

Hyperbaric Oxygen Therapy for Sudden Hearing Loss: An Audiologist's Experiential Perspective

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Abstract: The author, a semi-retired audiologist, offers his perspective on a personal episode of unilateral idiopathic sudden hearing loss consisting of a mild-to-severe high-frequency sensorineural component and a low-frequency conductive overlay. A review of pertinent literature is presented to facilitate an understanding of the possible causes of idiopathic sudden sensorineural hearing loss (ISSNHL), its treatments and their purported effectiveness, and the perceived risk-benefit ratio of hyperbaric oxygen therapy as a conventional treatment. A case-study approach is used to describe chronologically ordered events from the onset of the hearing loss through its resolution. The report is a journalistically styled, first-person account of the episode's onset, diagnosis, medical and rehabilitative interventions, and audiologic outcomes. The author details the sequential improvements in hearing levels in response to the combination of medical and rehabilitative treatments and describes his personal reactions to a series of hyperbaric oxygen sessions. Although hyperbaric oxygen therapy is associated with known risks and continues to warrant scientific scrutiny, it appears to be a viable and potentially effective treatment for ISSNHL. Despite experiencing symptoms of ear barotrauma during the treatments, the author emerged with a renewed appreciation for the advantages of binaural hearing, the acoustic redundancy of speech, and the potential for relatively mild hearing loss and tinnitus to lead to negative psychological impacts. Audiologists, otolaryngologists, and others who come in professional contact with patients who suffer from ISSNHL are encouraged to act aggressively to ensure that all potentially effective measures are employed to minimize its negative consequences.

Key words: hyperbaric oxygen therapy, idiopathic hearing loss, steroids, sudden hearing loss, tinnitus

Abbreviations: ATA = atmospheres absolute; HBOT = hyperbaric oxygen therapy; ISSNHL = idiopathic sudden sensorineural hearing loss; NIDCD = National Institute on Deafness and Other Communication Disorders; UHMS = Undersea Hyperbaric Medical Society

Introduction

My first formal exposure to the topic of sudden sensorineural hearing loss was in one of my master's-level courses in Vanderbilt University's Hearing and Speech Sciences program, where I completed the degree requirements in audiology in 1967. Recently, that early lesson took on new meaning when I experienced a sudden unilateral loss of hearing. I recalled the lecturer in that class long ago describing sudden sensorineural hearing loss as a potentially reversible condition, but only if treated shortly after onset. At that time, the administration of vasodilators was the preferred treatment for sudden hearing loss of unknown origin. Schuknecht and colleagues (1962) had published a paper signaling their belief that viruses are the most common cause of sudden hearing loss, and Jaffe (1967) suggested that the virus particles affect the blood flow through the capillary network of the inner ear to produce a hypercoagulation effect, which could be treated with vasodilators. Many years later, in 2009, Agarwal and Pothier reported on three clinical trials involving 189 participants in which the investigators concluded that vasodilators are unproven as an effective treatment for idiopathic sudden sensorineural hearing loss (ISSNHL).

Before July 2020, my hearing could have been described as relatively intact for a 77-year-old male. As a semi-retired audiologist, I had performed a self-administered test of my pure tone thresholds in a sound-treated room at Michigan State University in September 2019. At that time, air-conduction thresholds were within normal limits (≤ 25 dB HL) at all frequencies between 250-8000 Hz except at 4000 and 8000 Hz in the right ear and at 8000 Hz in the left ear; hearing levels were 30 dB in each of those instances (see Figure 1a). No substantive conductive component was present, as evidenced by the absence of any clinically significant air-bone gap.

In the early morning hours of July 27, 2020, I awoke experiencing several strange sensations. My first impression was one of ear fullness, followed by an awareness of hearing loss and a loud ringing tinnitus in my left ear and a brief pulsatile tinnitus that seemed localized in my right ear. Sitting up in bed, I experienced a mildly nauseating vertigo, which subsided after a few minutes. Following the shock of experiencing a sudden hearing loss and my ruling out of an emergency-room visit because of the COVID-19 pandemic, I knew that I needed to be seen by an otolaryngologist and an audiologist as soon as possible. In a call to the office of a local otolaryngology clinic, which is staffed by several audiologists, I was told that the first available appointment was two days later. I made the appointment but went immediately to a local urgent-care facility, where I was given an ECG (or EKG) as a check for ischemia, or inadequate blood supply to the heart, and overall heart health. The ECG results were normal and, regrettably, I was not administered an audiometric evaluation. The attending physician prescribed an antibiotic, presumably because his otoscopic examination revealed a possible ear infection. Although I was dubious about the diagnosis, I took the antibiotic as a precautionary measure.

Two days later, my visit to the otolaryngologist and audiologist revealed the audiogram in Figure 1b, which led to a diagnosis of idiopathic sudden sensorineural hearing loss (ISSNHL) in the left ear. An unexpected audiological finding was a 25-dB conductive component at the single frequency of 250 Hz, based on a masked bone-conduction threshold that was verified by masking the right ear using an insert earphone. Hearing loss at the other frequencies, including 500 Hz and 3000-8000 Hz, ranged from mild to severe. As shown in Figure 1b, the July 29 audiogram revealed hearing in the left ear to be significantly poorer than in the right, but fortunately, not so poor as to functionally degrade the speech recognition threshold (SRT) and



speech intelligibility. SRTs were 5 dB and 25 dB HL in the right and left ears, respectively, while word recognition scores (in quiet), established at 55 dB and 65 dB HL, were 96% bilaterally. Still, I was very conscious of the loss of left-ear hearing sensitivity in both the low and high frequencies, ear fullness, and high-frequency tinnitus. In the days that followed, I could easily detect deficits in understanding speech from my left side, in my ability to enjoy music, and in localizing high-frequency sounds.

