Classification of Tinnitus

Multiple Causes with the Same Name

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KEYWORDS

- Tinnitus causes • Tinnitus subtypes • Classification of tinnitus
- Hearing loss and tinnitus

KEY POINTS

- Tinnitus is associated with many different causes, likely many different mechanisms, and many different subtypes.
- Any change compromising the auditory system, in any location, can lead to tinnitus.
- This article focuses on identifying some of the different subtypes. One way to classify tinnitus is to parallel the way hearing loss is classified: conductive versus sensorineural.
- This article reviews a broad range of approaches to understand and demarcate different tinnitus subtypes based on hearing loss.
- Appreciating and understanding different subtypes will help lead the way to different treatments, and will be critical for exploring and finding cures for different subtypes.

TINNITUS: FROM PERCEPTION TO REACTIONS

Occasionally, after exposure to loud sounds such as concerts and parties, many people when in a quiet environment, such as a bedroom, perceive some hearing loss accompanied by tinnitus, a sound that is generated in the auditory system. In most cases, it disappears completely by the next morning. Almost any form of hearing loss, such as from ear wax, otitis, or even a foreign body in the ear canal, can cause transitory tinnitus.

Once the person recovers from the temporary hearing loss, tinnitus perception fades away. However, for some people, this noise in the ears or in the head does

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not disappear, and it becomes another dimension in their lives. It is present all the time and, even more, it interferes in thoughts, emotions, hearing, sleep, and concentration. It can cause intense suffering for a few.

In the first case, it is called tinnitus perception. In the second case it is it is called chronic tinnitus. This tinnitus can cause extreme distress in everyday life.

PERCEPTUAL CHARACTERISTICS OF THE TINNITUS

The tinnitus can be pulsatile, nonpulsatile, constant, or intermittent; it can be tonal, or sound like buzzing, whooshing, beeping, hissing, or ringing; and it can affect 1 or both ears.

It can be subjective (perceived only by the affected individual) or objective (also heard by an observer); constant or intermittent; perceived in 1 or both ears, or within the head; pulsatile (synchronous with the heart beat or asynchronous); loud or faint; and can manifest with a variety of pitches. Tinnitus can be acute (<3 months), subacute (3–6 months), or chronic (>12 months). The onset can be gradual or sudden, and can change characteristics over time.[1,2]

Tinnitus is associated with many different causes; this article provides an overview of clinical situations associated with tinnitus.

SUBTYPES OF PATIENTS WITH TINNITUS

One approach to classifying tinnitus is to parallel the way hearing loss is classified: conductive versus sensorineural. Conductive hearing loss results from obstruction or damage to the outer or middle ear, preventing sound from being conducted to the inner ear. Sensory hearing loss results from cochlea or the stria vascularis damage. Sensorineural hearing loss results from damage of the hair cells, cochlear synapses, spiral ganglion neurons, and/or more proximal auditory structures. Any change compromising the auditory system, in any location, can lead to tinnitus.

CONDUCTIVE TINNITUS

A change in blood flow, muscles, or physiology of the middle ear can lead to tinnitus.

Ear Infections

Any form of ear infection can be accompanied by tinnitus. Once the infection process is resolved, tinnitus disappears. However, some patients keep experiencing tinnitus. One possible explanation is what is called unmasking. Tinnitus was already present but without perception, and, during the temporary hearing loss, or its increase, the patient's attention to the symptom can increase and it can become noticeable.

Tympanic Membrane and Ossicular Chain

Middle ear disorders that disrupt sound energy passing from the tympanic membrane to the inner ear can cause conductive hearing loss and tinnitus, such as tympanic membrane perforation; disruption or fixation of the ossicular chain resulting from infections, trauma, otosclerosis, or Paget disease; chronic otitis media and cholesteatoma; and eustachian tube dysfunctions.

