ENTITLEMENT ELIGIBILITY GUIDELINES

HEARING LOSS

MPC 00646
ICD-9 389.1 (Sensorineural Hearing Loss), 389.0 (Conductive Hearing Loss)

The Hearing Loss Entitlement Eligibility Guideline will apply to claims where the 2006 edition of the Table of Disabilities would apply. Refer to the TOD Transition Protocols.

AN APPLICATION FOR ENTITLEMENT FOR A HEARING LOSS SHOULD PROPERLY BE SUBMITTED AS "HEARING LOSS" REGARDLESS OF THE TYPE(S) OF LOSS.

Please note: Entitlement should be granted for a *chronic* condition only. For VAC purposes, "chronic" means that the condition has existed for at least 6 months. Signs and symptoms are generally expected to persist despite medical attention, although they may wax and wane over the 6-month period and thereafter.

DEFINITION

There are two general types of hearing loss: sensorineural (sometimes called perceptive) and conductive hearing loss.

Sensorineural hearing loss is hearing loss due to a defect in the cochlea or the auditory nerve whereby nerve impulses from the cochlea to the brain are attenuated. **Conductive hearing loss** means the partial or complete loss of hearing due to defective sound conduction of the external auditory canal or of the middle ear. A **mixed hearing loss** is a combination of sensorineural and conductive.

DIAGNOSTIC STANDARD

For VAC purposes, normal hearing exists where there is decibel loss of 25 dB or less at all frequencies between 250 and 8000 hertz.

For VAC purposes, a hearing loss disability exists when there is a Decibel Sum Hearing Loss (DSHL) 100 dB or greater at frequencies of 500,1000, 2000 and 3000 Hz in either ear, OR 50 dB or more in both ears at 4000 Hz.

For VAC purposes, a non- disabling hearing loss exists when there is a decibel loss greater than 25 dB at frequencies between 250 and 8000 hertz (inclusively), and this loss is not sufficient to meet VAC's definition of a hearing loss disability.

A hearing loss disability can be considered to be partially caused by service factors, when there is decibel loss greater than 25 dB evident on the discharge audiogram in at least one of the frequencies between 250 and 8000 Hz (inclusively) AND a hearing loss disability is established after discharge.

The presence of a hearing loss and the type of hearing loss may be determined from an audiogram. Diagnosis of the type of hearing loss may be made by a clinical/licensed/certified/registered audiologist or a qualified medical practitioner.

The cause of the hearing loss cannot be determined from an audiogram alone. The history from the patient, the physical examination and relevant test results must be considered along with the audiogram findings.

It is preferred that audiograms submitted to the Department for entitlement or assessment purposes be performed by a clinical/licensed/certified/registered audiologist. However, audiograms submitted from other sources such as hearing instrument specialists (HIS), physicians or nurses may be considered by VAC if they meet the following standards.

For an audiogram to be considered acceptable by the Department, the following criteria should be met. The hearing should be tested in both ears at 250, 500, 1000, 2000, 3000, 4000, 6000 and 8000 htz. Air and bone conduction values in both ears should be recorded. Speech Reception Thresholds (SRTs) for each ear should be recorded. An indication of reliability of the audiogram should be indicated. A narrative description of the test results is also welcomed.

Psychogenic deafness may be conscious (feigned) or unconscious. The diagnosis can usually be confirmed by sophisticated audiometric testing including cortical-

evoked response audiometry.

Audiograms that do not meet the above standards should be considered by the adjudicator on a case-by-case basis. The determination of reliability is based on the interpretation of information provided on the audiogram, the age of the audiogram, and its consistency with previous audiograms.

It is important to note that the literature indicates that American audiometric data collected prior to 1969/1970 may be based on the ASA standard. As late as 1977 it was recommended that audiogram blanks be labelled as ANSI or ISO to ensure that all hearing levels in a report were actually ANSI or ISO (Hearing and Deafness, 4th ed., p. 287).

