THE ROLE OF THE MIDDLE EAR IN ACOUSTIC TRAUMA FROM IMPULSES.*†‡

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ABSTRACT.

Exposure to high intensity impulse noise may produce a wide range of audiometric and histological effects in experimental animals. The objective of this study was to assess the changes in the middle ear mechanism after impulse noise exposure and to relate these changes to the audiometric and histological effects.

Nine monaural chinchilla were exposed to either 161 or 166 db peak SPL impulses of 1 msec "A" duration, presented at a rate of 1 per minute for 50 minutes. The conductive mechanism of the chinchilla was assessed using standard clinical measures of static and dynamic impedance before and after the noise exposure. Auditory thresholds were measured before and after noise exposure using the average evoked response (AER) technique. At 30 days post-exposure, the animals were sacrificed for histology.

Pre-exposure tympanometry showed that: *1.* the total mean impedance of the chinchilla ear is considerably lower than that of man; *2.* a method related hysterisis effect is present in both the susceptance and conductance tympanograms; and *3.* sedation has a significant effect on the total impedance of the ear and on the shape of the tympanograms. After exposure to high level impulse noise: *1.* tympanograms become irregular and double peaked, indicating tympanic membrane stress; *2.* for the given exposure, 166 db is the impulse intensity needed to rupture consistently the tympanic membrane; and *3.* audiometric and histological data correlate with the tympanometric findings and demonstrate a protective effect of a tympanic membrane rupture on the cochlea.

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INTRODUCTION.

A consistent problem in noise research is a lack of consistency in the experimental results. The problem of variability or susceptibility is particularly acute in impulse noise research.[1] As early as 1946, Reid[2] reported a wide range of audiometric findings in humans after exposure to 180 db impulses. The conductive mechanism of the middle ear was implicated as one possible source of variability. Pearlman,[3] in a review of blast injuries to the ear, suggested that rupture of the tympanic membrane during the positive phase of the blast might tend to lessen inner ear damage by reducing the efficiency of transmission through the middle ear. More recently, Akiyoshi, *et al.*,[4] noted that in rabbits and guinea pigs exposed to sound pressures less intense than 640 gm/cm^2 (189 db SPL), injury to the tympano-ossicular system was slight, other than tympanic membrane perforation, whereas damage to cochlear hair cells was extensive. Animals exposed to pressures greater than 1 kg/cm^2 (193 db SPL) revealed extensive damage to the tympano-ossicular system, including ossicular dislocation and fracture; however, damage to the cochlea was small.

The concepts of a critical intensity[5] for variability and "tough" and "tender" ears[6] have been put forth in an effort to explain the problem of susceptibility. More recently, Ward[7] reviewed the concept of susceptibility to hearing loss following continuous noise exposure, and concluded that susceptibility was normally distributed in a population. Hamernik, *et al.*,[1] using 1 msec "A" duration impulses of 155, 161 and 166 db SPL, have shown an inverse correlation between the size of the hair cell lesions in the chinchilla and the sound pressure level of the impulses. The 161 db impulses appeared to be a critical intensity or the intensity which exhibited the widest range of histological effects, while the results of the 166 db exposure level were suggestive of a protective effect secondary to a conductive failure.

The purpose of this study was: *1.* to measure the normative tympanometric values of the chinchilla conductive mechanism; *2.* to correlate the impedance of the chinchilla conductive mechanism with the degree of TTS produced by impulse noise exposure, and *3.* to note the conductive changes (if any) following impulse noise exposure.

MATERIALS AND METHODS.

Chinchillas weighing between 600 and 900 grams were made monaural by means of surgical destruction of the left cochlea under sodium pentobarbital anesthesia (0.5 cc/kilo, i.p.). A chronic electrode was placed on the dura over the region of the inferior colliculus, and a reference electrode was inserted into the nasal cavity. The electrodes were attached to a two-prong transistor plug which was fastened to the skull with cranioplastic cement.

