

# Chapter 2

## Different Forms of Tinnitus

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### Keypoints

1. Subjective tinnitus has many forms and may be regarded as a group of disorders rather than a single disorder.
2. There are a few objective ways to distinguish between the different forms of tinnitus.
3. Tinnitus has been classified subjectively according to:
  - (a) Intensity: Often using a visual analog scale or loudness matching.
  - (b) Character: High frequency (like crickets), low frequency (rumbling), tonal, pulsatile, constant, or intermittent.
  - (c) Other features such as the ability to modulate the tinnitus by manipulating their jaw, moving their eyes, or applying pressure on neck regions.
  - (d) Whether referred to one ear, both ears, or perceived as being inside the head.
4. Some diseases, such as Ménière's disease, are accompanied with tinnitus; such tinnitus may be different from other forms of tinnitus.
5. Some forms of tinnitus are associated with affective disorders such as depression or phonophobia.
6. Subjective tinnitus is often accompanied by abnormal perception of sounds, known as hyperacusis (lowered tolerance for sounds) or hypersensitivity to sounds.

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**Keywords** Objective tinnitus • Subjective tinnitus • Somatosensory tinnitus • Modulation of tinnitus • Abnormal perception of sounds

### Abbreviations

AVM	Arterio-venous malformations
EEG	Electroencephalography
MEG	Magnetoencephalography
TMJ	Temporomandibular joint

### Introduction

Subjective tinnitus is a broad group of sensations that are caused by abnormal neural activity in the nervous system that is not elicited by sound activation of sensory cells in the cochlea. Subjective tinnitus is by far the most common kind of tinnitus. Subjective tinnitus is phantom sounds that have similarities with the phantom limb symptoms and central neuropathic pain (see Chap. 14) [1, 2].

It is a general problem that the same name (tinnitus) is used for so many different forms of subjective tinnitus with different characteristics, different severities, and different causes. Having the same name used for fundamentally different disorders, such as the different forms of tinnitus, is an obstacle in treatment as well as research. The fact that tinnitus is not a single disorder but many makes epidemiological studies difficult to interpret. Different epidemiological studies have come up with very different numbers for the prevalence of tinnitus to some extent, because different definitions of tinnitus and its severity were employed in different studies.

It is agreed that the incidence of tinnitus increases with age and is more common in people who have had exposure to loud noise. Studies of the prevalence of

tinnitus in individuals above the age of 50 years have shown values from 7.6% to 20.1% (see Chap. 5).

In general, subjective tinnitus has no physical signs, and there are no objective clinical diagnostic tests that can distinguish between the different forms of subjective tinnitus. Only the patient's own description can serve as a basis for a clinical evaluation. Only recently have laboratory research methods been developed that might provide some insight into the different anatomical locations of the abnormalities associated with different forms of tinnitus. Neuroimaging methods are now beginning to provide some information on the functional changes in the brain of individuals with tinnitus (see Chap. 18). Electrophysiologic tests (electroencephalography, EEG, and magnetoencephalography, also known as MEG) can provide some information about plastic changes in the brain associated with tinnitus (see Chap. 20). These methods may become the basis for future clinical tests that can make a differential diagnosis of the different kinds of tinnitus possible and then relate it to pathology.

## Subjective for Objective Measures of Tinnitus

Loudness matching and the use of a visual analog scale have been used for estimations of the loudness of an individual's tinnitus. However, loudness matching results in unrealistically low values [3–5]. The use of a visual analog scale seems to give more realistic values.

In the absence of objective tests, tinnitus has been classified according to its perceived severity. Reed classifies tinnitus into three broad groups: mild tinnitus, moderate tinnitus, and severe chronic tinnitus [3]. Mild tinnitus is defined as tinnitus that does not interfere noticeably with everyday life, moderate tinnitus may cause some annoyance and may be perceived as unpleasant, and severe chronic tinnitus affects a person's entire life. These classifications rely on the individual person's own description of their tinnitus. Similar classifications have been used for pain (see [6]).

## The Anatomical Location of the Physiological Abnormality

Like other phantom sensations, such as phantom limb syndrome, tinnitus is often referred to a different

anatomical location than that of the pathology. Since tinnitus has the character of sound, it is often referred to one or both ears. Naturally, tinnitus has been regarded as a pathology located in the ear. Therefore, individuals with tinnitus often seek medical assistance from an ear specialist. However, examination of the ear in most cases finds nothing to be wrong. Also much of the research conducted early had been directed to the ear for studies of the pathology of tinnitus.

The anatomical location of the physiological anomaly of subjective tinnitus is often unknown and is likely to be different from where the tinnitus is referred (one ear, both ears, or in the middle of the head). Instead, the anatomical location of the abnormality that causes tinnitus is the brain. However, it is not obvious which region of the brain the pathology is located, and the abnormal function is not necessarily restricted to regions that are normally activated by sound stimulation.

Many forms of tinnitus are caused by activation of neural plasticity, which makes it difficult to identify the cause and the location of the primary pathology.

Activation of neural plasticity may change many neural processes, re-route information, alter the relation between inhibition and excitation, and change temporal coherence of activity in the population of neurons that may be involved in different forms of tinnitus.