To **convert ASA hearing losses to ISO-ANSI** levels, the following decibels are added:

FREQUENCY	DECIBELS ADDED
125	9
250	15
500	14
1000	10
1500	10
2000	8.5
3000	8.5
4000	6
6000	9.5
8000	11.5

ANATOMY AND PHYSIOLOGY

Sounds are collected by the external ear (auricle) and transmitted down the external ear canal to set the eardrum in motion. The eardrum separates the canal from the middle ear with its 3 ossicles - malleus (hammer), incus (anvil), and stapes (stirrup). The eardrum's vibrations are picked up and amplified by the ossicles and conducted to the cochlea (organ of hearing). The whole system, from the auricle to the stapes, is the conducting apparatus of the ear.

Any abnormality in the system, from wax in the ear canal to fixation of the stapes by otosclerosis, can cause a **conductive hearing loss**. Conductive hearing loss is the result of sound waves not being transmitted effectively to the inner ear because of some interference in the external canal, the ear drum, the ossicular chain, the middle ear cavity, the oval window, the round window, or the eustachian

tube. In pure conductive hearing loss there is no damage to the inner ear or the neural pathway.

The ossicle's vibrations are transmitted indirectly to the fluid in the cochlea. Movement of the fluid stimulates the cochlea's hair cells. They transmit electrical impulses along the auditory nerve (of hearing) to the brain. The whole system, from the cochlea to the auditory cortex in the brain, is the sensorineural apparatus. Any abnormality in these structures such as - cochlear damage or acoustic neuroma can cause a **sensorineural hearing loss**.

As a general rule, sensorineural hearing loss affects mainly the high tones, and conductive hearing loss the low tones. The rule is not invariable. Early Meniere's disease classically causes a low tone sensorineural hearing loss; otosclerosis causes a conductive hearing loss that may affect the high tones as well as the low tones.

A complete audiogram shows hearing by both air and bone conduction. In a **conductive hearing loss**, hearing by bone conduction is normal, hearing by air conduction is diminished. The air conduction graph is therefore at a lower level in the audiogram than the bone conduction graph. The gap between the two graphs is known as the "air-bone gap". In a **sensorineural hearing loss**, air and bone conduction are affected equally; the two graphs are at approximately the same level, there is no air-bone gap. In a **mixed hearing loss**, air and bone conduction are both affected, but the loss by air is more severe than the loss by bone. The air conduction graph is lower than the bone conduction graph. There is an air-bone gap.

CLINICAL FEATURES

Two common causes of sensorineural hearing loss prominent in pension medicine are noise and age. These factors initially cause a high tone hearing loss, which gradually spreads to other frequencies. It may be difficult to determine whether noise, age or a combination of the two is to blame.

To cause a hearing loss, noise has to be loud enough and of sufficient duration. The louder the noise, the shorter the time it will take to damage hearing. Both a single intense sound and repetitive sounds can cause a **sensorineural hearing loss.** The hearing loss may be temporary (Temporary Threshold Shift) with complete recovery of hearing in a few hours to a few weeks or permanent. A single intense, sudden sound can damage the ear before the aural reflex can act (i.e. where sound is generated in a shorter time than 44 ± 11 milliseconds). For repetitive sounds, the contraction of the stapedial muscles is maintained when

repetition occurs less than once a second.

There is a clear tendency for the ear to be more tolerant of noise at the low frequencies, as opposed to the middle and higher frequencies. The ear appears to be particularly vulnerable to frequencies in the range of 2000 to 4000 Hz, or even 6000 Hz. These frequencies are likely to be generated in industrial settings by various hammering, stamping, pressing, shipping and rivetting operations, and in other settings by gunfire, explosions, and some types of aircraft noise. The loudness level or intensity of noise, measured in decibels, and the length of exposure are critical. Continued exposure to noise above 85 db over time will cause hearing loss. According to the National Institute for Occupational Safety and Health (1998), the maximum exposure time on a single episode at 85 db is 8 hours, and at 110 db it is one minute and 29 seconds. Noise levels above 140 db can cause immediate hearing damage. Frequency, measured in cycles per second or Hertz (hz), is also important as high frequency noise can cause more damage than low-frequency noise.

