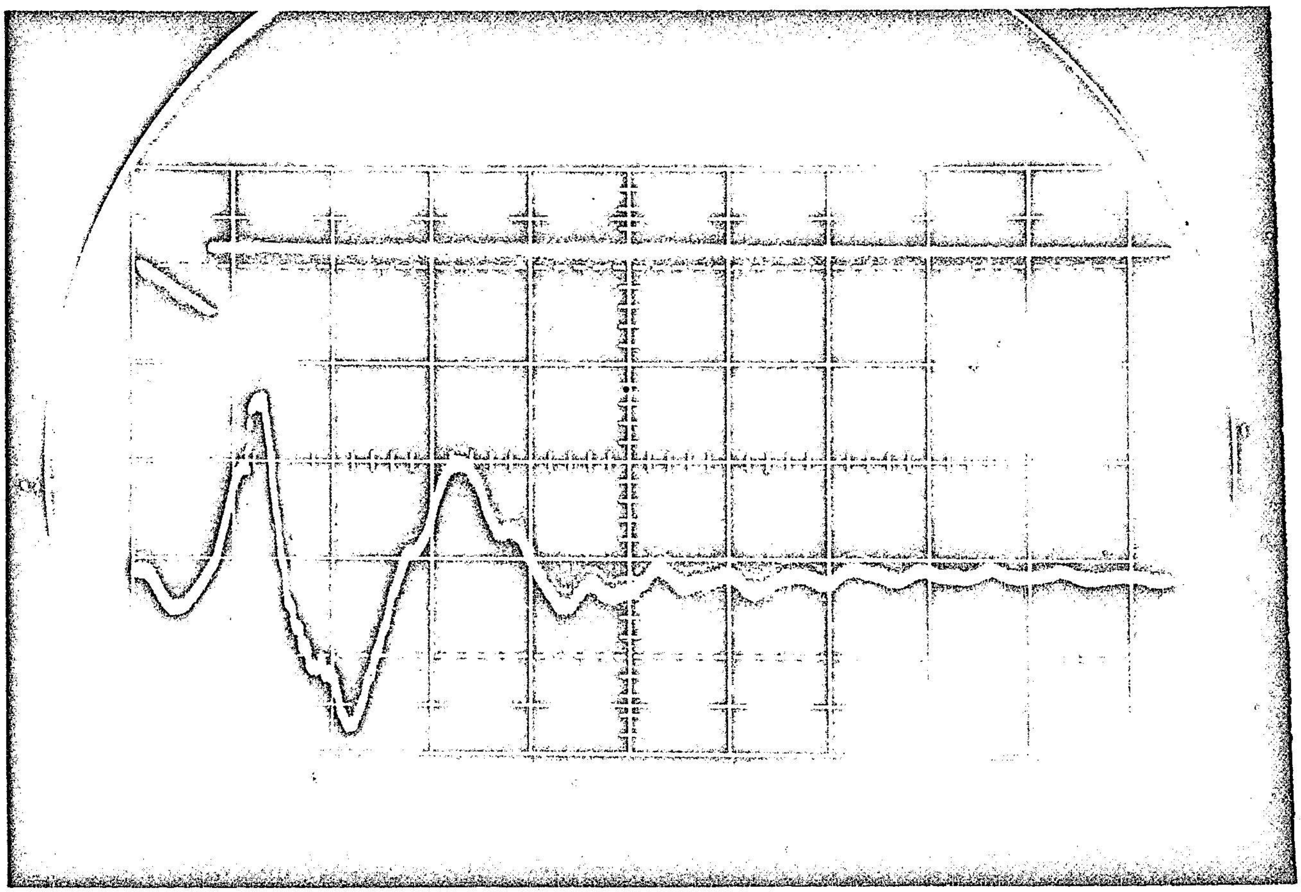


Figure 3. 125 msec duration N-wave

Spectral Analysis

The spectral analyses were carried out as follows: The output from the FM tape recorder was fed into a Spectral Dynamics Corporation (SDC) Dynamic Analyzer Model SD-101A. The beat frequency signal for heterodyning was obtained from a SDC sweep oscillator Model SD-104A-2. The crystal filter pass of the Dynamic Analyzer was 2 Hz. The signal was then put through a Hewlett-Packard Moseley Division Logarithmic Converter Model 7561A and from there to a Hewlett-Packard Moseley Division 2D-3 Time Base. The sweep was set at 1 Hz/sec. The output was graphically recorded on a Moseley Autograf Model 2 DR-2 X-Y Recorder. Figures 4-6 show the spectral power densities for the different types of sonic booms.