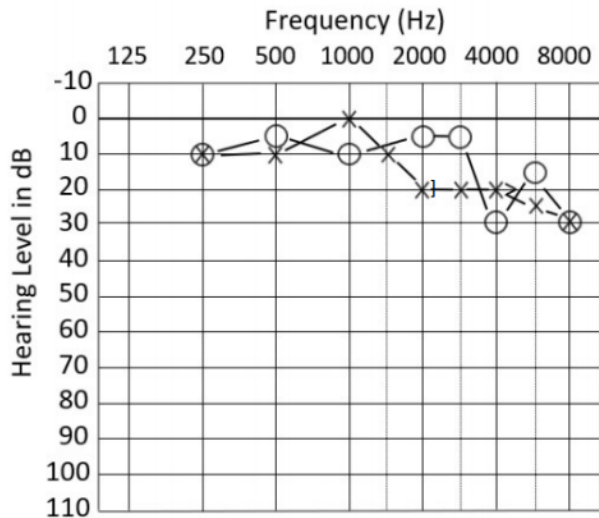


Figure 1a. Baseline pure tone audiometric thresholds obtained on September 25, 2019, prior to onset of sudden hearing loss (Symbols: O = unmasked AC, R; X = unmasked AC, L; > = unmasked BC, L;] = masked BC, L).

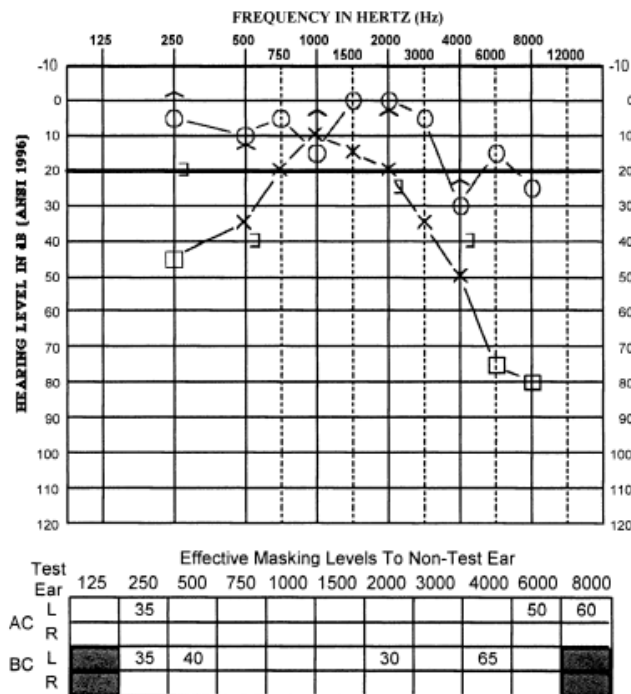


Figure 1b. Pure tone audiometric thresholds obtained on July 29, 2020, two days after onset of sudden hearing loss.

R Ear	L Ear	
O	X	masked AC
Δ	□	masked AC
Λ		unmasked BC
C	J	masked BC

Although a variety of treatments have been suggested for ISSNHL (Crigger & Wilson, 2016), the corticosteroid Prednisone, taken either orally in high doses or via intratympanic injections, is currently the major medical-treatment option (e.g., Kuhn et al., 2011; Anyah et al.,



2017). The otolaryngologist offered me either option, and I chose the oral version, largely to avoid the need to have several intratympanic injections, which would have required multiple visits. I took the tablets on a tapered-reduction schedule for a total of 12 days.

The otolaryngologist also recommended that I consider hyperbaric oxygen therapy (HBOT), as it is now considered a potentially viable treatment for ISSNHL (e.g., Lamm et al., 1998; Racic et al., 2003; Crigger & Wilson, 2016; Saesen et al., 2017). I visited a local wound clinic where HBOT is administered in a room containing two monoplace, pressurized chambers. Based on consultation with the physician in charge, we jointly decided to begin HBOT sessions immediately, as waiting is generally believed to lessen the chances for recovery (Stachler et al., 2012; Lloyd, 2013; Edizer et al., 2015; NIDCD, 2018). After the first treatment and a preliminary literature search of HBOT's potential benefits, I agreed to undergo a subsequent series of HBOT sessions, punctuated by audiologic exams to determine whether and when my hearing had stabilized.

Interspersed during the HBOT sessions, I was seen for four subsequent ENT visits, each of which also included an audiologic evaluation. A timeline of my visits, which includes a baseline audiogram for the left ear, the duration of Prednisone intake, and all HBOT sessions, is shown in Figure 2. Details related to the figure will be discussed in a later section (see *Timeline of Changes in Hearing Levels*).

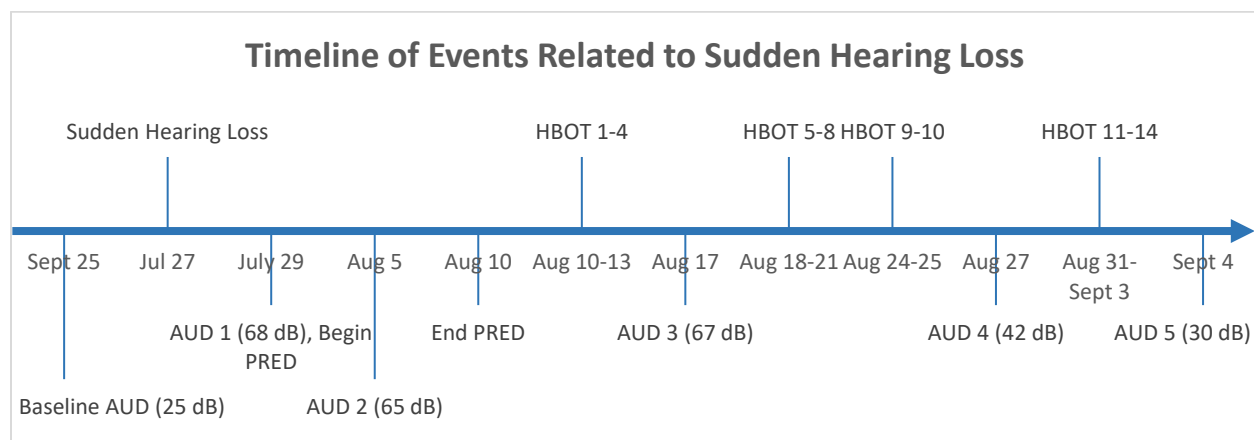


Figure 2. Timeline of events surrounding idiopathic sudden hearing loss. Events include audiometric evaluation at baseline (September 25, 2019), occurrence of sudden hearing loss (July 27, 2020), administration of medical treatment and HBOT, and follow-up audiological evaluations. (AUD = audiological evaluation, PRED = Prednisone medication, HBOT = hyperbaric oxygen therapy). Parenthesized values represent air-conduction pure tone thresholds in dB HL, averaged across 4000, 6000, and 8000 Hz, established at the indicated times.