Examples of approximate decibel (dB) levels are as follows:

- 85 handsaw
- 95 electric drill
- 100 factory machinery
- 105 snow blower
- 110 power saw
- 120 pneumatic drills, heavy machines, chain saw
- 120 jet plane (at ramp)
- 130 jackhammer, power drill, air raid, percussion section at symphony
- 140 airplane taking off
- 150 jet engine taking off, artillery fire at 500 feet
- 163 rifle
- 166 handgun
- 170 shotgun

Characteristically, the first sign of noise damage appears as a dip or notch at one of the higher frequencies, usually 4000 or 6000 Hz. The notching configuration is not, however, always present; for example, it may be obliterated by the effects of aging or continued exposure to noise. With continued noise exposure, the hearing loss becomes permanent because of irreparable damage to the cochlea's hair cells. As exposure continues, more hair cells are damaged and the hearing loss, although remaining most severe in the upper frequencies, extends to the lower frequencies.

Above a certain intensity, noise becomes explosive and causes blast-type injuries.

It may rupture the eardrum, causing a **conductive hearing loss**. If no further damage has been done, the hearing loss may be temporary; if the eardrum heals, complete restoration to normal is possible. The blast may damage or dislocate the ossicles of the middle ear, causing a **conductive hearing loss** that may be permanent unless the ear is successfully operated upon. The blast may also tear the sensitive part of the cochlea (the organ of Corti) from its moorings, causing a **sensorineural hearing loss** that is permanent and irreparable. Any combination of these injuries may occur, so that the hearing loss may be **conductive, sensorineural, or mixed.**

PENSION CONSIDERATIONS

SENSORINEURAL HEARING LOSS

A. CAUSES AND/OR AGGRAVATION

THE TIMELINES CITED BELOW ARE NOT BINDING. EACH CASE SHOULD BE ADJUDICATED ON THE EVIDENCE PROVIDED AND ITS OWN MERITS.

1. Exposure to *at least one episode of acoustic trauma* **just prior** to clinical onset or aggravation

Acoustic trauma means a condition of sudden aural damage resulting from short-term intense exposure or a single exposure to loud noise, such as that made at close quarters by:

- fireworks
- small arms fire
- gunfire
- artillery fire
- exploding grenades, mines or bombs
- blast injury

Most literature does not specify the range of noise levels likely to occur with any of these types of explosions. Noise levels are probably well in excess of 120 decibels. Extremely loud noises can rupture the ear drum and damage the ossicles. Less intense acoustic trauma may produce ear pain or a sensation of burning, temporary threshold shift, permanent hearing loss and tinnitus. The severity of the loss varies with the magnitude of the explosion and proximity to the blast.

There is evidence to support the notion that a single exposure to acoustic trauma may possibly be sufficient to induce a state of permanent threshold shift. This is, however, considered an unlikely event unless there is objective evidence of concomitant middle ear damage such as a ruptured ear drum or bleeding from the ear canal.

2. Exposure to noise, *other than acoustic trauma*, during or just prior to clinical onset or aggravation

   Consideration must be given to variables including, but not limited to, the following:
   - level of the noise hazard
   - continuity of noise hazard
   - duration of noise hazard
   - presence or absence of adequate ear protection
   - location of noise hazard (e.g. in enclosed or open area)

3. Presbycusis

   This is the sensorineural hearing loss that occurs as a part of normal aging. There is considerable variation in the onset of presbycusis. Changes that occur in the aging auditory mechanism may begin early or later in life, and progress slowly or rapidly.

4. Treatment with certain drug(s) **during or just prior** to clinical onset or aggravation

   Each drug is to be looked at on an individual basis as to length of time required to cause or aggravate hearing loss.