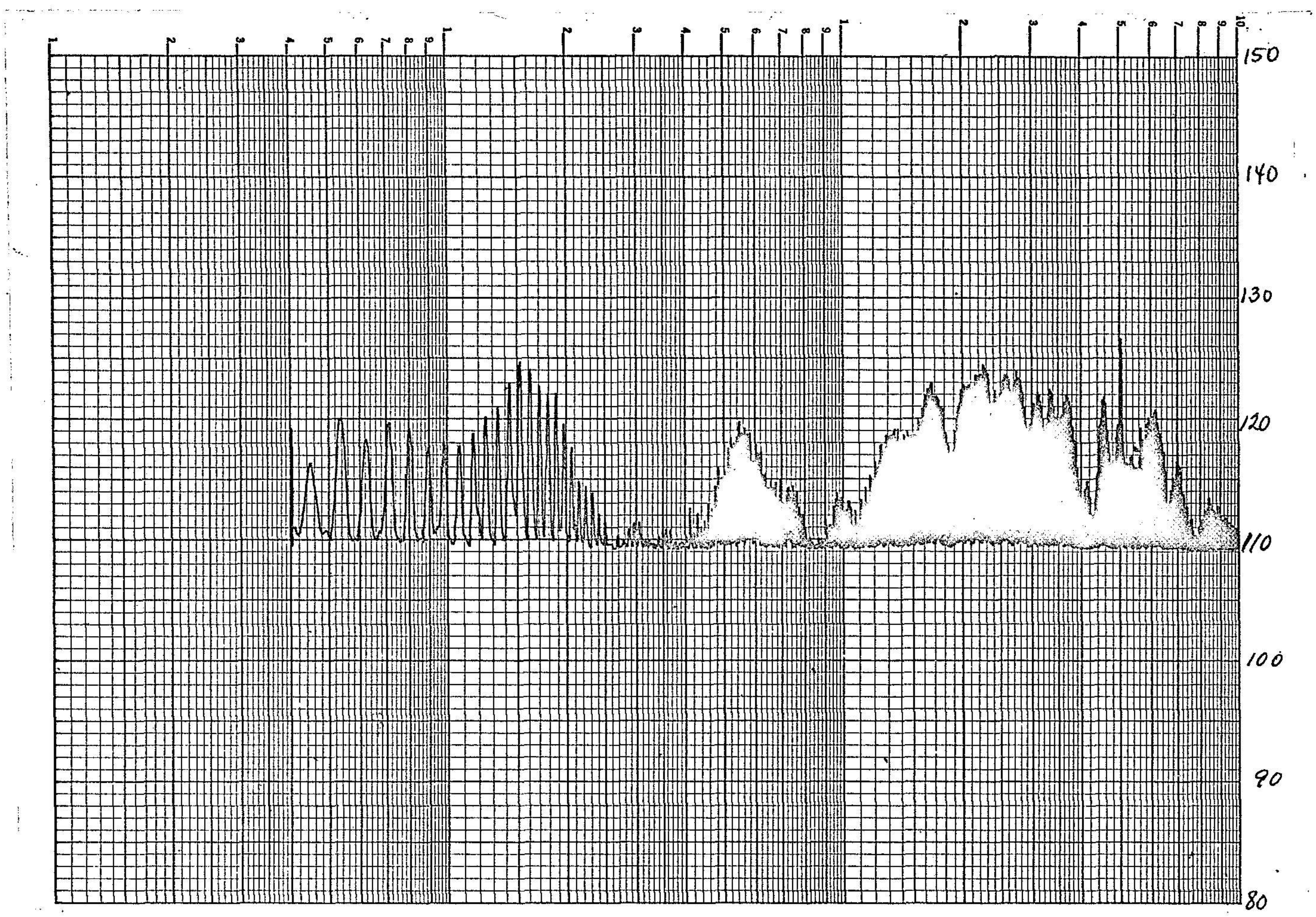


Figure 4. Spectral analysis of the 2 msec duration N-wave