Glomic Tumors

Glomus tympanicum tumors, or tympanic paraganglioma, are the most common benign tumors of the middle ear. Derived from the neural crest, the proliferation of

paraganglion cells is highly vascularized. They are rare, hypervascularized, benign neoplasms, but locally invasive.

Paraganglioma of the temporal bone originates from the tympanic plexus of the Arnold and Jacobson nerves (glomus tympanicum) or the adventitia layer of the jugular bulb (glomus jugular). Glomic tumors usually affect middle-aged women. Clinical manifestations are secondary to the tumor invasion on surrounding structures and include hearing loss (conductive and/or sensory), tinnitus (neurosensorial or pulsatile), dizziness, facial palsy, dysphagia, hoarseness, and pain. The otoscopic examination shows a red, pulsatile mass of the middle ear that can blanch during pneumatic otoscopy, the so-called Brown sign.[3]

Myoclonus

Middle ear myoclonus is defined as a rhythmic movement of the tympanic membrane secondary to repetitive contraction of the tensor tympani and stapedial muscles.[4] Tinnitus is usually unilateral, and the sound characteristic might give about a clue to the myoclonus's location. When the source is the tympanic tensor muscle, tinnitus sounds like a click and a butterfly, whereas, when produced by the stapedius muscle, it sounds like a buzzing noise.[5,6] The cause remains unknown, but has been associated with facial paralysis, trauma, vascular conditions, infections, demyelinating disorders, anxiety, and tumors.[4] Occasionally, rhythmic movements of the tympanic membrane can be visualized on otoscopic examination.

A clear mechanism for this rare subtype of tinnitus is not well understood but it is likely to be caused by the propagation of the muscle contraction noise, vibration of the tympanic membrane during contraction of these muscles, and alteration of cochlear microphony.[6]

Palatal myoclonus caused by the tensor veli palatini and levator veli palatini can also cause tinnitus. Tinnitus is heard in both ears and presents as clicks, resembling the noise made by the snapping together of 2 fingers.[7] The cause is unknown but could be associated with many clinical conditions, such as vascular lesions, trauma, infection, multiple sclerosis, or psychogenic, or it could be idiopathic. Palatal myoclonus can be diagnosed on direct oral cavity examination.[5]

The tinnitus sound could be a result of the eustachian tube snapping open or from the breaking of surface tension as the walls of the eustachian tube open under the action of peritubal muscles.[7]

Tonic Tensor Tympani Syndrome

Tinnitus is one of the symptoms of the tonic tensor tympani syndrome, initially described by Klochoff[8] as the tonic tensor phenomenon. Other complaints include ear fullness, tinnitus, dysacusis, tension headache, dizziness, and disequilibrium.

The tensor tympani muscle is 2.0 cm long, inserts in the neck of the malleus and the cartilage portion of the eustachian tube, and is innervated by the mandibular branch of the trigeminal nerve. Contraction of the tensor tympani muscle pulls the eardrum membrane inward and displaces the stapes into the scala vestibuli.

Clinically, the tonic tensor tympani syndrome is associated with acoustic shock injury, neck harm caused by whiplash, and temporomandibular disorders.[9] Myofascial disorders (trigger points) of masticatory muscles (eg, masseter and medial pterygoid, which are also innervated by the mandibular branch of trigeminal nerve) are also prevalent.

When the tensor tympani remains in a tonic contraction (fixed), it decreases tympanic compliance, causing attenuation of low-frequency acoustic energy transmission through the middle ear. The audiometric signature of this syndrome is (1) a decrease of

the acoustic tympanometry static peak compliance,[10,11] (2) low-frequency conductive hearing loss, and possibly (3) a small low-frequency sensorineural hearing loss.[11,12]

SENSORINEURAL TINNITUS

Sensorineural tinnitus is when the sensory or nervous system initiate the tinnitus. This initiation could take place in the cochlea, the auditory nerve, the temporal lobe, or perhaps even other parts of the neural system throughout the brain.

