Sudden sensorineural hearing loss

An urgent otological presentation

KIMBERLEY BRADSHAW MB BS, MS
MELVILLE DA CRUZ FRACS, MSc, MD

Sudden sensorineural hearing loss (SSNHL) is an otological condition that requires urgent assessment and treatment in consultation with an ENT surgeon. A structured approach to the assessment of patients presenting with sudden hearing loss can help differentiate between SSNHL and other causes of hearing loss.

KEY POINTS

- Sudden sensorineural hearing loss can be a debilitating condition; however, overall it has a good prognosis.
- History, examination, tuning fork tests and pure tone audiometry can help differentiate between sensorineural and other causes of hearing loss.
- Quality evidence for treatment regimens for sudden sensorineural hearing loss is limited, but the best recommended treatment is timely oral or intratympanic steroids.
- Early referral to an ENT specialist is important.

A sudden and dramatic drop in hearing is a reasonably common presentation to public hospital emergency departments, GP practices and ENT departments. It can be a frightening symptom for patients and requires urgent assessment when caused by sudden inner ear or sensorineural loss. This article focuses on sudden sensorineural hearing loss (SSNHL), including recognising symptoms to distinguish SSNHL from other forms of sudden onset hearing loss and the options for treatment. Management of SSNHL is controversial because the evidence for some treatments is limited and mild SSNHL has a high spontaneous recovery rate. Although no treatment is proven, there are guidelines for initial management. There is much still to learn about SSNHL and more clinical trials are needed to establish evidence-based management.

Mechanism of hearing

Normal hearing is dependent on an intact and functioning auditory pathway. The anatomy of the ear can be divided into the outer, middle and inner ear. The outer ear is composed of the pinna and external ear canal, which capture and focus the sound towards the tympanic membrane. Sound energy then vibrates the tympanic

MedicineToday 2022; 23(8): 23-33

Dr Bradshaw is a PhD candidate at The University of Sydney, Sydney; and an Ear, Nose and Throat Registrar at the Westmead Hospital, Westmead. Associate Professor Da Cruz is an Ear, Nose and Throat Surgeon at Westmead Public and Private Hospitals; and Associate Professor of Surgery at The University of Sydney, Sydney, NSW.

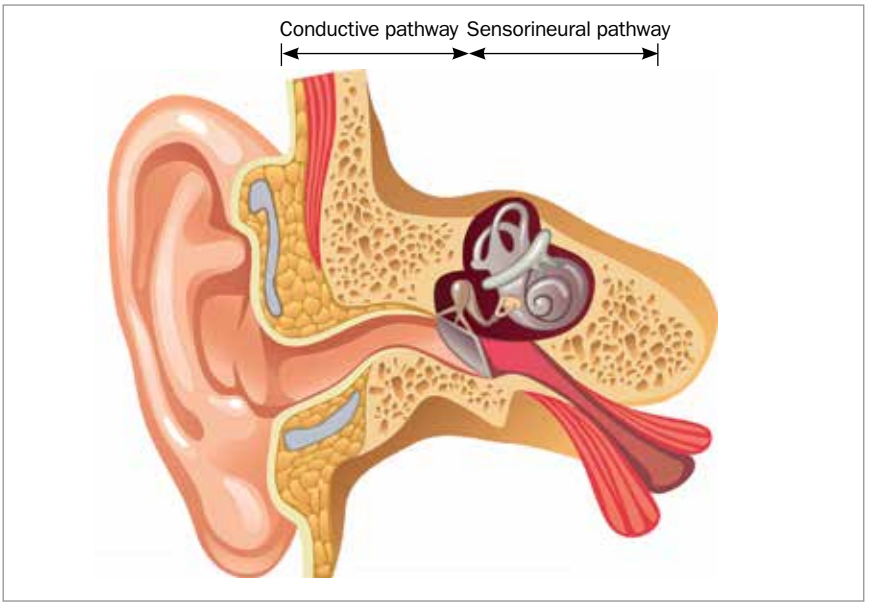


Figure 1. Causes of hearing loss may involve either the conductive pathway (external ear, tympanic membrane, hearing bones) or the sensorineural pathway (cochlea, hair cells, auditory nerves).

membrane, which transmits this energy to the middle ear space and hearing bones (ossicles). The ossicles connect via the stapes to the round window of the cochlear (inner ear), where this energy is transmitted to the fluid within the cochlear, and the hair cells are activated by the movement of fluid. Action potentials are created by hair cell movement in the adjacent auditory nerve fibres, producing electrical signals that travel to the auditory processing centres of the cerebral cortex. Thus, sound energy is converted into electrical energy via the conductive and sensorineural pathways of hearing. Disturbance of these pathways leads to hearing loss (Figure 1).

What is SSNHL?

Sudden hearing loss is a rapid onset of subjective impairment of hearing in one or both ears. This can be conductive, sensorineural, or mixed in nature, with a wide differential diagnosis. Physical examination, tuning fork testing and pure tone audiometry can help to distinguish SSNHL from other differential diagnoses.

SSNHL has been defined as hearing loss that is sensorineural in nature of at least 30 dB over at least three consecutive frequencies within a 72-hour window. The definition assumes premorbid hearing in the affected ear is similar or identical to hearing in the unaffected ear. SSNHL is considered idiopathic when no identifiable cause for hearing loss can be identified, and is the case in more than 90% of presentations.

Presentation

History and examination are important in the diagnosis of SSNHL to differentiate sensorineural hearing loss from conductive hearing loss and to ascertain which ear is affected.