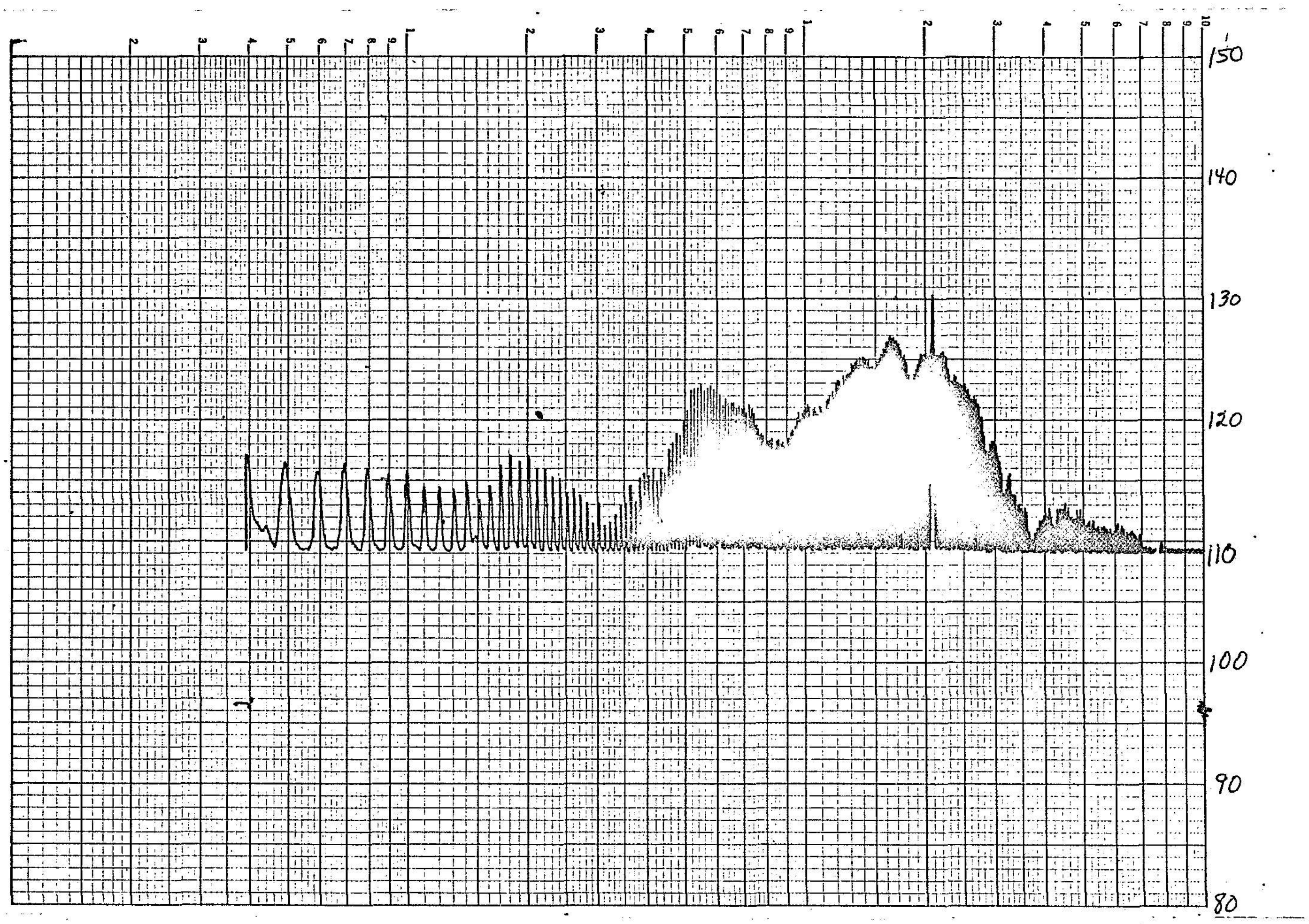


Figure 5. Spectral analysis of the 4.76 msec duration N-wave.

