Sudden Hearing Loss

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Sudden sensorineural hearing loss has been described as a medical emergency in search of appropriate diagnostic techniques and treatments [1]. The abrupt development of an unexpected sudden sensory deficit warrants consideration of this situation as an emergency by medical professionals and lay personnel. Despite the dramatic presentation, in most cases, sudden sensorineural hearing loss is the presenting symptom of a pathophysiology that has yet to be identified. In as many as 88% of patients, a battery of diagnostic testing fails to yield an identifiable cause [2].

Sudden sensorineural hearing loss is an active topic of discussion and research in the otolaryngology literature. Over the past 30 years, greater than 800 articles, or roughly one every 2 weeks, has been published on this topic in the English medical literature. Several excellent broad-based reviews of this topic have been written by respected clinicians [1–6], and courses on this subject are regularly presented at professional meetings.

The incidence of sudden hearing loss in the United States is often reported at between 5 and 20 cases per 100,000 people annually, based on a report by Byl [7]. Another commonly quoted figure is that there are roughly 4000 cases annually in the United States [8]. These numbers seem discrepant because 4000 cases annually calculates to 1.3 cases per 100,000 people per year in the United States, currently with a population of roughly 300 million people [9]. Even using the lowest end of Byl’s estimate produces 15,000 cases per year in the United States. A recent 5-year study in Thailand demonstrated the incidence to range from 6.49 to 10.21 cases per 100,000 population per year [10].

In its most nonspecific interpretation, the term sudden hearing loss could conceivably refer to any sudden loss of hearing (ie, sudden sensorineural hearing loss or sudden conductive hearing loss). Despite this, the term sudden hearing loss, and the abbreviation SHL in reference to sudden hearing loss, is common parlance.
sensorineural hearing loss is frequently found in the literature [5]. Sudden sensorineural hearing loss is also abbreviated as SSHL and sometimes referred to as sudden deafness [8]. For the purposes of this article, sudden hearing loss (SHL) refers to sudden sensorineural hearing loss. The National Institute on Deafness and Other Communication Disorders (NIDCD) [8] defines sudden sensorineural hearing loss as a rapid loss of hearing, occurring over a period of up to 3 days. The hearing loss must be of at least 30 dB in three connected frequencies. Further, the NIDCD indicates that sudden sensorineural hearing loss should be considered a medical emergency, although other investigators openly dispute this [11].

The definition of SHL as a minimum of 30 dB loss over three consecutive frequencies occurring in fewer than 3 days is not universally adhered to in the literature [6]. Some studies do not provide a specific definition for SHL [12,13], whereas others use alternate definitions (such as a 20-dB loss) [14]. The use of unclear or alternate definitions for SHL produces substantial difficulty in comparing patient populations and treatment outcomes among studies; however, there are some justifiable reasons for using an alternate definition of SHL. For example, the sudden development of a 25-dB hearing loss at three consecutive octaves in the range of human speech would likely be noticed and distressing to a normally hearing individual, yet the definition of SHL suggests that a 25-dB loss should not be considered pathologic. Further, in the authors’ experience, the presentation of a patient who reports truly progressive loss over 3 days is far less common than presentation of an abrupt loss of hearing marked by a specific point in time. It seems reasonable to propose that rapidly progressing hearing loss may be a different disease entity than hearing loss that occurs at a definable time point. Presumably, these reasons for using an alternate definition of SHL may be considered by some investigators who include patients who have lesser degrees of hearing loss or more rapid progression of symptoms in studies of SHL.

In addition, modern reports tend to treat the term SHL as a diagnosis and thus limit the report or study to only those patients who have idiopathic SHL. Older reports of SHL may be more likely to consider SHL as a symptom and thus include patients in whom the diagnostic evaluation reveals an obvious cause (eg, an acoustic neuroma). Although not all investigators adhere rigorously to the definition espoused by the NIDCD, most studies include patients who have suffered at least a loss of 20 dB at more than one frequency over less than a 72-hour period. Regardless of how one defines SHL, there is some agreement that the natural course of those who suffer idiopathic SHL involves a spontaneous recovery of function in a percentage of patients. Although the natural course of disease has not been established in a large number of patients, spontaneous recovery is variably reported to occur in up to 65% of patients who have idiopathic SHL [3]. Although the percentage of patients experiencing spontaneous recovery varies in different series, two of the larger series involving
patients followed without treatment reported spontaneous recovery rates of 58% (n = 52) [3] and 65% (n = 28) [15].