Sensorineural tinnitus is almost always accompanied by a sensorineural hearing loss, which is the most common type of tinnitus. It is important to remember how audiometric zero and normal hearing were established. After examining thousands of attendees of 1939 World's Fair in New York and San Francisco, the average hearing of 19-year-olds at 1 kHz was set to be 0 dB hearing loss. This level was considered to be the audiometric zero; the minimum sound pressure that the average young human with normal hearing could detect.[13] Normal hearing refers to a range about the average value. If someone had hearing thresholds at age 19 years of −10 dB hearing loss, and if that person's thresholds are now 0 dB hearing loss, they have a hearing loss. Even small changes on hearing thresholds can cause tinnitus. It is important to remember that human hearing extends up to 20 kHz, whereas hearing is typically tested only up to 8 kHz.

Patients with tinnitus may present normal audiometric thresholds but complain of difficulty hearing sounds when there is background noise.

Presbycusis Tinnitus

As people age, they lose their hearing gradually. Age-related hearing loss (presbycusis) is the number 1 communication disorder and is one of the top three chronic health conditions of the elderly. It reduces the capacity to understand speech, resulting in social isolation. For some, this might also include tinnitus as a result of the natural aging process, which might be difficult to distinguish from other unknown causes. Establishing presbycusis tinnitus is likely to require an insidious onset of a high-frequency sensorineural hearing loss, usually bilateral and symmetric, originating after some age (eg, 60 years or even 70 years), and a progression of loss over time, with no other likely causes. Physiologic changes in hearing with aging are associated with the damage of the cochlear sensory hair cells and morphologic changes of the stria vascularis leading to deficiency in the circulation and perfusion of the cochlea.[14]

Metabolic Tinnitus

The auditory system requires glucose and high energy availability to function. Maintenance of inner ear fluid homeostasis depends on the supply of oxygen and glucose by the stria vascularis. Changes in blood flow or metabolites cause impairment to the inner ear function, damaging the auditory system and possibly causing hearing disorders, including tinnitus. Some clinical situations that might affect inner ear homeostasis are discussed here.

Diabetes type II

The cochlea is a target of hyperglycemia. Abnormal blood glucose levels, even for short periods of time, might result in subclinical pathologic changes such as microangiopathy in the stria vascularis, damaging the cochlea and auditory neuropathy and causing hearing loss and tinnitus. Hearing evaluation among diabetics shows low-frequency hearing loss, associated or not with midfrequency and high-frequency hearing loss,[15] and most pronounced in the right ear.[16]

Hypothyroidism

Hypothyroidism, a common endocrinological disorder, results from reduced thyroid hormone actions at the peripheral tissues, slowing down the whole-body functions. It can also present in a subclinical form in patients with thyroid peroxidase antibodies and thyroid-stimulating hormone values in the upper normal range. Chances to develop hypothyroidism increases with age, and it is 10 times more common in women than in men.[17] Decrease of cerebral blood flow and glucose metabolism in hypothyroidism might affect the stria vascularis causing damage to the cochlea, hearing loss, and tinnitus. Hearing loss is usually bilateral mild to moderate, with a flat audiogram.[18]

Dyslipidemia

High serum low-density lipoprotein level is a vascular risk factor; in the inner ear it damages the cochlear microcirculation, causing hearing impairment and tinnitus.[19] Low high-density lipoprotein level increases the chances to develop low frequency/mid frequency hearing loss.[15]

Anemia

Anemia (low hemoglobin level) harms the cochlea because of deficiency of oxygen delivery, resulting in sensorineural hearing loss tinnitus.[20–22]

Vitamin and mineral deficiencies

Several reports have linked vitamin and mineral deficiencies to hearing loss and tinnitus, including vitamin B_{12},[23,24] B_1,[24] D_3,[25] folate,[26] zinc,[27–29] and magnesium.[30]

Causes include malabsorption syndromes, continuous use of proton-pump inhibitors, bariatric surgery, and vegetarianism.

