Rapid Recovery From Acoustic Trauma: Chicken Soup, Potato Knish, or Drug Interaction?

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Objectives: To describe the phenomenology and consider possible mechanisms mediating rapid and unexpected recovery from acoustic trauma after ingestion of a food substance (potato knish).

Study Design: Single subject with repeated test measures.

Setting: Regional Veteran’s Administration Medical Center, tertiary care medical center.

Methods: Pure-tone audiometry and distortion product otoacoustic emissions (DPOAEs) performed at 6 days, 21 days, and 1 year postexposure.

Results: Medical treatment with corticosteroids and a diuretic alone failed to improve auditory function and related symptoms (tinnitus and aural fullness) over a 2-week period. Rapid recovery of auditory function (dramatic improvement in pure tone thresholds; reappearance of DPOAEs) and abatement of related symptoms directly followed physiologic reactions from ingesting a food substance.

Conclusions: Rapid recovery from acoustic trauma was temporally correlated with urodynamic and cardiovascular reactions from ingesting food containing sulfite preservative, a substance to which the individual was allergic. Factors that may have contributed to recovery of function include massive diuresis, increased heart rate, release of biochemical mediators, mediator-induced vasodilatation, and changes in vascular or cell membrane permeability. Establishing relationships that lead to recovery of function from acoustic trauma may facilitate research and aid in the development of new treatment options for this condition.

It is well established that high-level sound exposure can produce temporary and/or permanent threshold shifts (TTS, PTS) in auditory sensitivity.1,2 In the United States, these concerns have impacted various occupational settings by mandating hearing conservation programs when workers are exposed to noise levels exceeding 85 dB(A) for 8 hours.3 In contrast, there are no mandated regulations covering recreational exposures to high levels of airborne sound. Acoustic overexposures resulting from amplified music (use of personal listening devices, attending concerts) from sporting events, through recreational use of firearms (hunting, target, skeet shooting), and so on can produce similar detrimental effects, and these nonoccupational-related events represent areas of additional concern.4 Whereas advancements in the area of noise induced hearing loss have been progressing for decades, mechanisms underlying TTS and PTS are still not completely understood. Incomplete knowledge of underlying anatomic, physiologic, and neurobiochemical effects, including reversible and nonreversible alterations to sensory and/or supporting cell structure and function, hypoxic/ischemic reactions, reperfusion-based generation of reactive oxygen species, excitotoxic effects on cochlear synapses, and individual differences in susceptibility,5,6 represent some of the domains that require further investigation. Moreover, because there is no psychophysical test or physiologic measure that can distinguish TTS from PTS,9 when clinicians are faced with acute acoustic trauma, counseling individuals with respect to the prospects of recovery is an uncertain proposition.
trauma is presented because the magnitude of hearing loss and presumed cochlear site of lesion did not offer a favorable prognosis for recovery. This report highlights the unique circumstances and offers insight into factors that may have importance to successful recovery from acoustic trauma including abatement of related symptoms.

CASE HISTORY, MEDICAL TREATMENT, AND RESULTS

A 48-year-old professional woman (physician/ophthalmologist) with no prior history of hearing loss experienced auditory and vestibular symptoms after attending a rock-music concert and being exposed to intense music for a period of several hours. When this event occurred, the individual was attending a national medical conference being held in a southern city in the United States. According to the patient, her left ear was in close proximity (within 3 feet) to a large amplified speaker system. The morning after the event, she awoke with decreased hearing sensitivity, tinnitus, aural fullness (all localized to her left ear), nausea, vomiting, and vertigo. The nausea and vomiting subsided within 1 day. When the auditory and remaining vestibular symptoms persisted for 6 more days, an otolaryngologist was consulted.

Otolaryngological Examination

At the time of initial medical examination (6 days postexposure), hearing loss, tinnitus, aural fullness, and vertigo were described. By history, auditory and vestibular symptoms were attributed to intense noise exposure without use of ear protection. Other contributing factors including traumatic events or excessive exertion unrelated to the event were denied. By otoscopic examination, tympanic membranes were normal in appearance, and standard tuning fork testing was consistent with sensorineural hearing loss in the left ear. Fistula testing was negative, and there was no detectable nystagmus or facial nerve weakness. Audiometric studies showed high-frequency hearing loss in the left ear (no air-bone gaps), with reduced amplitude or absent distortion product otoacoustic emissions in the mid- to high-frequency range; normal hearing sensitivity and intact distortion product otoacoustic emissions below 6.0 kHz were observed in the right ear (Figs 1 and 2). The remaining head, neck, and neurologic examinations were normal. Based on the degree of hearing loss and the communicative needs of the individual, medical treatment was instituted.

