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When alarm bells ring: emergency tinnitus

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Abstract. – OBJECTIVE: The aim of this study is to develop a diagnostic-therapeutic algorithm for those suffering from tinnitus who seek emergency aid.

MATERIALS AND METHODS: A literature review has been performed on articles from the last 30 years.

RESULTS: It is important to activate medical or surgical diagnostic and therapeutic strategies, in order to safeguard and rehabilitate the various functions affected. Psychiatric comorbidity is the most frequent pathological condition of those with serious or catastrophic tinnitus. In these cases, mortality risk is linked to suicide, morbidity to tinnitus-correlated distress.

CONCLUSIONS: Tinnitus, mainly linked to loss of hearing, is a frequent symptom among the population at large. About 7% of those affected by tinnitus turn to their doctor to solve their problem, while between 0.5 and 2% request urgent medical assistance. Their cry for help may be the result of an acute onset of tinnitus or the rapid impairment of an already chronic condition. Tinnitus is not considered an urgent ear, nose and throat (ENT) condition by the Associazione Otorinolaringologi Ospedalieri Italiani (AOOI) [Italian Association of Hospital ENT], even though there are many pathological conditions, sometimes serious, associated with tinnitus and emergency action is necessary to reduce the risk of morbidity and mortality.

Key words:
Tinnitus, Decompensated tinnitus, Urgency, Emergency, Psychiatric comorbidity, Suicide.

Introduction

Tinnitus can be defined as the perception of a sound in the absence of an external stimulus[1,2]. The word comes from the Latin "tinnire" (to ring)[3,4] and is usually described by the patient as a "ringing in the ears"[5].

Tinnitus is perceived as a subjective hearing sensation in one or both ears and/or in the head[5,6], not produced by an external source, and can be reported as a simple (whistling, hissing, buzzing, etc.) or as a complex sound[7]. Tinnitus may be continuous, discontinuous or intermittent and, according to its characteristics, pulsatile or non-pulsatile.

Tinnitus is a symptom, not a disease. Over the years, various classifications have been proposed: the most common differentiates between objective and subjective tinnitus.

Objective tinnitus is rare and is found in less than 1% of cases[8]. It is audible when a careful examination is made of the ear and the temporal or cervical regions[9,10]. The sound generally comes from internal biological activity such as vascular turbulence: pulsation[11]; convulsion of the muscles in the middle ear, Eustachian tube and soft palate; and temporomandibular joint dysfunctions[9].

Some authors claim that it is incorrect to term these phenomena "tinnitus", and prefer "somatosounds". For convenience, the term tinnitus will be used to include objective tinnitus in this study.

Subjective or internal auditory tinnitus is much more common than external auditory tinnitus and is found in approximately 20% of the population. It is perceived as a sound or noise and can be called true, intrinsic, auditory, non-vibratory tinnitus, or phantom sounds[1]. Unlike objective tinnitus, it originates in the acoustic pathways.

Depending on the time length, tinnitus may be acute, subacute or chronic. It is acute up to three months and subacute up to six months. If it persists for more than six months, it is chronic (even though some authors already consider it so after three months[8]). In 1999, Zenner and Pfister[12] proposed an innovative and systematic classification of tinnitus, based on knowledge of the anatomy and physiology of the hearing system. They made

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a first distinction between subjective and objective tinnitus, subsequently further distinguishing subjective tinnitus, based on its anatomical site, as conduction, sensorineural and central tinnitus[12].

According to some authors[7,13] and depending on the patient's sequelae, tinnitus can be divided into "clinical" (decompensated) or "nonclinical" (compensated).

Patients can cope with compensated tinnitus and suffer little or no psychological stress. Their quality of life remains more or less the same.

Decompensated tinnitus, found prevalently in 1-2.4% of the adult population[7,12], causes a high level of psychological stress and emotional overlay. Patients' quality of life is seriously compromised.

