ENTITLEMENT ELIGIBILITY GUIDELINES

TINNITUS

MPC No code
ICD-9 388.3

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

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

Tinnitus is defined as the perception of a sound in one or both ears or in the head when it does not arise from a stimulus in the environment.

DIAGNOSTIC STANDARD

Diagnosis by a qualified medical practitioner and/or clinical/licensed/certified/registered audiologist is required. A single indication or complaint of tinnitus is not sufficient for diagnostic purposes. The condition must be present for at least 6 months.

ANATOMY AND PHYSIOLOGY

The mechanism and pathophysiology of tinnitus remains obscure. There are basically two types of tinnitus: subjective tinnitus and objective tinnitus. Subjective tinnitus is perceived by the individual only, and cannot be heard by anyone else. This is by far the most common type of tinnitus. Objective tinnitus is not only heard by the individual, but may also be heard by others.

Tinnitus regularly accompanies such disorders as presbycusis, noise-induced hearing loss and otosclerosis. It is one of the three symptoms of Meniere's disease. For most people, tinnitus is less bothersome during the day when they are

surrounded by noise related to their job or activities. Tinnitus often becomes more noticeable at night and may, in many individuals, cause trouble with sleeping.

Tinnitus can occur as a result of certain abnormalities affecting the outer ear, the middle ear, the inner ear, the eighth cranial nerve or the brain. The most common origin of tinnitus, however, appears to be the inner ear.

CLINICAL FEATURES

Tinnitus can be constant, intermittent, or occasional. It may be pulsatile (synchronous with the heartbeat).

Individuals who experience tinnitus have provided many different descriptions of what the tinnitus sounds like to them. Descriptions include:

- high-pitched sound
- ringing sound
- whistle
- squealing sound
- steam
- wind
- rushing water
- roaring
- hum
- transformer hum
- television hum
- chirping sound
- muffled speech
- breathing sound (respiration)
- pulse-like sound

The loudness of tinnitus as perceived by an individual can vary dramatically. Some individuals perceive it as very quiet, and are usually aware of the sound only when they are in a quiet environment with no significant background noise, such as night time when going to sleep. Others perceive tinnitus as an extremely loud sound that can be heard even when in the presence of other extremely loud sounds. It is the perception of the individual with tinnitus that is important.

It is not unusual for tinnitus to change in loudness, depending upon the background noise present, exposure to loud sound, physical exertion, emotional pressure, medications, or other factors.

The presence of tinnitus can produce a number of detrimental effects, even when no hearing loss is present. If hearing loss is also present, in conjunction with the tinnitus, these negative effects are even more problematic. The effects can include:

- hearing problems
- communication distraction
- speech discrimination difficulty
- psychological/psychiatric implications
- lifestyle changes

PENSION CONSIDERATIONS

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 sufficient to have caused some decibel loss of hearing (permanent or temporary) **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

2. Exposure to noise, other than acoustic trauma, that is of sufficient intensity and duration to cause hearing loss of 25 decibels or more at 3000, 4000 or 6000 frequency [in the ear(s) with tinnitus], prior to clinical onset or aggravation

3. A head injury or head trauma **just prior** to clinical onset or aggravation

   This would include:
   - head injury/concussion with or without unconsciousness or skull fracture
   - direct trauma to all or part of the auditory apparatus

4. Treatment with at least one ototoxic drug which also produces, in the majority of cases, some sensorineural hearing loss of 25 decibels or more at 3000, 4000 or 6000 frequency (in the ear(s) with tinnitus), **during or just prior** to clinical onset or aggravation

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

5. Treatment with salicylate or quinine derivatives for a condition for which the drug cannot be ceased or substituted, **during or just prior** to clinical onset or aggravation

6. Otosclerosis **months to years** prior to clinical onset or aggravation

Otosclerosis is a primary disorder of the labyrinthine capsule characterized by new bone formation and often involving the footplate of the stapes.

Stapes mobilization and stapedectomy as surgical procedures for otosclerosis may cause tinnitus.

Otosclerosis itself or surgery for the condition can cause tinnitus.