Medical Treatment

Treatment included a course of corticosteroids (prednisone, 60 mg/d) to be tapered after a 3-week period and a trial dose of a diuretic (Lasix, Aventis Pharmaceuticals, Bridgewater, NJ), which was taken daily and discontinued after 3 days. Vertigo resolved within the first 24 hours after initiating medical treatment; hearing loss and other auditory-related symp-
toms localized to the left ear (aural fullness and tinnitus) persisted unabated. On the 15th day after medical treatment (21 days postexposure), the patient ingested a potato knish, which contained sulfite preservatives, a food substance to which she was allergic. Within 10 to 15 minutes after food ingestion, she developed physiologic alterations in body homeostasis, which were characterized by supraventricular tachycardia (measured heart rate, >270 beats per minute) and diuresis of several liters. During this episode, which lasted approximately 45 minutes, she experienced a loud “rushing noise” in the left ear. After heart rate returned to normal and diuresis subsided, marked subjective improvement in hearing sensitivity, resolution of tinnitus, and aural fullness were immediately realized. Audiologic testing confirmed the return to normal sensitivity (below 6.0 kHz, left ear), including the reappearance and relative normalization of DPOAEs (Figs 1 and 2). Frequency-specific threshold shifts from the affected (left) ear, calculated as the difference in decibels (dB) between pure tone audiometric thresholds obtained at 6 and 21 days postexposure, approximated 55 dB at 3.0 kHz, 55 dB at 4.0 kHz, 70 dB at 6.0 kHz, and 25 dB at 8.0 kHz. Threshold shifts for the right ear, determined over the same frequency range, averaged 5 dB. Retesting at ~1 year postexposure showed relative stabilization of pure tone hearing sensitivity with preservation of DPOAEs.

**DISCUSSION**

The unilateral loss of hearing in the left ear, absence of DPOAEs at selected frequencies, sensation of aural fullness, and perception of continuous tinnitus were all attributed to short-term high-level sound exposure from attending a rock music concert. Based on the magnitude of threshold shifts measured at 6 days postexposure, concomitant vestibular symptoms, and absence of damage to middle ear structures, we estimate that steady state sound pressure levels (SPLs) exceeded 110 to 120 dB, but impulsive sound levels were less than 160 dB. Although characteristics of the auditory impairment suggested a primary cochlear site of lesion, this information did not distinguish TTS from PTS. However, available research suggests that when threshold shifts exceed 40 dB at 6 days postexposure and when hearing loss has a relatively rapid onset, PTS is a much more probable event. Indeed, it is well established that noise-induced TTSs overwhelmingly recover to baseline levels within 12 to 24 hours post-exposure; our supposition that threshold shifts ranging from 25 to 70 dB (average 51 dB) at 6 days postexposure were permanent in nature is consistent with literature in this area. Thus, after initial medical examination and documentation of significant hearing loss by audiological tests, recovery of function was deemed unlikely.

Whereas medical treatment was directed toward reducing postinjury inflammatory reactions and/or ischemia, no subjective changes in auditory-related symptoms were realized within the initial 2-week period over which they were used. Neither the absence of high-level sound exposure over a 3-week period (i.e., so-called rest and chicken soup scenario), nor the pharmacological treatments alone, was
successful in reversing auditory-related symptoms in any positive way. Rapid and dramatic improvements in auditory sensitivity, including resolution of tinnitus and aural fullness, occurred concurrently with the food-induced physiologic reactions. Therefore, of several potential hypotheses that could explain our results, gradual recovery in the absence of excessive noise exposure cannot be considered as an effective explanation. Two remaining but related hypotheses are offered below.

Based on the patient’s response to the sulfites, food-induced anaphylactoid reaction is a viable option. In this scenario, supraventricular tachycardia and massive diuresis could be reflective of immunoglobulin E–mediated events; alternatively, this reaction could also have resulted from a hypersensitivity effect to a corticosteroid-sulfite interaction. Both phenomena fall under the rubric of anaphylaxis, which can present as a spectrum of reactions ranging from mild to severe. Certain foods, such as potatoes and/or food additives such as sulfites, can be powerful stimuli capable of triggering anaphylactic reactions in those individuals that have immunologic sensitivities to these compounds. In food-related anaphylaxis, activation of mast cells, basophils, monocytes, and macrophages and release of potent biochemical mediators can trigger a cascade of reactions in cardiovascular, respiratory, gastrointestinal, and/or cutaneous body systems. Involvement of particular biochemical mediators, such as nitric oxide (NO) may be significant. Available information suggests that NO and associated soluble guanylate cyclase and cyclic guanosine monophosphate-dependent protein kinase-I signaling pathways can target cochlear blood vessels of the lateral wall, spiral ligament, stria vascularis, and basilar membrane; supporting structures of the organ of Corti (Hensen’s cells, Deiters’ cells, and pillar cells); inner and outer hair cells; and afferent nerve endings near inner hair cells and efferent nerve endings near outer hair cells. Indeed, microvascular impairment of the inner ear secondary to high-level noise exposure is a potential mechanism that can lead to peripheral auditory dysfunction. Smooth muscle relaxation of the vascular network from NO-mediated effects can result in increased cochlear blood flow and improvement of inner ear function.