Patients with idiopathic SSNHL usually experience immediate or rapid hearing loss, often reported upon awakening. Hearing loss can involve any frequency range and severity can be mild, moderate or severe to profound. Other common associated symptoms include tinnitus, vertigo or dizziness and, less often otalgia and paraesthesia. However, associated symptoms are nonspecific and may not differentiate between conductive and sensorineural hearing loss.

Many patients experience a sensation of a blocked ear or aural fullness initially and do not attribute this to hearing loss, given it is common for wax impaction, otitis media or eustachian tube dysfunction to cause a similar sensation. For this reason, patients may not seek attention immediately or can be inappropriately reassured without investigation and treatment.

Most cases of SSNHL are unilateral with an equal distribution of right- and left-sided hearing loss. Bilateral SSNHL is rare (3%). However, an underlying cause is far more likely to be identified in bilateral cases, such as systemic autoimmune disorders or hypercoagulation disorders. There should be a high suspicion for SSNHL if a patient with a history of unilateral SSNHL presents with similar symptoms on the contralateral side.

Natural history

Spontaneous recovery occurs in 30 to 60% of patients with idiopathic SSNHL, generally within the first few weeks. If symptoms persist longer than three months, spontaneous recovery is unlikely.

Epidemiology

The incidence of SSNHL is estimated to be between five and 20 cases per 100,000 per year.[2] However, estimates vary due to spontaneous recovery and failure to seek medical attention. Idiopathic SSNHL can occur at any age but most commonly affects individuals in their 40s to 60s. Incidence varies from 11 per 100,000 in those younger than 18 years to 77 per 100,000 for people 65 years and older.[3,4] In general, similar numbers of men and women are affected.[5]

Risk factors

Most reported risk factors for SSNHL are related to the cardiovascular system, as the inner ear may be vulnerable to cardiovascular insults, as well as hypercholesterolaemia, smoking, increased alcohol consumption and low serum folate, but no definitive associations have been made.

Other risk factors include mutations in factor V Leiden and the methylenetetrahydrofolate reductase (MTHFR) genes, hyperhomocysteinaemia, elevated factor VIII (essential blood-clotting protein) and deficiencies of antithrombin and protein C and S, but screening for these is not cost effective.

Pathophysiology

The complete pathophysiology of SSNHL remains unknown and is likely to be multifactorial. The following theories have been proposed.[6]

The labyrinthine viral infection theory

Although the evidence that implicates viral infection in SSNHL is circumstantial, proposed mechanisms include:

- direct viral invasion of the fluid spaces of the cochlea (cochleitis)
- direct invasion of the cochlear nerve (neuritis)
- reactivation of a virus that is latent within the tissues of the inner ear.

Viruses that have been implicated include mumps, measles, rubella, herpes and varicella zoster. However, there are significant challenges in investigating the viral theory and such cases likely only account for a small fraction of idiopathic SSNHL.

The labyrinthine vascular compromise theory

Vascular occlusion, or ischaemia, is a possible mechanism for SSNHL because the cochlea is an end organ with respect to its blood supply. The labyrinthine artery branches into a cochlear and vestibular artery, both end vessels, which makes them more susceptible to thromboembolic events, reduced blood flow or vasospasm.

The intracochlear membrane rupture theory

Rupture of the thin membranes that separate the middle ear from the inner ear at the oval and round windows, as well as inside the cochlea between the perilymphatic and endolymphatic spaces, could theoretically produce hearing loss. Mixing of the the perilymph and endolymph may alter electrolyte homeostasis, endocochlear potential and hair cell function.

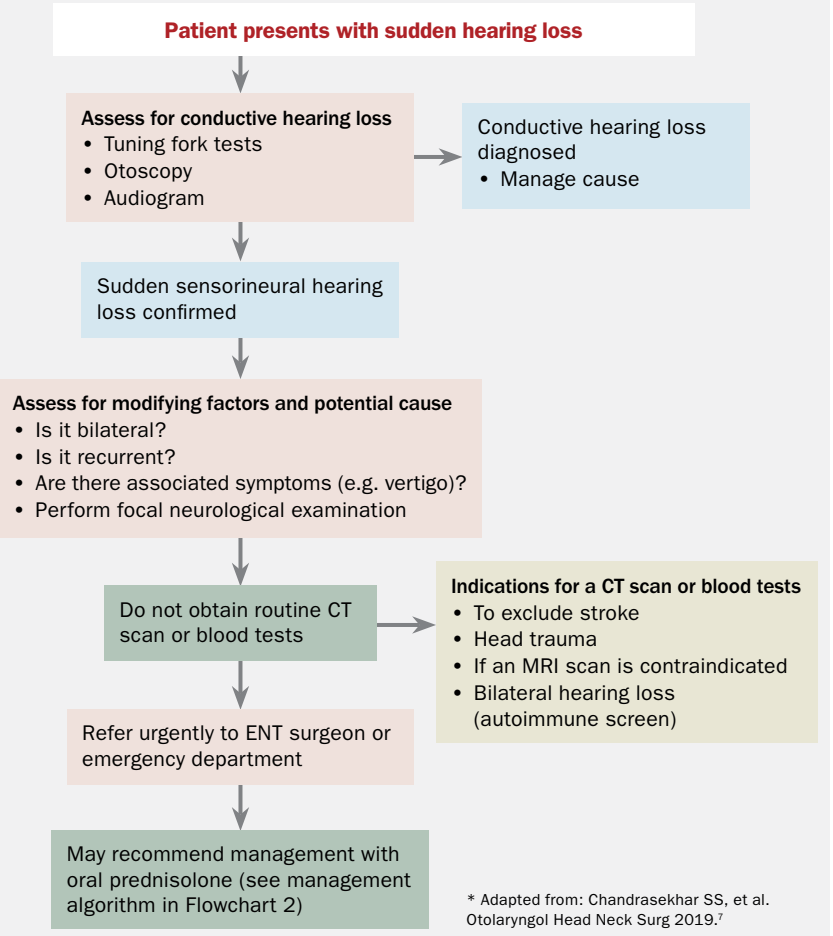
When the membranes repair and recover spontaneously, and if hair cells are still viable, hearing loss may be partial or fluctuating, which makes this an attractive theory, fitting with the clinical presentation.

