Beitrag wird präsentiert am 11.03.2011 um 08.30 Uhr im Rahmen der PV02.

Permanent cochlear nerve degeneration after "temporary" noise-induced hearing loss: evidence from two animal models.

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After acoustic injury, sensory cell loss can occur within hours, however loss of spiral ganglion neurons is not visible for weeks. This difference in degenerative time-course has suggested that hair cell loss is the “primary” event, and that neuronal loss occurs “secondarily”, perhaps due to lack of neurotrophins normally released by hair cells. In mice and guinea pigs, we show that noise exposures adjusted to produce a large, but reversible, elevation of cochlear thresholds (as measured by ABRs and DPOAEs) lead to rapid synaptic degeneration, even with no hair cells loss and full DPOAE recovery. By immunostaining for pre-synaptic ribbons and post-synaptic terminals, we demonstrate a 50% loss of synapses within 24 hrs post-exposure. Spiral ganglion cell loss approaches 50% in noise-exposed animals, but only after months to years. This primary neural degeneration, which is reflected in ABR amplitude reduction but not in ABR threshold elevations, may contribute to hearing difficulties in noisy environments, though it does not affect thresholds in quiet. If present results extrapolate to humans, the assumption that full threshold recovery indicates full cochlear recovery, on which noise exposure guidelines are based, is seriously flawed, and acoustic overexposure is even more dangerous than currently appreciated.