7. Severe or prolonged malnutrition

Severe malnutrition can result in segmental demyelination of the vestibular and/or cochlear nerves. This has been documented in beri-beri (thiamine deficiency) and nicotinic acid deficiency. This cause should be considered in all clients who have been Prisoners of War or in a malnourished state. The tinnitus develops before the malnourished state has been corrected.

8. Certain disorders of the face or teeth **just prior** to clinical onset or aggravation

Such disorders of the face and teeth would include:
- bruxism
- trigeminal neuralgia with associated spasm of the stapedius muscle
- myoclonus of the middle ear or palatal muscles
- Costen's syndrome - a temporomandibular joint dysfunction syndrome that can cause symptoms of pain and tinnitus or exacerbate existing tinnitus

9. Chronic eustachian tube disorder **during or just prior** to clinical onset or aggravation

Eustachian tube disorder includes, but is not limited to:
- eustachian tube obstruction from any cause
- chronic rhino-sinusitis with obstruction
- adenoid pathology
- chronic allergic disorders

10. Chronic tympanic membrane disease **during or just prior** to clinical onset or aggravation

Tympanic membrane diseases would include, but are not limited to:
- tympanic membrane perforation with or without surgery
- hemotympanium following blast trauma

11. Chronic middle ear disease **during or just prior** to clinical onset or aggravation

Middle ear disease would include, but is not limited to:
- middle ear cleft/air volume dysfunction
- serous, or chronic otitis media
- cholesteatoma with erosion into the semicircular canal
- ossicular chain discontinuity due to infection, surgery, or trauma
- tympanic membrane surgery with complications

12. Chronic inner ear disorder **during or just prior** to clinical onset or aggravation

Inner ear disorders would include, but are not limited to:
- chronic inner ear infections with co-existent sensorineural hearing loss This would include mumps, measles, varicella zoster and chronic labyrinthitis complicating chronic suppurative otitis media or bacterial meningitis.
- perilymphatic leaks from head injury, head trauma or barotrauma
- benign positional vertigo following head injury or labyrinthine disorders

13. At least one source of vascular sound in and around the affected ear prior to clinical onset or aggravation

Sources of vascular sound in and around the ear include, but are not limited to, the following:
- acquired arteriovenous fistulae
- glomus jugulare tumour
- Paget's disease
- benign intracranial hypertension
- carotid stenosis/occlusion including ischemia
- vertebrobasilar arterial occlusion including ischemia
- cerebral atherosclerosis
- intracranial aneurysm
- increased thyroid gland vascularity
- persistent stapedial artery
- hemorrhage, thrombosis and spastic changes of cerebral circulation
- glomus tympanicum
- cervical artery bruit
- congenital anomalies of cranial arterio-venous circulation

14. Immune disease with associated sensorineural hearing loss of 25 decibels or more at 3000, 4000 or 6000 frequency (in the ear(s) with tinnitus), prior to clinical onset or aggravation

An immune disease would include, but is not limited to:
- AIDS
- systemic lupus erythematosis
- rheumatoid disease
- polyarteritis nodosa
- scleroderma
- dermatomyositis

15. A primary or metastatic intracranial neoplasm prior to clinical onset or aggravation

A primary or a metastatic intracranial neoplasm is a benign or malignant neoplasm affecting the brain, meninges, skull or cranial nerves (e.g. acoustic neuroma).

16. Certain complications of a neurological disease at the time of onset or aggravation

This would include, but is not limited to:
- trigeminal neuralgia with associated stapedius muscle spasm
- myoclonus of the middle ear or palatal muscles
- lesions of the lateral pontine tegmentum
- multiple sclerosis and other demyelinating diseases usually with sensorineural hearing loss
- temporal lobe epilepsy with auditory hallucinations
- cerebral anoxia - due to ischemic changes in the auditory cortex
- raised intracranial pressure
- pseudotumor cerebri - administration of certain drugs in patients with this disease can aggravate pre-existing tinnitus
- carbon monoxide poisoning can cause tinnitus

17. Otic Barotrauma

Sensorineural hearing loss is a rare complication following otic barotrauma. Sensorineural hearing loss with tinnitus is more commonly seen following rapid ascent during diving whereupon gaseous nitrogen forms in the cochlea. Such damage is generally considered irreversible.