The immune-mediated inner ear disease theory

Immune-mediated inner-ear disorders, including both organ-specific inner ear disease (Cogan's syndrome) and systemic autoimmune disorders, may be a cause for SSNHL.

A distant viral infection may act as a trigger for an autoimmune response, which produces antibodies that cross-react with an inner ear antigen, activating cellular stress pathways within the cochlea.

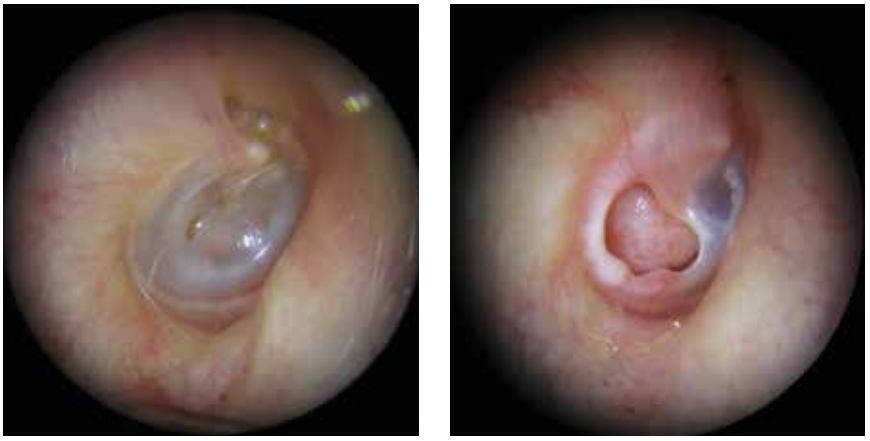
1. SUGGESTED ASSESSMENT OF PATIENTS WITH SUDDEN HEARING LOSS IN GENERAL PRACTICE*



* Adapted from: Chandrasekhar SS, et al. Otolaryngol Head Neck Surg 2019.[7]

Diagnosis

The diagnosis of SSNHL can be made based on findings from the patient's history, otoscopy, tuning fork tests (Weber and Rinne) and pure tone audiometry, followed by imaging. It is important to exclude causes of conductive hearing loss,



Figures 2a and b. a (left). Middle ear effusion associated with otitis media. b (right). A central perforation of the ear drum due to repeated infections. Both commonly produce a conductive hearing loss.

which may present similarly with reduced hearing or a blocked ear. A suggested approach to the assessment of sudden hearing loss in general practice is outlined in Flowchart 1.

Otoscopy

Otoscopic examination of the external ear canal and tympanic membrane can diagnose or exclude many causes of conductive hearing loss. A patent ear canal with no occluding mass excludes wax impaction, foreign bodies, tumours, exostoses and stenosis. Visualisation of a healthy intact drum that is mobile on pneumatic insufflation excludes cholesteatoma, otitis media and middle ear effusions (Figure 2). In patients with SSNHL, the otoscopic examination is generally normal.

Tuning fork tests

The Weber and Rinne tests distinguish between sensorineural and conductive hearing loss (Figure 3). They are performed with a 512 Hz tuning fork and compare symmetry between the two ears (Weber), as well as the efficiency of hearing via air conduction and bone conduction (Rinne). The technique for how to perform a tuning fork test is important and will improve with practice. Poor technique can lead to a false result.[8]

3. USING TUNING FORK TESTS TO EVALUATE HEARING LOSS

The Weber and Rinne tests are the tests most commonly used in the office setting to assess hearing loss. Results from the two tests can be used to determine the presence and severity of hearing loss.

The Weber test

The Weber test is used to determine whether a patient has symmetrical hearing. It is performed using a 512 Hz tuning fork. Place the vibrating tuning fork in firm contact with the midline of the forehead and then on the vertex of the skull (Figure 3a). A patient who has:

- normal hearing – will hear the sound equally in both ears, 'in the middle' or 'all over'
- a conductive hearing loss – will refer the sound to the affected ear
- a sensorineural hearing loss – will refer the sound to the unaffected ear.

Weber test

Figure 3a. To test the symmetry of hearing between the two ears, the tuning fork is placed firmly on the forehead and then on the vertex of the skull.

The Rinne test

The Rinne test is used to compare air and bone conduction. The 512 Hz tuning fork is the most useful fork to use. Bone conduction is assessed first.

Place the fork firmly over the mastoid bone (Figure 3b). Tell the patient to listen to the sound, not to the vibration. When the patient claims not to hear the sound any longer, assess air conduction by quickly moving the fork and placing it 2 to 3 cm from the meatus of the external canal, with the tines of the fork parallel to the plane of the canal (Figure 3c).