Following a two to four-day recovery period, audiograms were obtained for each animal, using the auditory evoked response (AER) technique.[8] Thresholds were determined at 250, 500, 1k, 2k, 4k and 8k Hz, the test signal being a 20 msec tone burst with a 5 msec rise-fall time. The threshold was considered to be the half-way point between the lowest intensity eliciting a definite N_1-P_1 response and the highest intensity producing no response. Three pre-exposure thresholds were obtained at each of the six test frequencies, the accepted pre-exposure value being the mean of the three tests. Following noise exposure, thresholds were obtained at log intervals from 0.5 to 775 hours.

Pre-exposure tympanometric data were obtained from the intact ear of each animal at least twice, using the Grason-Stadler model 1720 Otoadmittance meter. The test probe was fitted with a small, inflatable tracheotomy tube cuff to facilitate an airtight seal between the probe and the external ear canal. During the tympanometric testing, the animals were lightly sedated with sodium pentobarbital (0.2 cc/kilo, i.p.). Impedance in acoustic ohms was determined from the tympanometric curves. After noise exposure, tympanograms were obtained on the same day and at varying time intervals until either the tympanogram returned to normal, or the animal was sacrificed.

The traumatizing impulses were generated by a shock tube (a compressed air driven source) with the animal oriented at normal incidence in an anechoic chamber. The exposure consisted of 50 impulses at an intensity of 161 or 166 db peak SPL with a 1 msec "A" duration delivered at a rate of one per minute.

For tympanometric control purposes, one animal underwent myringotomy twice for creation of different sized tympanic membrane ruptures. Another animal underwent incus removal to establish ossicular discontinuity.

At the conclusion of the experimental period, the noise-exposed animals underwent exploratory tympanotomy under general anesthesia, the approach being via an opening in the lateral bulla. The tympanic membrane was examined and then retracted to allow direct observation of the ossicular chain. Following this examination, the animals were sacrificed and prepared for the surface preparation histology.

RESULTS.

A. Pre-Exposure AER Thresholds and Tympanometry.

A total of 10 experimental animals was used. One of these was used for tympanometric purposes only, five were exposed to the 161 db impulse noise level, and four were exposed to the 166 db level. The mean

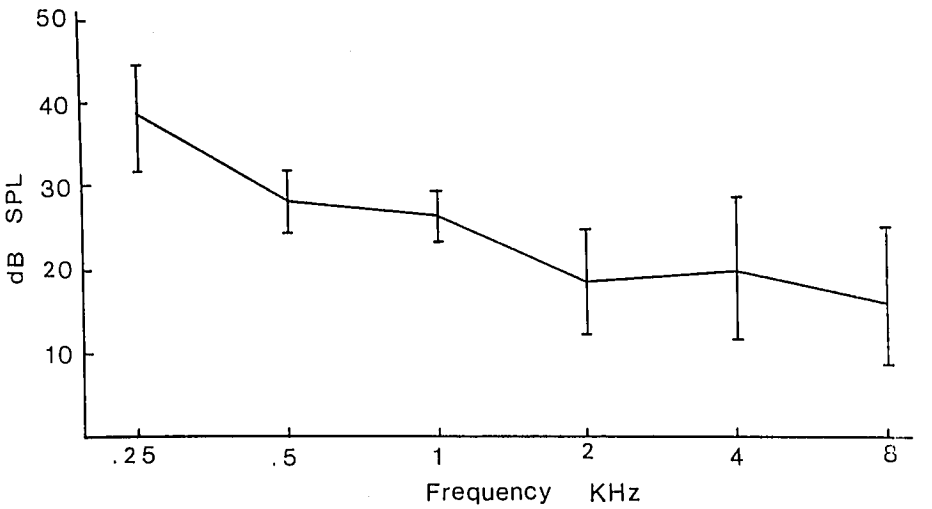

Fig. 1. Mean pre-exposure AER thresholds for 10 animals (Bar = ± 1 standard deviation).