The likelihood that future studies will include large numbers of untreated patients is small. Various forms of treatment have become widely accepted as beneficial in most locations, and the idea of not providing such treatments could possibly be viewed as unethical. Typically, much of the recovery is thought of as occurring within the first month after the SHL event; however, a recent report found that nearly 22% of patients showed improvement in their audiometric studies beyond the first month following the onset of symptoms [16].

The specific definition of what constitutes “improvement” or “recovery” after an SHL is not uniform among studies and reports. Vague subcategories of recovery, such as partial recovery or minimal recovery, are presented in the literature without universally accepted definitions. Perhaps the most lenient and possibly the most commonly encountered definition of improvement is an improvement of 10 dB in pure-tone average (PTA) or an improvement of 10% or 15% in speech discrimination score (SDS). An alternate, stricter interpretation is improvement of 20 dB in PTA or 20% in SDS. Further, some investigators use a mathematic formula to calculate recovery as a percentage of hearing recovered [17]. Each of these definitions has advantages and disadvantages. Using absolute values of improvement has the advantages of being relatively simple and being able to be performed without knowledge of prior hearing function or a normally hearing contralateral ear. The disadvantage is that absolute values can be misleading in certain patients. For example a patient who has a 90-dB PTA and a 10% SDS could be considered improved at an 80-dB PTA and a 10% SDS, even though the patient may not notice any subjective improvement. Further, patients who experience improvement in PTA but a worsening SDS (or vice versa) may be classified as improved. Presenting recovery as a percentage is more complex but provides consideration for a varying spectrum of severity of sudden loss. A major disadvantage to presenting recovery as a percentage is the need to know or assume the function of the ear before the SHL event. In most cases, an audiogram before the SHL event is not available and must be assumed. Typically, the ear is assumed to be normal or similar to the contralateral ear. The validity of these assumptions has an impact on the calculation of a recovery percentage.

Most important, the lack of a uniform definition of recovery limits the ability to compare or combine data across studies. In a recent article, the authors and colleagues [18] presented data using four different definitions of recovery to compare their data to those of other studies. The recovery rates between their study and three other studies were found to be reasonably similar when identical definitions were applied. It seems reasonable to speculate that perhaps the applied definition of recovery is the single most important factor in determining what percentage of patients who have SHL recover.
Beyond the differing definitions of SHL and the dispute over how to best evaluate the presence or absence of recovery from a sudden loss, several important questions regarding sudden sensorineural hearing loss remain unanswered. The cause of SHL remains obscure in most cases. The role and value of diagnostic testing is uncertain and, thus, the evaluation of patients presenting with SHL is not standardized. Perhaps most pertinent to its consideration as an emergency is this question: Is there any treatment that provides better patient outcomes than simply observing the natural course of the disease?

Etiology

The list of agents linked to the development of SHL ranges from snake venom to oral contraceptives [4]. Some of these agents may legitimately cause SHL, whereas others are likely the result of simple association. Box 1 presents a list of some causes associated with the development of SHL. Some conditions such as vestibular schwannoma are known to present with SHL and can be readily diagnosed with an appropriate evaluation. SHL in most patients, however, has no identifiable cause. A review of 837 patients diagnosed with SHL between 1989 and 1993 found that 88% were ultimately deemed idiopathic [2].

Although the list of potential etiologies is lengthy, the more substantial evidence seems to support that idiopathic SHL is most commonly the result of viral infection, vascular disruption, or an autoimmune processes. None of these etiologies has undisputed evidence supporting its role in the cause of SHL; therefore, these cases remain idiopathic. Reviews on the potential etiologies of SHL are readily available [4,19,20].