If air conduction is heard longer than bone conduction (i.e. a Rinne positive result), the patient has either normal hearing or a sensorineural hearing loss. If bone conduction lasts longer than air conduction (i.e. a Rinne negative result), a conductive hearing loss is present.

Rinne test for bone and air conduction

Figure 3b. To assess bone conduction, the tuning fork is placed firmly over the mastoid bone.

Figure 3c. To assess air conduction, the fork is then placed 2 to 3 cm from the external canal, with the tines of the fork parallel to the plane of the canal.

Interpreting the tests

Results from the Weber and Rinne tests can be used to distinguish between conductive and sensorineural types of hearing loss. This is described in Figures 3d to f.

Combining results from the Weber and Rinne tests

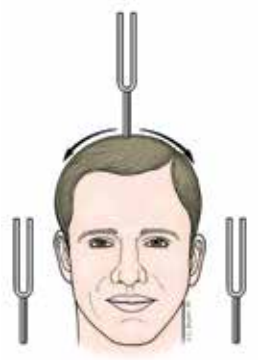


Figure 3d. Symmetrical Weber test and positive Rinne test in each ear.

Hearing referred equally to each ear in the Weber test with positive Rinne results in both ears indicates symmetrical hearing with normal middle ear function.

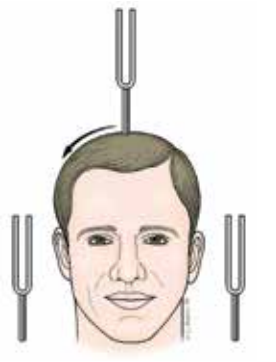


Figure 3e. Asymmetrical Weber test and unequal Rinne tests. The patient complains of right-sided hearing loss.

Hearing is referred to the right ear in the Weber test, with the Rinne test negative for the right ear and positive for the left ear, indicating conductive deafness in the right ear.

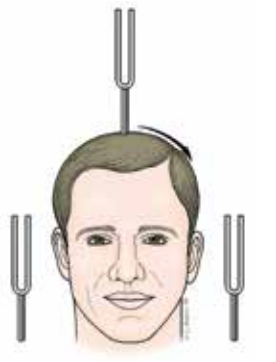


Figure 3f. Asymmetrical Weber test and positive Rinne test in each ear. The patient complains of right-sided hearing loss.

Hearing is referred to the left ear in the Weber test with a positive Rinne result in each ear, indicating sensorineural deafness in the right ear.

Adapted with permission from: Atlas MD, Lowinger DSG. Office evaluation of hearing loss in adults. Med Today 2000; 1(6): 117-119.

Figure 3. Tuning fork tests: an important part of the assessment of sudden hearing loss.[8]

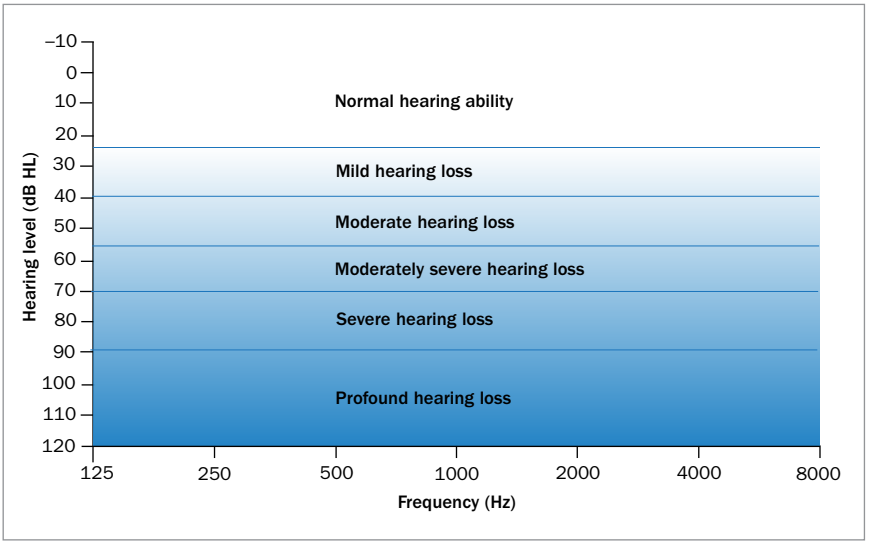


Figure 4. Severity of hearing loss on audiogram.

Audiometry

A pure tone audiogram and tympanometry are essential in the diagnosis of SSNHL. Air conduction thresholds and bone conduction thresholds recorded under calibrated conditions are needed to distinguish between sensorineural and conductive hearing loss. Air conduction thresholds are obtained using headphones and bone conduction by directly stimulating the cochlea with a vibratory stimulator on the mastoid. They are charted on an audiogram using symbols, measuring intensity (loudness) of sound heard in decibels against the frequency (pitch) of the sound (250 to 8000 Hz) (Figure 4).

A basic understanding of how to interpret an audiogram is important. Figure 4 shows ranges of hearing loss severity on an audiogram chart. An audiogram can determine if the patient has normal hearing, sensorineural, conductive or a mixed loss and whether it is symmetrical or asymmetrical (Table 1, Figure 5).

More complex tests of inner ear function and the central auditory pathways, such as otoacoustic emissions and auditory evoked brainstem potentials, require specialist interpretation.

