

Case Report

Tinnitus in an active duty navy diver: A review of inner ear barotrauma, tinnitus, and its treatment.

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Duplessis C, Hoffer M. Tinnitus in an active duty navy diver: A review of inner ear barotrauma, tinnitus, and its treatment. *Undersea Hyperb Med* 2006; 33(4):223-230. This case elucidates subtle cues that must be appreciated by the examiner in diving related injuries, who may not have experience with barotrauma-mediated pathology. Inner-ear barotrauma (IEBT) does not mandate ostensible hearing loss or vertigo; tinnitus may be the sole manifestation. Symptoms may present hours or even days post-dive. A common misconception exists that there are no efficacious treatment options for IEBT short of surgery for an overt perilymphatic fistula (1). Treatment options are available including acute high dose steroid administration, as prescribed for acute noise-induced or idiopathic hearing loss, optimally administered within three weeks of the acute insult. Tinnitus does not necessarily constitute a chronic untreatable symptom, which the patient must learn “to live with” (6,7).

BACKGROUND

Tinnitus, derived from the Latin word *tinnire* (“to ring”), is defined by the unwanted auditory perception of internal origin (1,2). Tinnitus affects up to 30% of the adult population, with incapacitating symptoms reported in 6% of those afflicted, and significant depression experienced in 40% (2,3). Given similar psychoacoustic descriptions of tinnitus, individuals differ radically in the perceived intrusiveness of symptoms. A sobering statistic is that 94% of patients seeking medical care are not offered treatment for their affliction, exacerbating symptoms (3). Reassurance and hope are as appropriate and the cognitive appraisal of tinnitus is an integral component of the disease (4). There are efficacious

treatments for tinnitus, with more novel treatments on the horizon. However, these are not effectively promulgated in the literature.

This case elucidates the subtle cues that must be appreciated by the examiner in diving related injuries, who may not have experience with barotrauma-mediated pathology. Inner-ear barotrauma (IEBT) does not mandate ostensible hearing loss or vertigo; tinnitus may be the sole manifestation. Symptoms may present hours or even days post-dive. A common misconception exists that there are no efficacious treatment options for IEBT short of surgery for an overt perilymphatic fistula. However, treatment options are available including acute high dose steroid administration, as prescribed for acute noise-induced or idiopathic hearing loss, optimally administered within three weeks of

the acute insult (6,7).

CASE

A 20-year old white male, AD, US Navy diver without significant prior medical history, presented several days after the onset of tinnitus sustained after performing a no-decompression dive on air (20 feet for 40 minutes). The dive was uneventful, except for difficulty clearing his ears, requiring a forceful Valsalva. Ringing commenced several hours post-dive, described as low level (rated as 1 on a scale of 10), high pitched, unilateral (right ear), and constant since inception. He denied other symptoms during or post-dive; notably, vertigo, hearing loss, nausea, vomiting, headache, visual changes, ataxia, or dizziness. He endorsed having a “cold” and recent attendance at a rock concert.

On examination, MMSE was without deficit. Exam findings were significant for mobile tympanic membranes bilaterally, without evidence of middle ear effusions, TM perforation, or hemotympanum. Nasal and oral mucosa revealed mild congestion consistent with the reported upper respiratory infection. There was no discernible evidence of nystagmus, and the rest of the neurological exam was normal.

The patient was educated on noise-induced hearing loss, instructed to avoid loud noise exposures, and to return for continued or worsening symptoms. The patient was re-evaluated six weeks later, prior to engaging in further diving, and he reported continued tinnitus. Subsequent audiological testing revealed an isolated new right-sided 60-dB threshold shift at eight kilohertz with normal speech reception testing, word recognition, acoustic reflex testing, and tympanometry (type A tympanograms). Distortion Product Otoacoustic Emission Testing (DPOAE) revealed emissions consistent with the audiogram and normal cochlear function. ENT

consultation rendered the diagnosis of IEBT. The patient was started on a course of high dose steroids (60mg per day for 10 days).