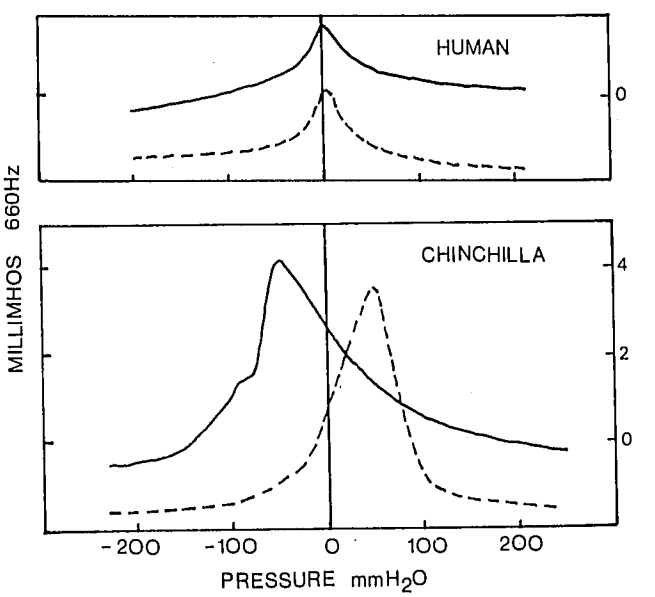

Fig. 2. Schematic of normal human and chinchilla tympanograms. Six hundred sixty Hz susceptance curves, traced from positive to negative pressure (————) and from negative to positive pressure (- - - - -).

pre-exposure AER thresholds for the 10 experimental animals are shown in Figure 1.

A typical pre-exposure chinchilla tympanogram is illustrated in Figure 2, and is compared with a normal human tympanogram. Tympanograms were obtained for both 220 and 660 probe tones; however, the 660 Hz tracings were more diagnostic and, therefore, the discussion and illustra-

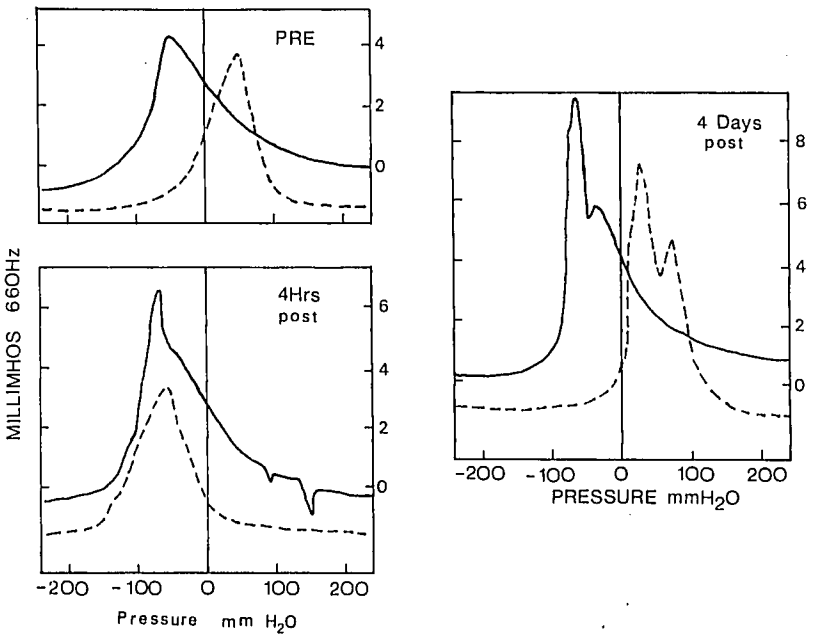

Fig. 3. Post-exposure chinchilla tympanograms compared with pre-exposure tracing. Six hundred sixty Hz susceptance curves.

tions refer only to 660 Hz curves. The pressures applied to the ear canal ranged from +400 to −400 mm H_2O. The ordinates of the tympanometric illustrations are in arbitrary units.