A run was recorded and analyzed of the complete system but without any noise presented to the microphone. This served as control.

In order to establish intensity parameters for the y-axis, a calibrated 124.7 dB puretone of 125 Hz was recorded on the FM recorder through the B&K system using the same microphone amplifier settings as during the actual experiment (120 dB large knob, -10 dB small knob), and also the same recorder input setting (3 volt.) The tape recorded stimuli were then passed through a B&K frequency analyzer Type 2107 which was set for maximum filter slope. The output of this device led to a Tektronix 545B oscilloscope for display. By

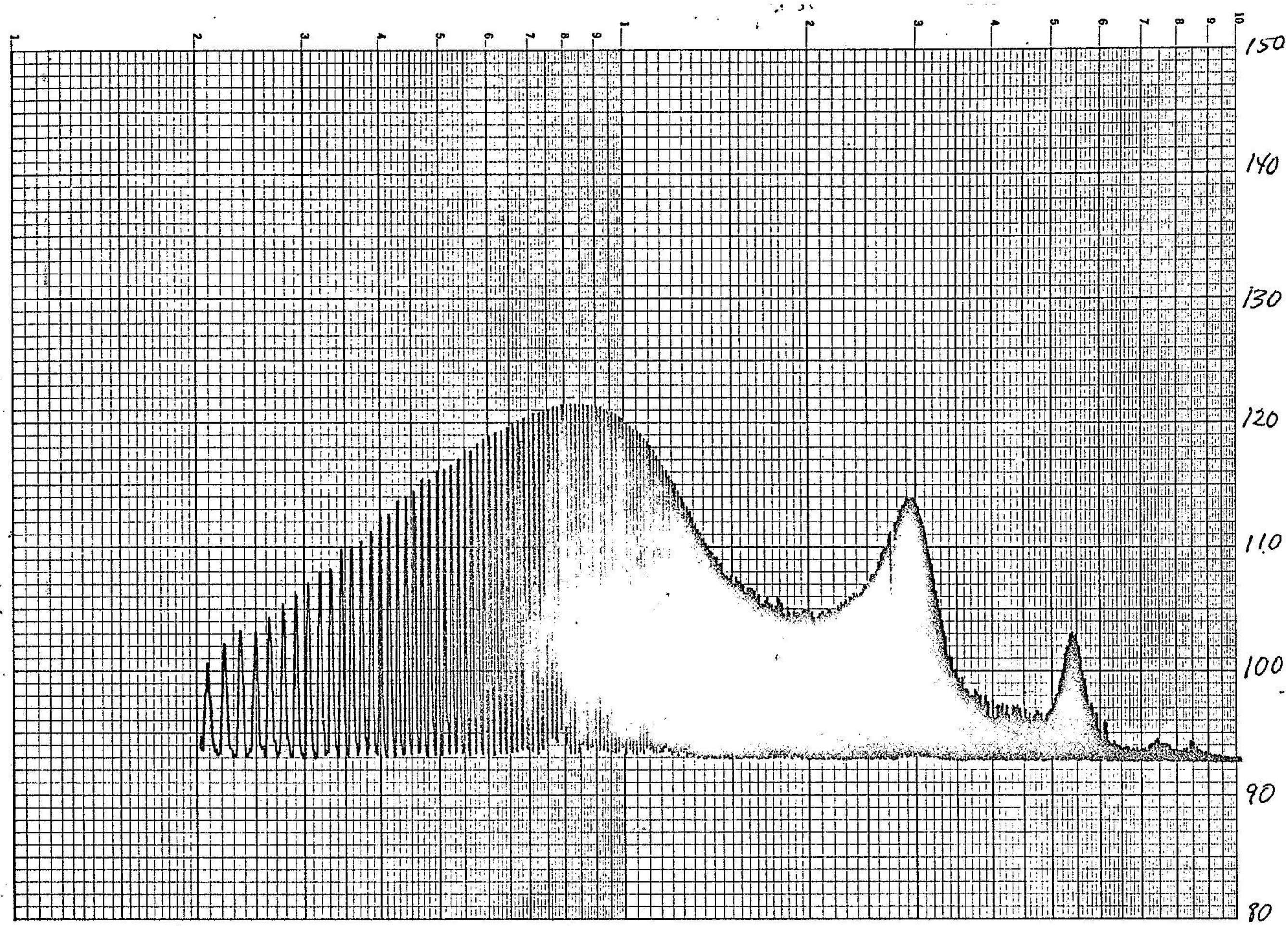


Figure 6. Spectral analysis of the 125 msec duration N-wave

manually tuning through the spectrum, the point of greatest intensity was found. The value of amplitude on the scope was recorded. The recorded 125 Hz pure tone was then fed into the frequency analyzer (but without the internal filtering) and the output led into the oscilloscope. The amplitude of this known intensity signal was recorded and compared with that found for the stimulus signal. Since the stimulus frequency selected corresponded to the greatest peak on the