Differential Diagnosis

Tinnitus is caused by ischemic (stroke), congenital, traumatic (otosclerosis, noise, barotrauma), inflammatory (autoimmune, mastoiditis), surgical, medicinal (chemotherapeutics), malignant (acoustic neuroma) (1), neurologic, metabolic (diabetes, hypothyroidism, vitamin B₁₂ deficiency), idiopathic (presbycusis), infectious (labyrinthitis), and psychologic disorders. Diving related etiologies include inner ear barotraumas (IEBT), inner ear decompression sickness (DCS), and arterial gas embolism (AGE). The dive profile, a no decompression dive, maximum depth of twenty feet, without gas switching, is inconsistent with a diagnosis of inner ear DCS. AGE is ruled out given the normal neurological exam and MMSE. Isolated tinnitus post-dive, without associated symptoms, decreased the suspicion for IEBT. The presentation is most consistent with viral labyrinthitis, and/or noise-induced tinnitus.

DISCUSSION

Classification

Tinnitus may be broadly classified as either subjective or objective. Subjective tinnitus is a phantom auditory perception without physical auditory or vibratory activity in the cochlea, perceived only by the patient (3). Although most commonly associated with hearing loss, it may occur with normal hearing. In objective tinnitus the examiner can appreciate the objectionable noise experienced by the patient, by placing a stethoscope over the head and neck structures near the patient's ear (1). Additional classification measures include magnitude, quality, timing (progressive, fluctuating, intermittent, or constant), and pitch.

Objective tinnitus is caused by sound emanating from the body either reaching the cochlea by bone conduction, or conducted to the middle ear cavity without documented dysfunction of the auditory system. Sources include turbulent blood flow or muscle contractions (2).

Tinnitus quality may be tonal (ringing or whistling), or noise-like (roaring or hissing) (5). Tinnitus severity may be mild (audible only when in a quiet place) or severe (disturbing symptoms degrading the quality of life) (6). Pitch assessment may help differentiate etiologies. Low frequency tinnitus (< 250 Hz) may reflect Menière's disease. Higher pitch tinnitus is associated with noise induced hearing loss (7).

Pathophysiology

Tinnitus-related neuronal activity is not governed by mechanically-derived acoustics (8), but the brain's perception and interpretation of novel or abnormal neural activity (1). The altered perception inevitably stems from some insult to the cochlear apparatus, the cochlea's outer hair cells (OHC), and inner hair cells (IHC). Injury may lead to repetitive discharge from the hair cells, spontaneous activity in auditory nerve fibers, hyperactivity of cochlear nuclei, or a reduction in normal suppressive activity of the central auditory cortex on peripheral auditory nerve activity (1).

A leading theory posits discordant dysfunction of the OHC and IHC, postulating that tinnitus related neuronal activity is generated in the dorsal cochlear nucleus from unbalanced activity transmitted by type I and type II auditory nerve fibers (derived from the IHC and OHC respectively). This attractive theory reconciles various subcategories of tinnitus; tinnitus experienced in the absence of hearing impairment, and hearing impairment unaccompanied by tinnitus (8).

The role of Central Nervous System (CNS) Plasticity

The abnormal, novel or dearth of neural activity from the ear may induce CNS neural plasticity (4)], including a redirection of information to regions of the CNS that normally do not receive auditory input, including the autonomic and limbic systems. This activation yields many of the deleterious symptoms of tinnitus, including depression, hypertension, anxiety, and insomnia, which correlate better with reported severity, than psycho-acoustical measurement (8).

Normally, the auditory system receives and processes a large volume of acoustic information, transmitting a small fraction for conscious perception. Exposure to familiar and unimportant sound is blocked at the subconscious level, "habituated", without activation of the limbic and autonomic systems. However, in various types of inner ear injury, the abnormal, novel or even dearth of discharge activity may be incorrectly categorized as important, and thus processed for enhancement, amplification, and transmission. More importantly, the processed information may be transmitted erroneously to the limbic and autonomic systems (via neural plasticity), and the higher cortical auditory centers for conscious perception (tinnitus).

Furthermore, when the CNS plastically activates a "conditioned reflex arc" (8) linking the auditory with the limbic and autonomic nervous systems, "it establishes a detrimental, self-perpetuating, negative reinforcement, leading to tinnitus enhancement, and prevention of habituation" (8).