Several points can be made about the normal tympanometric curves of the chinchilla: *1.* the calculated overall acoustic impedance in ohms is significantly lower for the chinchilla than for the human. The mean impedance for 21 ears was 705 Ω with the 220 Hz probe tone and 144 Ω with the 660 Hz probe tone. This compares with normal human median impedance values of 1856 Ω and 409 Ω. The lower impedance of the chinchilla ear may possibly be the result of the chinchilla's having a lighter and more delicate middle ear mechanism; *2.* the chinchilla tympanogram constantly exhibits a hysterisis pattern not seen in the human; *i.e.*, the curves, whether traced from an initial positive pressure applied to the external canal to negative pressure, or vice-versa, do not overlap; and *3.* the normal sedated chinchilla was noted to have a mean negative intratympanic pressure of approximately −43 mm H_2O. The negative pressure is a consequence of the sedation, because several animals were tested awake, and, after correction for the normal hysterisis effect, no negative pressure was observed.

B. Post-Exposure Tympanometry.

Figure 3 illustrates some of the characteristic changes in the tympanogram after impulse noise exposure. The normal hysterisis pattern was

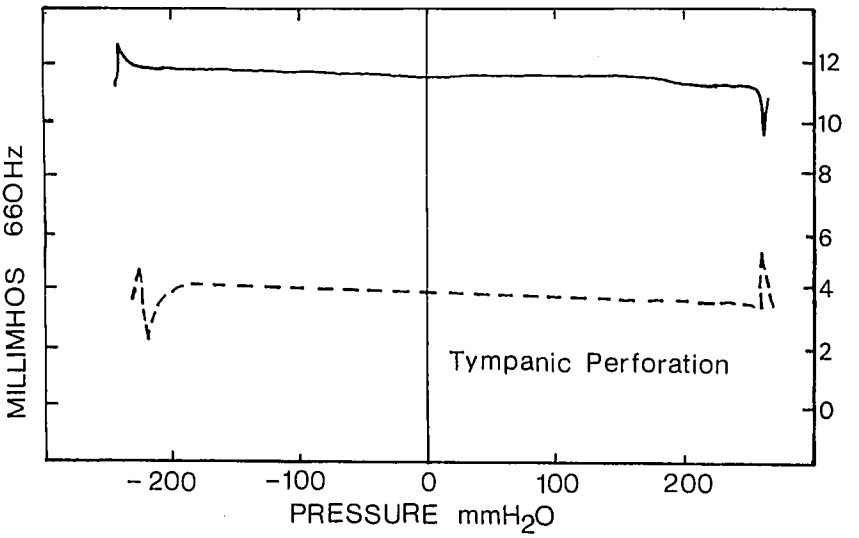

Fig. 4. Typical tympanograms of chinchilla with TM perforation.

TABLE I.

Summary of Acoustic Impedance, Threshold Shift and Histological Data.

Exposure	Animal	Pre-Exposure |Z| 660	Two-Hour Post-Exposure |Z| 660	Final |Z| 660	Average 1K, 2K, 4 kHz db Shift Two Hours	Average 1K, 2K, 4 kHz db Shift 30 Days	Hair Cell Loss
	2	140	121	142	+ 1	+ 1	Small
	3	141	—	187	+32	+ 2	Moderate
161 db	4	144	113	188	+10	− 3	Small
	5	149	156	130	+71	+47	Severe
	1	186	134	160	+60	+24	Severe
	7	75	—	113	+24	+ 3	Small
	8	106	—	140	+25	− 8	Small
166 db	6	163	—	116	+33	+ 9	Small
	9	168	—	123	+ 8	+ 2	Small

generally lost for 24-72 hours after the noise exposure. In addition, the normal smooth character of the curves was replaced by a tendency toward choppiness with a dominant "double-peak" or "W" pattern, especially with the 660 Hz probe tone. The rough pattern gradually disappeared in five to 15 days.

Figure 4 illustrates the typical pattern seen with tympanic membrane perforations. The tympanometric curve was characterized by a flat response throughout the range of pressures applied to the external canal. Table I briefly summarizes data obtained for the experimental animals. A comparison of changes in calculated acoustic impedance between pre-exposure,