Definition, Prevalence, and Etiologies of ISSNHL

There is no universal definition of ISSNHL but it is often defined as an increase in pure tone thresholds of greater than 30 decibels (dB) in at least three adjacent frequencies occurring within 72 hours. Although my sudden hearing loss might not adhere strictly to this widely used definition, in that it involved both a low-frequency conductive component and a high-frequency



sensorineural component and the three adjacent frequencies affected by sensorineural loss were the non-octave frequencies of 4000, 6000, and 8000 Hz, the symptoms I experienced were those described by many researchers (see, for example, Murphy-Lavoie & Mutluoglu, 2020).

Prevalence estimates vary, but the NIDCD (2018) estimates that between one and six people per 5000 every year are affected by ISSNHL, indicating that the actual number of new cases each year could be much higher because the condition often goes undiagnosed. A retrospective analysis by Edizer et al. (2015) conducted on cases of sudden hearing loss between 2009 and 2013 revealed that the condition was accompanied by tinnitus in 52% of patients and by vestibular symptoms in 27%.

There are many possible etiologies of sudden sensorineural hearing loss. They include vascular occlusion, hemorrhage, or disease (including viral infections); vasospasm; change in blood viscosity; labyrinthine membrane breaks; autoimmune disorders; trauma; toxins; demyelinating disease; stroke; or schwannoma (Kuhn et al., 2011; Murphy-Lavoie & Mutluoglu, 2020). For most patients, however, an etiologic factor is not identifiable. The etiology has been reported to be idiopathic in 71% of cases, and the condition, whose onset typically occurs upon awakening, has variously been described as affecting males and females equally (Chau et al., 2010) or affecting males predominately (Kuo et al., 2020). The condition typically affects adults in their late 40s and early 50s, although it can occur at any age (Kuhn et al., 2011; Lloyd, 2013; NIDCD, 2018). Ballesteros et al. (2012) concluded that lack of recovery from ISSNHL may be related to a specific genetic risk factor, but they found no relationship between ISSNHL and classical cardiovascular risk factors. Ciorba et al. (2015) have shown that cardiovascular risk factors, if they exist, do not have any significant influence on hearing threshold recovery in ISSNHL.

Current Treatments for ISSNHL

Full or partial spontaneous recovery rates, which are difficult to determine because many patients do not seek medical care, have been found to vary widely from 25% to 89% across studies (Lamm et al., 1998). Piper et al. (2021) indicate that "... as many as 65% of cases may resolve spontaneously." The prognosis for hearing recovery appears to be dependent on the severity of hearing loss, age, the presence of vertigo, and audiometric configuration (Kuhn et al., 2011; Murphy-Lavoie & Mutluoglu, 2020). Spontaneous recovery and recovery due to any specific treatment regimen may be difficult to distinguish definitively, especially because ISSNHL is often treated as early as possible to maximize the chances for recovery (Stachler et al., 2012; NIDCD, 2018). The effects of any treatment, therefore, are likely to be combined with any degree of spontaneous recovery that may occur. Pragmatically, many treating physicians wait a short period of time before initiating an active treatment regimen to allow for at least some degree of healing to occur naturally.

Lamm et al. (1998) found that if the delay in treatment of ISSNHL ranged between two and six weeks, one-half of the cases showed a substantial hearing gain of more than 20 dB in at least three frequencies, one-third showed a moderate improvement (10-20 dB) and 13% showed no hearing improvement at all. They recommended that HBOT be administered no later than three months after onset for patients with ISSNHL, acoustic trauma, or noise-induced hearing loss. More recently, Murphy-Lavoie et al. (2012) indicated that the best and most consistent outcomes are obtained when HBOT is initiated within two weeks of symptom onset and



combined with corticosteroid treatment. Lloyd (2013) has expressed a viewpoint favoring the earliest possible treatment:

The earlier the treatment is commenced the higher the chance of obtaining a significant improvement in hearing thresholds. By 2 weeks the chances of gaining any significant benefit are very low. (p. e593)

The medical and non-medical treatments that are now being used to expedite recovery are corticosteroids and hyperbaric oxygen therapy, respectively. Based on a review of 68 clinical studies that investigated the effect of HBOT in the last couple of decades, Bayoumy and de Ru (2019) recommended that therapy for ISSNHL be initiated as early as possible, preferably within 48 hours of onset, and to use a combination of therapies consisting of HBOT and corticosteroids.

A panel of 19 experts has provided an evidence-based guideline for managing patients presenting with ISSNHL (Stachler et al., 2012). The panel recommended that physicians first rule out conductive hearing loss and educate the patient regarding the natural history of ISSNHL, benefits, risks, and efficacy of medical interventions. It recommended that physicians then consider offering corticosteroids as initial therapy and hyperbaric oxygen therapy within three months of an ISSNHL diagnosis. The panel also advised physicians regarding specific tests and therapies to avoid as routine measures.

Regenerative therapy techniques to restore inner ear hair cell functioning have been discussed recently as potentially effective in reversing ISSNHL and other acquired hearing losses (McLean & Franck, 2021). Gene therapy techniques are also being investigated as applicable to that sub-population of individuals whose ISSNHL is due to genetic causes (McKenna, 2021).

