Arch Otorhinolaryngol (1981) 230: 285–288

Archives of
Oto-Rhino-Laryngology

Cochlear Vascular Histology in Animals Exposed to Noise* **

D. Vertes and A. Axelsson

Dept. of Otolaryngology and Audiology, University of Göteborg, Sahlgren's Hospital, S-413 45 Göteborg, Sweden

Summary. The regularly occurring cochlear vessels in the external wall and spiral lamina were studied in the guinea pig and chinchilla following exposure to various types and durations of noise. A soft-surface specimen technique with or without injection of a contrast medium into the vascular system was used, and the occurrence of specified vascular parameters was assessed using phase-contrast microscopy. Noise does not seem to result in any extraordinary vascular pathology, but a slight, overall decreased blood supply to the cochlea and localized changes depending on cochlear turn are suggested.

Key words: Cochlear vasculature – Histology – Acoustic trauma – Guinea pig – Chinchilla

Introduction

There are two mechanisms by which noise may affect inner ear vessels. First, sound may influence cochlear vessels directly by its mechanical or hydrodynamic actions on cochlear membranes. Second, even at moderate sound levels, changes in inner ear vessels may result from a general circulatory response. These effects may vary with spectral content, intensity, and duration of the noise exposure as well as its duty cycle (continuous or impulse noise). Noise can differentially influence cochlear vasculature across animal species. Noise can also have a selective effect on different cochlear vascular systems which in turn may vary along the length of the cochlea. Immediate and late effects may also vary. That all these factors differentially influence the resulting histopathology is evidenced by the wide variation in type, extent, and location of vascular change reported to follow pure-tone or noise overexposure.

* This investigation was supported by the Labour Environment Protection Fund (78/69:2)
** Presented at the 17th Workshop on Inner Ear Biology in Stockholm, June 23–25, 1980

0302-9530/81/0230/0285/$ 01.00

Methods

Details regarding our techniques for studying inner ear vasculature may be found in previous publications (Axelsson et al. 1975; Vertes and Axelsson 1979). In the studies to be reported we have employed a soft-surface specimen method with or without injection of a contrast medium into the vascular system. With this technique cochlear membranes and soft tissues are studied as surface preparations in decalcified specimens. In most of our experiments we have examined all of the regularly occurring cochlear vessels in the external wall and spiral lamina. Each vessel is studied with respect to the occurrence of certain vascular parameters. Control and experimental animals are mixed, and specimens are randomly examined in a double-blind fashion. The results are statistically analysed for differences between experimental and control groups.

To date we have completed four noise vascular studies. The exposure paradigms are shown in Fig. 1. In our first investigation (Lipscomb et al. 1977) we used 20 chinchillas, ten of which were exposed to a 110-dB SPL broad band noise for 8 h. Hearing was tested using behavioural measures before and after exposure, sensory cell counts made and cochlear vasculature studied using contrast injection techniques. Animals were killed 8–16 days after exposure. Findings included (1) greater hair cell damage apically than basally which decreased from the third row of outer hair cells to the inner hair cells; (2) poor agreement between hearing impairment and hair cell loss; (3) poor injection of contrast media in the vasculature; (4) no remarkable changes in spiral lamina vessels; and (5) changes in stria vascularis, which were most common in turn 3, including vacuoles at the attachment of Reissner's membrane and intercellular gaps between cells in stria epithelium.

In another study using seven test and four control chinchillas, experimental animals were exposed to pink noise of varying intensities from 110-dB for 8 h to 125-dB for 15 min (Vertes et al. 1979). The time factor decreased by one-half for each 3-dB increase in noise intensity. After a 3-week survival period animals were examined for histopathological changes using decalcified preparations and surface techniques. In this case no contrast was injected in the vessels. We found (1) minimal scattered hair cell loss; (2) vascular changes regardless of type, most common apically and in spiral lamina vessels; (3) more variability in the vascular pattern of experimental animals; (4) mean values in experimental animals statistically different from controls including: (a) decreased red blood corpuscle (RBC) density in the radiating arterioles and collecting venules of the spiral lamina and limbus vessels, (b) increased occurrence of perivascular cells in the vessel of the spiral prominence, limbus vessels, and the vessel of the tympanic lip, and (c) decreased occurrence of perivascular cells in the vessel of the basilar membrane.

In another experiment nine guinea pigs were exposed to a 120-dB SPL 4 kHz narrow band noise for 30 min and six animals served as controls in a study of the acute effects of noise (Axelsson et al.

ANIMAL	NOISE	INTENSITY	DURATION	SURVIVAL
CHINCHILLA N=10 C=10	BROADBAND	110-dB SPL	8 H	8-16 DAYS
CHINCHILLA N=7 C=4	PINK NOISE	110-125-dB SPL	8-1/4 H	3 WEEKS
GUINEA PIG N=9 C=6	4 kHz NARROW BAND	120-dB SPL	30 MIN	NO
GUINEA PIG N=21 C=12	4 kHz PURE TONE	124-140 dB SPL	5-80 MIN	NO

Fig. 1. Summary of the experimental details of four noise-vasculature studies

1981). Since the animals were killed immediately after exposure, hair cell counts were not made and histological observations were limited to the vasculature. Results included (1) few vascular effects; (2) large individual variations; (3) vascular changes, regardless of type, dependent on location in the cochlea, with no changes in turn 2 and an approximately equal number of changes in turns 1, 3, and 4; (4) vascular changes, regardless of type, more frequent in spiral lamina than external wall vessels; (5) vascular changes, regardless of type, more frequent in vessels facing perilymph than in those facing endolymph; and (6) mean values in experimental animals statistically differing from controls including: (a) more longitudinal orientation of RBCs and (b) more spaced (less dense) RBCs in some vessels apically.