Noise-Induced Tinnitus

Noise exposure is one of the most common causes of tinnitus. Focusing on it as subtype could therefore be important for many patients. Noise-induced hearing loss (NIHL) is accompanied by a notched audiogram, worse hearing at 3 to 6 kHz, and then an improvement at 8 kHz as an example. Subgrouping noise-induced tinnitus could require a history of noise exposure. It might also be helpful to distinguish between several years of noise exposure, versus a sudden NIHL, perhaps resulting from an impulse noise; for example, a gunshot. Air bag explosions can also result in impulsive noise-induced tinnitus but may also include head trauma, so that would have to be excluded in the subgrouping. If the noise-induced tinnitus affected only 1 ear, this would allow an important within-subject control. In addition, with a sudden-onset noise-induced tinnitus (eg, following an explosion or gunshot), it would be possible to distinguish immediate effects related to tinnitus, in contrast with long-term noise effects, which could include many parts of the nervous system.

Noise-induced hearing loss

NIHL is typically from long-term, continuous exposure to noise. The extent of the damage to the inner ear depends on the degree of sound intensity, the duration of the exposure, and genetic susceptibility. Tinnitus is usually bilateral, continuous, and high-pitched.[31,32]

Acoustic trauma

Acoustic trauma hearing loss results from a single or repeated sudden intense noise exposure, such as firearm shooting, car airbag release, and recreational music. It has been reported to immediately cause tinnitus.[33]

Acoustic shock

Acoustic shock injury (ASI) results from of a brief exposure to sudden unexpected loud sounds causing tinnitus, ear pain, ear pressure, hyperacusis/phonophobia, and vertigo. Nonotological symptoms include insomnia, headaches, and disorientation. High levels of emotional trauma and anxiety may be present. Usually, symptoms are temporary and disappear within hours to days following exposure. ASI has been described mostly among call center staff using a telephone headset or handset.[34–37] Clinical examination and audiometric testing are usually normal. The proposed neurophysiologic mechanism is an exaggerated startle response with contraction of the tensor tympani muscle.[35]

Sudden Sensorial Hearing Loss and Tinnitus

Sudden sensorineural hearing loss (SSHL) is defined as a loss of hearing of 30 dB or more in 3 consecutive frequencies in 1 or both ears that occurs within 72 hours. Tinnitus associated with SSHL is usually unilateral with an abrupt onset. As a comorbidity, it may become chronic and become the patient's primary concern.[38]

Viral inflammation, vascular occlusion, and immune diseases are possible causes, causing damage to the inner ear, the cochlea, or auditory pathways.[39] Some causes require urgent diagnosis, such as vestibular schwannoma (acoustic neuroma), stroke, malignancy, noise, and ototoxic medication, which may present as SSHL.[38] Although the presence of tinnitus causes greater emotional reactions in these patients,[40] its presence is a positive outcome factor for hearing recovery.[41]

Rapidly Progressive Bilateral Sensorineural Hearing Loss and Tinnitus

Rapidly progressive bilateral sensorineural hearing loss is defined as a shift of 15 dB or more at any frequency or 10 dB or more at 2 or more consecutive frequencies, or a significant change in discrimination score that occurs within 3 months or more apart.[42] Tinnitus is usually associated with the sensorineural hearing loss and is more frequent among women 30 to 60 years old. If the patient presents a positive response to steroid therapy, hearing loss is classified as immune mediated. In about 15% to 30% of cases, there is an association with autoimmune diseases such as systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, multiple sclerosis, ulcerative colitis, and Cogan syndrome. Pathophysiologic findings include cochlear inflammation, noninflammatory vasculopathy, and type II to IV hypersensitivity reactions.[43]