The concept that a viral infection can cause SHL seems reasonably well supported. Congenital infection with certain viral agents (cytomegalovirus, rubella, herpesvirus) is associated with hearing loss [19]. Viral labyrinthitis induced in animals can create a reversible SHL [21]. Epidemiologic evidence has linked a specific viral illness (Lassa fever) to SHL [22,23]. There is a strong reason to suspect that viral infections can affect the facial nerve and vestibular nerve, causing acute facial palsy or vestibular neuronitis. These entities bear striking similarity to SHL, in that they begin suddenly, often without warning, and spontaneous recovery of function is seen in a variable percentage of patients. A similar process could occur in the cochlear nerve, causing potentially reversible hearing loss.

Similar to the concept that viral infection may cause SHL, the concept of vascular disruption is also reasonably well supported. Ischemic events affecting the auditory pathway have been demonstrated in some patients who have SHL [24]. Certain prothrombotic risk factors and genes have been associated with SHL [25,26]. Plasmapheresis and other treatments designed to alter blood viscosity have been reported to help recovery [27].
Box 1. Causes associated with development of hearing loss

*Infectious causes*
Meningitis (streptococcal, meningococcal, cryptococcal)
Mumps
Rubeola
Rubella
Syphilis
Herpesvirus (simplex, zoster, varicella)
Lassa fever
HIV/AIDS
Mononucleosis
Mycoplasma
Toxoplasmosis
Cytomegalovirus

*Toxic causes*
Snake bite
Ototoxic agents

*Immunologic causes*
Wegener’s granulomatosis
Cogan’s syndrome
Primary immune inner ear disease

*Other causes*
Meniere’s disease
Hyperostosis cranialis interna
Pseudohypoacusis

*Neoplastic causes*
Acoustic neuroma
Meningioma
Lymphoma
Leukemia
Myeloma
Meningeal carcinomatosis

*Neurologic causes*
Multiple sclerosis
Neurosarcoidosis

*Circulatory causes*
Cerebrovascular accident
Sickle cell disease
Cardiopulmonary bypass
Vertebrobasilar insufficiency

*Traumatic causes*
Temporal bone fracture
Acoustic trauma
Barotrauma
Perilymph fistula
Otologic surgery
The idea that hearing loss may be the result of an autoimmune process is attributed to McCabe [28]. Subsequent studies have supported this premise, and specific autoantibodies have been identified and proposed to cause hearing loss [29]. Further, the association of SHL with known autoimmune diseases such as Cogan’s syndrome, Wegener’s granulomatosis, and temporal arteritis has been well documented [30].

All of these proposed etiologies seem reasonable; however, they all have significant flaws when applied broadly to all cases of SHL. After much discussion, many investigators might concede that SHL likely represents the common symptom of a number of different pathologies, but on the whole, it is not known what causes most cases of SHL.

Lastly, one should carefully consider cochlear membrane rupture as a cause of SHL. Practical clinical experience with temporal bone trauma, stapedectomy surgery, and other aspects of common otology has shown that damage to the internal membranes of the cochlea can result in hearing loss. Thus, in the appropriate clinical setting, accepting cochlear membrane rupture as an etiology for SHL seems appropriate. The concept of spontaneous cochlear membrane rupture is different. Unlike the notion that SHL may be caused by viral, vascular, or autoimmune causes, there seems to be little objective evidence to support the idea that a substantial percentage of idiopathic SHL is caused by a spontaneous cochlear membrane rupture.

Evaluation

Evaluation of patients who have SHL varies substantially among clinicians. Overtly, the goal of the evaluation should be to detect known causes of SHL. To this end, the evaluation should begin with history taking. The main elements of the history include the time of onset, progression or fluctuation since onset, and the presence or absence of any other noticed neurologic deficits. The presence of vestibular symptoms, tinnitus, or a feeling of aural fullness should be elicited and may suggest onset of Meniere’s disease. Diagnostic criteria for this disorder are readily available [31]. The presence of aural pain may be seen with infectious causes; however, a few patients report pain in the presence of a normal examination. In older reports, the history of an antecedent upper respiratory tract infection was often reported and thought to possibly correlate with a viral cause of SHL. Although this approach seems logical, an informal evaluation of this concept by Mattox and Simmons [3] found that roughly 25% of patients who had SHL reported an antecedent upper respiratory tract infection, which was very similar to a control population.