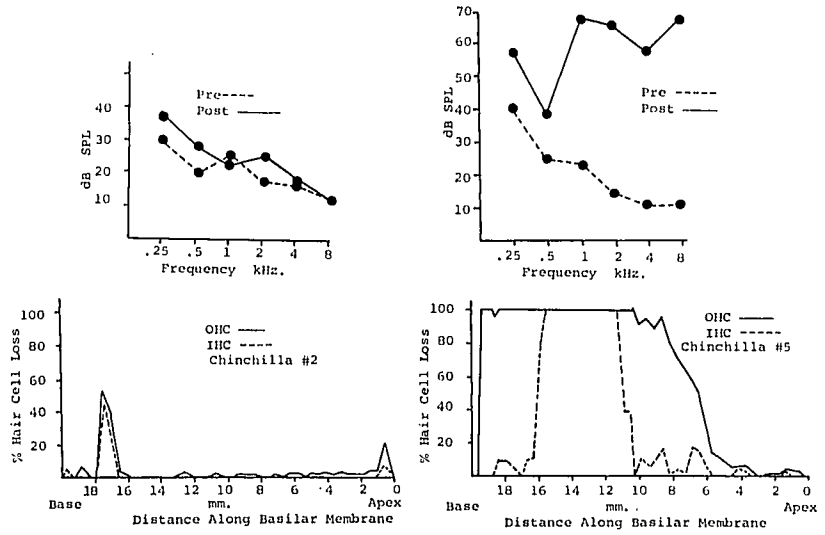

Fig. 5. Pre-exposure and PTS audiograms and corresponding cochleagrams for extreme examples of the 161 db group.

day of exposure, and termination evaluations indicates that there were no consistent overall changes in the 161 db group. The 166 db group showed perforations on the day of exposure, but by 30 days, the impedances had returned to normal; furthermore, there was no correlation between the acoustic impedance before exposure and either the level of TTS or the size of cochlear lesions.

C. Audiometric and Histological Results.

1. One Hundred Sixty-One db Exposure. The five animals exposed to the 161 db impulse noise exhibited the widest range of variability in the histologic and audiometric data (Table I). The pre- and post-exposure audiograms for the two extremes (animals 2 and 5) of the 161 db group are presented in Figure 5 with their corresponding cochleagrams. Animals 1 and 5 suffered a large TTS at all frequencies. For low frequencies, $.25 \leq f \leq 1$ kHz, the TTS varied between 45 and 65 db, while at the higher frequencies, $2 \leq f \leq 8$ kHz, TTS varied from 50 to more than 70 db. In both animals, the recovery curves resolved to a severe PTS of from 10 db at the lower frequencies, to nearly 60 db at the higher frequencies ($1 \leq f \leq 8$ kHz). There was a concommitant loss of both inner and outer hair cells throughout the basal one-half to one-third of the cochlea in this animal. Animals 2 and 4, on the other hand, suffered relatively small (generally less than 30 db at all frequencies) TTS, essentially no PTS (less than 10 db at all frequencies), and only relatively small cochlear lesions. Animal 3 suffered a tympanic membrane perforation of moderate size which healed spontaneously in 15 days; this animal had less than 10 db TTS below 1 kHz and 40-60 db of TTS at 2-8 kHz. This loss resolved to normal pre-ex-

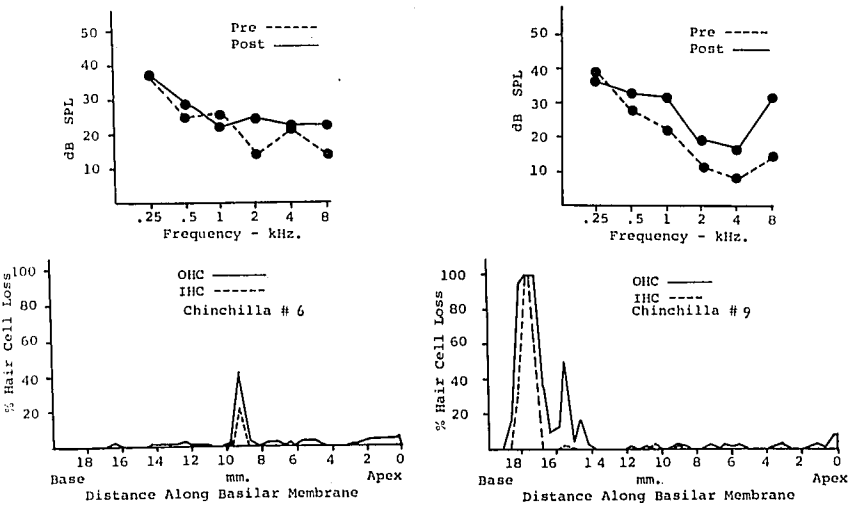

Fig. 6. Pre-exposure and PTS audiograms and corresponding cochleagrams for extreme examples of the 166 db group.