It is possible that different characteristics of tinnitus distinguish the different kinds of tinnitus. There is recent evidence that the pathology of tinnitus that is pulsating is different from tinnitus that is not pulsating (see Chap. 59).

The pathology of tinnitus that is caused by external factors may be different from tinnitus that occurs without external factors being involved.

Deprivation of sensory input may constitute such external factors. It is known to be powerful in turning on neural plasticity, and there are many examples of how restoring input to the auditory nervous system can alleviate tinnitus [7] (see Chaps. 74 and 77). The fact that these methods provide relief from tinnitus supports the hypothesis that neural plasticity has been activated by the absence of signals to the nervous system.

Tinnitus occurs together with age-related hearing loss (see Chap. 36) and noise-induced hearing loss (see Chap. 37), as well as after administration of ototoxic antibiotics, some diuretics (furosemide), and quinine [8].

Tinnitus caused by noise exposure may normally abate after ending the exposure, but the tinnitus may sometimes remain present after ending exposure and may last indefinitely, which indicates that generation of tinnitus is caused by a stable pathologic state of neural circuits. These neural networks, which generate that kind of tinnitus, have bistable properties: one normal and another pathologic.

Exposure to loud sounds can cause tinnitus (see Chap. 37), and so can administration of ototoxic drugs. It is not known if the cause is the reduction in input to the auditory nervous system that turns neural plasticity on, or if it is overstimulation or possibly the morphological damage from overstimulation that activates neural plasticity.

There is evidence that the pathology of subjective tinnitus that occurs in Ménière's disease (see Chaps. 38 and 60) is different from other forms of tinnitus because it can be reduced or eliminated by sympathectomy [9], which has not been shown effective in other kinds of tinnitus. Tinnitus in Ménière's disease may therefore be a specific form of tinnitus that is different from other forms.

Tinnitus almost always occurs together with vestibular schwannoma (earlier known as acoustic tumors) (see Chap. 39). There are reasons to believe that the pathology of these forms of tinnitus is also different, although studies have not been published that could support this hypothesis. It has also been shown that there are other specific differences between the tinnitus that accompanies vestibular schwannoma and other forms of tinnitus. Thus, Cacace (1994) found some specific signs that occurred regarding tinnitus after operations for vestibular schwannoma [10], consisting of gaze-evoked or gaze-modulated tinnitus (see Chap. 39). He ascribed it to a phenomenon of deafferentation-induced plasticity. Acoustic schwannoma is one of the few risk factors for tinnitus that is almost 100%. The tinnitus does not normally disappear after removal of the tumor [11]. Injury of the auditory nerve from trauma, surgical operation, or viral infection (neuritis) is also associated with a high risk of tinnitus.

Traumatic head injuries are often associated with tinnitus.

Tinnitus often occurs with migraine headaches. It seems likely that the pathology of these forms of tinnitus differs from each other, although studies have not confirmed this hypothesis.

Tinnitus also accompanies disorders such as temporomandibular joint (TMJ) [12, 13]. Tinnitus, in connection with TMJ disorders, often disappears when the TMJ disorder is treated successfully (see Chap. 95). The pathology of these forms of tinnitus may be related to the anatomical connections between the trigeminal caudal nucleus and cochlear nuclei [14] (see Chap. 9).

Tinnitus often accompanies neck disorders (see Chap. 80) [15]. However, there is often no known cause to the tinnitus (idiopathic tinnitus). These forms of tinnitus are known as "somatosensory tinnitus," and the reason for this abnormal cross-modular interaction may be the involvement of the nonclassical pathways. The pathology of these forms of tinnitus is likely to be different and therefore require different kinds of treatment. The pathology may be related to the anatomical connections between the upper spinal cord ( $C_{2-4}$ ) and cochlear nuclei (see Chap. 9).

Subjective tinnitus is often accompanied by an abnormal perception of sounds, such as hyperacusis (decreased tolerance for sounds in general, see Chap. 3), phonophobia (fear of sound), and misophonia (dislike of specific sounds) (see Chap. 4). Some individuals with tinnitus hear sounds as being distorted, spoiling the enjoyment of music. This distortion may also make it difficult to understand speech.

Many individuals who have tinnitus (about two-thirds) can modulate their tinnitus by signals from the somatosensory system, such as from eye movements [16], manipulations of their jaw, and applying various pressure on specific neck regions [17–19]. These forms of tinnitus can be managed by somatosensory-oriented treatment [20], and such individuals may be a subgroup with a different pathology.

Affective symptoms accompany some forms of tinnitus [21]. It seems likely that such forms of tinnitus are different from other forms and that their pathology may differ as well (see Chap. 62).

## Conclusion

Tinnitus is not a single disorder and the symptoms vary substantially. The causes of different individual's tinnitus also have wide variants. The fact that a disorder with such differences has the same name is an obstacle in studies of tinnitus and patient management.

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Textbook of Tinnitus

Møller, A.R.; Langguth, B.; DeRidder, D.; Kleinjung, T.  
(Eds.)

2011, XXX, 786 p. 157 illus., 57 illus. in color.,

Hardcover

ISBN: 978-1-60761-144-8