graph, the intensity could be ascribed to a known point on the graph. The filter passes of the B&K and the Dynamic Analyzer were about the same, so the intensities recorded on the oscilloscope and X-Y plotter were about equal. The excursion of the pen on the oscillograph was kept as small as possible to minimize errors in amplitude linearity due to pen inertia.

Histology

The temporal bones were removed and fixed in 4% gluteraldehyde for 24 hours. The osseous labyrinth was carefully removed and the membranous labyrinth stained for half an hour with 1% osmium tetroxide. The tissue was next dehydrated in successive steps of 50, 70, and 95% ethanol. The stria vascularis was removed and the tectorial membrane peeled off of the organ of Corti. The basilar membrane and organ of Corti were removed one coil at a time and placed on a glass microscope slide. Glycerin was added over the tissue and a cover slip was mounted and sealed with wax. The specimen could then be studied under oil immersion with a phase contrast microscope.

RESULTS AND DISCUSSION

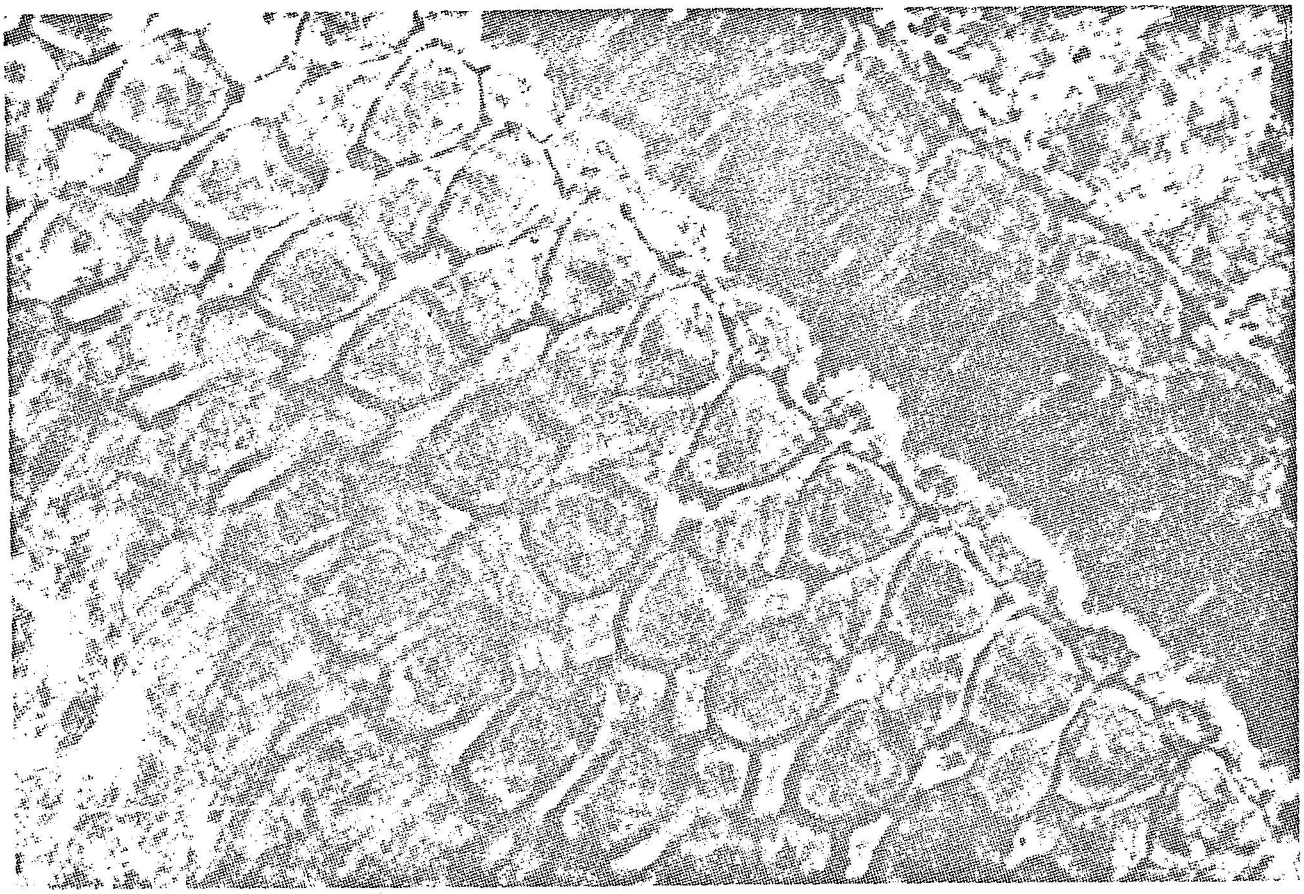
In all 24 guinea pigs exposed to the sonic booms, damage was restricted to the apical turn, leaving the other turns normal (Figures 7 and 8.)

