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Tinnitus: a multidisciplinary clinical approach

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Abstract. *Tinnitus: a multidisciplinary clinical approach.* This article provides a clinical step-by-step approach for assessing a patient with tinnitus as primary complaint. The medical diagnosis of the disease provoking the tinnitus has to be made first in a comprehensive evaluation, including imaging. The psycho-acoustic characteristics and the influence on health-related quality of life is a compulsory complementary assessment to establish a complete picture of the patient.

Definitions

Tinnitus is the medical term for the auditory perception of sounds in the absence of surrounding sounds. The name is derived from the Latin word 'tinnere', which translates as 'to ring'.

These auditory experiences present in a great variety: a rustling, whistling, ringing, murmuring or humming sound which can come in high or low tones, be loud or soft and be continuous or interrupted. It can even be synchronous with heartbeat or breathing. Different types of sound can be heard simultaneously (polyphonic sensation) and there may be diurnal and nocturnal variation.

Tinnitus should be distinguished from auditive hallucinations, words or songs, a symptom that can occur in some psychiatric disorders (e.g. schizophrenia). Music sensations, however, may occur in tinnitus patients,[1] where they are predominantly related to hearing deprivation and not to psychosis.[2]

Tinnitus may be the only or most important symptom, or it may be a component of an otovestibular complaint (impaired hearing, distortion of sound, recruitment, sensation of pressure or pain in the ear, vertigo, instability or blurred vision). Furthermore, tinnitus can be accompanied by unpleasant sensations and be associated with anxiety and depression.[3,4]

Hyperacousis, or sound and noise intolerance, can accompany tinnitus but it should be considered as a separate feature since the correlation between tinnitus and hyperacousis is weak.[5] People who suffer from hyperacousis report discomfort from sounds that would be acceptable to most normally hearing people. They find day-to-day sounds to be too loud and disturbing. These patients often suffer from painful sensitivity in one or both ears when exposed to loud sounds.[6] Hyperacousis should be differentiated from recruitment, which is an abnormally rapid increase of loudness.

The consequences of tinnitus may be confined to this auditory perception, but more often it interferes with health-related quality of life[7,8] and with cognitive performance.[9-11]

Tinnitus is a non-pathognomonic sign of the involvement of the auditory system. It can fulfil a signal function for an underlying organic pathology and arise from common causes such as an earplug of wax or ventilation difficulties of the middle ear. It can also result from cochlear involvement or from rare yet important medical conditions such as brain tumours. The medical cause of tinnitus therefore needs to be determined precisely.

Assessing a patient with a primary complaint of tinnitus requires a comprehensive medical aetiological evaluation and a functional tinnitus assessment.[12]

Epidemiology

Tinnitus as such is not an abnormal sensation. Most people will experience tinnitus after a couple of minutes in a silent anechoic room.[13] Tinnitus will also occur after intense noise exposure for some hours in the majority of normal hearing people. Tinnitus that occurs every day for more than 5 minutes is reported by 10-15% of the population, and for 1

to 2% it affects their quality of life considerably.[14] In a recent survey of a population-based study assessing persons of 55-65 years of age, nearly 12% reported daily tinnitus sensations, and more than 15% of them described the tinnitus loudness as loud or very loud (6 or more on a scale of 0-10).[15]

Medical aetiological evaluation of tinnitus complaints

Two major categories can be distinguished: objective tinnitus, in which there is a real sound source, and subjective tinnitus, which is a phantom sensation.

Table 1

Causes of objective tinnitus originating from bodily sounds

arterial pulsatile tinnitus due to vascular pathology
carotid stenosis
dissecting carotid aneurism
resonance in the petrosal bone
arterial loop in the internal auditory canal
elevated cardiac output.
enhanced bone conduction with perception of arterial beat
semicircular canal dehiscence
arterio-venous malformation (AVM)
glomus tympanicum tumour
humming or murmuring of venous origin (“*venous hum*”)
intracranial hypertension (with or without obesity)
Arnold Chiari malformation
venous turbulence (with or without malformation)
muscle contractions and tube-opening sounds
m. tensor tympani (enhanced during tube dysfunction)
palatoclonus
cervical arthrosis sounds (while rotating head)
temporomandibular joint clicking
clicking of auricular cartilage or tympanic membrane