Systemic vs. intratympanic steroids

Systemic steroids or intratympanic steroid injections are currently the preferred medical treatment for ISSNHL (Kuhn et al., 2011; Rauch et al., 2011; Lai et al., 2017), and Prednisone appears to be the drug of choice among the medical community. Side effects, most of them rare or non-serious, are well known (see WebMD, 2021).

Several studies, all based on a review of pooled data and studies using meta-analysis, provide a synopsis of the status of the relative efficacy of these treatments. Rauch et al. (2011) found that two months after treatment of 250 patients diagnosed with ISSNHL and who had presented with a pure tone average of 50 dB or higher, hearing levels in the affected ear improved by approximately 30 dB HL under either oral or intratympanic-injection treatment conditions. They concluded that intratympanic treatment was not inferior to oral Prednisone treatment and thus that both were equally effective. In patients who had failed initial treatment with systemic steroids, Ng et al. (2015) found that additional treatment with salvage intratympanic corticosteroid injections demonstrated a statistically significant improvement in hearing thresholds as compared to controls, with no serious side effects reported.

Lai et al. (2017) agreed that both systemic and intratympanic treatments produce similar short-term efficacy for improving pure tone averages in patients with ISSNHL, noting that intratympanic therapy may reduce the side effects associated with systemic steroid use. Based on a review of six articles in the scientific literature, Qiang et al. (2017) concluded that intratympanic steroid treatment groups exhibited better outcomes in pure tone average improvement and recovery rate than systemic steroid therapy groups.



Clearly, these studies suggest that factors associated with the outcomes of systemic vs. intratympanic steroid injection may need further clarification before the question of their relative efficacy can be resolved. They also require caution in interpreting the significance of gains in pure tone thresholds, as participants in different studies often exhibit highly varying degrees of ISSNHL at onset.

Hyperbaric oxygen therapy

According to Kate McKenney (Younghans, 2020), administrative manager for the Michigan Medicine Comprehensive Wound Care Clinic, HBOT was first used in the 1930s by the U.S. Navy to treat decompression sickness, and today's safety protocols are still based on Navy research. HBOT has been performed in healthcare settings since around the 1950s. McKenney notes that a nationwide rule requires that a trained and certified healthcare professional be present onsite during the entirety of each treatment.

HBOT is aimed at treating a wide variety of conditions, including: severe anemia, brain abscess, air bubbles in blood vessels, burns, carbon monoxide poisoning, crushing injury, sudden hearing loss (often termed *sudden deafness*), decompression sickness, gangrene, skin or bone infection that causes tissue death, nonhealing wounds (such as diabetic foot ulcer), radiation injury, skin graft or skin flap at risk of tissue death, traumatic brain injury, and sudden (painless) vision loss (Mayo Clinic, 2021). Understandably, HBOT requires a physician's prescription.

Numerous systematic reviews have been performed to determine the most effective treatments for ISSNHL. For many decades, the three most promising treatments have included vasodilators, corticosteroids, and hyperbaric oxygen therapy. Racic et al. (2003) showed a particularly positive impact of HBOT on ISSNHL when compared to the vasodilator—or hemorheologic agent—pentoxifylline. Their retrospective study included 115 patients with idiopathic sudden hearing loss and compared hearing improvement in those administered HBOT (N=51) and those administered pentoxifylline infusions (N=64). Results showed a statistically significant mean hearing improvement of 46 dB in those treated with HBOT, compared with an average 21.5-dB improvement in the group treated with pentoxifylline infusion. Furthermore, 47% of the HBO-treated patients showed hearing recovery to pre-morbid levels, compared to 6% of the pentoxifylline-treated patients. Based on the several studies reviewed above, it is not surprising that a vasodilator was found to be less effective than HBOT in treating ISSNHL.

Among vasodilators, corticosteroids, and hyperbaric oxygen therapy, hyperbaric oxygen has been described as the only option that has sufficient randomized controlled trials to have a positive meta-analysis Cochrane review (Murphy-Lavoie & Mutluoglu, 2020). Bennett et al. (2012) found that in persons with acute ISSNHL, there was an improvement of 15.6 dB in average pure tone audiometric thresholds after HBO treatment, although they advocate for an appropriately powered trial to define those patients for whom HBOT is most beneficial. Based on their pooled analysis, they were unable to establish any benefit of HBOT in the treatment of tinnitus.

HBOT is a non-invasive procedure aimed at supplying 100% oxygen under high pressure to areas of the body that require more oxygen to heal wounds and fight infection. The pressurization facilitates the intake of oxygen into the lungs, thereby increasing the amount of oxygen in the blood. In addition to assisting with fighting infection and healing, HBOT also stimulates the release of stem cells and growth factors (Younghans, 2020).

Piper et al. (2014) provide the following rationale for the use of HBOT to treat ISSNHL, based on the position of the Governing Body of the Undersea Hyperbaric Medical Society



(UHMS):

The rationale for the use of hyperbaric oxygen to treat ISSHL is supported by an understanding of the high metabolism and paucity of vascularity to the cochlea. The cochlea and the structures within it require a high oxygen supply. The direct vascular supply, particularly to the organ of Corti, is minimal. Tissue oxygenation to the structures within the cochlea occurs via oxygen diffusion from cochlear capillary networks into the perilymph and the cortilymph. The perilymph is the primary oxygen source for these intracochlear structures. Unfortunately, perilymph oxygen tension is decreased significantly in patients with ISSHL. To achieve a consistent rise of perilymph oxygen content, the arterial-perilymphatic oxygen concentration difference must be extremely high. This can be restored with hyperbaric oxygen therapy. (p. 139)

HBO treatments are called *dives* to compare them to diving under seawater. Treatment depths are usually 2.0 ATA (atmospheres absolute) or 2.5 ATA. Any time a patient dives at pressures above 2.0 ATA, periodic air breaks are necessary to avoid an oxygen toxicity event. Air breaks are normally provided on a scheduled basis but may be administered whenever needed. A monoplace chamber can be pressurized to 3 ATA, the equivalent of being 66 feet deep in seawater. In my case, a pressure of 2.5 ATA was applied, which is equivalent to a seawater depth of approximately 50 feet. The treatment protocol recommended by the UHMS (2014) is 100% oxygen at 2-2.5 ATA for 90 minutes daily for 10-20 treatments. The effect of pressurization on the ears is analogous to what happens while descending in an airplane, and depressurization is analogous to the effects while ascending.