Trauma-induced, ionic-based osmotic change (cellular swelling) is another potential mechanism by which functional and structural alterations in sensory or supporting cell morphology can adversely affect cochlear function. With respect to outer hair cells, swelling can alter the internal turgor of the cell and adversely affect electromotility. The absence of DPOAEs would be consistent with such an effect. Similarly, cellular swelling of the supporting cells can also lead to permanent hearing loss and other complications if left untreated. Moreover, high-level sound exposures can also induce excessive release of glutamate from inner hair cells, which may produce permeability changes, swelling of postsynaptic dendritic membranes, and impairment of neural transmission. Thus, if swelling was present in sensory cells, supporting cells, and/or primary auditory neurons, then massive diuresis may have contributed to reversing this condition.

Because reversibility of function was in large part complete, permanent damage to cochlear hair cells, supporting cells, or primary auditory neurons was avoided. Therefore, reversible changes in supporting cell structure and function is another potential factor, which may have contributed to our results. Based on in vitro laser scanning confocal microscopy, Flock et al found that intense sound exposure produced reversible changes in Hensen and Deiter cell morphology and that these alterations correlated with changes in cochlear microphonic measurements in their preparation. Additionally, using the survival-fixation technique, Nordmann et al and Srokowski et al found that buckling of supporting pillar cells with decoupling and subsequent recoupling of stereocilia to the tectorial membrane was the only abnormality, which correlated with threshold shifts that were temporary in nature. In these experiments, acoustically driven buckling of supporting cells was interpreted as a protective mechanism that the cell uses to guard against permanent injury. Decoupling of stereocilia from the tectorial membrane has also been postulated as a mechanism for producing threshold elevations, tinnitus, and other related symptoms. Because tinnitus com-
pletely resolved after hearing sensitivity improved, reversible changes in supporting cell function would be consistent with our results.

In summary, several possible scenarios were presented that may account for the rapid reversible changes in auditory functions we observed. Whereas the series of events described was most likely attributed to sulfite-induced anaphylactoid reaction, we cannot with certainty delineate between a possible immunoglobulin E reaction or a sulfite-corticosteroid-mediated sensitivity effect. At the time of the episode, no medical assistance or laboratory workup was obtained. However, we are unaware of any published corticosteriod-sulfite interactions producing similar auditory system changes noted herein. Given the patients’ allergic history and reaction to sulfites, the former explanation seems more likely. Therefore, we postulate that 2 main factors were involved in the recovery process: massive diuresis and effects of biochemical mediators, either working alone or in combination.

CONCLUSION

Rapid recovery of inner ear function after acute acoustic trauma was concurrent with food-induced anaphylactoid reaction. Based on the close temporal relationship between food ingestion, reactive physiologic events, and immediate recovery of auditory function by subjective account, the dramatic improvements were not considered fortuitous in nature or as a coincidental event. The resolution of symptoms and time course of recovery was rapid and in large part complete. There was no evidence for gradual recovery of function, which might have occurred if the absence of high-level sound exposure alone over the 3-week period was the main contributing factor mediating these effects. Furthermore, the direct accounts of a highly trained physician/scientist not only enhances the validity of these observations but served as the motivating reason to report this case. Therefore, factors that may have contributed to recovery of function include massive diuresis, release of biochemical mediators, mediator-induced vasodilatation, vascular and/or cell permeability changes, and increased heart rate. Whereas our test results were consistent with a primary cochlear site of lesion, these findings could not delineate TTS from PTS. Therefore, from a clinical perspective, predicting recovery of function was compromised and uncertain. Presently, the main tools available for avoiding permanent auditory damage from high-level sound exposures are prevention and education. Noninvasive physiologic markers targeting those individuals at higher risk for potential injury from high-level sound exposure may facilitate future preventive measures.28

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