Objective tinnitus

Objective tinnitus is caused by real sound produced internally by the body and perceived by a normal healthy ear. These bodily sounds can be enhanced e.g. with occlusion of the outer ear canal or when there is facilitated bone conduction, as in a dehiscent semicircular canal.[16] A non-exhaustive overview of possible causes for objective tinnitus is given in Table 1. For many of these causes, therapeutic options are available, including surgery.[12,17]

Subjective tinnitus

Continuous tinnitus is caused by a dysfunction in the peripheral or central auditory system. Sustained focal cortical gamma-band hyperactivity in the primary auditory cortex is considered to be the neural code of tinnitus.[18] According to many authors it can be considered as an auditory phantom phenomenon[19-22] similar to phantom pain. It is caused by a reorganisation of the central auditory tract and auditory cortex, with a loss of suppression of neural activity. Some hypotheses suggest an initial reversible phase and a later, more irreversible, phase after two to three years.[23,24] This fits in with clinical data that demonstrate that tinnitus becomes more intractable for microvascular decompression surgery[25] and transcranial magnetic stimulation[26] after 3-4 years.

Any permanent effect on the auditory system resulting in increased activity in the auditory cortex can result in tinnitus. Table 2 gives a non-exhaustive list of conditions leading to subjective tinnitus.

It is emphasised that loudness and the level of suffering are not related to the extent of lesions or the seriousness of the underlying condition.

Medical evaluation of patients presenting with tinnitus

When assessing a patient presenting with tinnitus, a cascade of possible explanations of tinnitus-provoking physiopathological mechanisms is checked and the contribution of each factor estimated (Figure 1).

In first instance the focus is on whether or not the perceived sound is a real one, and therefore an objective tinnitus, and which part of the body is responsible for the generation of this sound: is it a bodily sound or not?

There are different hypotheses concerning the appearance of subjective tinnitus:

1) Physiological tinnitus. This can be considered as a normal type of tinnitus.

2) Cochlear origin. Cochlear tinnitus arises when there is a sustained higher firing rate of the inner hair cells, such as after loud noise exposure. Experimentally, this was proven for the intake of aspirin by Guitton.[27] This is most probably the case in Ménière’s disease during attacks.

3) Tinnitus related to sensorineural hearing loss. Auditory deprivation generally leads to a fall in peripheral output and an

Table 2
Causes of subjective tinnitus

physiological tinnitus
temporary tinnitus after exposure to loud noise
conductive hearing loss
cerumen plug
otosclerosis
otitis serosa
perceptive cochlear hearing loss
Ménière's disease
permanent noise trauma
presbyacusis
auto-immune inner ear disease
genetic sensorineural deafness
labyrinthitis or cochleitis
retrocochlear neural hearing loss and cerebellopontine angle pathology
acousticus neurinoma (vestibular schwannoma)
cochleovestibular compression syndrome
brainstem and auditory cortex dysfunction
brain tumours
white matter lesions
general or focal cortical dysfunction
including depression
toxicological or pharmacological effects
among other drugs, salicylate intoxication
extra-auditory somatic influences
temporomandibular dysfunction
whiplash trauma
cervical pathology