HBOT sessions are conducted either in a pressurized chamber or under a pressurized oxygen hood that envelops the head. The monoplace pressurization chamber in which I received hyperbaric oxygen treatments is shown in Figure 3.

Rhee et al. (2018) compared outcomes after HBOT plus medical treatment versus medical treatment alone in 2401 patients with ISSNHL. Pooled odds ratios for complete and incomplete hearing recovery and absolute hearing gain were significantly higher in the HBOT plus medical-treatment group than in the medical-treatment-alone group. The benefit of HBOT was greater in groups with severe-to-profound hearing loss at baseline, when HBOT was used as a salvage treatment (following medical treatment), and when the total duration of HBOT was at least 20 hours. In agreement with other researchers, Rhee et al. suggested that future trials are needed to investigate maximal treatment benefit.

Financial Costs of HBOT

HBOT is relatively expensive. The costs, whether covered by insurance plans or by patients themselves, vary widely depending on: (1) the designation of the treated condition by the Food and Drug Administration as on-label or off-label, (2) the type and location of the provider (i.e., hospital-affiliated vs. private clinic), and (3) the severity of symptoms (Katz, 2017). Those costs must cover the intervention of medical staff and technical specialists over multiple sessions, which vary widely depending on the treated condition. Approval of conditions eligible for HBOT is determined by the Centers for Medicare and Medicaid Services and UHMS,





Figure 3. HBOT environment in which pure oxygen is delivered in a pressurized hyperbaric chamber. (Photograph provided by and used with permission of Sparrow Wound and Hyperbaric Clinic, Lansing, Michigan, USA, <https://www.sparrow.org/departments-conditions/all-departments/wound-hyperbaric-clinic>).

and HBOT for sudden sensorineural hearing loss is approved by the UHMS. In some cases, providers offer discounts to veterans or when several treatments are administered consecutively, and they apply a sliding scale for patients who pay out of pocket. If not covered by insurance plans, the average out-of-pocket cost per treatment is about \$250 per typical treatment and, if delivered at a large medical hospital, can exceed \$1000 (CostHelper Health, 2021). Currently, Part A or Part B of Original Medicare does not routinely cover HBOT for ISSNHL, as indicated by the absence of ISSNHL from the National Coverage Determination for Hyperbaric Oxygen Therapy (NCD 20.29). Some secondary insurers, however, do cover most of the costs.

Initiated in a hospital-based wound clinic on the day of intake, my treatments began before obtaining assurance that health insurance would apply. The hospital voluntarily agreed to cover the costs of a specified number of initial treatments, after the attending physician recommended that treatments begin as soon as possible to facilitate an optimum outcome. Ideally, it is advisable that patients with ISSNHL attempt to obtain definitive, preferably written, information about insurance coverage before initiating HBOT.



HBOT Precautions and Risk Factors

HBOT sessions are normally conducted on an outpatient basis on weekdays only, but some facilities operate around the clock to address emergent issues. Facilities commonly maintain a list of prohibited materials that cannot be brought into the chamber; anything that goes into the chamber must be approved by the Hyperbaric Safety Director. Preparation for each session requires that the following are *strictly prohibited*: flammable materials, including matches, lighters, and newspapers or other reading materials; synthetic materials such as nylon or rayon (only supplied, hospital-grade pants and gown must be worn); use of oil-based or petroleum products prior to a given session (including hairspray, hair gels, mousse, lipstick, lotions, creams, and nail polish); electronic items (anything with a battery such as cell phones, fitness trackers, watches and hearing aids); and food (although water is allowed for hydration).

In each HBOT session, patients first undergo a period of gradual pressurization (lasting, in my case for approximately 22 minutes) before receiving pressurized, 100% oxygen for 30 minutes. While still under pressurization, they are then allowed to breathe normal (21%) oxygen for 10 minutes through an oxygen mask to prevent oxygen toxicity. Two more 30-minute cycles of pressurized, 100% oxygen, separated by another 10 minutes of breathing normal air, are followed by a period of gradual decompression. A hyperbaric technologist continually controls the chamber settings and monitors the patient during the entire session. Throughout each treatment session, two-way audio and visual communication is maintained between the technologist and the patient, and the patient can view a preferred TV channel or DVD movie throughout the procedure.

A noteworthy paradox for patients with ISSNHL is the significant potential impact of pressurization on the eardrums. Irrespective of the condition being treated, many studies indicate a major side effect of HBOT to be middle ear barotrauma, with most cases resolving in the absence of repetitive trauma (e.g., Lima et al., 2014). According to Heyboer et al. (2017), eardrum retraction occurs in some patients, and in rare cases conductive hearing loss resulting from tympanic membrane rupture is also possible. Rarely, middle ear barotrauma can be transmitted to the inner ear, with risk of rupture of the round or oval window membranes and impairment of inner ear function, causing vertigo and sensorineural hearing loss. Though quite rare, other potential risks include barotrauma of the sinus and paranasal regions, dental structures, and lungs; oxygen toxicity of the central nervous system and lungs; and, with prolonged exposure, ocular side effects that damage the lens and retina, some of which are reversible after treatments are discontinued. Heyboer and colleagues state that HBOT can impact blood pressure and that there is a theoretical risk of pulmonary edema in patients with compromised left ventricular function and a risk of hypoglycemia in patients with diabetes. Those investigators also note that there is a risk of claustrophobia in some patients when HBOT is administered in a hyperbaric chamber.