   The drugs include, but are not limited to, the following:

   (A) a **parenteral** aminoglycoside antibiotic:
   - gentamicin
   - streptomycin
   - kanamycin
   - amikacin
   - netilmicin
   - tobramycin

   B) **intravenous** administration of:
   - ethacrynic acid
   - furosemide
   - bumetanide
   - ancomycin
   - erythromycin

   C) chemotherapeutic agents:
   - nitrogen mustard
   - bleomycin

- cisplatin
- a-difluoromethylornithine
- vincristine
- vinblastine
- misonidazole
- 6-amino nicotinamide
- carboplatin

D) salicylate or quinine derivatives at time of clinical onset or aggravation

Sensorineural hearing loss may follow long periods of therapy with these drugs at pharmacological doses, although the effects are often reversible.

5. Suppurative labyrinthitis of the affected ear within approximately 30 days prior to clinical onset or aggravation

Suppurative labyrinthitis means inflammation of the labyrinth (a system of interconnecting canals in the inner ear), characterized by the formation of pus.

6. Meniere's disease at time of clinical onset or aggravation

7. Systemic immune mediated disorder at time of clinical onset or aggravation

Systemic immune mediated disorder means a group of disorders thought to be mediated by the deposition of immune complexes in various body tissues. The disorders associated with sensorineural hearing loss are:

(a) systemic lupus erythematosus
(b) periarteritis nodosa
(c) Wegener's granulomatosis
(d) Cogan's syndrome
(e) Behçet's syndrome

8. Leprosy prior to clinical onset or aggravation

Leprosy is a slowly progressive chronic infectious disease caused by *Mycobacterium leprae*.

9. An acute vascular lesion involving the arteries supplying the cochlea on the affected side at time of clinical onset or aggravation

A rare cause of sensorineural hearing loss is a vascular lesion, i.e. any local process causing a sudden decline in the supply of blood to the cochlea and includes ischemia, emboli and hemorrhage affecting these arteries.

This involves an **acute onset** of sensorineural hearing loss which is described in relation to hemorrhage, thrombosis and vasospasm of the terminal branch of the anterior-inferior cerebellar artery or cochlear vessels. These events may be caused by diseases such as diabetes mellitus, and degenerative (mainly atherosclerotic) vascular disease.

Spontaneous haemorrhage into the inner ear has been described as a complication of leukemia, Wegener's granulomatosis, subarachnoid hemorrhage and temporal bone trauma.

Persons would need a history of diabetes mellitus with associated vascular complications as well as an acute sensorineural hearing loss to be considered.

For arteriosclerosis, evidence of atheroma (cholesterol plaque) in other organs is necessary for it to be considered as a possible cause of **sudden** hearing loss.

10. Hyperviscosity syndrome within approximately 30 days prior to clinical onset or aggravation

Hyperviscosity syndrome is any blood disorder causing a significant increase in the viscous properties of the blood, such as Waldenstrom's macroglobulinemia, polycythemia, the leukemias, and sickle cell trait.

11. An acute infection from a particular virus within approximately 30 days prior to clinical onset or aggravation

The viruses include, but are not limited to, the following:

- mumps virus
- measles virus
- rubella virus
- pertussis virus
- varicella-zoster virus

12. Bacterial meningitis within weeks or months prior to clinical onset or aggravation

    Bacterial meningitis is an inflammation of the lining of the brain and spinal cord caused by bacteria, common types of which are *Hemophilus influenzae m., Meningococcal m., Pneumococcal m.,* and *Tuberculous m.*

13. Neurosyphilis prior to clinical onset or aggravation

    Neurosyphilis means central nervous system manifestations of syphilis, which is a sub-acute chronic infectious disease caused by the spirochaete *Treponema pallidum*, characterized by episodes of active disease interrupted by periods of latency. Hearing loss should develop during an active phase of the illness.

14. Tuberculosis involving the temporal bone on the affected side of the head prior to clinical onset or aggravation

15. Paget's Disease of bone affecting the skull at time of clinical onset or aggravation

    Paget's disease of bone (also known as osteitis deformans) means a disease of bone marked by repeated episodes of bone resorption and new bone formation resulting in weakened deformed bones of increased mass.