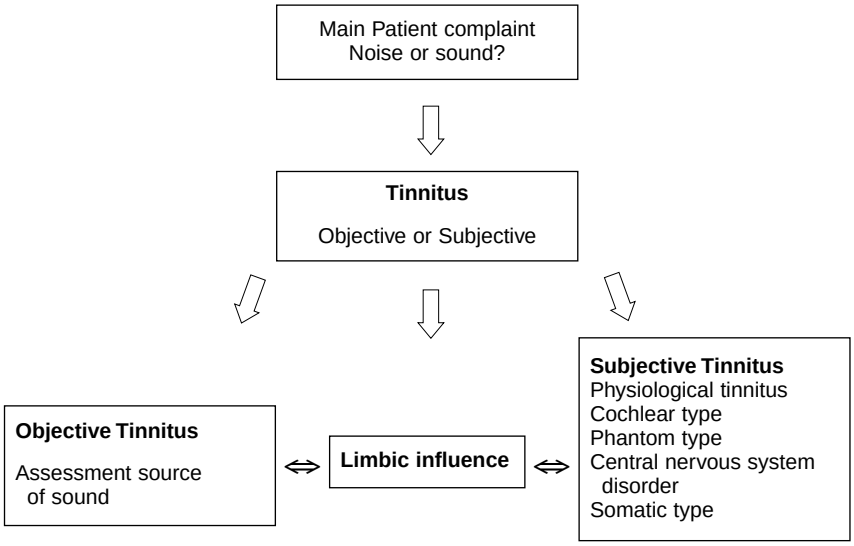

Figure 1
Diagnostic flow chart for tinnitus

increase in cortical hyperactivity, with functional changes in the auditory cortex.[28] This type of tinnitus is described as a "phantom auditory sensation", and it is associated with a reorganisation of the tonotopic cortical map. Moreover, the decrease in lateral inhibitory masking[29] is a concomitant effect of decreased cochlear output.

4) Somatic origin. Somatosensory modulation of the auditory system can be caused by influences emanating from, in particular, the jaw and masticatory muscles and from the neck.[30,31]

5) A final mechanism involves dysfunction or lesions in the brainstem and central nervous system. An underdiagnosed but important pathology is the cochleovestibular compression syndrome (CVCS). In CVCS, there is contact between the blood vessels that are normally in the proximity of the cochleovestibular and facial nerves. Diagnostic criteria have been proposed implementing a research classification based on increasing certainty, similar to the criteria for Ménière's disease (Table 3).[32] A particular subtype of CVCS is described as typewriter tinnitus, with bursts perceived as noises made with an old-fashioned typewriter.[33] In classical CVCS, the vascular conflict is in the cisternal segment of the eighth nerve; in typewriter tinnitus the conflict is located in the internal auditory canal.

A separate mechanism involves the limbic system. This is linked to the suffering experienced as a result of tinnitus. It is the cornerstone of Jastreboff's neurophysiological model of tinnitus suffering.[34] Neuro-imaging[35] and superselective pharmacological anaesthesia of the amygdalo-hippocampal region[36] could clearly demonstrate the involvement of the limbic system in tinnitus patients.

As is clear from Table 1 and 2, many pathologies leading to tinnitus are neurological or neurosurgical in origin. However, temporomandibular joint abnormalities can also result in tinnitus, and a multidisciplinary approach is

Table 3
Diagnostic criteria of cochleovestibular compression syndrome (CVCS)

Condition: other causes excluded. **Possible CVCS**: initially intermittent unilateral tinnitus spells without associated symptoms. **Probable CVCS**: possible CVCS with associated symptoms (otalgia, vertigo or hemifacial spasms) or MRI demonstrating vascular compression of cochleovestibular nerve (using high resolution heavily T2-weighted CISS images) or abnormal ABR (long I-III, absent wave II) **Definite CVCS**: probable CVCS with associated symptoms and/or abnormal ABR and/or abnormal MRI **Certain CVCS**: definite CVCS with surgical proof.

therefore required which allows a complete evaluation of the auditory and non-auditory causes and influences of tinnitus.

The Tinnitus Research Initiative group (TRI) proposed a guideline recommending examinations. A level of significance accompanies each of the recommended examinations (Table 4a).[37]

In general the auditory system is evaluated clinically, functionally and morphologically.

All available diagnostic means will be used, starting from a specific and general history, followed by the clinical ear, nose, throat and head and neck examination. Particular attention is paid to the auditive, vestibular and masticatory systems and infections of the upper airway.[38]

A general neurotological examination is conducted.[39]

Blood pressure is taken, both supine and standing.

Neuro-imaging, including MRI, should be performed. In a recent survey of patients with cochleovestibular complaints, about 6% of the patients were found to have an organic pathology and 50% of the patients over 50 years and 21% of patients under 50 years had white matter lesions and neurovascular conflicts.[40]

Additional diagnostic tests are performed for specific indications based upon the findings of the medical evaluation.

Attention is paid to neuropsychological (and sometimes psychiatric) aspects, and in particular to feelings of anxiety and depression.[10] The use of questionnaires supports this evaluation (Table 4b).