Because of the likelihood of barotrauma, each HBOT session is staffed by a physician who performs an otoscopic inspection before and after treatment. The Teed Scale (see Revolution Health & Wellness, 2021) is used to classify gradations of barotrauma, based on the condition of the eardrums, for the purpose of indicating when further treatments can be safely administered. If, before a session, the otoscopic inspection reveals an abnormal eardrum, a longer period of compression and decompression may be needed or the session for that day may be canceled. An abnormal eardrum after treatment, such as redness or retraction, can suggest the need to skip a day or more before the next treatment. These effects are minimized by the



technologist’s instructions to patients on techniques that are most effective in equalizing middle ear pressure. Those techniques, which are quite familiar to hearing-care professionals, include swallowing and yawning. In my case, I found swallowing while holding my nose (without performing a Valsalva maneuver), to be necessary for effective equalization during pressurization.

Timeline of Changes in Hearing Levels

The timeline in Figure 2 identifies the sequence of events from baseline audiometry through the initiation and termination of the medical and non-medical treatments. It also shows the timing of post-episodic audiologic evaluations conducted to evaluate treatment effects. That timeline is accompanied by Figure 4, which presents the pure tone air-conduction audiometric thresholds for the affected left ear on the dates indicated in both Figures 2 and 4. The hearing thresholds depicted in Figure 4 are indicative of a sensorineural loss, with the exception that a 10-dB air-

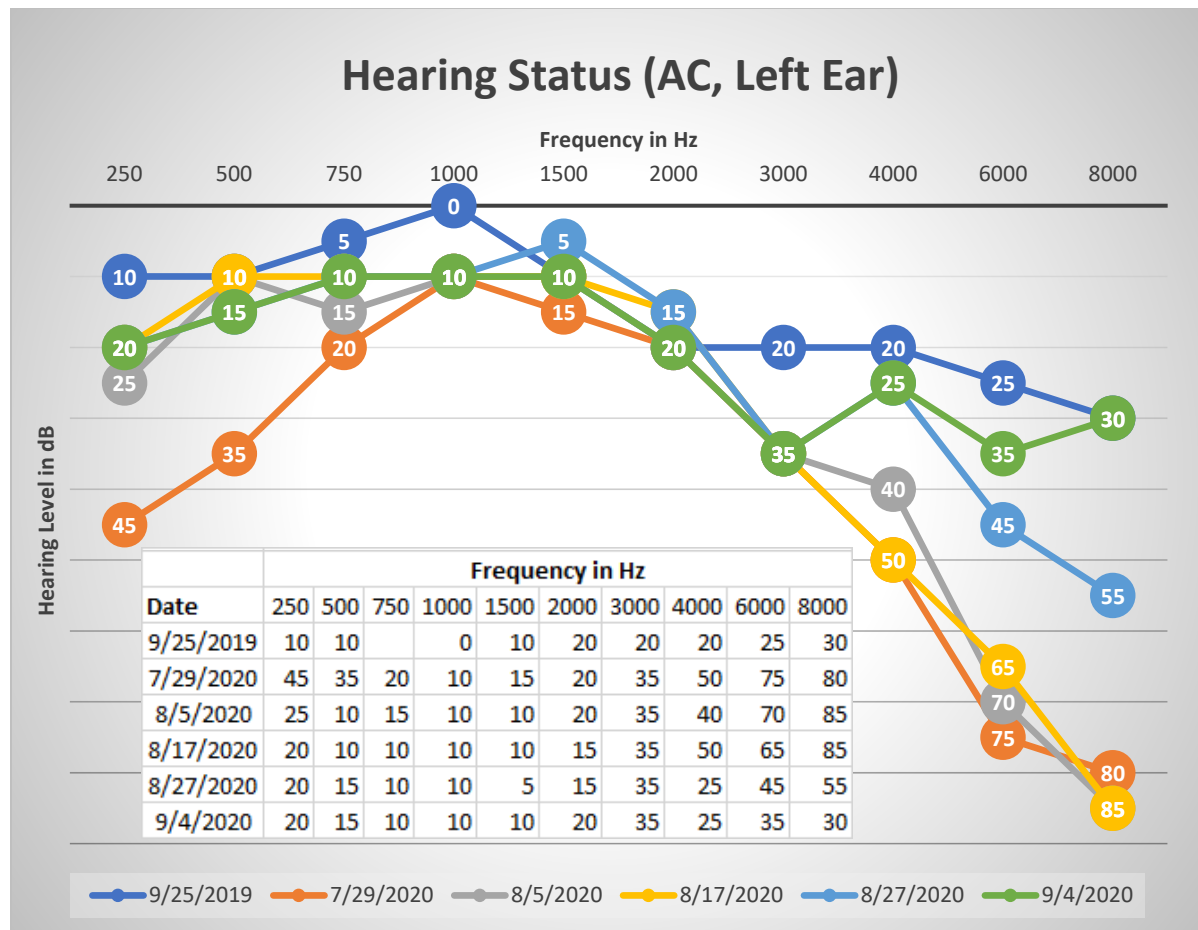


Figure 4. Air-conduction pure tone audiometric thresholds for the affected left ear, tracked over time from baseline on September 25, 2019, through September 4, 2020. Tabular data are included to augment graphed hearing levels, some of which overlapped at various times at some frequencies.



bone gap persisted at 4000 Hz in the audiometric findings through August 5; no gap was present in later audiograms. In combination, Figures 2 and 4 reveal the sequential relationship between hearing thresholds and the medical and hyperbaric oxygen treatments.