16. Head injury with temporal bone fracture within a few weeks of onset or aggravation of hearing loss

    20% of fractures to the temporal bone are transverse fractures which may cause injury to the cochleovestibular nerve resulting in a sensorineural hearing loss.

17. At least one episode of otic barotrauma within approximately 30 days prior to clinical onset or aggravation

    Otic barotrauma means damage to the middle ear from inequalities in the barometric pressure on each side of the tympanic membrane.

    Sensorineural hearing loss is a rare complication following otic barotrauma and is usually associated with a perilymph fistula.

18. A benign or malignant neoplasm affecting the auditory apparatus on the affected side prior to clinical onset or aggravation

    A benign or malignant neoplasm means a primary or secondary neoplasm of the auditory nerve, inner ear, temporal bone, cerebellopontine angle or posterior cranial fossa (that part of the skull that lodges the hindbrain which includes the cerebellum, pons, and the medulla oblongata).

19. A course of therapeutic radiation to the head or neck region within approximately 18 months prior to clinical onset or aggravation

    Therapeutic radiation means medical treatment by irradiation with gamma rays, x-rays, alpha particles or beta particles.

20. Surgery to the middle ear, inner ear or posterior cranial fossa region prior to clinical onset or aggravation

21. Cholesteatoma at the time of clinical onset or aggravation

22. Inability to obtain appropriate clinical management

    See Introduction to Entitlement Eligibility Guidelines.

B. MEDICAL CONDITIONS WHICH ARE TO BE INCLUDED IN ENTITLEMENT/ASSESSMENT

- Hearing loss of all types.

C. COMMON MEDICAL CONDITIONS WHICH MAY RESULT IN WHOLE OR IN PART FROM SENSORINEURAL HEARING LOSS AND/OR ITS TREATMENT

- otitis externa from the wearing of hearing aids
- tinnitus
- vertigo

CONDUCTIVE HEARING LOSS

A. CAUSES AND/OR AGGRAVATION

THE TIMELINES CITED BELOW ARE NOT BINDING. EACH CASE SHOULD BE ADJUDICATED ON THE EVIDENCE PROVIDED AND ITS OWN MERITS.

1. Otosclerosis at time of clinical onset or aggravation

2. Exposure to at least one episode of acoustic trauma **just prior** to clinical onset or aggravation

   Acoustic trauma means a condition of sudden ear damage resulting from short-term intense exposure or a single exposure to loud noise such as that made at close quarters by fireworks or small arms fire or gun fire or artillery fire or exploding grenades, mines or bombs. It usually produces immediate symptoms such as deafness, pain, bleeding or tinnitus in the affected ear. The immediate symptoms may abate over a few days. Blast injury may cause damage to the tympanic membrane and ossicles, with a variable degree of injury to the cochlea.

3. Head injury with temporal bone fracture within a few weeks of onset or aggravation of hearing loss

   80% of fractures of the temporal bone result in conductive hearing loss as a result of ossicular chain injuries.

4. Otic barotrauma on the affected side within approximately 7 days prior to clinical onset or aggravation

   A ruptured tympanic membrane is the most common result of barotrauma. The hearing loss from a ruptured tympanic membrane may be as large as 60 decibels if the rupture was caused by a force severe enough to impair the ossicular chain, but usually the loss is less than 30 decibels and involves practically all frequencies. It is not possible to predict the degree of permanent hearing loss from a ruptured tympanic membrane.

5. A penetrating injury to the middle ear on the affected side within approximately 7 days prior to clinical onset or aggravation

   Such an injury means the intrusion of a foreign body such as a weapon, implement, stick, bullet or shrapnel fragment into the tympanic cavity.

6. Permanent obstruction of the external auditory canal on the affected side at the time of clinical onset or aggravation

   Such obstruction means at least 90 percent blockage of the external auditory canal **but not from wax or small foreign bodies that can be removed.**

7. A surgical procedure involving the middle ear or the external auditory canal on the affected side within approximately 7 days prior to clinical onset or aggravation

   The procedure includes a myringotomy, myringoplasty, mastoidectomy, ossiculoplasty, fenestration, middle ear prostheses. Complications can occur during or after surgery. Conductive hearing loss may be seen in persons due to bone debris and other mastoid contents that enter the ear.