Ototoxicity

Ototoxic drugs and chemicals might damage the auditory system, causing functional harm to the cochlear hair cells. As a consequence, they might cause tinnitus and hearing loss. The symptoms develop during or after the end of use. Audiometry shows neurosensory bilateral hearing loss, symmetric or asymmetric with 1 ear being affected later. At present, more than 150 drugs are known to be ototoxic.[44] The most common drugs are aminoglycosides (irreversible), vancomycin (irreversible), macrolide antibiotics (reversible), platinum-based anticancer drugs (irreversible), loop diuretics (reversible), and quinine (reversible). Of interest is the widespread use of salicylate as a pain medication. It might induce mild to moderate hearing loss when used in high doses. Hearing loss is usually bilateral and symmetric and is associated with tinnitus; both the hearing loss and the tinnitus usually disappear within 24 to 72 hours after cessation of the drug.

Auditory Neuropathy Spectrum Disorder

Auditory neuropathy spectrum disorder (ANSD) is a retrocochlear hearing condition in which the patient has normal functioning outer hair cells (expressed by normal otoacoustic emission response) or cochlear microphonics, absent middle ear muscle reflexes, and an absent/abnormal auditory brainstem response.[45]

Clinically, ANSD is manifested by poor speech recognition severely affected by background noise, which does not correspond with pure tone thresholds. Bilateral low-pitched tinnitus (<1000 Hz) is a common complaint and can be severe enough to affect the quality of life of these individuals.[46] Mechanisms include injury to the inner hair cells and their synapses, the synaptic transmission to spiral ganglion, and neural signal transmission from the auditory nerve to the brainstem. Causal factors in adults include noise exposure, aging,[45] and genetic causes. There are at least 13 genes that affect the synaptic transmission or central auditory signaling pathway, including presynaptic (inner hair cell), postsynaptic spiral ganglion, and the auditory nerve spiral ganglion cell bodies and proximal axons.[47]

Vestibular Schwannomas

Vestibular schwannomas (VSs) are benign slow-growing intracranial tumors of Schwann cells of the vestibulocochlear nerve originating from the internal auditory canal or the cerebellopontine angle. VS has a prevalence of 8% among all intracranial tumors and 90% among cerebellopontine angle and internal auditory canal neoplasms.[48] They are classified in 2 physiologically distinct types: sporadic VS and neurofibromatosis type 2.

Sporadic VSs (90%) are unilateral, usually manifest around age 50 to 55 years, without gender predilection. Patients have progressive unilateral hearing loss and tinnitus that might be associated with facial numbness and facial paralysis, balance problems, and vertigo. Sudden hearing loss is the first clinical presentation in some cases.[49]

Neurofibromatosis type 2 is an autosomal-dominant inherited disorder, which predisposes affected individuals to develop a multiple neoplasm syndrome. It results from mutations in the *NF2* tumor suppressor gene located on chromosome 22q. It mostly occurs in young adulthood (age 20–30 years), with bilateral vestibular nerve schwannomas associated with other tumors of the nervous system, visual problems, skin tumors, and peripheral neuropathy. Initially, hearing loss is often unilateral, and is associated with or preceded by tinnitus. Dizziness and imbalance problems, facial paresthesia, and facial nerve palsy can also occur.[50,51]

Tinnitus and hearing loss are likely caused by compression of the auditory nerve, secondary to spasm or occlusion of the labyrinthine artery and potentially toxic substances secreted by the tumor to the inner ear or the cochlear nerve.[49]

Ménière's Disease

Tinnitus is a cardinal symptom in Ménière's disease (MD), a disorder of the inner ear. A typical clinical picture of MD presents as intermittent episodes of vertigo lasting from minutes to hours, fluctuating hearing loss, tinnitus, and aural fullness.[52] MD is more common among women aged 40 to 50 years. The symptoms are usually unilateral but can become bilateral over time.