The patient should be questioned about trauma to the ear and about the presence of autoimmune diseases in the patient or family members. A history of sexually transmitted disease exposure should be reviewed in an attempt to screen for neurosyphilis. Exposure to ototoxic agents should be explored. Travel to exotic locations may suggest etiologies that are less often
encountered by the treating physician. A surprising number of patients report that the hearing loss was noticed immediately on awakening, suggesting that the hearing loss occurred during sleep; the significance of this is not known.

One particularly unresolved area of evaluation involves Lyme disease. Lyme disease has been associated with the development of SHL and with the development of other cranial neuropathies, including facial paralysis [32,33]. It is tempting to ask the patient about previous tick bites and possibly obtain Lyme titers in an attempt to evaluate the patient for zoonotic causes for SHL. Any such evaluation should be undertaken with caution, however, because a percentage of the asymptomatic population is positive for Lyme titers, and the incidence and prevalence of the disease can vary dramatically in different geographic areas. Beyond these diagnostic difficulties, the appropriate treatment for those who have SHL associated with Lyme disease is disputed. The input of an infectious disease specialist or neurologist who has a particular interest in Lyme disease may be appropriate in patients in whom this diagnosis seems pertinent. At present, there does not seem to be satisfactory evidence in the literature with which to resolve this issue.

Although not generally useful for diagnostic purposes, the duration of time from the onset of symptoms until presentation at the otolaryngologist’s office may be one of the most important factors in determining a patient’s prognosis. Most patients do not seek treatment immediately at the onset of symptoms, and the typical presentation is generally delayed 48 to 96 hours. As is discussed later, substantial evidence that treatment rendered by the otolaryngologist is primarily responsible for an improvement in outcome over the natural course of the disease is not in abundance. Thus, it has been suggested that a certain percentage of patients who develop symptoms have spontaneous improvement within a few days of onset and are less likely to present for evaluation. Patients whose symptoms last beyond a few days are more likely to present for evaluation, and these patients may be less likely to improve [3].

Many studies acknowledge that some degree of vertigo or imbalance is present in a percentage of patients who have SHL [2,3]. The criteria distinguishing SHL with vertigo from acute labyrinthitis are not formally defined. To a rough approximation, it seems that a patient who presents due to hearing loss and acknowledges a mild degree of imbalance is more likely to be diagnosed with SHL, and a patient who presents with a chief complaint of vertigo and is found to have hearing loss is more likely to be diagnosed with labyrinthitis. The use of objective tests of vestibular function to distinguish the two diagnoses does not appear to be commonplace.

The physical examination of the patient who has SHL should include a standard otolaryngologic examination, with specific attention to known causes of SHL. A neurotologic examination with otoscopic examination to evaluate for effusion, infection, and certain neoplasms is essential, along with thorough evaluation of the cranial nerves and cerebellar function. The nasal septum should be examined for evidence of autoimmune processes.
Following the history and physical examination, the audiometric evaluation should be reviewed. For the purposes of comparison, the audiometric evaluation should include PTA (0.25 kHz, 0.5 kHz, 1 kHz, 2 kHz, 4 kHz [or 3 kHz], and 8 kHz) and SDS to evaluate severity of initial loss and the degree of recovery.

In most instances, MRI of the brain and internal auditory canals enhanced with gadolinium contrast should be obtained in most patients to identify treatable, serious causes of sudden loss (eg, acoustic neuroma, cerebrovascular accident). For patients in whom MRI scanning is contraindicated, a contrasted CT scan of the head with thin cuts may be an appropriate substitute and should be able to identify larger lesions. Consultation with a neuroradiologist may allow such a CT scan to be optimized to detect smaller lesions.