Our group recently proposed a clinical-neuroplastic classification of tinnitus, called Tinnitus Holistic Simplified Classification (THoSC)[14], which focuses on a more rational diagnostic and therapeutic management that is, in our view, nearer to clinical reality.

We classified tinnitus as:

- Audiologic or deafferentation tinnitus – caused and sustained by hearing loss;
- Somatosensory tinnitus – caused, sustained and modulated by cross-modal somatosensory tinnitus in patients without a clear profile of hearing loss, where a tinnitogenic trigger can be recognized from non-auditory devices;
- Psychogenic tinnitus – caused, sustained and modulated by psychological disorders or psychopathological conditions;
- Combined classes – tinnitus caused and sustained by at least two of the three previous factors.

Tinnitus is frequently triggered by conditions that cause a reduction in auditory acuity (presbyacusis, acute and chronic acoustic trauma, otosclerosis, Ménière's disease, chronic otitis, sudden deafness, VIII cranial nerve schwannoma, etc.).

About 70% of hypoacusic patients are aware of tinnitus and about 95% of patients who suffer from tinnitus are hypoacusic. These data support the hypothesis whereby the sensory deafferentation of the auditory cortex constitutes, in many cases, the main risk factor for the creation of tinnitus, a phenomenon activating the deafferented areas.

An ear without a brain is unable to transmit and transduce or perceive sounds; a brain without an ear can be devastated by the perception of sounds.

Ten to fifteen% of the population is affected by chronic tinnitus[4,6], which may at times cause great stress[15], with anxiety, depression, difficulty in concentrating and sleep disorders[16].

There is a significant link between more serious tinnitus, anxiety and sleep disorders and general psychopathological level[17,18].

Patients who complain higher discomfort than the average person affected by tinnitus are the very ones who have a higher stress level.

Numerous studies highlight a link between the gravity of the tinnitus and psychometric parameters, particularly anxiety, depression and somatization[19,20].

Data from the literature highlight that between 26.70[21] and 77%[22] of patients with tinnitus manifest psychiatric comorbidity.

The risk of suicide in these patients is ten times more than that of the average person[23].

Psychiatric comorbidity and the risk of suicide are not the only elements that can lead patients with tinnitus to seek emergency counselling.

Tinnitus is a frequent symptom, principally linked to loss of hearing[24]. About 7% of patients affected by tinnitus ask for help in resolving the problem; between 0.5 and 2% require urgent medical assistance[7,25].

This need for help may be the result of an acute onset of tinnitus or the rapid impairment of an already chronic condition.

The aim of this study is to develop a diagnostic-therapeutic algorithm for those suffering from tinnitus who seek emergency aid.

Materials and Methods

A literature review has been performed on articles retrieved from PubMed from the last 30 years. Research was carried out using keywords such as "tinnitus and emergency", "tinnitus as urgency", "tinnitus and psychiatric comorbidity", in a time span from 1984 to 2015.

Results

Pathologies associated with tinnitus

The medical pathologies exhibiting tinnitus in their symptomatological characteristics are numerous, and are both of otological and non-otological origin (see Table I for further details). When the cause of this phenomenon is unknown, the term "idiopathic tinnitus" is used.

Table I. Common systemic neuro-otological risk factors for developing tinnitus.

Aetiopathogenesis	*Pathological characteristics*
Otological, infectious	Otitis externa, Otitis media, labyrinthitis, mastoiditis
Otological, neoplastic	Vestibular schwannoma, meningioma, vascular tumours
Otological, labyrinthine	Sensorineural hearing loss, Ménière's disease, vestibular vertigo
Otological, other	Impacted cerumen, otosclerosis, presbyacusis, noise exposure, barotraumas, genetic deafness, sudden hearing loss
Neurological	Meningitis, migraine, multiple sclerosis, epilepsy
Traumatic	Head or neck injury, loss of consciousness
Otofacial	Temporomandibular joint disorder
Cardiovascular	Hypertension, vascular disorders, cerebrovascular disorders
Rheumatological	Rheumatoid arthritis
Immune mediated	Systemic lupus erythematosus, systemic sclerosis
Endocrine and metabolic	Diabetes mellitus, hyperinsulinaemia, hypothyroidism, hormonal changes during pregnancy
Ototoxic medications	Analgesics, antibiotics, antineoplastic drugs, corticosteroids, diuretics, immunosuppressive drugs, non-steroidal anti-inflammatory drugs, steroidal anti-inflammatory drug
Psychiatric disorders	Depression, anxiety and somatization

Some of these pathologies may constitute urgent cases, and this is why it is particularly important for a patient with acute tinnitus to be thoroughly assessed.

