was impaired by effusion and pressure in the cavity. Recently, it was reported that auditory brainstem response (ABR) measures in childhood were significantly altered with children's experiences of OME. As the critical source of ABR, cochlea mechanics change is related to ABR changes. in this paper, an animal OME model was created by inducing lipopolysaccharide into middle ear of guinea pigs. the vibration of the basilar membrane at apex and basal turn were measured in both control and experimental ears by a laser Doppler vibrometer after 3 days and 14 days of inoculation. the amplitude and latency of ABR in response to click and pure tone sound at selected frequencies were measured in the control and experimental ears. the results show that the displacements of basilar membrane at apex and basal turn in response to 80 dB sound input in the ear canal decreased across frequency range of 200~40k Hz. the amplitude of ABR signal was reduced and the latency increased at 500, 1k, 2k, 4k, 8k, 16k and 32k Hz. the basilar membrane mobility and ABR measured in two experiment groups show different responses induced by OME at early and chronic stages. the cochlea mechanics-ABR coupled analysis suggests that the cochlea function change is related to the ABR change in the animal OME model. This study gives us a better understanding of the relationship between cochlea function and ABR change in the middle ear disease. (Work supported by OCAST HR06-036 and NIH/NIDCD R01DC006632)

507 The Relationship Between Outer Hair Cell Loss and Hearing Loss in Rats Exposed to Styrene

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Outer hair cells (OHCs) are known to contribute to the cochlear amplifier. However, the relationship between cochlear amplification and number of OHC is still unclear. There are examples of normal thresholds with missing OHCs and conversely, elevated thresholds with a normal population of OHCs. Styrene targets OHCs starting from the third row, resulting in apoptotic cell death of the targeted cells. However, the remaining OHCs and IHCs appeared intact. IHC death did not occur until all 3 rows of OHCs were missing. Thus, the styrene-injured cochlea may be a good model for study of the relationship between cochlear amplification and OHC number. Our results showed that loss of OHCs up to 1/3 caused only a slight permanent threshold shift (PTS). in some cases, 1/3 of OHCs were missing without any observable biological consequences, indicating that normal cochlear amplification does not require a complete set of OHCs and 2/3 of normally functioning OHCs are sufficient to maintain normal cochlear sensitivity. When OHC loss increased from 1/3 to 2/3, PTS increased gradually to about 40 dB. the hearing loss did not continue to increase with OHC loss in the range from 2/3 to 100%. the data indicate that the gain of the cochlear amplifier is not eall or none f. in a certain range (1/3 to 2/3 OHC loss), the gain is OHC number dependent.

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508 Modulation of the Endothelial Barrier Permeability

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The permeability properties of microvascular barriers in the ear lie between those of the tight blood brain barrier and the more leaky endothelial barriers in peripheral tissues including muscle and mesentery. This review is based mainly on investigations in individually perfused microvessels from mammalian mesentery. the approach enables measurement of microvessel permeability under conditions where ultrastructure of the vessel wall, signaling pathways in endothelial cells, and organization of the cytoskeleton and cell adhesion complexes are directly measured. the aim is to stimulate ideas for new approaches to understand regulation of the microvascular barriers in the ear. the permeability to macromolecules is determined by a quasi-ordered surface glycocalyx which restricts access of plasma proteins to the endothelial cell surface, the inter-endothelial cleft, and entrance to caveolae. Loss of ordering in the glycocalyx may be one of the earliest events in endothelial barrier dysfunction, increasing macromolecule permeability and adhesion of inflammatory cells to the endothelial cell surface. the cleft pathway between adjacent endothelial cells regulates water and small solute permeability through selective pores or channels within tight junction strands and infrequent breaks in these strands. the integrity of this pathway is determined by a balance between cell-cell adhesion and resting tension within the endothelial cell. Acute increases in permeability result from decreased adhesion without additional tension development. in contrast, endothelial cells in microvessels exposed to sustained inflammatory states express a phenotype with upregulated contractile mechanisms. Some cultured endothelial cell monolayers express a similar contractile phenotype. Microvascular beds of the ear exposed to repeated low level injury (transient hypoxia or loud noise) may develop a similar inflammatory phenotype.

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509 Cochlear Blood Flow and Loud Sound Damage to the Lateral Wall: a Tribute to Merle Lawrence

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Lateral wall blood flow of the cochlea has a critical role in maintaining cochlear fluid homeostasis and oxygenation. the natural functions of endothelial cells, pericytes and smooth muscle cells are to form the blood/lymph barrier and adjust blood flow. Cochlear microcirculation can be impaired through exposure to intense sound; such impairments include increase in vascular permeability, reduction in circulation (ischemia), aggregation of leukocytes and injury to endothelial cells. the high-energy use of the cochlea requires adequate blood circulation but also enhances the vulnerability of lateral wall cells via oxidative stress. Subsequently, lateral wall inflammation is a resulting hallmark of the sound damage, including the expression of vascular adhesion molecules and the

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