Chapter 58
Clinical Description of a Different Form of Tinnitus: Intermittent Tinnitus

Miguel J.A. Láinez, Anna Piera, and Alejandro Ponz

Keypoints

1. Intermittent (paroxysmal) tinnitus is a form of non-pulsatile tinnitus.
2. An intermittent nature can be the only sign that intermittent tinnitus is different from other forms of tinnitus.
3. Intermittent tinnitus may be accompanied by irregular symptoms of other neurotologic disorders.
4. Both objective and subjective tinnitus may be intermittent.
5. A wide range of pathologies may cause intermittent tinnitus, but the cause of most forms is unknown.

Keywords Paroxysmal (intermittent) tinnitus • Myoclonus • Temporomandibular joint changes • Cerebellopontine angle changes • Migraine • Auditory hallucinations • Audiogenic seizures

Abbreviations

ABR	Auditory brainstem response
AGS	Audiogenic seizures
CPA	Cerebellopontine angle
CSF	Cerebrospinal fluid
EEG	Electroencephalography
GABA	Gamma amino butyric acid
IC	Inferior colliculus
TMJ	Temporomandibular joint

M.J.A. Láinez (✉)
Department of Neurology, Hospital Clínico Universitario, University of Valencia (Spain), Avenida Blasco Ibáñez, 17, 46010 Valencia, Spain
e-mail: jlaineza@meditex.es

Introduction

Suddenly occurring non-pulsatile tinnitus (intermittent tinnitus) can be the only symptom or it can be accompanied by neurotologic symptoms like vertigo, headaches, visual changes, and disturbances of consciousness.

Like constant tinnitus, intermittent tinnitus can be objective or subjective, depending on the pathology; objective tinnitus is caused by physical sounds generated in the body, which can also be heard by an observer. Subjective tinnitus is caused by abnormal neural activity, and only the patient can hear the tinnitus.

Objective Intermittent Tinnitus

Some important pathology should be ruled out in patients who present with intermittent objective tinnitus. The most important disorders that may occur together with objective tinnitus are palatal and middle-ear muscle myoclonus and temporomandibular joint (TMJ) disorders.

Palatal and Middle-Ear Muscle Myoclonus

Tinnitus produced by middle-ear myoclonus is objective intermittent tinnitus, and is rare; only a few cases are reported in the literature. In middle-ear myoclonus, otoscopic examination shows visible rhythmic movements of the eardrum, and weak clicking sounds are heard in the ear by auscultation. Tympanometry confirms rhythmic changes in middle-ear compliance. Middle-ear myoclonus can be accompanied by palatal myoclonus or can be the only manifestation. Palatal

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myoclonus is an uncommon rhythmic "shock-like" involuntary movement of the muscles of the soft palate, throat, and other structures derived from the branchial arcs. Objective intermittent tinnitus associated with palatal myoclonus can be related to hearing impairment; however, this relation is not always present. Examination of muscles of the soft palate and throat shows rhythmic involuntary movements and, in some cases, spontaneous clicking sounds by auscultation near the ear [1–3].

Temporomandibular Joint Changes (Synchrony with Joint Movements)

The TMJ is a complex, sensitive, and highly mobile joint. Millions of people suffer from temporomandibular disorders. Tinnitus associated to TMJ changes is synchronic with joint movement and is easy to provoke during the examinations. Several disorders of TMJ cause tinnitus: luxation, condyle malposition, bruxism, degenerative arthropathy, capsulitis, and many others. These disorders are also very common in inflammatory arthropathies. Regardless of the cause of a TMJ disorder, all of them can be common causes of intermittent tinnitus, and a routine examination for these disorders is needed in all tinnitus clinics [4–6].

Subjective Intermittent Tinnitus

Subjective intermittent tinnitus, which is much more common than objective tinnitus, can occur together with pathologies such as cerebellopontine angle (CPA) disorders.

Cerebellopontine Angle Disorders

CPA disorders may be suspected in patients with unilateral hearing loss and unilateral intermittent tinnitus with or without dizziness. Audiological and imaging studies of the posterior fossa are used to rule out disorders of the CPA. Lesions of the CPA are frequent and represent 6–10% of all intracranial tumors. Vestibular schwannoma (acoustic neuroma) and meningioma are the two most frequent lesions and account for approximately 85–90% of all CPA tumors. The other 10–15% encompasses a large variety of lesions including aneurysms, epidermoid cysts, arachnoidal cysts, Arnold–Chiari malformations, lipoma, and melanomas. Such lesions are now detected more frequently because of the sensitivity and accuracy of magnetic resonance imaging (MRI) [7–10].