Beyond the previously described measures, the evaluation of patients who have SHL is extremely variable from one clinician to another. Laboratory evaluations including complete blood count, basic or complete metabolic panels, electrolyte levels, and sedimentation rate are undoubtedly reasonable, although many clinicians no longer obtain these studies routinely because the diagnostic yield is low. Rheumatologic workup including sedimentation rate and antinuclear antibodies are indicated when the history is suggestive of autoimmune causes. Fluorescent treponemal antibody testing may be appropriate when suspicion for neurosyphilis is high, although some investigators have pointed out that the false-positive rate in an otologic population may be high [34]. Large panels of viral titers seem to have been largely abandoned, although selected titers may be appropriate based on the clinician’s impression. Laboratory testing of vestibular dysfunction is generally not necessary for patients who have SHL. Patients who have balance complaints significant enough to warrant objective testing may often be relegated to an alternate diagnosis.

Management

The management of SHL is perhaps the most controversial aspect of this entity. The medical literature contains seemingly innumerable reports touting various treatment agents and regimens. The use of more than one agent in treatment is exceedingly common, and the choice of agents used varies substantially among clinicians. In the United States, oral steroids remain the mainstay of treatment. Certain other treatments are commonly employed and enjoy relatively widespread support; specifically, transtympanic steroids perfusions and oral antiviral agents. The use of treatments such as carbogen gas inhalation, hyperbaric oxygen treatment, diuretics, plasma expanders, and agents designed to alter blood flow or viscosity is not unusual, although these agents are perhaps less commonly employed in the current environment. In regions outside the United States, other therapies enjoy much wider acceptance. For example, in central Europe, hypervolemic
hemodilution is one of the more commonly employed treatment regimens [35]. To date, no treatment agent or scheme is universally accepted, and no single agent has been irrefutably determined to improve or worsen patient outcomes beyond the natural history of the disease. A partial list of treatments advocated in the literature is presented in Box 2.

**Oral steroids**

In 1980, Wilson and colleagues [15] presented the results of a double-blind placebo-controlled trial evaluating the efficacy of steroids in treating SHL. Most saliently, the study found an improvement rate of 61% for those treated with steroids compared with 32% for those treated with placebo. This demonstration of efficacy has, at least in part, been responsible for the widespread implementation of oral steroids as treatment for SHL in the United States. A few important aspects of this study bear mentioning. First, in addition to the treatment and placebo groups, there was a third group of patients—untreated control subjects (presumably patients who did not receive placebo or steroids)—who showed a recovery rate almost identical to those treated with steroids (58%). Second, two clinical sites contributed patients to the study. The steroid regimens were not the same at the two clinical sites: patients at one site were treated with 10 days of dexamethasone starting at 4.5 mg twice daily, whereas those at the second site were treated with 12 days of methylprednisolone starting at 16 mg, three times daily.

Within recent years, several investigators have reported their own refinements to the treatment of SHL with oral steroids. Slattery and colleagues [36] presented a review from the House Ear Institute in 2006 that found that optimal results were achieved with a 14-day prednisone taper, starting at 60 mg orally daily. Others have advocated more aggressive regimens. Narozny and colleagues [37] presented a group of patients treated with 1000 mg methylprednisolone intravenously for 3 days combined with an oral prednisone taper starting at 60 mg daily and found that this treatment combined with other treatments produced improved outcomes compared with other regimens tried at their center.

Other studies fail to confirm the efficacy of oral steroids in treating SHL [38,39]. A randomized double-blind placebo-controlled trial presented in 2001 found that 60% of those who took oral steroids improved compared with 63% of those who received a placebo [38]. Further, the association of worse clinical outcomes with increasing steroid doses has been reported in a retrospective review of 250 patients [39]; thus, the concept that oral steroids are to be universally accepted as a benign, yet potentially helpful intervention, that can cause no morbidity, is challenged.