In our fourth noise-vasculature study, 33 guinea pigs, 12 of which were controls, were employed in experiments which also included electrophysiological recordings of cochlear microphonic (CM) potentials (Vertes et al. 1981). Twenty-one animals were exposed to a 4 kHz pure tone at levels ranging from 124-dB to 140-dB SPL for 5–80 min. Animals were killed following CM recording. The results were as follows: (1) changes in CM sensitivity supported the equal energy principle; (2) overall diminished blood supply to the cochlea evidenced by a decreased RBC density and an increased occurrence of aggregations and plasma gaps; (3) vascular changes, regardless of type, dependent on location in the cochlea, with least changes in turn 2 and an approximately equal number of changes in turns 1, 3, and 4; and (4) increasing vascular changes with increasing noise dose, but depending more on exposure duration than intensity.

General Conclusions

Based on our evaluations of different species of animals, including primarily guinea pigs, rats, and chinchillas, we have assembled a great deal of information (some normative) on the cochlear blood supply. Analyses of those data lead to several general conclusions regarding the effects of noise on the cochlear vasculature. These are as follows:

1. Noise does not seem to result in any extraordinary vascular pathology which is immediately evident using light microscopy.

2. Marked variability has been noted between: (a) species, (b) individual animals, (c) cochlear turns, and (d) vessels within each turn. As with hair cell damage, we have found that some, but not all, of this variability can be explained by the type, intensity, and duration of the noise used. For these reasons, it has become evident that studies of this type must involve a large sample size.

3. We have, on occasion, found a small perilymphatic hemorrhage present on the inside of the round window. Since similar observations have repeatedly been made in control animals, these hemorrhages do not seem to be attributable to noise.

4. In the evaluation of vascular pathology, when searching for differences between experimental and control animals, we have found that such differences tend to occur repeatedly with respect to certain parameters. We have summarized these as follows: (a) *red blood corpuscle (RBC) parameters,* including density, columns, aggregations and plasma gaps, and orientation; (b) *perivascular cell (PVC) parameters,* including PVC frequency, PVC size, and the frequency of PVCs compressing the vessel lumen; and (c) *vessel lumen parameters,* including lumen irregularity and lumen diameter. The most common change in the stria is an overall degeneration of the stria epithelium together with changes in pigmentary cells. In the studies performed thus far, in which only small differences are apparent between noise and control animals,

the existence of perivascular spaces and vessels lacking blood corpuscles (previously called avascular channels) contribute little to the differentiation of experimental and control animals. Using more severe and destructive noise exposures, however, these parameters might well be important.

5. We have found that to determine the effects of noise on the vasculature, *all* the cochlear vessels are important, not just the commonly studied vessel of the basilar membrane (outer spiral vessel) and the capillaries of the stria vascularis. Which vessel(s) show(s) the greatest number of changes after noise exposure is influenced by the type, intensity, and duration of noise exposure used. Thus far, the vessel which seems to be most resistant to change regardless of noise exposure is the vessel of the spiral prominence.

6. In general, at least two of our studies have suggested that noise results in an overall decreased blood supply to the cochlea.

7. Finally, in agreement with other authors, we have found that vascular change following noise exposure does not seem to be limited to the area corresponding to greatest sensory cell damage. Our results have suggested a shunting (perhaps compensatory) of the blood supply from one turn and/or vessel of the cochlea to another (or others). This shunting is reflected as a maintenance of "normal" vascularity in one turn at the expense of the blood supply in other turns. In our studies thus far, results have suggested that this area of more "constant circulation" is located toward the cochlear base.

Acknowledgements. Charlotte Hornstrand, Gunilla Jönsson, and Inga-Britt Christoffersson assisted in the preparation of the manuscript.

References

Axelsson A, Miller J, Larsson B (1975) A modified "soft surface specimen technique" for examination of the inner ear. Acta Otolaryngol (Stockh) 80: 362

Axelsson A, Vertes D, Miller J (1981) Immediate noise effects on cochlear vasculature in the guinea pig. Acta Otolaryngol (Stockh) 91: 237

Lipscomb DM, Axelsson A, Vertes D, Roettger R, Carroll J (1977) The effect of high level sound on hearing sensitivity, cochlear sensorineuroepithelium, and vasculature of the chinchilla. Acta Otolaryngol (Stockh) 84: 44

Vertes D, Axelsson A (1979) Methodological aspects of some inner ear vascular techniques. Acta Otolaryngol (Stockh) 88: 328

Vertes D, Axelsson A, Lipscomb D (1979) Some vascular effects of noise exposure in the chinchilla cochlea. Acta Otolaryngol (Stockh) 88: 47

Vertes D, Axelsson A, Miller J, Lidén G (1981) Cochlear vascular and electrophysiological effects in the guinea pig to 4 kHz pure tones of different durations and intensities. Acta Otolaryngol (Stockh) (in press)

Received July 28, 1980/Accepted February 6, 1981