Multifactorial factors such as genetics, metabolism, allergies, autoimmune reactions, and stress are associated with the cause of MD. Excessive endolymph accumulation in the cochlea, vestibule, and semicircular canals leads to endolymphatic

hydrops, causing damage to the ganglion cells,[53] which causes hearing loss and tinnitus. Usually, tinnitus sounds like low-pitched buzzing.[31,32]

The audiometric curve is characteristic, an up-sloping low-frequency sensorineural hearing loss with better hearing at 2000 Hz and worse hearing at frequencies greater than 2000 Hz. Fluctuations may lead to a flat sensorineural hearing loss.[52]

Vascular Conflict of Cranial Nerve VIII: Typewriter Tinnitus

This characteristic unilateral intermittent tinnitus is a form of tinnitus that occurs when a blood vessel (anterior-inferior cerebellar artery) is in close contact with the auditory nerve at its entrance into the brainstem. Tinnitus is described as sounding like a typewriter, Morse code, or machine-gun. Hearing loss usually manifests at tinnitus frequencies. If the vascular conflict affects the facial and/or the vestibular nerve, associated ipsilateral hemifacial spasms, otalgia, or vertiginous spells might occur.[54] Because typewriter tinnitus responds very well to carbamazepine,[55] the medication response is proposed to be used as diagnostic tool.[56] Tinnitus probably results from hyperactivity in the cochlear nerve generated by the conflict with the artery.[57]

VISUAL SNOW SYNDROME

Visual snow syndrome (VSS) is the perception of continuous television-static–like tiny flickering dots in the entire visual field. Visual snow has been called the tinnitus of the eyes.

Clinical criteria for the definition of VSS have recently been proposed[58]:

1. Visual snow: dynamic, continuous, tiny dots in the entire visual field lasting more than 3 months.
2. Presence of at least 2 additional visual symptoms of the 4 following categories: (1) palinopsia (persistent recurrence of a visual image after the stimulus has been removed); (2) enhanced entoptic phenomena (images produced by the eye's own structures); (3) photophobia; (4) nyctalopia (night blindness).
3. Symptoms are not consistent with typical migraine visual aura.
4. Symptoms are not better explained by another disorder.

There is a high prevalence of bilateral persistent tinnitus among patients with VSS (>60%). Possibly, both disorders share a common pathophysiologic mechanism that could involve thalamocortical loops secondary to dysfunctional neuronal excitability and impaired habituation response.[59]

Other symptoms include migraines, tremors, balance problems, vertigo, and fatigue.[60]

TINNITUS AND THE VESTIBULAR SYSTEM

Vestibular Migraine and Tinnitus

Tinnitus is present in about 46% to 68% of cases of vestibular migraine.[61]

The diagnosis of vestibular migraine is based on an association of recurrent vestibular symptoms that precede, occurs with, or occur after migraine. Symptoms include headache, visual aura, and/or photophobia and phonophobia in at least 50% of the episodes, in the absence of another diagnosis.[62] Vestibular migraine attacks can last from minutes to days.[63]

Fluctuating hearing loss and aural fullness can also be part of the clinical history, with a mild sensorineural hearing loss on the audiogram. Vestibular migraines occur at any time in life, but middle-aged women are the most commonly affected. Episodes might be triggered by stress, lack of sleep, dehydration, certain foods, and physical

activity.[61] Among women of childbearing age, attacks are associated with the menstrual cycle.[63] One possible explanation for the presence of tinnitus is that migraine may cause damage in the inner ear secondary to release of neuropeptides such as calcitonin gene–related peptide.[63]

Benign Paroxysmal Positional Vertigo and Tinnitus

Benign paroxysmal positional vertigo (BPPV) is a common peripheral vestibular disorder of the semicircular canals elicited by specific head movements causing recurrent sudden brief vertigo episodes. Most cases result from abnormally displaced otoconia into the semicircular canals producing a false sense of head rotation and nystagmus. The diagnosis is made using provocative maneuvers leading to a distinctive pattern of nystagmus observed in the plane of the affected canal. In general, BPPV is a self-limited condition but it can interfere with daily activities and contribute to the risk of falls in elderly patients.[64]