Recently, Levine has described a subtype of intermittent tinnitus, called typewriter tinnitus, with an excellent response to treatment with carbamazepine, in which vascular compression of the auditory nerve was suspected to be the cause in five of six patients that were studied. This suggests that surgical decompression may also be effective in such patients [11].

Audiogenic Seizures and Epilepsy

The inferior colliculus (IC) plays an important role in many pathophysiological conditions that involve hearing (including tinnitus, age-related hearing loss, and audiogenic seizures (AGS)). AGS occur frequently in rodents and can be genetically mediated. AGS can also be readily induced in experimental animals [12]. AGS can be induced in normal animals by administration of drugs that are GABA receptor antagonists. Glutamate-mediated excitation is a critical element of neurotransmission in IC neurons, and excessive activation of glutamate receptors in the IC is implicated in AGS. Such neurotransmitter abnormalities cause excessive firing of IC neurons that act as the critical initiation mechanism for triggering seizures in response to intense acoustic stimuli, thus AGS. The IC plays a role in the integration of acoustic-motor and acoustic-limbic integration, as well as in acute and chronic AGS. García-Caraisco et al. [13] have demonstrated in animal experiments that chronic kindled AGS change behavioral expressions in a similar way as those that occur in temporal lobe epileptic seizures mixed with audiogenic seizure activity, which is known to be dependent on brainstem networks. This form of AGS involves subcortical intermittent pattern of tinnitus manifestation [14, 15].

Tinnitus, as an intermittent pathologic cortical manifestation, has been described in only a few patients while electroencephalography (EEG) was monitored, and was determined that the tinnitus originated from the contralateral mid-temporal area [16].

Auditory Hallucinations

Auditory hallucinations can be simple or complex. Therefore, tinnitus could be considered an auditory hallucination. Several studies with transcranial magnetic stimulation have reported a benefit in improving both auditory hallucinations and tinnitus by modulating cerebral cortex activity. New results of studies and new questions about the neurobiological basis of mental and neural disorders have concerned whether there is a common substrate in tinnitus and auditory hallucinations [17, 18].

Migraine with Basilar Aura

Vertigo, dysarthria, and tinnitus may occur together with basilar aura in individuals with migraine. It has been reported to occur in 50% of individuals with basilar aura. Other symptoms are diplopia, bilateral visual symptoms, bilateral paresthesia, hearing loss, decreased level of consciousness, and ataxia. For management of patients with tinnitus and headache, it is important to obtain information about how the tinnitus may be regarded as a symptom that precedes headache [19–21].

Cerebrospinal Fluid Pressure Changes

Both intracranial hypotension and hypertension (pseudotumor cerebri) have been suggested as possible causes of intermittent tinnitus. A lumbar puncture is needed to measure pressure changes in cerebrospinal fluid (CSF), and in most cases, a neuroimaging technique (MRI or CT) is necessary to rule out other brain lesions [22–24].

Phantom Sensations Without Evidence of Cortical or Auditory System Dysfunctions

In spite of the many different pathologies that may cause intermittent tinnitus, in some patients no pathology can be found and the tinnitus remains a phantom intermittent sensation – an expression of an abnormally high correlation of activity in many nerve cells in cortical and subcortical parts of the auditory system [25, 26].

Techniques used in Diagnosis of Intermittent Tinnitus

Several different techniques are useful in the diagnostic workup of patients with intermittent tinnitus; the most important are MRI, auditory brainstem responses (ABR), and in some patients, EEG.

Magnetic Resonance Imaging

MRI is performed in almost all patients with intermittent tinnitus in order to rule out CPA disorders, cortical ectopias, and indirect signs of benign intracranial hypertension of licuoral hypotension.

Electroencephalography

EEG is only indicated if there are further signs of seizure and when tinnitus is accompanied by symptoms of consciousness disturbance.

Basal EEG recordings performed with provocation maneuvers like flashing light and hyperventilation in some cases may be useful. EEG can help determine if temporal lobe discharges are present.

Role of Auditory Brainstem Responses

ABR are indicated only in intermittent tinnitus for screening auditory nerve compression and can provide prognosis for microvascular compression [27].

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