posure levels by 774 hours. His cochleagram shows a low level loss (less than 20 percent) of primarily outer hair cells throughout most of the cochlea. At sacrifice, tympanotomy of these animals revealed no significant middle ear findings, with the exception of a healed tympanic membrane perforation in animal 3.

2. One Hundred Sixty-Six db Exposure. The four animals exposed to the 166 db SPL peak pressure impulses were a much more homogeneous group. The two extremes are presented in Figure 6. TTS ranged from 20 to 40 db at the lower frequencies ($\leq$ 1 kHz) and from 20 to 60 db at the higher frequencies. Recovery from the TTS was nearly complete at most frequencies, and final audiograms showed relatively small amounts of PTS (less than 20 db) at the higher frequencies. Each chinchilla was found to have relatively small hair cell lesions in the basal one-half of the cochlea. All animals in this group were found to have either a single large perforation or multiple smaller perforations of the tympanic membrane, the latter giving the appearance of a "shotgun" effect. The perforations closed spontaneously in 12-25 days. All perforations were demonstrated both tympanometrically and by direct observation. Tympanotomy at sacrifice revealed all ossicular chains to be intact, the only deviations from normal being the healed tympanic membranes.

3. Experimental Controls. The tympanic membrane of one animal was surgically ruptured (no noise exposure) to determine the effect of a perforation on the AER threshold. The averages of his 1, 2 and 4 kHz thresholds are shown in Table II. Following a small tympanic membrane rupture, no discernible deterioration in threshold was noted. After the drum had

TABLE II.

Summary of Acoustic Impedance and Threshold Shift Data on Experimental Control Animal Number 10.

	Pre-Perf	Small Perf	Small Perf Healed	Large Perf	Large Perf Healed
|Z| 660	195	—	163	—	155
Average threshold at 1, 2, 4 kHz (db SPL)	40	32	37	46	32

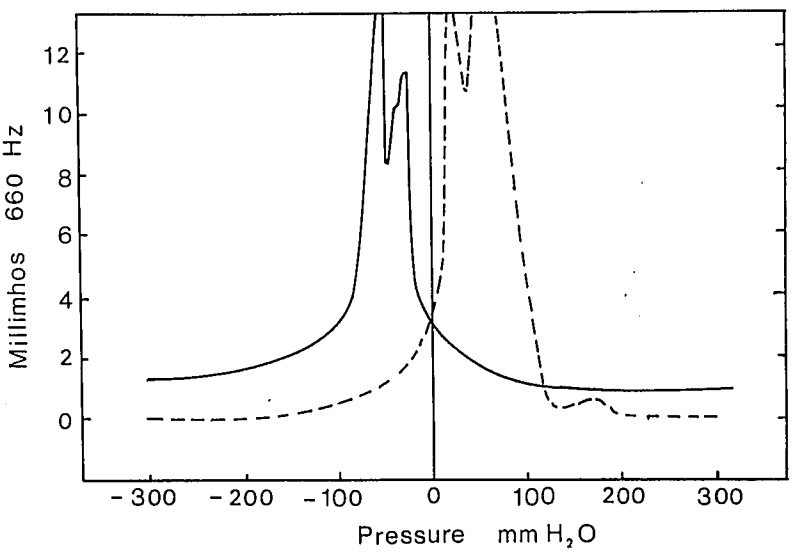

Fig. 7. Tympanogram of chinchilla with ossicular discontinuity. Note prominent double peak and that tracing excursion exceeds instrument capability.

healed, a second, larger defect was created. The larger defect produced an average increase in threshold of 10 db, with the largest increases in the lower frequencies. The thresholds gradually returned to normal levels as the perforation healed spontaneously over a 35-day period. The tympanometric course of this animal was similar to that of the animals suffering large perforations following the 166 db exposure.