Pathophysiology of Diving-Induced Tinnitus

Diving related inner ear pathology includes DCS, AGE, and IEBT (9). IEBT is commonly ascribed to labyrinthine window ruptures forming a perilymphatic fistula (5). Significantly, symptoms may not develop for

hours or even days after the incident. The mechanism may be either implosive or explosive (10). These injuries may be compounded by gas entry to the inner ear (11).

Perilymphatic fistulas may heal spontaneously with conservative measures that mitigate middle and inner ear over-pressurization. If this fails, it may be amenable to surgical correction, although the timing remains controversial.

What is perhaps not widely appreciated is that IEBT may occur without window ruptures, and without overt otoscopic signs (10), manifest by inner ear hemorrhage, and intralabyrinthine injury, resulting in deficits to discrete tonal frequencies. Cochlear injury may involve degeneration of IHC and OHC (typically more severely damaged) (11), suggesting a role for otoacoustic emissions (OAE) testing in the diagnostic work-up. This mechanism of injury has been associated with a more sudden onset of symptoms not amenable to treatment (10).

The diving profile helps to differentiate inner ear DCS from IEBT. Inner ear DCS manifests itself during the ascent phase of the dive, or shortly afterwards, and is associated with deep diving, breathing gas changes, and other signs of DCS. This differentiation is important, as recompression treatment is indicated for inner ear DCS, but not IEBT, although ongoing research may alter this opinion (11).

MEDICAL EVALUATION OF TINNITUS

History and Physical Examination

Pertinent history includes alcohol and caffeine consumption, smoking, illicit drug abuse, medications, and herbal use. Establish the tinnitus as objective or subjective. Identify its location, onset, timing, intensity, pitch, quality, severity, and associated symptoms. A record of the dive profile should include the

depth and duration of the dive, breathing gas, use of gas switching, decompression schedule, work performed, phase of diving for symptom onset and prior hyperbaric exposures.

A complete head and neck and neurologic examination should be performed including an otoscopic and cranial nerve examination, and Weber, Rinne, Romberg's, nystagmus, gait, Dix-Hallpike, and dysmetria testing.

Diagnostic Studies

Middle ear studies include tympanometry, and stapedial reflex, reflex decay and recruitment testing. Evaluate the cochlear-vestibular apparatus via audiologic testing (pure tone and bone levels, speech reception thresholds, speech discrimination, and uncomfortable loudness levels). For severe cases, consider assessing pitch and loudness matching (matching the frequency of the tinnitus and estimating loudness with pure tones or noise), minimum masking levels, and residual inhibition (achieving resolution of symptoms for a defined period of time after exposure to a masking tone in the pitch and intensity of the tinnitus (1), to identify those who may benefit from tinnitus masking therapy. Additional testing may include auditory brainstem response testing, otoacoustic emissions (OAE) testing, MRI and CT imaging, tone-decay testing, and electronystagmography (2). For unilateral tinnitus of unknown etiology, laboratory analysis may include CBC, lipid profile, glucose level, fluorescent treponema antibodies (FTA), Lyme titer, thyroid and autoimmune panel.

Treatment

Development of efficacious treatment has been handicapped by several issues, including the subjective nature of the disorder and its multifaceted etiology impeding study and targeted therapy (6). Fortunately, the majority of patients with tinnitus require simple reassurance and the possible recommendation

of white noise masking; very few patients have symptoms severe enough to require extensive intervention. Treatment modalities should incorporate education, counseling, reassurance and support. Conservative treatment includes lifestyle and dietary modification. Avoid loud noise, and potentially ototoxic medications. Dietary modifications include abstinence from nicotine, caffeine and alcohol.

Other treatments include transcutaneous electrical nerve stimulation (TENS), sound therapy (tinnitus maskers), biofeedback, cognitive-behavioral therapy, tinnitus retraining therapy (TRT), medications (oral and transtympanic), alternative therapies, and surgery. A brief synopsis of each follows:

TENS Therapy

TENS utilizing continuous or modulated alternating current applied to various points on the external pinna and tragus has proven to be a safe and effective modality in certain populations, with efficacy ranging from 33-82% (12). The physiological mechanism remains elusive, but has been theorized to involve increased cochlear blood flow, direct cochlear stimulation, and an adaptation of the pain gate theory. Electric current may restore the deprivation of neural activity in the auditory nerve, which engenders the phantom sensation of tinnitus(7).