Otoacoustic emissions

Otoacoustic emissions (OAEs) are relevant in the evaluation of sensorineural hearing loss. They are sounds given off by part of the cochlea, either spontaneously or when it is stimulated by soft clicking sounds (distortion product evoked OAEs). Present distortion product evoked OAEs indicate normal functioning of the outer hair cells. Absence of OAEs indicates a problem with the sensorineural pathway. The presence of OAEs in measurements early in idiopathic SSNHL shows a good prognosis for hearing recovery.[9]

TABLE 1. TYPES OF HEARING LOSS ON AUDIOGRAM

Type of hearing loss	Audiogram findings
Normal hearing	Air conduction and bone conduction thresholds between 0 and 25 dB
Sensorineural hearing loss	Air conduction and bone conduction thresholds are both equally reduced to >25 dB
Conductive hearing loss	Bone conduction thresholds are normal; air conduction thresholds are reduced with an air-bone gap of at least 10 dB
Mixed hearing loss	Both bone conduction and air conduction thresholds are reduced, but there is still an air-bone gap

Speech audiometry

Speech audiometry is important to assess functional deficit and determine which forms of auditory rehabilitation will be beneficial.

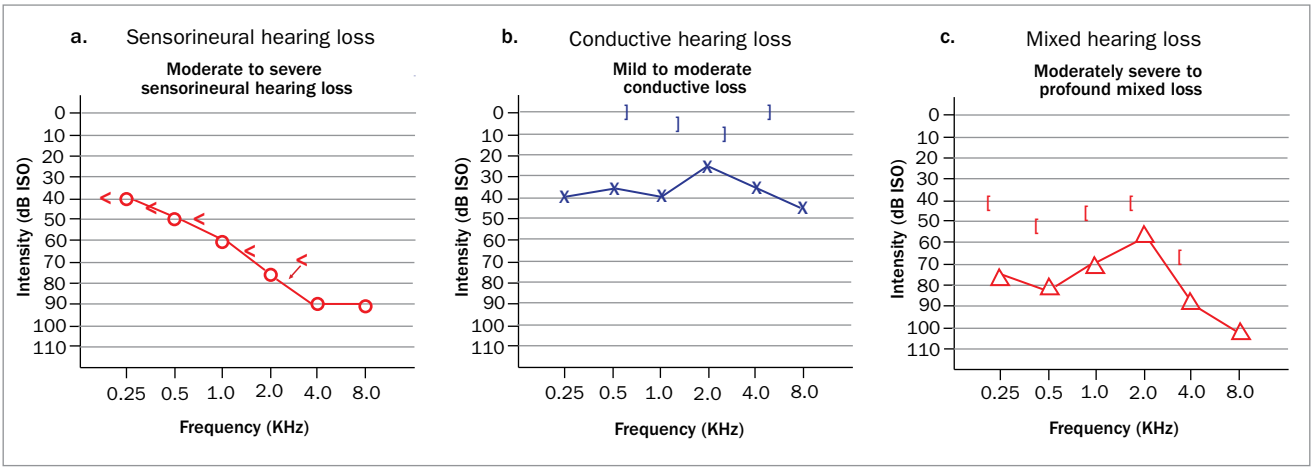
Imaging

MRI of the petrous temporal bone with contrast is the most important imaging study in sensorineural hearing loss. It is indicated in all patients with neuro-otological symptoms, particularly in patients with SSNHL that is unilateral or idiopathic. Special attention is paid to the inner ear, looking for potential causes of SSNHL, such as an acoustic neuroma.

CT scans are not recommended routinely for SSNHL. A CT scan may be indicated if there are focal neurological findings, a history of trauma or chronic ear disease or in patients in whom MRI is contraindicated (e.g. patients with pacemakers and cochlear implants).

Blood tests

Commonly performed blood tests include full blood count, coagulation studies, serum electrolytes, erythrocyte sedimentation rate, autoimmune screening, metabolic panel and thyroid function tests. However, the yield is generally low and blood tests are not recommended by the 2019 American Academy of Otolaryngology – Head and Neck Surgery Foundation *Clinical Practice Guideline: Sudden Hearing Loss (Update)* unless there is suggestion of an underlying systemic illness.[7]



Figures 5a to c. Types of hearing loss on audiogram. (a) left. Sensorineural hearing loss (right ear) shows a reduction in both air conduction (O) and bone conduction (<) thresholds. (b) middle. Conductive hearing loss (left ear) shows a gap between air conduction (X) and bone conduction (]). (c) right. Mixed hearing loss (right ear) is a combination of both sensorineural and conductive hearing loss, with reduced air conduction (Δ) and bone conduction ([).

Differential diagnosis

There is a large differential diagnosis for the symptom of acute unilateral sensorineural hearing loss (Box 1). Sudden hearing loss should only be termed idiopathic when no other cause can be identified or considered probable. Most of the causes listed in Box 1 can be excluded using a simple battery of tests that are available in general practice.

Management of idiopathic SSNHL

Patients with idiopathic SSNHL should be educated about the natural history of the condition and the risks and efficacy of available treatments to facilitate a joint decision-making process between the clinician and the patient. A suggested approach to management is outlined in Flowchart 2.