The following description of changes in my hearing thresholds assumes test-retest reliability to be ± 5 dB. A preliminary observation is that hearing had become substantially worse at 250 and 500 Hz between the baseline results of September 25, 2019, and when measured two days after the sudden hearing loss (AUD 1). At approximately the midpoint of the Prednisone treatment, the audiogram (AUD 2) indicated that hearing at those frequencies had improved substantially. This suggests that the medical treatment was likely effective in partially restoring hearing to its baseline level at 250 Hz, and effective in fully restoring hearing to its baseline level at 500 Hz. Hearing thresholds in the frequency range of 750-3000 Hz in the left ear, which were within normal limits at baseline, were minimally affected throughout the series of medical and HBO treatments.

In the higher frequencies, hearing was substantially affected by the sudden loss. Air-conduction pure tone averages at 4000, 6000, and 8000 Hz, tracked in Figures 2 and 4 with respect to the consecutive audiograms from baseline through audiogram 5 (AUD 5), reveal a 43-dB average drop at those frequencies between baseline and AUD 1. That drop represents the extent of the sudden hearing loss in that high-frequency region. There was essentially no improvement at those frequencies through four HBOT sessions. Between HBOT sessions 5 and 10, however, an average improvement of 25 dB occurred. An additional 12-dB improvement occurred after HBOT session 14, leading to a termination of the sessions based on the observation that hearing thresholds had largely been restored to their pre-episodic levels.

Over the period from July 29 through September 4, 2020, averaged hearing thresholds at 4000, 6000, and 8000 Hz, respectively, had improved by 25, 40, and 50 dB. In addition, the conductive component seen audiometrically for the left ear at the time of the sudden loss had completely resolved by the time of AUD 3, after completion of the Prednisone treatment and HBOT session 4. During the same period of late July through early September, however, Type A or A_s (A shallow) tympanograms were obtained for the right ear through the period ending at AUD 4. A Type B tympanogram was observed in that ear at AUD 5, indicating a stiff eardrum, a sign of fluid in the middle ear. For the left ear, tympanograms were Type A_s through AUD 2, and Type C—indicating negative middle ear pressure—through the final two audiometric evaluations. Both negative pressure (in the left ear) and build-up of fluid (in the right ear) were likely associated with the HBO treatments, and appeared subjectively to have resolved several days following the treatments.

Although it is not possible to rule out at least some degree of natural recovery during either the medical or hyperbaric oxygen treatments, the strong association between the two treatments and hearing recovery suggest that Prednisone intake and HBOT were likely responsible for improvements in different frequency regions. In a summary of the effect of the treatments on my hearing, the lead physician of the hyperbaric clinic offered his professional opinion:

The initial treatment with oral Prednisone was no doubt helpful in recovering low-frequency hearing, and HBO was helpful in arresting and improving high-frequency hearing between 4000-8000 Hz (Donato Borrillo, M.D., personal communication, September 8, 2020).



At this writing, my hearing has stabilized, although I continue to experience an almost constant ringing tinnitus that worsens with fatigue. The tinnitus is localized primarily in the left ear but at times is perceptible as a non-localized head noise. The frequency of the tinnitus is slightly above 10 kHz, estimated by using a frequency generator from a smartphone app to produce an air-conducted pure tone in my right ear as a reference, given that I cannot detect such high frequencies in my left ear. I think of myself as a *Category 1* tinnitus patient, as classified in the 0-4 categorization scheme of Jastreboff and Jastreboff (2000). Although I have not sought any formal rehabilitative measures for the tinnitus, I have engaged in a form of self-administered tinnitus retraining by listening to electronically streamed rain sounds or the sounds of a babbling brook before falling asleep during nights when the tinnitus is most disturbing. I use that exercise to match the overall level of the external sounds as closely as possible to that of the tinnitus—to achieve *blending*, which is the ability to hear the external sound and tinnitus separately but to experience them as interfering or intertwining with one another. Also, during the day, directing my thoughts to what is going on around me provides substantial relief from the tinnitus simply by allowing me to ignore it, as environmental sounds rarely mask the tinnitus. When being exposed to relatively loud noises, I always wear hearing protection (i.e., earmuffs or earplugs).

Personal Reactions to HBOT

Despite the risks of HBOT described above—some of which I was not fully aware at the beginning of my HBOT sessions—I consider the benefits of improved hearing well worth the risks. That is probably true in large part because of my long career as an audiologist, coupled with the sense of loss I experienced in late July 2020 when I awoke, having suddenly lost the reasonably good hearing I have enjoyed for almost eight decades. The experience was shocking, and I was relieved to know there might be interventions to recover at least some of what I had lost.

While undergoing HBOT, I experienced middle ear barotrauma consisting of eardrum retraction and (in one instance) eardrum redness, which lasted until the following day. When that occurred, that day's treatment was canceled. I also experienced a temporary myopia and, during the first several treatments, a feeling of claustrophobia, even though I have never regarded myself as being claustrophobic. The experience was accompanied by imagined scenarios that raised concerns such as what happens while being in an enclosed chamber if: (1) there is a fire, (2) there is a power outage, or (3) I should need to use the restroom. The technologist addressed these concerns by assuring me that: (1) she could safely get me out of the chamber within a minute in case of an emergency, (2) there was a backup power source in the hospital-affiliated facility, and (3) a gender-appropriate urinal could be provided before entry into the chamber if desired. (The latter concern is best addressed by the patient's using the restroom before each session.)

In my case, one technologist typically staffed the two hyperbaric chambers. The second chamber was not always occupied, however, and a second technologist occasionally relieved the primary technologist when a break was needed. A concern that I quietly contemplated during the initial HBOT sessions was what might happen if the technologist should become incapacitated while I was inside the chamber, which is locked from the outside. I found relief in the thought that help might be summoned in that unlikely event either if the technologist were able to ask for help by telephone or if I were able to use the microphone inside the chamber to summon help from other staff members who were working near the hyperbaric area. Although there have been



no reports that patient security has ever been affected by such incidents, that issue is one that ideally should be addressed routinely to assure patients that they are completely safe while in the chamber.