Figure 7

Apex - Many scars following sonic boom

Figure 8

2nd Turn - Normal structures, same guinea pig after sonic boom exposure



Since the time of sacrifice of the guinea pigs ranged from 24 hours to two weeks after exposure, it was felt that the damage to the hair cells was permanent. The tissue shown in Figure 7 was taken from an animal sacrificed one week after sonic boom exposure. It can be seen that many of the damaged hair cells have been replaced by scars which appear as a straight line across the space occupied by the cell. Hair cell damage is mainly restricted to the outer two rows of cells with the inner row escaping damage completely.

A very interesting observation is that the Preyer reflex remains unchanged after exposure to sonic booms even when tested within 15 minutes of exposure. This is very important because it demonstrates that no audiologic test can uncover the damage even though it is very evident when examined histologically. Thus, sonic boom experiments conducted with human subjects might reveal no impairment of auditory function while, in fact, damage may be present. This is a result of the organization and function of the basilar membrane. Low frequencies cause small displacements of the entire basilar membrane so that there are many cells capable of responding to the stimulus even though the cells located in the region where the maximum displacement occurs are destroyed. For this reason, low frequency hearing may not be affected even though the apical turn is damaged. However, a weakening has occurred which could in time affect other areas, thereby aiding the development of a hearing loss.

A valid question could be raised that presenting sonic booms every second could be more detrimental than exposure of once or twice a day, which would be more realistic in the normal community. Repetitive exposure could well be more destructive as a result of the addition of damage before the cell could have a chance to repair itself; thus, reversible damage could be converted to irreversible damage by rapid consecutive stimulation. Additional research would have to be undertaken to clarify that point.

We noted that damage was similar regardless of the pulse signature. Destruction always occurred in the apical turn of the cochlea. This is indicative of the massive displacement of the basilar membrane created by pulses of such magnitude. The usual discrete point of maximum displacement produced by sounds of moderate intensity (85 dB) is replaced by a broad area in which the membrane is distended to its maximum.

Thus, it would appear that no design of an airplane wing would be acceptable for reducing the harm of sonic boom exposure except one which would considerably reduce the intensity.

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