1. DIFFERENTIAL DIAGNOSES OF SUDDEN SENSORINEURAL HEARING LOSS[10]

Tumours
- Vestibular schwannoma (acoustic neuroma)
- Metastases
- Tumours of the central nervous system and petrous temporal bone

Disorders of the inner ear
- Cochlear hydrops
- Labyrinthine fistula
- Cholesteatoma
- Temporal bone fracture
- Ménière's disease

Inflammatory and infectious causes
- Viral infection (e.g. herpes zoster, herpes simplex, influenza, EBV, measles, mumps, CMV, coxsackie, HIV)
- Bacterial infection (e.g. borreliosis, syphilis)
- Labyrinthitis

Cardiovascular diseases
- Hypertensive crisis
- AICA infarction

Immunological conditions
- Autoimmune disease of the inner ear
- Cogan's syndrome
- Antiphospholipid syndrome
- Multiple sclerosis
- Systemic lupus erythematosus
- Wegener's granulomatosis

Anatomical malformations
- Large vestibular aqueduct syndrome

Haematological conditions
- Leukaemia
- Aplastic anaemia or sickle cell anaemia
- Waldenstrom macroglobulinaemia

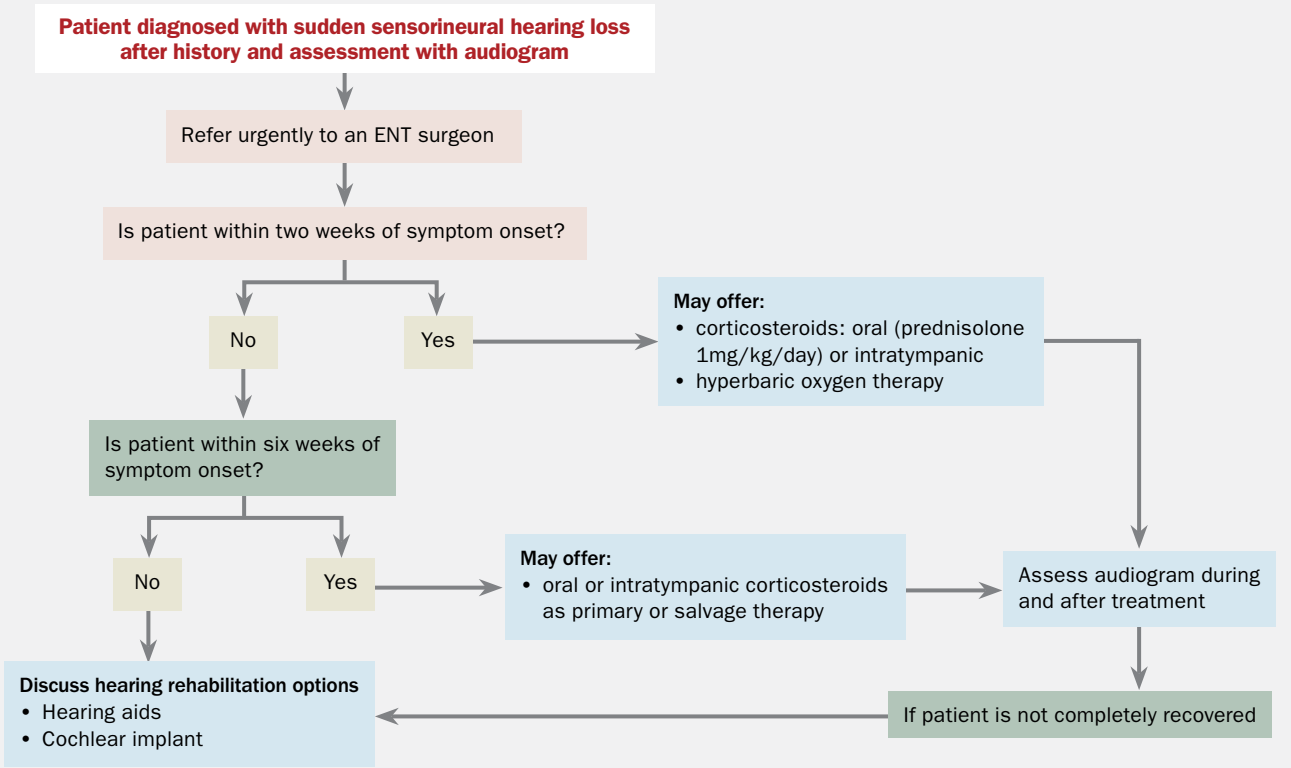
Abbreviations: AICA = anterior inferior cerebellar artery; CMV = cytomegalovirus; EBV = Epstein Barr virus; HIV = human immunodeficiency virus.

Systemic corticosteroids

Corticosteroids are currently the most widely accepted first-line therapy for SSNHL. However, the evidence-base for efficacy of corticosteroids remains unclear. The 2019 *Clinical Practice Guideline: Sudden Hearing Loss (Update)* recommends that the clinician may offer oral corticosteroids as a first-line therapy within two weeks of symptom onset after discussing the limited documented evidence for their efficacy, although corticosteroid therapy should be considered up to six weeks following onset of symptoms.[7] Options for systemic corticosteroids include oral prednisolone or oral or intravenous methylprednisolone or dexamethasone.

The dose of corticosteroids, the effect of timings and method of systemic administration remain contentious. Intravenous administration of corticosteroids has not been shown to be any more efficacious than oral prednisolone. Dosage and

2. SUGGESTED MANAGEMENT OF PATIENTS WITH SUDDEN SENSORINEURAL HEARING LOSS*



* Adapted from: Chandrasekhar SS, et al. Otolaryngol Head Neck Surg 2019.[7]

duration recommendations can be found in Table 2. Generally, a high dose (e.g. prednisolone 1mg/kg/day) is used for seven to 14 days, followed by a weaning course over a similar timeframe.