At times, the characteristics of tinnitus may direct a clinician towards the possible underlying pathology (see Table II for further details).

Tinnitus may be pulsatile or non-pulsatile. This differentiation is the one mainly adopted in the literature or diagnostic-therapeutic algorithms. We have chosen to follow this model for its practical implications in a clinic.

Pulsatile tinnitus

Pulsatile tinnitus synchronized with heartbeat is rare[26], potentially disabling, and can have both vascular and non-vascular aetiology. When it is vascular, it may be caused by turbulence in the bloodstream from increased volume or an irregular vascular lumen.

Tinnitus is probably of arterial origin if it gets worse with light compression of the ipsilateral jugular vein while it is probably venous if it improves with compression or by rotating the head in the direction of the tinnitus – both these move-

Table II. Characteristics of tinnitus in relation to pathologies.

Otoneurological pathology	*Tinnitus characteristics*
Tubal dysfunction	Murmuring, synchronous with respiration
Myoclonus of muscles in soft palate and middle ear	Jerks, lasting several seconds or minutes
Congenital vascular malformation or malformation acquired through head and neck, reverberating circulation in vessel near ear	Throbbing, synchronous with heart beat
Ménière's disease	Less frequent tinnitus, preceding vertigo attacks, then giving way to moment of calm
Otosclerosis	Continuous tinnitus more often at low frequency, sometimes pulsatile and intermittent
Presbyacusis, ototoxicity, noise trauma	Bilateral and high-frequency tinnitus
Schwannoma VIII CN	Unilateral and high-frequency tinnitus
Middle or outer ear	Usually low-frequency or "white noise" tinnitus, associated with transmissive hearing loss
Cochlear	Usually high-frequency tinnitus, often corresponding to damaged frequency, associated with neurosensory hearing loss
Central nervous system	Tinnitus with varying frequencies, often with accompanying neurological signs and symptoms

ments cause a reduction in jugular flow. Cases described in the literature usually pertain to benign buzzing in the veins, but sometimes severe conditions may be present, such as arteriovenous malformations, glomus tumours and carotid stenosis (see Table III for further details).

Arteriovenous fistulae with pulsatile tinnitus are common[27]. In 2008, Ali et al[28] described the case of a 66-year old patient who not only had pulsatile tinnitus, but also pain in the left ear, exophthalmos, conjunctivitis, diplopia and decreased visual acuity. Computed tomography (CT) and magnetic resonance (MRI) showed a cavernous left fistula. The patient was treated with detachable platinum coils and n-butyl cyanoacrylate and symptoms cleared up completely.

Pulsatile tinnitus may be the warning of possible carotid dissection, for which an urgent diagnosis is important in order to help long-term prognosis and prevent ischemic complications. In this case, symptoms normally are face and neck pain, headache, unilateral pulsatile tinnitus, amaurosis fugax, Horner's syndrome, retinal infarction and cerebral ischaemia of the anterior circulation[29].

In 2007 Nakagawa et al[30] described the case of a 48-year old woman who, as a result of a road accident, suffered from diplopia and pulsatile tinnitus in the left ear. An angiogram revealed a cavernous carotid fistula and an extracranial dissection of the internal carotid artery. The patient underwent endovascular stent angioplasty, with complete resolution of the symptoms.

An acute ischaemic stroke of the anterior inferior cerebellar artery can be associated with pulsatile tinnitus, hearing loss, ataxia, nystagmus and hypoalgesia.