Tinnitus is associated with the onset of positional vertigo in about 19% of patients affected by BPPV. Tinnitus is intermittent, slightly intense, mostly unilateral, and localized in the same ear as the BPPV, which disappears or decreases immediately or shortly after the repositioning maneuver.[65,66] A possible explanation for this finding was proposed by Barozzi and colleagues[66] in 2013 based on Gussen's[67] previous work. She examined human temporal bones with cochlea-vascular degeneration related to heredity, aging, or viral causes. Otoconial displacement was observed within the ductus reuniens and cochlear duct as far as the basal turn of the cochlear spiral. Tinnitus in BPPV can be triggered by cochlear changes secondary to the presence of displaced otoconia.

SOMATOSENSORY MODULATION OF TINNITUS

Cross-Modal Interaction Among the Sensory Cortex

In their natural environments, humans and animals receive multimodal sensory stimuli. The sensation of external stimuli in the environment is taken to the brain by specialized sensory organs such as the eyes, the ears, or the skin. These inputs are processed in primary sensory cortices, such as the primary auditory cortex, the primary somatosensory cortex, or the visual cortex. Anatomic connections suggest that there is a multimodal functional interaction among sensory cortical regions, which means that they all interact in the brain in order to acquire a faster perception of the external world and its stimuli.

The deprivation of a sensory modality is likely to induce compensatory changes, named cross-modal plasticity.[68] For example, congenitally deaf individuals have superior visual abilities, and blind individuals present better auditory perception. Neurons from the deprived cortex are recruited from another sensory modalities. It has been shown that the auditory cortex can be activated by visual stimuli in deaf individuals. Cross-modal interaction in the presence of sensory deprivation of 1 of the senses is a plastic phenomenon in which the brain often compensates for loss with supranormal performance in 1 or more of the intact sensory systems.

Tinnitus Modulation

A subgroup of patients with tinnitus are able to change their tinnitus perception, intensity pitch, and location as a result of some trigger activity. These changes are mainly evoked with head, jaw, and eye movements or by applying pressure to a muscle in the head, neck, jaws, or a limb,[69,70] and trigger-point palpation.[71] These findings provide more evidence of interactions among the somatosensory and the auditory pathway.

The changes on tinnitus central percept are defined as tinnitus somatosensory modulation and are common among people with tinnitus.

Somatosensory Tinnitus

Somatosensory tinnitus should be suspected when the patient can modulate the loudness or intensity of the tinnitus with eye movements; cutaneous stimulation of skin on the hand region; through movements of the head, neck, and limb; and passive palpation of myofascial trigger points.[69–73]

Somatosensory tinnitus affects people of any age and is not necessarily related to hearing loss and degree of tinnitus severity.[74] Tinnitus can be pulsatile or nonpulsatile and the localization is often associated with the ear ipsilateral to the somatic disorder.[75]

Clinical criteria suggesting the presence of somatic tinnitus have recently been reported,[76] taking into account tinnitus characteristics and associated clinical symptoms. They include:

1. Tinnitus modulation: if movement of the head, neck, jaw, or eyes activates tinnitus modulation, use somatic maneuvers and digital pressure on myofascial trigger point.
2. Tinnitus characteristics: simultaneous appearance or intensification of tinnitus with neck or jaw complaints/pain, trauma, and bad posture.
3. Associated symptoms and signs, such as frequent pain in the cervical spine, head, or shoulder girdle; myofascial trigger points; muscle tension in the suboccipital muscles and extensor muscles of the cervical spine; temporomandibular disorders; bruxism and dental diseases.

Specific maneuvers performed using forceful movements of the head and neck, and eye movements, have been used to evaluate the presence of tinnitus modulation, but their absence does not exclude somatosensory tinnitus.