There are numerous side effects of systemic corticosteroid administration that need to be weighed with the potential benefits as they can occur even in short-term use (Box 2). Given deafness can be an extremely debilitating condition, however, if there is even a small possibility of restoring or improving hearing then it is a reasonable treatment to offer. As such, this has become standard practice. However, there is a large role for shared decision making with patients.

When systemic corticosteroids are contraindicated, such as in pregnancy, severe psychiatric illness or extreme hypertension, an ENT surgeon should be consulted for consideration of intratympanic corticosteroids as a first-line treatment (Table 2).

Intratympanic corticosteroids

Intratympanic corticosteroids are administered either by direct injection through the tympanic membrane or insertion of a tympanostomy tube (grommet) for repeated administrations. The main indications for intratympanic corticosteroid injections are: salvage therapy after systemic corticosteroids or when systemic corticosteroids are contraindicated.

Intratympanic corticosteroid administration has several advantages over systemic corticosteroids, including targeted drug delivery resulting in possibly higher inner ear concentration, minimal systemic absorption or side effects and that they are a good option in patients in whom systemic corticosteroids are contraindicated (e.g. pregnancy, extreme hypertension, diabetes or psychiatric illness).[11-13]

Other therapies

Hyperbaric oxygen therapy (HBOT) has been proposed based on the theory of vascular compromise as the aetiology for SSNHL, with the purpose of increasing the oxygen supply to the possibly ischaemic cochlear structure. The 2019 *Clinical Practice Guideline: Sudden Hearing Loss (Update)* states that HBOT could be used as an optional therapy within two weeks of onset of symptoms or as salvage therapy in combination with corticosteroids therapy within one month of onset of SSNHL.[7]

The potential limited benefits of HBOT need to be weighed against the costs,

TABLE 2. MANAGEMENT OF SUDDEN SENSORINEURAL HEARING LOSS WITH CORTICOSTEROIDS

	Systemic corticosteroids	Intratympanic corticosteroids
Commencement	• Can be initiated by a GP or emergency department physician in consultation with an ENT surgeon	• Requires an ENT surgeon to perform the injection
Timing of treatment	• Immediate • Ideally within 14 days of symptom onset • Offer up to six weeks after symptom onset	• Immediate • Salvage therapy after two weeks of oral corticosteroids without improvement
Dose†	• Prednisolone 1 mg/kg/day (maximum 60 mg) • Alternatives: – methylprednisolone 48 mg/day, or – dexamethasone 10 mg/day	• 0.4 to 0.8 mL into middle ear space; up to four injections over a two- to four-week period • Various preparations available: – methylprednisolone 4 mg/mL, 8 mg/mL, 10 mg/mL, 30 mg/mL, 40 mg/mL – dexamethasone 24 mg/mL, 10 mg/mL, 4 mg/mL
Duration	• Full dose for seven to 14 days, then weaning dose over a similar time period	
Monitoring	• Audiogram at presentation, at completion, and at intervals up to six months after treatment	• Audiogram at presentation, one week apart during the course of injections, at completion and six months after treatment
Adverse effects	• Discuss adverse effects prior to commencing treatment, including: – insomnia – hyperglycaemia – hypertension – avascular necrosis of the hip	• Less side effects than systemic treatment • Side effects include: – local pain – transient vertigo – a small risk of otitis media – tympanic membrane perforation (if ventilation tube is used)

* Adapted from: Chandrasekhar SS, et al. Otolaryngol Head Neck Surg 2019.[7]
† No dose or preparation has shown increased benefit over another. The choice is based on clinician preference and experience as well as local policy. Consult an ENT surgeon for local guidelines.

availability and side effects, including barotrauma, oxygen toxicity, worsening of cataracts, fatigue and seizures. It is also costly, time consuming and usually requires travel on the patient's part. Good patient selection is important if considering this therapy.

A number of other therapies have been proposed. Antiviral medications have been trialled based on a possible aetiological role of subclinical viral labyrinthitis. However, antiviral medications in combination with oral corticosteroids have not been shown to give any additional outcome benefit and so are not routinely recommended. If sudden hearing loss is accompanied by a facial nerve palsy, such as in Ramsay Hunt Syndrome, then antiviral medications are indicated. Other therapies that do not have a significant evidence-base include volume expanders, vasodilators, carbogen therapy, calcium channel blockers, pentoxifylline, prostaglandin E1, thrombolytics, antioxidants, histamines, magnesium, diuretics and other herbal remedies.

Auditory rehabilitation

An audiogram should be obtained three to six months after the onset of symptoms to quantify the extent of permanent hearing loss. There is unlikely to be any improvement in hearing after this time and it is reasonable to assume this will be the patient's long-term level of hearing.