During the initial HBOT sessions, I had quietly entertained the thought that my hearing in the left ear, or perhaps both ears, might eventually worsen to the point of justifying the need for a CROS or BiCROS hearing aid system, or perhaps conventional hearing aids. It was only after obtaining the audiometric results of AUD 4 that I was able to dismiss that concern. Also, the otolaryngologist at that point was able to rule out the presence of an VIIIth nerve tumor, given that hearing would likely have worsened over time, not improved, if such a tumor were present.

In addition to the risks directly posed by hyperbaric oxygen treatments, the risks associated with the COVID-19 pandemic added anxiety to my overall experience, given the number of HBOT sessions and doctor visits. During all the visits, appropriate precautions—including the use of temperature checks, hand sanitizer, and face masks—were always taken to protect patients. (Understandably, masks are not worn inside the HBOT chamber.) That these precautions were effective is supported by the fact that I never contracted COVID-19.

During most sessions, I was able to distract from my various concerns by the ability to watch TV or a movie while in the chamber, activities that were noticeably comforting after the first several sessions. The continued hearing improvement in my left ear provided further relief from any anxiety, and during subsequent treatments, I found myself optimistically anticipating another session because of the prospect that it might facilitate further hearing improvement.

A lingering concern is what caused my sudden hearing loss and whether it might occur again, and whether a similar loss could possibly affect the opposite ear. If so, might it be worse next time? Because blood supply to the cochlea has long been associated with sudden sensorineural hearing loss, and such episodes often occur during sleep, the question might be asked: Can such a loss be caused while sleeping in a bodily position that restricts blood supply? Experiencing this episode has also made me feel more vulnerable to other adverse health effects, especially those that commonly accompany aging.

The literature reveals that many unknown factors determine the extent to which spontaneous recovery occurs in ISSNHL. The distinction between spontaneous and treatment-related recovery, therefore, is far from definitive. If we arbitrarily assume that as many as two-thirds of patients with ISSNHL may experience full or partial spontaneous recovery, as stated by Piper et al. (2021), and that the spontaneous recovery rate is higher for those having milder degrees of hearing loss—as claimed by Leung et al. (2016)—the recommendation of both lead physicians that I undergo HBOT was undoubtedly a precautionary decision. Even if a spontaneous recovery rate of approximately two-thirds were accurate, I believe that it is justifiable for an individual with ISSNHL to consider a 1:3 odds of sustaining a permanent hearing loss as unacceptable and, therefore, to choose a treatment option over no treatment.

For a person of my age, I currently have a relatively mild hearing loss that is limited to 3000-8000 Hz bilaterally. I occasionally self-administer informal, practical tests of high-frequency differences in hearing sensitivity between ears. Those checks on my hearing include listening for differences while rubbing a thumb against the fingers held near the ear, gently rubbing a finger against each tragus, or listening for differences in speech fricatives or music while closing one ear at a time. Despite some notable high-frequency deficiencies, these exercises remind me to appreciate the advantages of binaural hearing and the acoustic redundancy of speech, both of which allow me to understand speech well under quiet and even under moderately noisy conditions.



Conclusions

This experience has led me to have a renewed appreciation for the sense of hearing. It has also reminded me that there are potentially effective treatments for sudden sensorineural hearing loss, despite general acceptance of the notion, even among audiologists, that all sensorineural deficits tend to be permanent.

Although my speech recognition thresholds and speech recognition scores (in quiet) are within normal limits bilaterally, there is no question that even a partial unilateral loss of hearing negatively impacts overall functional hearing ability, as well as psychological health (see Trevis et al., 2018). I have also become more starkly aware of the need for hearing healthcare professionals, including audiologists, to take seriously their patients' complaints of tinnitus, which remains a severely underfunded medical condition (American Tinnitus Association, 2021a).

Those who have never suffered from tinnitus are likely to underestimate its deleterious impacts on quality of life. Audiologists and other healthcare workers should not be satisfied with simply checking a box or line on an intake-interview form indicating that a patient experiences tinnitus; they should feel obligated to question those patients about the effect tinnitus is having on their everyday lives. Minimally, they should routinely administer a tinnitus questionnaire (American Tinnitus Association, 2021b) to those patients and, if warranted, refer them to a professional who can successfully manage the tinnitus. A coherent set of recommendations for audiological assessment and rehabilitative management of patients with tinnitus has been provided recently by Henry et al. (2020). Spankovich and colleagues (2021), in a recent summary of tinnitus research that addresses the probable anatomical and physiological underpinnings of tinnitus, characterize the condition as an "attempt of the brain to fill in the reduced peripheral input (Spankovich, 2019)." Interestingly, recent research suggests that bimodal stimulation techniques, in which tonal stimuli are synchronously combined with tactile stimulation, can be used to reduce tinnitus, as well as restore equilibrium in persons with imbalance disorders (Eagleman, 2021).

Despite the potential risks of the use of systemic steroids and hyperbaric oxygen therapy, I consider my rehabilitative journey well worth the associated risks because of the benefits of the substantial degree of hearing that has been restored. Although hyperbaric oxygen therapy may have been used as a precautionary treatment in my case, it appears to have had a substantial—and likely a major role—in restoring my unilateral high-frequency hearing to functionally normal levels. This experience has left me with a deeper appreciation of the need for audiologists, otolaryngologists, and others who come in professional contact with patients who suffer from hearing loss to be empathetic to their needs and to provide all rehabilitative measures aimed at lessening its negative consequences.

Finally, my encounter with unilateral sudden hearing loss has served as a reminder of the importance of the interprofessional role audiologists play through their participation in a team approach aimed at diagnosing and determining a course of intervention in cases like mine. That encounter also suggests that it is desirable and appropriate for audiologists—including student interns, as well as emerging and seasoned professionals—to acquire a thorough understanding of ear anatomy and physiology and to become familiar with the basic terminology and impact of pharmacological medications on auditory and vestibular functioning. Those goals can best be achieved by targeted curricular enhancements, where needed, in academic Au.D. programs.



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