Sound Therapy

Sound therapy, via tinnitus maskers, has been efficacious in almost 90% of patients (13). Devices can be designed on an individual basis, providing frequency selective masking. Future masking devices may utilize high frequency bone conduction, circumventing the need for ear occlusion (13). Evidence suggests that some patient's achieve an accumulation of residual inhibition with chronic usage (5). There are also bedside maskers, which may be utilized for symptoms interfering with sleep (13).

In patients with hearing loss at frequencies < 4 kHz, a hearing aid may improve both hearing and tinnitus by raising the background sound level (3).

Biofeedback

In biofeedback, surface electrodes are placed on muscles in the upper quadrant, neck and face. Patients are instructed in methods to relax muscle tension identified on a computer output, visualizing real-time success through computer screen monitoring (12).

Tinnitus Retraining Therapy (TRT)

Although repair of a damaged cochlea is not yet possible, the brain's detection and interpretation of signals can be modified, exploiting its inherent plasticity (8). TRT is a method based on the neurophysiological model of tinnitus, predicated upon the model of neuroplasticity. The method attempts to induce sustained habituation of reactions and perceptions to intrusive tinnitus and external sounds (8). It eliminates neuroplastic connections linking the auditory with the limbic and autonomic nervous systems. Specifically, the brain is trained to filter abnormal signals, preventing them from activating the limbic and autonomic nervous systems. This leads to habituation of tinnitus-evoked reactions, and reductions in signals reaching higher cortical levels involved in perception (habituation of tinnitus perception) (8). TRT has been reported to produce significant and sustained improvement in symptoms in over 80% of patients.

Surgical Options

For the special case associated with traumatic induced perilymphatic fistula formation, surgical correction, performed early is a viable option. Timing remains controversial (10).

Alternative Therapies

Alternative therapies of anecdotal efficacy include early institution of hyperbaric oxygen therapy (HBO₂), and low level laser therapy (14). Both may increase oxygen delivery and ATP synthesis in stressed or injured cochlear tissues (14).

Medications

Medications of anecdotal efficacy include vitamins, herbs, antioxidants, carbogen inhalation (14,15), alprazolam (in patients with insomnia and anxiety)(13), and lidocaine. Recent evidence suggests that anti-oxidants may have some impact on tinnitus (19).

Perhaps not promulgated in the medical literature is that steroids (6) may be a highly efficacious treatment for barotraumatically mediated tinnitus-when administered early. Furthermore, transtympanic medicinal administration is increasing in prevalence, and transtympanic steroidal administration has proven efficacious for the treatment of sudden sensorineural hearing loss (SSNHL), autoimmune inner ear disease (AIED), and Menière's disease (20).

Oral steroids are proven to improve the symptoms of sudden sensorineural hearing loss (SSNHL) (16,17). Standard practice increasingly incorporates the acute administration of high dose oral steroids for a variety of cochlear maladies including acute noise and barotraumatically induced hearing loss.

Transtympanic Therapy

Transtympanic (intratympanic) perfusion therapies are emerging for targeted diffusion of medications across the round window membrane into the inner ear fluids, reducing systemic side effects, and achieving higher localized concentrations (16, 18). Transtympanic medical therapy (using gentamycin) has become standard and accepted therapy for vertigo, hearing loss, and dizziness

associated with Menière's Disease (19,20).

Transtympanic steroidal administration has proven efficacious for the treatment of sudden sensorineural hearing loss (SSNHL), autoimmune inner ear disease (AIED), and Menière's disease (18). Transtympanic steroidal delivery has successfully improved hearing in patients with SSNHL unresponsive to oral steroid administration (17), or in whom oral steroids are contraindicated (18). Transtympanic steroid therapy has also proved effective for primary tinnitus. Sakata's group utilized intratympanic steroids on 1214 patients with cochlear tinnitus and reported a 71% response rate (21). Shulman's group reported a similar success rate of 70% when using transtympanic steroids for tinnitus (22). There are still a number of questions with regards to the use of transtympanic medicines, in general, and steroids in particular for the treatment of inner ear disorders. The best route of administration, best total dose, best dosing frequency, and the best end point of therapy still need to be determined. Nevertheless, transtympanic steroids are now a common treatment for tinnitus (23).

