

Stria Vascularis in Acoustic Trauma

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Summary. Mechanical energy of noise destroys not only the organ of Corti, Reissner's membrane, and the basilar membrane but also the lateral wall of cochlear duct. The damage is characterized by ruptures running parallel to the attachment of the basilar membrane in the prominentia spiralis or sulcus spiralis externus. The other signs of injury after acoustic overstimulation are blisterlike detachments of the stria vascularis from the spiral ligament. The mechanism of injury is interpreted as the result of interaction of given mechanical properties of exposed tissues and of the kinetic energy of sound in narrow segments of cochlear duct.

Key words: Acoustic trauma – Stria vascularis – Mechanism of noise injury

It is generally accepted that overstimulation by noise affects the hearing organ in two different ways. The long-term overstimulation causes an exhaustion of metabolic reserves of sensory cells and leads to irreversible changes and finally to the necrosis of sensory elements. Noise of extreme intensity, however, can mechanically destroy the tissues of the organ of Corti by the acoustic energy of sound reaching the ear.

The acoustic trauma is manifested in several steps: (1) Ruptures in the reticular lamina; (2) destruction of the organ of Corti and mesothelial cells forming the tympanic cover layer; and finally (3) ruptures in Reissner's and/or the basilar membrane [8]. The highest degree of traumatization (i.e., ruptures in Reissner's and/or the basilar membrane) is also usually accompanied by mechanical injury of the lateral wall of the cochlear duct.

Morphological changes in the lateral wall after noise-exposure were studied, e.g., by Ward and Duvall [10] and by Duval et al. [1]. They describe structural changes in all three types of stria cells. Many authors have found a relation between the stria damage and changes in cochlear microvasculature [2, 7].

Spoendlin [6] noticed stria alterations when Reissner's membrane was ruptured. We found heavy mechanical injury to the lateral wall in cases when Reissner's and/or the basilar membrane were mechanically damaged.

Material and Methods

Guinea pigs weighing 300–320 g were exposed for 3, 10, 20, and 60 min to one-third octave band of noise centered at 1 kHz of different intensities (130–145 dB SPL). The animals were killed immediately (i.e., within 5 min) and 1, 2, and 5 days after the exposure. On the whole 347 ears were used.

After decapitation, the temporal bones were removed, bullae opened, and the inner ears fixed by slow perfusion with 10% formaldehyde solution and submerged for a few days in the fixative. After a thorough rinse with water and before removing the bony cochlear wall, the membranous labyrinths were stained briefly in toto by perfusion with Ehrlich hematoxylin and then perfused again with water. With this method of staining, uninjured tissue is stained only superficially, whereas mechanically injured tissue is conspicuously stained because the stain penetrates deeply through ruptures into the injured tissue. Mechanically damaged deeply colored tissue is clearly visible during the microdissection of the membranous labyrinth and the difference in staining facilitates the differentiation of sound-induced lesions from preparation artifacts.

After the examination under the stereomicroscope, segments of the lateral wall, i.e., spiral ligament with stria vascularis, were peeled off from the bony cochlear wall and separated from the cochlear partition. Then they were mounted as surface specimens in glycerol for examination under the light microscope. Other parts of the injured lateral walls were dehydrated in alcohol, embedded in paraffin, and sectioned. Ten noise-exposed ears were double-fixed with 2.5% buffered glutaraldehyde and 2% buffered osmium tetroxide and prepared for transmission electron microscopy.

Results and Discussion

Together with ruptures in Reissner's and/or the basilar membrane we found mechanical injury of the lateral wall. After 20-min exposure to 142-dB SPL the injury occurred in 5%–10% of exposed ears, after 60-min exposure to 142-dB SPL the injury occurred in 50%. The occurrence of traumatic changes in the lateral wall of the cochlear duct corresponds to the occurrence of ruptures in the basilar and Reissner's membrane [8].

The pathomorphological finding was quite characteristic. In surface specimens, the damaged epithelium of stria vascularis had the appearance of a blister. The stria epithelium was detached from its base, i.e., from the spiral ligament and the detachment had a round or oval shape. In the center, the detached epithelium was usually torn and, together with sections of blood capillaries, protruded into the scala media (Fig. 1). By staining in toto, the stain penetrated easily through the ruptured epithelium into the tissue and underneath the stria and the extent of injury became quite evident.

