goggle tests were performed and above lab tests were reevaluated.
30 patients (7.1%) complained vestibular symptoms. Among them, only 17 patients (4.1%) showed objective laboratory findings of vestibulopathy. No patients dropped into total bilateral vestibulopathy in follow-up caloric test. No patients could be restored caloric response, but symptom was improved after cessation of injection. 12 patients (2.8%) suffered from hearing symptoms such as tinnitus and subjective hearing impairment. However, no patient had decrease in threshold in pure tone audiogram even at high frequency. 6 of these patients (1.4%) suffered from cochlear symptoms along with vestibular dysfunction. Vestibular symptoms were occurred earlier (within 38 days) than cochlear symptoms (137 days) ($p<0.05$). Nobody suffer from cochlear symptoms after completion of injection.
In summary, our 10 year experience of SM injection for tuberculosis show 4.1% incidence of vestibular toxicity with 38days, and no cochlear toxicity. Early detection of ototoxicity could minimize the risk, preserve the function, and use enough therapeutic doses in 95.9% of patient.

597 T-817MA Attenuates Inner Ear Barotrauma in the Guinea Pig

Hitoshi Maekawa[1], Takeshi Matsunobu[1], Masaya Nakagawa[2], Noboru Iwakami[2], Akihiro Shiotani[1]
[1]Department of Otorhinolaryngology, National Defense Medical College, [2]Research Laboratories, Toyama Chemical Co., Ltd.
Objective: Inner ear barotrauma (IEB) that is caused by acute pressure changes can often lead to permanent severe sensorineural hearing loss (SNHL). However, the mechanism that causes IEB is still unknown and its treatment is not established. 1-{3-[2-(1-benzothiophen-5-yl)ethoxy]propyl}-azetidin-3-ol maleate (T-817MA) is a newly synthesized agent developed for treatment of neurodegenerative disease such as Alzheimer's disease. T-817MA not only has neurotrophic effects but also reduce oxidative stress. In the present study, we assessed the possibility of new drug treatment using T-817MA for IEB.
Methods: Healthy female guinea pigs with a normal Preyer's reflex were used in this study. Animals were randomly assigned to one of three groups: (1) group 1 received T-817MA-enhanced water with 0.7mg/ml (2) group 2 received T-817MA-enhanced water with 0.2mg/ml (3) group 3 received normal water (control group). Treatment was begun10 days prior to the intense pressure loading, which induced acute SNHL, and continued until day 21. To assess the efficacy of T-817MA, auditory brainstem responses (ABR) were measured on day 1 (10days pre-exposure), just after the pressure loading, and then at 3 day, 1, 2, and 5 weeks after the pressure loading. ABR threshold shifts were compared between the three groups at each time point.
Results: T-817MA significantly attenuated hearing loss at 1 and 2 weeks after the intense pressure loading ($p < 0.05$, Mann-Whitney's U test).
Conclusions: Treatment for IEB using T-817MA was proved to be effective. These findings indicate that T-817MA might be a promising therapeutic drug in IEB induced SNHL.

598 JNK Activation and Up-Regulation of Thioredoxin Reductase (TrxR) in the Rat Cochlea Following Styrene Exposure

Manna Li[1], Guang-Di Chen[1], Donald Henderson[1]
[1]SUNY at Buffalo
Styrene, a widely used industrial chemical, is ototoxic. It causes death of auditory hair cells after about 7 days of exposure (800 mg/kg/day) through apoptosis (Chen et al., 2007). This study was designed to determine styrene-induced alterations in the cochlea underlying the apoptotic cell death. Thioredoxin(Trx)/thioredoxin reductase(TrxR) system plays an important role in maintaining cellular redox balance and inhibiting JNK pathway, which leads to apoptotic cell death. Long Evans rats were exposed to styrene by gavage at a dose of 800 mg/kg in oil for 4 days or as controls by oil gavage alone. Cochlear tissues were sampled 1 hour after the last exposure. TrxR and p-JNK were determined by Western blot analysis. The results showed a remarkable increase of p-JNK in the styrene exposed group compared to the oil control group. However, TrxR level in the styrene exposed level was also up-regulated. The data indicated that styrene did not cause damage to the Trx/TrxR redox system. We speculate that styrene exposure results in over-production of reactive oxygen species (ROS), which activates stress signaling pathways leading to apoptotic cell death. The over-produced ROS can also oxidize Trx and the overloaded oxidized Trx may stimulate synthesis of TrxR.
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599 Changes in the Structure and Function of the Inner Ear Caused by Burow's Solution

Mitsuya Suzuki[1], Akinori Kashio[1], Takashi Sakamoto[1], Tatsuya Yamasoba[1]
[1]University of Tokyo
We examined the changes in the hair cells of the inner ear following intratympanic injection of Burow's solution. Under general anesthesia, physiological saline was placed with a dropper upon the left round window membrane (RWM) of albino guinea pigs through a small hole made in the tympanic bulla. For experimental animals, Burow's solution (pH 3.7) was placed on the RWM for 30 min (30-min group), 1 h (1-h group) or 2 hs (2-h group). Five days later, animals were anesthetized and sacrificed by decapitation. ABRs at 4, 8 and 20 kHz were recorded immediately before surgery and decapitation. The left bony labyrinth was removed and then fixed with 4% paraformaldehyde at 4ºC. The cochlea and utricle were dissected were stained with rhodamine-phalloidin, and examined under a fluorescence microscope. The left RWM of additional animals in the 2-h group were embedded in Epoxy resin. On the operation day, all animals temporarily had a head tilt to the side of the surgery. In 30-min groups, no significant difference was observed between the postoperative ABRs and base-line thresholds. The post operative ABR thresholds at 20 kHz in the 1-h group and at 8 and 20 kHz in the 2-h group were significantly increased compared to base-line thresholds. Surface preparation of the organ of Corti showed no hair cell loss

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Peter A. Santi, PhD
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Association for Research in Otolaryngology
19 Mantua Road, Mt. Royal, NJ 08061 USA