18. Meniere's Disease

Tinnitus may be a symptom of Meniere's disease and should be claimed as such.

19. Inability to obtain appropriate clinical management

See Introduction to Entitlement Eligibility Guidelines.

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

- Hyperacusis

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

REFERENCES FOR TINNITUS

1. Australia. Department of Veterans Affairs: medical research in relation to the Statement of Principles concerning Tinnitus, which cites the following as references:

    1) Atlas MD.(1994) Tinnitus: a rational approach to management. Modern Medicine. Vol 37, No 12 December 1994.p24.
    2) Bailey. Q.Tinnitus. Australian Prescriber ,Vol 10 no 4. P69.
    3) Ballenger JJB (1991). Diseases of the Nose, Throat, Ear, Head and Neck. Fourteenth edition. Lea & Febinger: London. p1209.
    4) Bochner F, et al.(1978). Handbook of clinical Pharmacology. Little, Brown:Boston. p99.
    5) Brown RD, Penny JE, Henley CM et al (1981) Ototoxic drugs and noise.in Tinnitus, Ciba Foundation Symposium 85. Pitman, London 1985. P156-7.
    6) Christiansson AC, Wintzell KA.(1993) An Audiological survey of Officers at an Infantry Regiment. Scand Audiol:22 p147-152.
    7) Coles.RRA.Epidemiology of Tinnitus (1991) Ciba Foundation Symposium,Pittman , London,1985. p 20-23.
    8) Cooper JC jr (1994) Health and nutrition examination survey of 1971-75: Part 11. Tinnitus, subjective hearing loss and well being. J Am Acad Audiol 5: 37-43 (1994) p37 at 41.
    9) Dierhoff HG Meiber W (1987) Prevalence of Tinnitus in noise induced hearing loss Paper in Feldmann H (ed) Proceedings 111 International Tinnitus Seminar. Harsch Verlag, Karlshule, Germany, 1987, p 159-161.
    10) Domenech.J, Carulla m, Traserra j(1987) Tinnitus in patients treated with Cis-Platinum. III International Tinnitus Seminar. Harsch Verlag, Karlsruhe,1987.P168.
    11) Encyclopaedia of Occupational Health and Safety.(1983) 3rd (revised) edition,Ed. Luigi Parmeggiani. International Labour Office: Geneva p593-596.
    12) Evans EF (1981) General discussion- predisposing or aggravating factors Tinnitus, Ciba Foundation Symposium 85, Pitman,, London, 1985 p232.
    13) Feldmann H (ed) Proceedings 111 International Tinnitus Seminar. Harsch Verlag, Karlshule, Germany, 1987.
    14) Goodey (1981) Drugs in the treatment of tinnitus Tinnitus, Ciba Foundation Symposium 85, Pitman,, London, 1985 p 264.
    15) Hazell JWP.(1987) Tinnitus. Churchill Livingstone,London, 1987. p54-55.
    16) Hoover S(1984). Tinnitus and allergy III International Tinnitus Seminar.
    17) Harsch Verlag, Karlsruhe,1987.p110.
    18) Hoover.S (1987)Tinnitus and allergy.Paper in Feldmann H (ed) Proceedings 111 International Tinnitus Seminar. Harsch Verlag, Karlshule, Germany, 1987, p 42.House JW Brackmann (1981) Tinnitus:Surgical treatment. Tinnitus, Ciba Foundation Symposium 85, Pitman,, London, 1985 p215.

19) Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL.(1994) Harrison's Principles of internal medicine, 13th edition, New York,1994, p109.
20) Jones MP. (1988) Masking in tinnitus patient management, a comparison of two masking devices.Master of Arts (Audiology) dissertation, Macquarie University, December 1988. p5.
21) Kanda Y, Shigeno K, Kinoshita N, Nakao K, Yano M, Matsuo H (1994) Sudden hearing loss associated with interferon. Lancet. 1994;343:p1134-35.
22) Katz.J (ed) (1985) Handbook of Clinical Audiology. 3rd edition.Williams and Wilkins, Baltimore,1985. p31.
23) Ludman H (1988) Mawson's diseases of the inner ear, 5th edition, Edward Arnold,London, 1988. p607.
24) Luxton. L(1993) Tinnitus:its causes,diagnosis , and treatment. BMJ. No681 Vol 306 5th June 1993. p1490.
25) Mantz GJ (1993) Aminoglycoside cochlear toxicity. Otolaryngology Clinics of North America26 (5) p705-712.
26) Martindale: The extra Pharmacopoiea.(1989) .Twenty-ninth edition. Ed.
27) Mc Shane DP, Hyde ML,Alberti PW (1987) Tinnitus prevalence in occupational hearing loss claimants,Paper in Feldmann H (ed) Proceedings 111 International Tinnitus Seminar. Harsch Verlag, Karlshule, Germany, 1897, p152-7.
28) Miekle M, Greist S (1987) The percieved localisation of tinnitus.Paper in Moller AR. (1987) Can injury to the auditory nerve cause tinnitus? III International Tinnitus Seminar. Harsch Verlag, Karlsruhe,1987.p61.
29) Nardol JB (1993) Hearing loss. NEJM, October 7th 1993. p1098.
30) Neuberger M, Korpert K, Raber A, Schwert F, Bauer P (1990) Hearing loss from industrial noise, head injury and ear disease, Audiology , 1992; 31:45-57
31) Plinkert.PK, Gitter AH, Zenner HP. (1990) Tinnitus associated spontaneous otoacoustic emissions. Acta Otolaryngol (stockh) 1990: 110. p342-344.
32) Reynolds. JEF. The Pharmaceutical press. London. p985.
33) Sataloff. J. Sataloff RT, Vassallo L.(1980) Hearing Loss. 2nd ed, JB Lippincott Co, Philadelphia.p292.
34) Shulmann.A.(1984) Electrophysiological stimulation of tinnitus. III International tinnitus seminar. Harsch Verlag, Karlsruhe,1987.p139.
35) Sing. H, Rebentisch E, Poustka F, Curio I. (1989) Annoyance and health risk caused by military low altitude flight noise. Int Arch Occup Environ Health. (1990) 62: p357-363.
36) Spoendlin H(1987) Inner ear pathology and tinnitus.Paper in Feldmann H(ed) Proceedings 111 International Tinnitus Seminar. Harsch Verlag, Karlshule, Germany, 1987, p 42.
37) Stouffer JL, Tyler RS.,Kileny PR, Dalzell LE (1991) Tinnitus as a function of duration and etiology:counselling implications. The American Journal of Otology Volume 12, Number 3 3rd May 1991. p188-194.

38) Taylor, W., et al. (1965). Study of noise and hearing in jute weaving. *J. Acoust. Soc. Am.* **38**,p113.
39) Vernon JA.(1994) Tinnitus induced by head injury. Arch Otolaryngol head neck surg.Vol120, May 1994.p547-551.
40) Vernon.J Meikle(1981) Tinnitus masking:unresolved problems. Tinnitus, Ciba foundation symposium 85, Pitman,, London, 1985 p168.
41) Walton J (ed)(1993)Brain's diseases of the nervous system. Oxford University Press, Oxford, 1993 p114.
42) Yetiser. S. (1993) Concussive blast-type aural trauma, eardrum perforations, and their effects on hearing levels:An update on military experience in Izmir, Turkey. Military Medicine.158, 12:803, 1993. p803-806.
43) Zenner HP (1987) Modern aspects of hair cell biochemistry , mobility and tinnitus. III International Tinnitus Seminar. Harsch Verlag, Karlsruhe,1987.p154.

2. Canada. Department of Veterans Affairs. Medical Guidelines on *Hearing Loss*.

3. Canada. Department of Veterans Affairs. *A Review of Noise Induced Hearing Loss, Tinnitus and Evoked Response Audiometry,* Oct. 1997.

4. Fauci, Anthony S. and Eugene Braunwald, et al, eds. *Harrison's Principles of Internal Medicine*. 14th ed. Montreal: McGraw-Hill, 1998.

5. Paparella, M. M., et al, eds. *Otolaryngology*, vol II. 3rd ed. Philadelphia: W. B. Saunders, 1991.