Patients with permanent hearing loss should be referred to an audiologist for fitting of a traditional in-ear hearing aid or Bilateral Contralateral Routing of Signals (BiCROS) hearing aid, which transmits the sound from the affected ear to the unaffected ear and is for people with single-sided deafness. The level of functional impairment depends on several factors, including the severity of hearing loss, the frequencies affected, whether the hearing in the contralateral ear is normal or whether the affected ear was the only hearing ear, and the patient's age, occupation and social circumstances. Tinnitus and vertigo may also be bothersome symptoms that accompany hearing loss. Some audiologists can provide specialised tinnitus training and counselling.[6]

For patients with profound hearing loss that cannot be aided, a cochlear implant may be considered. A cochlear implant is an electronic prosthesis that works by bypassing the residual damaged hearing elements within the organ of Corti and directly stimulating the intact nerve endings in the auditory nerve.[14]

2. SIDE EFFECTS OF SYSTEMIC CORTICOSTEROIDS

- Suppressed hypothalamic-pituitary-adrenal axis function
- Avascular necrosis of the hip
- Hyperglycaemia and poor glycaemic control in people with diabetes
- Psychological disturbance (e.g. altered mood)
- Hypertension from sodium and fluid retention
- Peptic ulcer and gastrointestinal bleeding
- Increased intraocular pressure
- Impaired wound healing
- Increased risk of infections

Prognostic factors

The overall prognosis of SSNHL is good; about two thirds of patients recover some degree of hearing, although not necessarily back to normal or the same as the unaffected side. Most patients with idiopathic SSNHL will recover within three months, with 30 to 60% of patients experiencing spontaneous recovery. This proportion may be improved by the timely use of corticosteroids, but we do not have good data to support the impact of this.[1]

The most important prognostic factor is the degree of hearing loss at presentation. The less severe at presentation, the greater the likelihood of recovery. Other factors indicating a poor prognosis for hearing recovery include associated vertigo, late recovery with or without treatment and associated systemic features (i.e. a high ESR or CRP).[15]

Features suggestive of a favourable prognosis of hearing recovery include:

- isolated high or low frequency loss (compared with a flat pattern); isolated low frequency has the greatest chance of recovery
- presence of tinnitus
- absence of vertigo
- absence of underlying systemic illness.[16]

Conclusion

SSNHL is an otological condition that requires urgent treatment and specialist referral. It is important for clinicians to have a high index of suspicion and an understanding of the diagnosis and need for prompt management. In the initial assessment, simple tuning fork tests and otoscopy can exclude conductive causes, such as middle ear effusion, wax impaction and infection, which might have similar presentations and do not require specialist referral. If there is uncertainty about the diagnosis, it is important to arrange an urgent audiogram with air and bone conduction, after clearing the ear canal of any obstruction.

There remains a lack of consistency in treatment regimens due to low efficacy evidence; however, the 2019 American Academy *Clinical Practice Guideline: Sudden Hearing Loss (Update)* should be followed. Once the sensorineural nature of the hearing loss is established and a diagnosis of SSNHL is suspected, patients require urgent referral to an ENT specialist for consideration of corticosteroid treatment. MT

References

1. Mattox DE, Simmons FB. Natural history of sudden sensorineural hearing loss. Ann Otol Rhinol Laryngol 1977; 86: 463-480.
2. Lin RJ, Krall R, Westerberg BD, Chadha NK, Chau JK. Systematic review and meta-analysis of the risk factors for sudden sensorineural hearing loss in adults. Laryngoscope 2012; 122: 624-635.
3. Rauch SD. Idiopathic sudden sensorineural hearing loss. N Engl J Med 2008; 359: 833-840.
4. Alexander TH, Harris JP. Incidence of sudden sensorineural hearing loss. Otol Neurotol 2013; 34: 1586-1589.
5. Fetterman BL, Luxford WM, Saunders JE. Sudden bilateral sensorineural hearing loss. Laryngoscope 1996; 106: 1347-1350.
6. Da Cruz M. Alleviating the distress of tinnitus: a phantom sound. Med Today 2012; 13(6): 16-22.
7. Chandrasekhar SS, Tsai Do BS, Schwartz SR, et al. Clinical Practice Guideline: Sudden Hearing Loss (Update). Otolaryngol Head Neck Surg 2019; 161: S1-S45.
8. Da Cruz M. The essentials of managing hearing loss. Med Today 2015; 16(4): 31-42.
9. Chao TK, Chen TH. Distortion product otoacoustic emissions as a prognostic factor for idiopathic sudden sensorineural hearing loss. Audiol Neurootol 2006; 11: 331-338.
10. Young Y-H. Contemporary review of the causes and differential diagnosis of sudden sensorineural hearing loss. Int J Audiol 2020; 59: 243-253.
11. Singh A, Irugu DVK. Sudden sensorineural hearing loss - a contemporary review of management issues. J Otol 2020; 15: 67-73.
12. El Sabbagh NG, Sewitch MJ, Bezdjian A, Daniel SJ. Intratympanic dexamethasone in sudden sensorineural hearing loss: a systematic review and meta-analysis. Laryngoscope 2017; 127: 1897-1908.
13. Ermutlu G, Süslü N, Yılmaz T, Saraç S. Sudden hearing loss: an effectivity comparison of intratympanic and systemic steroid treatments. Eur Arch Otorhinolaryngol 2017; 274: 3585-3591.
14. Da Cruz M. Cochlear implants – the best option for sever hearing loss in adults. Med Today 2021; 22(9): 30-40.
15. Lin RJ, Krall R, Westerberg BD, Chadha NK, Chau JK. Systematic review and meta-analysis of the risk factors for sudden sensorineural hearing loss in adults. Laryngoscope 2012; 122: 624-35.
16. Wen YH, Chen PR, Wu HP. Prognostic factors of profound idiopathic sudden sensorineural hearing loss. Eur Arch Otorhinolaryngol 2014; 271: 1423-1429.

COMPETING INTERESTS: None.

ONLINE CPD JOURNAL PROGRAM

What is the usual prognosis for patients with sudden sensorineural hearing loss?

Review your knowledge of this topic and earn CPD points by taking part in MedicineToday's Online CPD Journal Program. **Log in to www.medicinetoday.com.au/cpd**