VASCULAR TINNITUS

Tinnitus associated with vascular structures has a characteristic sound synchronized to the heartbeat, which is produced by the turbulence of blood flow transmitted to the cochlea. The examiner in same cases can also hear it. Changes in tinnitus can be observed during head rotation and on compression of the internal jugular vein. The prevalence of this type of tinnitus is about 4% among people with tinnitus. Tinnitus may be caused by vascular anomalies or variants and is classified according to the site of generation. Some of them are described here; for a comprehensive review, see Sismanis[77] (2003).

Arterial Tinnitus

Atherosclerotic carotid artery disease

Pulsatile tinnitus may be the first manifestation of carotid artery occlusive disease; tinnitus is secondary to bruits produced by the turbulence of the blood flow in the compromised artery. Carotid artery occlusion may result in stroke, neurologic disability, or loss of life. Carotid atherosclerosis is an inflammatory disease that progresses to an eventual rupture of the atherosclerotic plaque. It is more frequent in men more than 50 years old. Risk factors include arterial hypertension, diabetes mellitus, dyslipidemia, cigarette smoking, and hyperhomocysteinemia. It is possible to auscultate a bruit on the carotid during the physical examination; its presence increases the risk of stroke and transient ischemic attacks.[78]

Arteriovenous Tinnitus

Pulsatile tinnitus can also be a clinical manifestation of arteriovenous malformations (AVMs) of the brain, an abnormal tangle of blood vessels connecting arteries and veins, and disrupting normal blood flow and oxygen circulation. Patients with these congenital lesions, which may enlarge over time, are at risk of serious intracerebral hemorrhage, stroke, or brain damage, depending on size and vascular anatomy location. Symptomatic presentation is most commonly intracerebral hemorrhage, focal seizures, neurologic deficits, and headaches. Aggressive therapy is used in most patients with AVMs, even if discovered incidentally.[79]

Depending on the localization, a bruit can also be heard when performing an auscultation on the scalp.

Venous Blood Flow Tinnitus

Idiopathic intracranial hypertension

Idiopathic intracranial hypertension is a syndrome characterized by increased intracranial pressure of unknown cause in the absence of clinical and radiological evidence of intracranial disorder. The mechanism responsible has been suggested to be a dysfunction of the cerebrospinal fluid dynamics caused by various hormonal and metabolic medical conditions and the use of certain medications.[78] Symptoms associated with a pulse beat–synchronous tinnitus include headaches, visual dysfunctions, hearing loss (often low-frequency sensorineural hearing loss), dizziness, aural fullness, and papilledema.[77]

Venous hum

This term is used to describe pulsatile tinnitus of unclear cause. Diagnosis of this condition should be made only after appropriate evaluation and elimination of other disorders.

GENETICS OF TINNITUS

The identification of genetic factors implicated in tinnitus will represent an important development. Candidate genes responsible for tinnitus may provide insights into the pathogenesis, novel gene-based diagnostic approaches, and therapy development.

However, because of the complexity and heterogeneity of tinnitus, genetic research is still in its infancy. As suggested by Lopez-Escamez and colleagues[80] in 2016, a possible methodological approach to advance tinnitus genetic subtyping is to cluster a few variables that could configure a phenotype. For example, a recent twin-cohort study has provided initial evidence that bilateral tinnitus is likely to be influenced by genetic factors and might constitute a genetic subtype.[81]

So far, no specific genetic locus has been identified; tinnitus is probably a polygenic condition. A recent genome-wide association study has identified potential metabolic pathways and has provided new insights into moderate genetic influences for tinnitus.[81,82]

SUMMARY

It is clear that there is not one single type of tinnitus. There are many causes and many different mechanisms. Appreciating and understanding different subtypes will lead to different treatments. This article focuses on identifying some of the different subtypes. It is hoped that clinical trials can focus on specific subtypes, which should increase the likelihood that treatments can be found for some patients with tinnitus.

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