In 2001 Lee et al[31] described the case of a 66-year old man who, a week before the onset of dysarthria, facial paralysis and ataxia had displayed sudden bilateral hearing loss, unilateral tinnitus and vertigo. The severity and persistence of the hearing loss caused the authors to conclude that loss of hearing was probably due to hypoperfusion of the inner ear artery, with relative labyrinthine infarction.

Kotan et al[32] described the case of a 42-year old stroke patient who came to the emergency room complaining of tinnitus and vertigo and who, after magnetic resonance angiography, was diagnosed with bilateral vertebral stenosis. About 25% of strokes are caused by posterior circulation or the vertebrobasilar system. The symptoms of a vertebrobasilar ischaemia may be clinically observed as tinnitus, vertigo, diplopia, migraines, hypokinesis and hearing difficulties. Despite its

Table III. Pulsatile tinnitus.

Correlated pathologies	Symptoms
Arteriovenous fistulae	Pulsatile tinnitus, earache, exophthalmos, conjunctivitis, diplopia, loss of visual acuity
Carotid dissection	Face and neck pain, headache, unilateral pulsatile tinnitus, amaurosis fugax, Horner's syndrome, retinal infarction, cerebral ischaemia in anterior circulation
Acute ischaemic stroke of anterior inferior cerebellar artery	Pulsatile tinnitus, hearing loss, ataxia, nystagmus, hypoalgesia
Aneurysms of dural venous sinuses	Pulsatile tinnitus, homolateral pain above ear
Aneurysm of intrapetrous carotid artery	Pulsatile tinnitus, hypoalgesia, signs of obstruction in Eustachian tube
Ruptured aneurysm of the noncoronary sinus of Valsalva in the right atrium	Unilateral tinnitus, dyspnoea, palpitations
Duplicated internal carotid arteries (ICA)	Pulsatile tinnitus, loss of hearing
Benign vascular tumours	Pulsatile tinnitus, otorrhagia, unilateral hypoalgesia
Glioneuronal tumour	Pulsatile tinnitus, non-specific broad symptomatological spectrum, generally depending on size and extent of tumour
Cerebellopontine angle angiosarcoma	Pulsatile tinnitus, unilateral tinnitus, slight headache, hearing loss, hemiparesis, numbness, ataxia
Bilateral vertebral stenosis	Tinnitus, vertigo, diplopia, headache, hypokinesis, hearing disorders
Hypertensive crisis	Tinnitus, vertigo and headache, or severe such as dyspnoea, chest pains, coma or death
Glomus jugulare tumour	Pulsatile tinnitus, unilateral paralysis of the accessory nerve palsy homolateral to tumour

low risk of morbidity and mortality, prompt diagnosis and relative treatment are of fundamental importance.

Feitosa-Filho et al[33] underline how hypertensive crises and emergencies are the clinical situations representing more than 25% of all medical emergencies. Hypertensive crises are clinical situations followed by clinical signs and symptoms. About 3% of all visits to the emergency services are due to significant increases in blood pressure. Consequently, a hypertensive emergency is the most critical clinical situation, needing particular attention and care. Signs and symptoms may be slight, such as tinnitus, vertigo and migraine or severe, such as dyspnoea, chest pains, coma and/or death.

Aneurysms of dural venous sinuses may be considered rare. Only eight cases have been published, five of which were treated surgically and three by endovascular approach. Notably, Mehanna et al[34] described an additional case, treated successfully by endovascular coiling, of a 46-year-old woman affected for six years by pulsatile tinnitus that started after physical exercise, associated with a sharp pain above her right ear. Her symptoms worsened progressively to the point that she needed to compress her right jugular vein, ipsilateral to the tinnitus, or turn her head to the right in order to follow a conversation. The only sign noted was a murmur heard on auscultation over the right temporal bone. An angiographic evaluation revealed a high right jugular bulb, a dominant right lateral sinus with an irregular venous aneurysm at the proximal right sigmoid sinus. Endovascular treatment led to an 80% improvement in the patient's tinnitus and complete resolution of the temporal murmur.