4. Ossicular Discontinuity. It was noted that none of the exposed animals suffered obvious damage to the ossicular chain. For documentary purposes, one animal underwent removal of the incus through a posterior bulla approach. Following complete healing of the defect, tympanometry was performed. The tympanogram is illustrated in Figure 7. These curves are considerably different from any of those obtained in the impulse noise exposed animals, thus lending additional evidence that there was no severe ossicular damage in the noise-exposed group.

DISCUSSION.

Several differences have been noted between the normal chinchilla and human tympanograms: *1.* the lower value of compliance in the chinchilla is probably largely explained by the reduced mass of the chinchilla's conductive mechanism as well as by the bullar middle ear structure of this animal, and *2.* its lower resistance is probably explained by the shorter cochlear of the chinchilla as compared to human (*i.e.*, 20 mm/35 mm).

The flat tympanograms obtained in animals with tympanic membrane perforations suggest loss of eustachian tube function in that no applied air pressure was lost through the tube into the pharynx when a +400 mm H_2O pressure was applied to the open middle ear cavity for sustained periods. The explanation for this probably lies in the use of sedation during measurement as well as in tubal edema of traumatic origin.

It was noted that the post-exposure tympanograms from animals without perforations were irregular and choppy in nature. A similar pattern is known to occur in humans with healed tympanic membrane perforations; thus, these curves were interpreted as characteristic of tympanic membrane stress with probable rupture of the radial fibers of the middle drum layer in the absence of an actual perforation. The tympanograms gradually returned to normal over a period of 10-15 days, a time compatible with the reorganization and repair of such stressed fibers.

The previously recorded[1] inverse correlation between hair cell loss and noise level between 155 and 166 db SPL peak pressure impulses seems to be accounted for in a protective effect of a large tympanic membrane rupture. This mechanical damage most likely occurs relatively early in the exposure sequence, at an intensity of 161 db or higher, thus protecting the cochlea from a significant amount of the acoustic trauma.

The effect on the AER threshold of sizeable tympanic membrane rupture appears to be relatively minor, since: *1.* the thresholds of the control animal with experimental perforations were not significantly altered, and *2.* the thresholds of animals with noise-induced tympanic membrane ruptures recovered to normal pre-exposure levels before the perforations were healed; thus, the TTS observed following the impulse trauma were considered to be of sensori-neural rather than of conductive origin.

The previously recorded extreme variability at the 161 db exposure level cannot be explained on the basis of middle ear differences or changes, because tympanometry, acoustic impedance comparison, and direct observation failed to reveal any significant correlations between pre- and post-exposure states among the animals. The 161 db level seems to be a transition level between cochlear damage and cochlear protection on the basis of tympanic membrane rupture; it is also the intensity at which individual variability is, for unexplained reasons, extreme. The differences in sus-

ceptibility across subjects at this level appear to be post-stapedial rather than conductive. In any event, because of the marked variability, the 161 db SPL peak pressure 1 msec A-duration exposure appears ideal for the further exploration of the problem of the physiologic basis for susceptibility to acoustic trauma.

On the basis of both tympanometry and observation through the operating microscope, tympanic membrane perforation, is essentially the only structural damage occurring to the conductive mechanism in these experiments. Neglecting species differences (rabbits vs chinchilla), a comparison of the impulse noise peak SPL used here with those employed by Akiyoshi, *et al.*, suggests that there is a considerable dynamic range (161-193 db) over which tympanic membrane rupture occurs alone before the ossicular chain becomes significantly involved. When the chinchilla is compared to man, von Gierke[9] reported that the human tympanic membrane ruptures at blast waves of 180 db; therefore, if one neglects the signatures of the impulse, the chinchilla may be 15-20 db more susceptible to tympanic membrane rupture than man. The net effect of the chinchilla vulnerability is that, for high level blasts (>160 db), the chinchilla may be less susceptible to sensori-neural hearing loss from noise trauma because of the protective effect of the tympanic membrane rupture.

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