Several well-written reviews of the use of oral steroids in the treatment of SHL have been presented [40–42]. A 2006 Cochrane database review concluded that the value of steroids in the treatment of SHL remains unclear [40]. Conlin and Parnes [41,42] similarly concluded that no valid randomized
Box 2. Sensorineural hearing loss treatments

Oral steroids
- Prednisone
- Dexamethasone
- Methylprednisolone
- Betamethasone

Transtympanic steroids
- Dexamethasone
- Methylprednisolone

Intravenous steroids
- Methylprednisolone

Oral antivirals
- Acyclovir
- Valacyclovir

Hemodilution
- Dextran
- Hydroxyethyl starch

Vasodilators
- Histamine
- Papaverine
- Verapamil
- Procaine hydrochloride
- Cyclandelate

Carbogen gas inhalation

Hyperbaric oxygen therapy

Vitamins
- B₁
- B₃
- B₆
- B₁₂
- E

Diuretics
- Mannitol, others

Antibiotics
- Magnesium
- Betahistine
- Pentoxifylline
- Vinpocetine

Thrombolytics
- Tissue plasminogen activator
- Batroxibin

Anticoagulants
controlled trial exists to determine effective treatment of SHL, and that systemic steroid use cannot be considered the gold standard of treatment for SHL. Despite these systematic reviews, clinicians practicing in the United States are cautioned to consider offering steroids to those who present with SHL because certain practitioners and publications may consider this treatment to represent a gold standard in treatment.

**Transtympanic steroids**

Transtympanic steroid injection (also called intratympanic steroid perfusion) was applied to the treatment of Meniere’s disease in 1991 [43]. Based on the acceptance of systemic steroid therapy for treatment of SHL, the use transtympanic steroids for the treatment of SHL was proposed by Silverstein and colleagues [44] in 1996. Animal studies conducted by Parnes and colleagues [45] and Chandrasekhar [46] demonstrated that transtympanic steroid perfusion results in dramatically higher concentrations of steroids in the labyrinth. Based on these studies and on favorable initial clinical evaluations, clinicians have embraced transtympanic steroid perfusion as a treatment modality. The advantages and disadvantages of transtympanic steroid administration compared with systemic steroid administration are presented in Box 3.

The use of intratympanic steroids has evolved into three main protocols for treatment of sudden SHL: (1) initial or primary treatment for sudden SHL without systemic steroids, (2) adjunctive treatment given concomitantly
with systemic steroids for sudden SHL, and (3) “salvage therapy” after failure of systemic steroids for sudden SHL.

Beyond these three general protocols, there is substantial diversity among clinicians with regard to the type and amount of steroid used and the timing and frequency of administration.

A number of investigators have presented series of patients who have SHL treated with transtympanic steroids [18,45–50]. Most of these studies are retrospective reviews of clinical experience and none have definitively established the efficacy of transtympanic steroids as superior to the natural rate of recovery. A large multicenter trial is underway to better resolve this issue, and results are expected within the next few years.

**Oral antiviral agents**

The use of antiviral agents to treat SHL is relatively widespread. Contemporary literature suggests that most practitioners are using oral antiviral agents; however, the use of intravenous agents, including interferon, has been reported [51]. Oral antiviral agents have been described as potentially beneficial [52]. Two randomized double-blind placebo-controlled trials have been conducted, one evaluating valacyclovir [53] and the other evaluating acyclovir [54]. Both studies failed to show significant improvement with the use of an oral antiviral agent. Despite these negative trials, oral antiviral agents remain commonly employed in the treatment of SHL, and not without reason. Animal studies have shown that animals that have

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**Box 3. Transtympanic steroids**

**Advantages**
- Assured compliance
- May be suitable for patients in whom systemic steroids are contraindicated or declined
- Therapy directed to the affected ear
- Higher concentration of steroids in the ear
- Few side effects, complications
- Office-based procedure, accomplished without general anesthetic
- Well tolerated