In histologic sections, the stria vascularis was obviously detached from the spiral ligament (Fig. 2a). The rupture usually ran between the basal cells of the stria vascularis and cells of the ligament connective tissue. Sometimes, the rupture even ran through the basal cell and fragments of basal cell remained

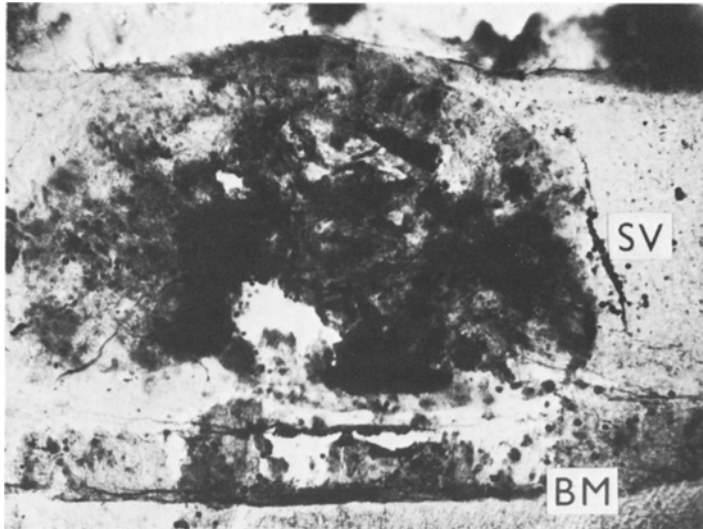


Fig. 1. Lateral wall of cochlear duct in the second turn of guinea pig, exposed to $\frac{1}{3}$ octave band of noise with centre frequency 1 kHz, 144 dB SPL, 60 min. The detached epithelium has the shape of an oval blister in the center of which the epithelium is torn out. *SV*, stria vascularis; *BM* basilar membrane

attached to the spiral ligament (Fig. 2b). In the stereomicroscope, the traumatized lateral wall gives the impression that the stria epithelium is torn from the spiral ligament by a negative pressure and that in the center of damage, in the place of maximum rapid pressure changes, the force is so great that fragments of tissue and blood capillaries are extracted from the epithelium. The center of injury to the lateral wall as well as the ruptures in Reissner's and/or the basilar membrane are in the same narrow segment of cochlear duct.

Except for morphological changes in the stria vascularis we found less striking damage located immediately above the insertion of the basilar membrane. In the epithelium of the sulcus spiralis externus or in the prominentia spiralis there are fine ruptures running parallel to the attachment of the basilar membrane. Regularly, they are in the places where the basilar membrane is ruptured.

These short, longitudinal ruptures can be explained as a result of extreme deviation of the basilar membrane. The radial fibers, which are the firmer components of the basilar membrane, pull a part of spiral ligament in which they are anchored [9].

The effect of noise on the cochlear lateral wall develops in several degrees dependent on the intensity and time of noise exposure. After exposure to 110–120-dB SPL changes in the diameter of blood vessels and in the endothelial cells have been observed by various authors [2, 5]. Lipscomb et al. [5] after 8-h stimulation to broad-band noise of 110-dB SPL found vacuolization of the vascular stria. Also, Lim and Dunn [4] have reported so called empty spaces in