Functional evaluation of tinnitus

The perception of tinnitus and tinnitus suffering should be considered separately. This is consistent with the conceptual framework of a comprehensive multimodal network of neurons involved with attention, memory, cognition and the limbic system with emotion.[24,35,41]

The various aspects of tinnitus are described as precisely as possible on the basis of the patient's experiences. We make a distinction between the description of the perceived sound itself, the impact of testing methods, the effect of tinnitus on quality of life, and the influence of environmental factors or circumstances. Many different test methods are used, with the most common being mentioned here.

In conclusion, we describe two specialised examination methods that affect the tinnitus sensation. These are electrical transtympanic promontory stimulation and transcranial magnetic stimulation (TMS).

Anamnesis data

Sudden or progressive onset, permanent or intermittent?

Duration of first tinnitus experience.

Determination of the site where the tinnitus is perceived: the left or right ear, both ears or centralised within the head.

Temporal characteristics: continuously the same volume of sound, irregularly fluctuating, pulsatile murmur of vessels (sharp arterial, dull venous murmur).

Quality of sound: pure tone, noise, polyphonic sensations. Registration of exact description given by the patient, including comparison with existing sounds or, alternatively, including mimicking of the sound by the patient.

Psycho-acoustic characteristics

Pitch matching

In unilateral tinnitus, sounds are directed to the contralateral ear in such a fashion that comparable pitch and sound characteristics (murmur, tone) are obtained. In bilateral tinnitus, each ear is tested separately. The perception of the tinnitus can be a combination of many frequencies or can consist of a set of frequencies. Matching the tinnitus to the external sound source can be a difficult process and results can vary from one test session to another.[42]

Loudness expressed on a Visual Analogue Scale (VAS) with equal-appearing interval scales.[43] The patient is asked to indicate the loudness of the noise on a scale of 0 (no tinnitus) to 10 (maximum intensity).

Table 4a

Patient assessment

TRI Consensus for patient assessment and outcome measurements (following TRI workshop 2006).[37]
In each category, recommendations are ordered according to their level of significance:
A: Essential B: highly recommended C: might be of interest
Physical Examination
A: Otological examination by a specialist
A: Examination of the neck (range of motion, tenderness, muscle tension...)
B: Examination of the temporomandibular function
Audiological Assessment
A: Audiometry (pure tone threshold; up to 8 kHz)
B: Immitance Audiometry
B: High-Frequency Audiometry (at least up to 12 kHz)
B: Otoacoustic Emissions
B: Loudness Discomfort Level
C: Auditory Evoked Potentials
Psychophysical Measures of Tinnitus
B: Loudness match
B: Pitch match
B: Maskability (MML)
B: Residual Inhibition

Table 4b

Questionnaires

TRI Consensus for patient assessment and outcome measurements (following TRI workshop 2006).[37]
In each category recommendations are ordered according to their level of significance:
A: Essential B: highly recommended C: might be of interest
A: Validated questionnaire for the assessment of tinnitus severity, which at present can be THI, THQ, TRQ or TQ
B: Assessment of tinnitus severity by additional questionnaires, and especially by the THI, because it is believed that the THI has been validated in most languages
C: Assessment of depressive symptoms
C: Assessment of anxiety
C: Assessment of quality of life
C: Assessment of insomnia.

Tinnitus level in dB HL

Sounds with a similar frequency and characteristics are first presented to the contralateral ear. This makes it possible to determine in HL a loudness similar to that perceived in the tinnitus. This stimulus is then presented to the ipsilateral ear in order to determine the ipsilateral hearing threshold. The difference equals the sensation level (SL). Both SL and the absolute value expressed in dB HL are given. The correlation between tinnitus severity and the SL is poor.[44]

Influence of testing situations

Inhibition after ipsilateral masking sound exposure.

No, partial or complete residual inhibition or a rebound phenomenon: it is assumed that, while peripheral tinnitus can be masked partially or completely, this is not the case with central tinnitus.

Length of residual inhibition when present.

Factors that influence tinnitus loudness

- teeth clenching and grinding
- muscle contractions in the head-neck area
- stress
- exposure to loud noise
- physical exercise
- influence of airway infections (rhinitis, sinusitis, pharyngitis)
- fatigue
- the use of caffeine, alcohol, salt or tobacco
- changes in position (lying down or standing up)
- waking up
- environmental silence

Influence on health-related quality of life

The impact of tinnitus on quality of life is evaluated using validated questionnaires. These can be generic (meaning that they are applicable to various disorders) or disease-specific (*in casu* for tinnitus).