8. Chronic otitis externa on the affected side

   Malignant otitis externa is one example of a chronic otitis externa which could produce a permanent conductive hearing loss. Chronic malignant otitis externa may be associated with diabetes mellitus or develop in an immunocompromised state (see EEG on Chronic Otitis Externa).

9. Chronic otitis media on the affected side **just prior** to clinical onset or aggravation

   Chronic otitis media may result in any of the following:
   - perforation of tympanic membrane (if small, the perforation generally heals spontaneously).
   - medial meatal fibrosis.
   - erosion of ossicles and tympanic membrane

10. Acute suppurative otitis media on the affected side just prior to clinical onset or aggravation

    The main complaint is pain. There is pus accumulation in the middle ear and bulging of the eardrum. If the ear drum ruptures, a conductive hearing

loss is present immediately. With proper treatment hearing usually returns completely to normal. But with or without treatment, the perforation may persist. Hearing may or may not return to normal, depending on the size and location of the perforation.

11. Non suppurative otitis media on the affected side just prior to clinical onset or aggravation

    This has been called “catarrhal otitis media”, “serous otitis media”, “otitis media with effusion” and other names. The middle ear in this variety contains serous or mucoid fluid. There is a conductive hearing loss which can be reversed by treatment; however, if left untreated the condition may progress to chronic adhesive otitis media, i.e. scarring of the middle ear, with a permanent conductive hearing loss.

12. Paget’s disease of the bone (osteitis deformans) affecting the skull at time of clinical onset or aggravation of conductive hearing loss

13. Rheumatoid arthritis involving any synovial joint of the head and neck prior to clinical onset or aggravation

    The joints between the incudomalleolar and incudostapedial joints are synovial and thus in rare instances may develop rheumatoid arthritis.

14. A granuloma that is invading the middle ear or is causing at least 90 percent obstruction of the external auditory canal on the affected side at the time of clinical onset or aggravation of conductive hearing loss

    Examples include a granulomatous foreign body, tuberculous and sarcoid granulomata.

15. A primary or secondary neoplasm that is invading the middle ear or is causing at least 90 percent obstruction of the external auditory canal on the affected side at the time of clinical onset or aggravation of conductive hearing loss

    Neoplasia produce conductive hearing loss by interfering with the motion mechanics of the middle ear and ossicles or by obstruction of the auditory canal. Tumours may involve the middle ear, mastoid, and the temporal bone primarily.

16. Therapeutic radiation of the head or neck where the temporal bone of the affected side was in the radiation field prior to clinical onset or aggravation of conductive hearing loss

    Osteoradionecrosis of the temporal bone may produce chronic infection which may result in conductive hearing loss. Conductive hearing impairment due to secretory otitis media following radiotherapy is a consequence well known to radiation oncologists. Published studies show conflicting results, with damage occurring immediately or subsequent to the radiation. Hearing loss may improve or may be permanent. There are a number of variables which confound the effects of radiation, but it has been demonstrated to cause dryness and scaling of the skin of the external auditory canal, leading to build up of debris which may result in conductive hearing loss.

17. Cholesteatoma at the time of clinical onset or aggravation

18. Inability to obtain appropriate clinical management

    See Introduction to Entitlement Eligibility Guidelines.

B. MEDICAL CONDITIONS WHICH ARE TO BE INCLUDED IN ENTITLEMENT/ASSESSMENT

- Hearing loss of all types.
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C. COMMON MEDICAL CONDITIONS WHICH MAY RESULT IN WHOLE OR IN PART FROM CONDUCTIVE HEARING LOSS AND/OR ITS TREATMENT

- otitis externa from the wearing of hearing aids
- tinnitus
- vertigo

REFERENCES FOR SENSORINEURAL HEARING LOSS

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