**Disadvantages/complications**
- Pain
- Tympanic membrane perforation
- Otitis media
- Vertigo (usually temporary)
- Hearing loss
herpes simplex virus labyrinthitis treated with acyclovir combined with prednisolone suffer less cochlear damage than animals treated with either agent alone [21]. If one assumes that a small minority of patients suffer SHL due to a viral infection caused by a virus susceptible to acyclovir or valacyclovir, and that treatment of patients with these agents results in a percentage of patients having an improvement that is better than the natural course of disease, then the number of patients required to conduct a study with sufficient power to resolve the treatment effect is high. One trial was designed to detect a 30% improvement in hearing with 90% power. The investigators concluded that 127 patients would be required for the study. Despite recruiting 45 clinical sites for the study and conducting the study over a 32-month period, the number of patients enrolled fell short of the goal. It seems reasonable to postulate that oral antiviral agents may have a beneficial effect that is difficult to prove experimentally without a large sample size. As an analogy, consider that the use of oral antiviral agents in the treatment of Bell’s palsy was not supported in initial randomized placebo-controlled trials but was recently supported in a trial involving a large number of patients [55]. Generally, for healthy patients, a short course of oral antiviral agents presents minimal risk and potential benefit, although the benefits have yet to be proved in clinical trials.

Hyperbaric oxygen

The use of hyperbaric oxygen therapy in the treatment of SHL dates back to at least 1979 [56]. Although little appears in the literature for much of the 1980s and early 1990s, there seems to be renewed interest in this treatment modality, with several reports in the past 10 years. Most of this literature comes from centers in Europe [37,57,58], with relatively few studies from the United States [59], suggesting a geographic difference in the application of this treatment. From a Cochrane database review, it was concluded that in certain patients, the application of hyperbaric oxygen therapy significantly improved hearing loss, but the clinical significance of the level of improvement is not clear [60]. In their review of the literature, Conlin and Parnes [41,42] concluded that no valid randomized controlled trial exists to determine effective treatment of SHL, including hyperbaric oxygen treatment.

Practical management

The lack of suitable scientific data to base treatment regimens on leaves the clinician in an undesirable position when faced with a patient who has SHL. In most instances, the treatment is patient specific, with the goal of maximizing benefit and minimizing risk: more aggressive treatment is offered to those who have more severe losses, those whose hearing loss may present substantial lifestyle impact or employment difficulties (e.g,
professional musicians), and those who do not have other medical conditions that might be worsened by potential treatment agents (eg, normal renal and hepatic function and the absence of diabetes). In 1996, Hughes and colleagues [4] provided a respected and useful review of this topic, which concluded by suggesting how the senior investigator might treat himself if he were to have SHL. The current authors present here their current protocol for treatment of SHL and how the senior author might treat himself were he to suffer SHL: the evaluation would include a history and physical examination as described earlier, contrast-enhanced MRI, and complete audiometry. Treatment would begin with a prednisone oral taper, starting at 60 mg daily, tapered over 2 weeks, along with concurrent administration of an oral antiviral agent, given for 1 week. If no objective improvement had been obtained after completion of medical therapy, then a transtympanic dexamethasone perfusion would be offered as salvage therapy. If presentation for treatment had been delayed, oral steroid therapy would potentially be offered up to 6 weeks from the onset of symptoms. Transtympanic steroids could also potentially be employed up to 6 weeks from the onset of symptoms. Although 6 weeks may seem late, it is not uncommon to have a patient present to the clinic in this time frame without having received systemic or transtympanic steroids. Although the authors would treat a previously untreated patient for up to 6 weeks, patients who receive a short or inadequate course of steroids early in the course of the disease are generally not re-treated with a more standard course of steroids when seen after 2 to 3 weeks. Carbogen inhalation, vasodilators, and other previously described therapies are not currently offered.

Summary

SHL remains one of the more interesting and disputed topics in otolaryngology. It is a topic of seemingly boundless discussion and active research. In most cases, SHL is the presenting symptom of an emergency for which we have yet to find a convincing cause or reliable treatment. Treatment varies dramatically among practitioners and regions; however, in the United States, many practitioners generally consider offering a course of oral steroids for patients in whom no other contradictory medical condition exists. The use of other treatments has been well described and should be carefully considered. Ongoing large-scale research projects may soon provide a greater understanding of how to more optimally manage SHL.

References