An internal carotid aneurysm in the petrous temporal bone is a rare lesion: there are but 54 cases described in the literature. The most common symptoms are pulsatile tinnitus, hearing loss and signs of obstruction in the Eustachian tubes. Rupture of an aneurysm may provoke bleeding so intense that the common carotid artery may need emergency ligature[35]. In the case of an intrapetrous carotid artery aneurysm, it is necessary to intervene at the first ontological symptoms such as hearing loss and tinnitus, in spite of the surgical complexity[36]. Guenther et al[37] described the case of a 32-year-old woman with left-sided tinnitus, dyspnoea and sudden onset of palpitations. At the Emergency Department, echocardiography revealed a ruptured aneurysm of the noncoronary sinus of Valsalva in the right atrium. The patient subsequently underwent immediate surgery with a patch repair of the ruptured aneurysm, which enabled her to be discharged in good health after a few days.

Among vascular anomalies are duplicated internal carotid arteries (ICAs). Gartrell et al[38] presented the case of a 15-year-old male with an unusual ICA anomaly, where there was no evidence of tinnitus, vertigo or aural pressure. The patient's medical history was marked by recurrent acute otitis media and subsequent bilateral tympanostomy, with no consequent complications. He had been fitted with hearing aids since his preschool years. CT and MRI angiography revealed bilateral masses in both middle ear spaces, which could be visualized through the anterior inferior quadrant of the tympanic membrane, together with a long-standing mixed unilateral hearing loss. In these cases, establishing a correct diagnosis is paramount to avoid possible catastrophic haemorrhagic complications such as bleeding, hearing loss, or neurologic deficits.

An aberrant ICA is rare: there are only approximately 45 cases recorded to date. The majority of these cases, presented as a unilateral anomaly without duplication, were associated with pulsatile tinnitus and hearing loss. Bilateral aberrant ICAs are extremely rare, with only 14 existing reports. Only one of these cases was with duplicated ICAs. The case described by the authors represents the only example known of a bilateral duplicated ICA not associated with persistent stapedial arteries.

Conservative management of an aberrant ICA is generally advised, seeking to avoid middle ear surgery where possible. The patient should be followed closely and if, despite conservative treatment, bleeding occurs, embolization may be attempted. This is preferable, where possible, over ligation of the ICA. Embolization is a definitive treatment for arteriovenous malformation, aneurysms and angiomatous malformations. In inoperable tumours, embolization can relieve pain, bleeding, pulsatile tinnitus or discomfort due to mass effect[39].

Topal et al[40] report the case of two sisters in which they diagnosed fibromuscular dysplasia of the carotid artery at the same age and had the same symptom: pulsatile tinnitus. This is a non-inflammatory atherosclerotic vascular disease that can affect the arteries of the cervix, kidney and gut. The family presentation of this rare disease is indicative of a possible genetic etiology, but, at the moment, the exact pathophysiology is not yet known. In both cases a conservative treatment with aspirin was proposed.

With regard to benign vascular tumours, Nouri et al[41] describe the case of a 60-year-old woman with a capillary haemangioma, with symptoms of pulsatile tinnitus, otorrhagia and unilateral hearing loss. A clinical test revealed a reddish polypoid mass at the bottom of the ipsilateral external auditory canal, covered by a thin inflamed tympanic membrane. Conductive unilateral hearing loss was present. CT and MRI tests showed a vascular mass in the middle ear. The tumour was subsequently removed. Haemangiomas are benign vascular tumours that are relatively common in the head and neck. Their occurrence in the temporal bone, especially in the middle ear, is exceptional; they represent less than 0.21% of all temporal bone tumours. The tumour is usually ipsilateral, and symptoms may vary from an asymptomatic mass to a tumour with pulsatile tinnitus, otorrhagia, hearing loss, vertigo, recurrent otitis media, or paralysis of the facial nerve[42-44]. An otoscopy usually shows a reddish mass at the back of the tympanum or, more rarely, a polypoid mass in the external auditory canal. The main differential diagnosis is with paraganglioma of the middle ear[43,44]. Surgery is required for a complete resection of the tumour because of its destructive and haemorrhagic potential. The relatively high recurrence rate varies from 16 to 23%. It depends directly on the quality of the surgical resection[43] and demonstrates the importance of follow-up for these patients. Surgical excision is not always necessary, as shown in the literature, since congenital haemangiomas can regress spontaneously[43,45]. The surgical technique depends on the size of the tumour, degree of hearing loss and position of the jugular bulb[43,46,47]. Laser CO_2 treatment can be an interesting alternative to conventional surgery, allowing a better visualization of the middle ear structures and reducing bleeding[41,42,45].