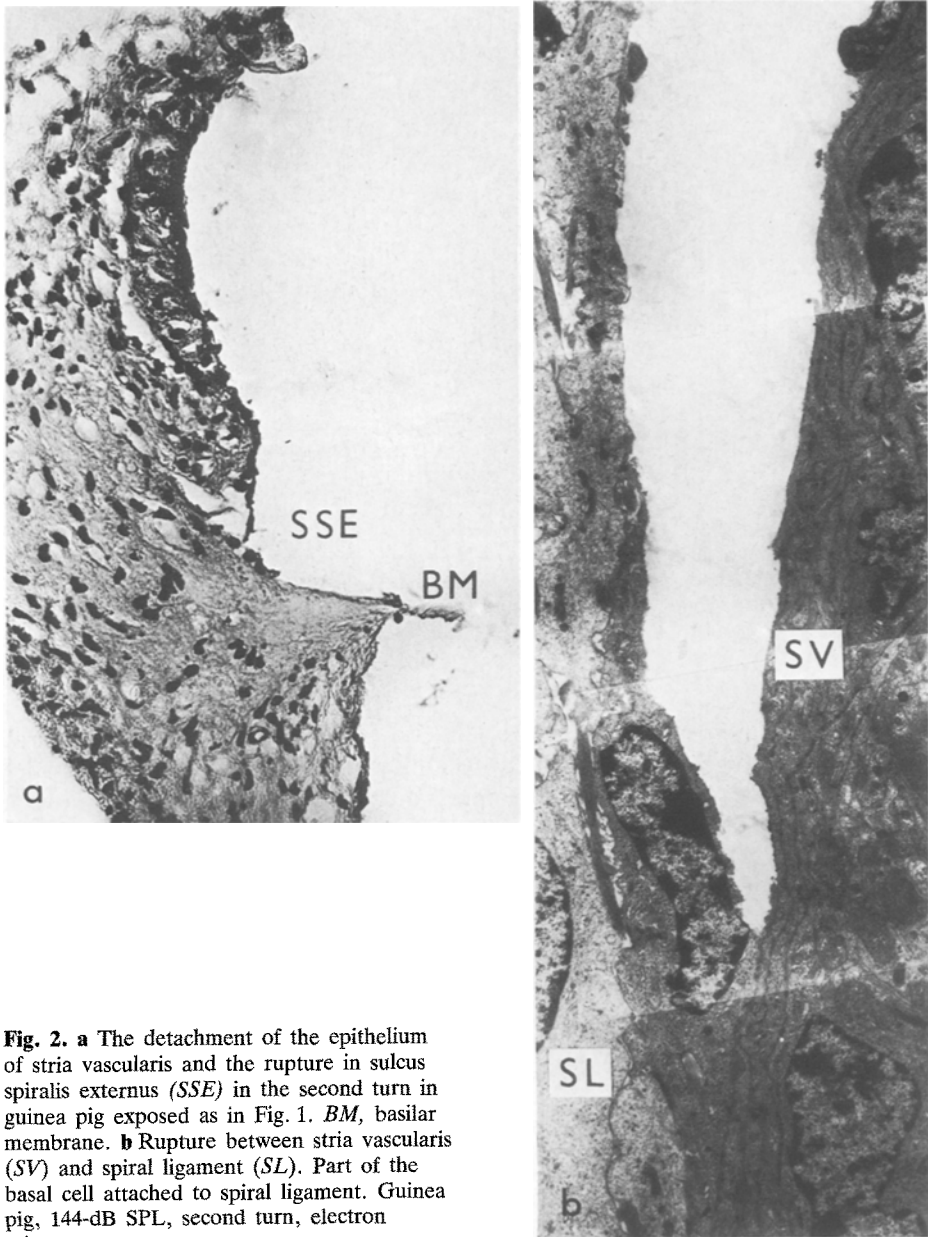


Fig. 2. **a** The detachment of the epithelium of stria vascularis and the rupture in sulcus spiralis externus (*SSE*) in the second turn in guinea pig exposed as in Fig. 1. *BM*, basilar membrane. **b** Rupture between stria vascularis (*SV*) and spiral ligament (*SL*). Part of the basal cell attached to spiral ligament. Guinea pig, 144-dB SPL, second turn, electron microscopy

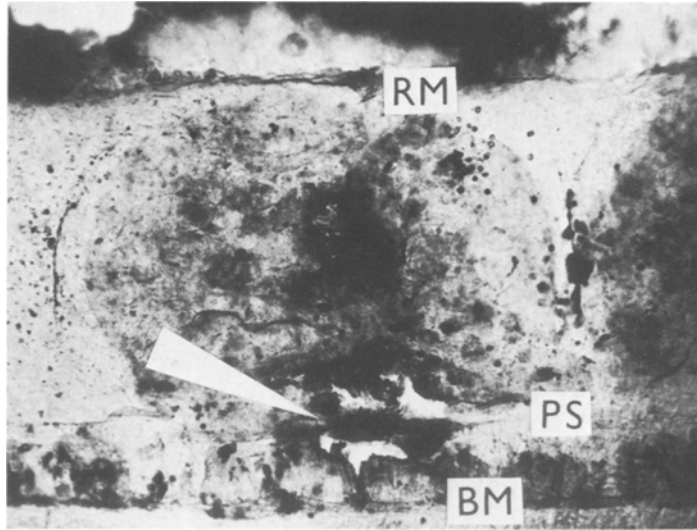


Fig. 3. Lateral wall of cochlear duct in the second turn of guinea pig, 144-dB SPL, 60 min. Ruptures (*arrow*) in prominentia spiralis (*PS*), running parallel to the attachment of the basilar membrane (*BM*). *RM* = Reissner's membrane

the stria vascularis and vacuolar degeneration in the area of the spiral prominence following 5-min exposure to 140-dB SPL. Johnsson and Hawkins [3] after 30-h exposure to 118–120-dB SPL found clearly visible changes in the stria vascularis with pyknotic nuclei and loss of mitochondria in the marginal cells. Duvall et al. [1] have reported degeneration in the spiral prominence 24 h after 15-min exposure to 123 dB. All the findings mentioned above show the increasing degree of damage correlated to the increasing intensity and/or duration of noise exposure.

In our experiment, high intensities were used as a surgical instrument that mechanically affected intravital inner ear tissues. The direct mechanical injury caused by intense sound indicating the way and place of action of kinetic energy of sound helps toward a better understanding of dynamic processes in the cochlear duct during sound stimulation.

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