Treatment for Diving-Induced Tinnitus

The diving literature supports initial conservative treatment of suspected IEBT, endorsing caution against CSF/intracranial pressure elevation, which may propagate to the inner ear. ENT referral is suggested for persistent suspicion of perilymphatic fistulas. Not publicized in the literature, diving or otherwise, is the potential role of steroid administration (oral and intratympanic) for acute IEBT, as touted for acute noise induced hearing loss.

The crux of treatment is early intervention. Tinnitus, whether isolated, or associated with other symptoms, temporally related to diving, suggests the possibility of

IEBT. Symptoms may present many hours or days post-dive. A perilymphatic fistula is not a pre-requisite to diagnose IEBT, which we now realize may be occult, and which may be treatable with high dose oral steroids, or intratympanic steroid administration.

Prevention of Diving Related Inner Ear Barotrauma

The best treatment for diving-related barotrauma is prevention. This includes ensuring patency of the Eustachian tubes prior to diving, and early and frequent autoinflations to equilibrate middle ear pressures during diving. The diving candidate should be instructed in the maneuvers, which may be attempted to open the Eustachian tubes including, the Valsalva, the Toynbee, the Frenzel, the Lowry, and the Edmonds techniques. Additional preventive strategies include descending in the vertical head up position, avoiding diving when unable to equilibrate the middle ear (due to upper respiratory tract infections, allergies, irritant exposures, and medications), and avoidance of alcohol, and allergic triggers prior to diving (such as dairy products).

PROGNOSIS

Without treatment, cochlear low frequency hearing may improve for a few weeks, but the remaining high-frequency hearing loss is usually permanent (10). In permanent vestibular injury, central compensation may, after some time, render the patient asymptomatic. Unfortunately, similar mechanisms do not exist for permanent cochlear injury (11). Tinnitus often improves over six to twelve months, presumably as damaged sensory endings are repaired or die (10). A residual high pitch tone accompanied by a residual high frequency hearing loss often remains (11).

CONCLUSION

The patient in this report had some improvement in symptoms after therapy despite its administration some six weeks after the insult. The audiogram improved in the threshold shift at 8k from 60 to 40 dB after treatment, with a commensurate decrease in symptoms, remaining stable on serial testing twelve weeks later. Whether this improvement is related to the steroid treatment, or the natural evolution of tinnitus resolution, is debatable. Earlier recognition of IEBT, with steroid administration, may have garnered a better outcome. Fortunately, this diver did not experience any decrement in pure-tone audiometric thresholds in the frequency ranges of 500-2000 Hz, or speech discrimination scores that may have undermined future diving.

Divers experience significant high-frequency sensorineural hearing loss, exceeding matched controls (10). Divers may be vulnerable to insidious sub-clinical inner-ear damage from exposures to hyperoxia, IEBT, and noise predisposing synergistically to permanent sensorineural hearing loss, which is not apparent on contemporary pure-tone audiometry, and which may be otherwise mitigated or circumvented with timely steroid treatment.

Otoacoustic emission (OAE) testing is a more sensitive measure than pure tone conventional audiometry in assessing inner-ear injury (particularly high frequency injury), from myriad insults including impulse noise exposure from firearms affording more lead time to administer interventions (steroids)(24,25). The author is currently investigating the utility of OAE testing in identifying sub-clinical inner ear injury after repetitive diving exposures.

This case elucidates the subtle cues, which must be appreciated by the examiner in diving related injuries. It is prudent to engage ENT consultation early in the evaluation of

diving related inner-ear injury. IEBT does not mandate ostensible hearing loss or vertigo; tinnitus may be the sole manifestation. Symptoms may present hours or even days post-dive. Treatment options may be available including acute high dose steroid administration, as prescribed for acute noise-induced or idiopathic hearing loss, optimally administered within three weeks of the acute insult. The effectiveness of steroid treatment in diving related injuries has not been studied, and should be subject to future clinical trials. Tinnitus does not inexorably dictate a chronic untreatable symptom, which the patient must learn “to live with” (6,7).

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