The rosette-forming glioneuronal tumour (RGNT) of the fourth ventricle is considered rare. The symptomatological spectrum is wide, non-specific and generally depends on the size of the tumour and its extent. Despite benign histological features and favourable post-operative progress, there is still limited clinical experience with regard to this tumour, which needs careful differential diagnosis of possible posterior cranial fossa masses in order to avoid undue surgical aggressiveness[48].

Guode et al[49] describe the case of a 16-year-old girl with right-sided tinnitus, ipsilateral hearing loss and mild headache, which had gone on for a year. These subsequently worsened and, together with vomiting and other neurological symptoms (hemiparesis, numbness, ataxia) led to a diagnosis of angiosarcoma in the cerebellopontine angle, with haemorrhaging and oedema. The patient underwent an emergency suboccipital craniectomy to remove the tumour and had subsequent radiotherapy treatment. Primary intracranial angiosarcomas are rare and few cases are reported in the literature, mostly located in the supratentorial areas. This case highlights how important it is for clinicians to be aware of the characteristics of this type of tumour and the need to include it in the differential diagnosis of rare lesions located in the cerebellopontine angle[49].

Glomus jugulare tumours are rare, hypervascular tumours that are generally benign and, because of their slow and insidious clinical manifestation, are diagnosed late. They represent a significant diagnostic and management challenge to the clinician[50,51]. Auscultation of the cervical and temporal regions can reveal murmurs that help to localize the lesion. In these cases, MRI, CT or angiography are recommended, and the patient should be referred immediately to an ear, nose and throat specialist. Primary manifestations involve the ear apparatus and the lower cranial nerves caused by mass effect. In this regard, Seymour, Lloyd and Harcourt[51] describe the case of a subject with a glomus associated with a previous accessory nerve palsy on the same side as the tumour. Glomus tumours, as part of the chromaffin cell system, may also secrete catecholamines, with complications that may arise related to inappropriate catecholamine release[52]. Treatment options can include surgery, radiotherapy and embolization. Surgery, followed by radiotherapy[53] if necessary, is the treatment of choice. However, it can have serious potential complications linked with the position and the vascular nature of the tumour.

The clinician should always suspect a vascular lesion in patients with a retrotympanic mass; careful diagnostic imaging investigations should always be carried out before any middle ear surgical exploration.

Non-pulsatile tinnitus

Non-pulsatile tinnitus is usually caused by a non-vascular tinnitogenic source: hearing impairment, palatal and middle ear myoclonus, dysfunctions of the Eustachian tube, tympanic membrane, Temporo-Mandibular Joint (TMJ), cervical spine, etc. In the majority of cases, it is bilateral, while unilaterality may indicate a more serious clinical condition such as a vestibular or acoustic neuroma, or Menière's syndrome.

Table IV. Non-pulsatile tinnitus.