The disadvantage of using generic questionnaires is that they are relatively insensitive in certain afflictions.[7] A frequently used questionnaire in medicine is the SF-36.[45]

Two widely used questionnaires are the Tinnitus Questionnaire (TQ)[46] and the Tinnitus Handicap Inventory (THI).[47] These questionnaires approach multiple dimensions of quality of life.

The Tinnitus Questionnaire has the following subscales: emotional and cognitive distress, intrusiveness, hearing difficulties, sleeping difficulties and somatic complaints.

The Tinnitus Handicap Inventory has an emotional, functional and catastrophic subscale. The total score of the scales shows a very good correlation (Pearson correlation coefficient 0.9).[48]

The TQ has been validated in Dutch and a French version is provided with a back-translation.[43]

Recently, the multiple activity scale for hyperacousis (MASS), a validated questionnaire for hyperacousis assessment in daily circumstances, was designed as an interview-based instrument.[5] The hyperacousis questionnaire, on the other hand, is a self-assessment questionnaire. It contains 14 questions with a maximum score of 42 (maximum hyperacousis), with a score greater than 28 appearing to represent strong auditory hypersensitivity.[6]

Figure 2
Transcranial Magnetic Stimulation (TMS) of the auditive cerebral cortex.

Specialised tinnitus tests

Electrical transtympanic promontory stimulation

This test determines whether electrical stimulation of the promontory can influence the tinnitus.

A needle electrode is positioned through the eardrum and onto the promontorium, close to the round window. The positioning is done under micro-otoscopical control and after local anaesthesia of the eardrum. The set-up is comparable to that for electrocochleography. An alternating current with frequencies varying between 50 and 1600 Hz is applied by means of the electrode. The intensity of the current varies between 0 and 500 µA. The patient indicates if he perceives a sound sensation and if he notices a difference in the tinnitus sensation.

Psycho-acoustic characteristics like dynamics, adaptation and frequency discrimination, as well as auditory perception, are also measured.[49] A normal audiogram does not exclude "dead zones" and therefore further screening using the Threshold Equalising Noise (TEN) test may be mandatory.[50]

Transcranial magnetic stimulation (TMS)

The aim is to suppress cortical hyperactivity and the concomitant tinnitus. TMS has been used successfully to influence the loudness of tinnitus.[51] TMS appears to be more effective if the tinnitus is of shorter duration.[26]

A strong pulsating magnetic field is selectively applied over the auditory cerebral cortex with a coil, after which it is determined if this procedure can influence the tinnitus. Both the amount of suppression and the optimal stimulation frequency to obtain this suppression is determined. The aim of this procedure is to influence the cortical hyperactivity.

A specially designed apparatus generates magnetic field pulses through the use of a field intensity of between 1.5 and 2.5 Tesla with a coil. By positioning this coil over a region of the skull, the underlying cerebral tissue will be modulated over an area of approximately 3 cm in diameter and anywhere between 2 and 3 cm in depth (Figure 2).

Sound-proof earplugs are given to the patient because the device generates sound clicks with every magnetic stimulus.

First of all, the stimulation threshold of the motor cortex is determined (muscle contractions are induced by stimulation). Subsequently, the upper part (area A2) of the temporal auditory cortex is stimulated at 90% of this intensity.

Successive stimulations of 1, 5, 10, 20 and 40 Hz are applied. The patient is asked to indicate the percentage of tinnitus reduction or exacerbation at the different stimulation frequencies. A placebo stimulation is also performed (the apparatus still generates the clicking sound, but a magnetic field is completely absent or directed away from the patient). This allows the investigator to determine the true level of suppression.

The stimulus can be of a sinusoidal or burst type and the influence of the clear tone variant and the murmur variant are tested.

Epilepsy is a contra-indication for this examination.[52]

Conclusion

In case of tinnitus, first a medical diagnosis has to be made of the underlying disease. The specific underlying pathophysiological processes then have to be understood. The assessment of tinnitus characteristics is complementary to the medical diagnosis and allows for a quantification of long-term follow-up. As well the psycho-acoustics of the tinnitus – in particular frequency and loudness – as the burden and distress associated with the tinnitus must be determined. The TQ is recommended for the latter.

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