Correlated pathologies	Symptoms
Meningioma and vestibular neurinoma	Non-pulsatile tinnitus, acute loss of hearing, ataxia
Ménière's disease	Non-pulsatile tinnitus, vertigo, fullness, loss of hearing
Petrous apex cholesterol granuloma	Non-pulsatile tinnitus, loss of hearing, vertigo, headache, facial spasms, diplopia
Occipital neuralgia syndrome	Non-pulsatile tinnitus, migraine, pain in the distribution of the occipital nerve, vertigo, nausea, visual disturbances, scalp paraesthesia
Lumbar puncture	Non-pulsatile tinnitus, postural headache, nausea, vomiting, ocular disturbances
Subdural haematoma	Non-pulsatile tinnitus, persistent headache in homolateral region behind ear
Pneumolabyrinth	Non-pulsatile tinnitus, neurological and/or somatic symptoms
Idiopathic sudden sensorineural hearing loss (ISSNHL)	New onset unilateral non-pulsatile tinnitus, unilateral hearing loss
Autoimmune vestibulo-cochlear disorders (AVCD)	Non-pulsatile tinnitus, vertigo, fullness
Multiple sclerosis	Persistent non-pulsatile tinnitus, associated with reversible loss of hearing
Lightning or electrical damage	Non-pulsatile tinnitus, blindness, confusion, amnesia, cardiac arrhythmias, vascular instability
Myiasis of the external and middle ear	Non-pulsatile tinnitus, otalgia, otorrhea
Acute quinine toxicity	Non-pulsatile tinnitus, nausea, vomiting, prolungation of QT interval
Carbon monoxide (CO) poisoning	Tinnitus, hearing loss, vestibular disorders, cognitive sequelae, anxiety, depression, headache, insomnia, fatigue, visual acuity disturbance, peripheral neuropathies and Parkinsonian-like syndrome
Overdose of salicylates, tricyclic antidepressants and benzodiazepine	Non-pulsatile tinnitus, dizziness, numbness of the tongue, visual disturbances, muscle spasms, convulsions, reduced consciousness, coma and respiratory arrest
Heart attacks (particularly anterior)	Non-pulsatile tinnitus, dizziness, headache, weakness, cough
Acute acoustic trauma (AAT)	Non-pulsatile tinnitus, frequent selective hearing loss
Auricular foreign body	Unilateral non-pulsatile tinnitus, dysgeusia
Auricular trauma	Non-pulsatile tinnitus, hearing loss, vertigo, pain

Meningiomas and vestibular neuromas are the most common benign intracranial tumours, in first and second place, respectively. In the cerebellopontine angle, these tumours represent 6-15%[54,55] and 80%[54,56,57] respectively, of all tumours.

The simultaneous presence of two kinds of tumour in the cerebellopontine angle, in the absence of neurofibromatosis type 2 or history of cerebral irradiation, is extremely rare. In the literature, there are few cases of intracranial meningioma combined with neuroma in the same patient[58,59] and only five cases of meningioma and vestibular neuroma in the same cerebellopontine angle.

Grauvogel et al[60] report the case of a 46-year-old woman with sudden left hearing loss, tinnitus and homolateral ataxia, with coexistent meningioma and vestibular neuroma, which were not distinguishable radiologically. The diagnostic process and imaging studies showed an intra- and extrameatal lesion. The neuroradiological diagnosis was vestibular neuroma. The patient underwent surgery via retrosigmoid approach, which showed up two distinct tumours: a small intrameatal neuroma and a larger meningioma, originating from the dura of the petrous bone. The patient experienced no neurological deficit after surgery: in particular, the facial nerve function was completely preserved. The definitive histopathological examination revealed "fibromatous meningioma and neuroma". It is thus clear that a careful interpretation of imaging studies before surgery is crucial[60]. Radiosurgery is an alternative treatment option, particularly for smaller neuromas, which do not press on the brainstem or cause hydrocephalus[61]. Various theories attempt to explain the coincidence of two quite distinct primary brain tumours in the absence of conditions such as phacomatosis (for example, neurofibromatosis type 2) or a previous cerebral irradiation. Tumours may develop through pure chance, or the first tumour may act as a stimulus on the surrounding brain parenchyma or brain tissue and induce a new tumour in a different tissue. Alternatively, a cancerogenous stimulus may develop tumours in different tissues at the same time, or a residual embryonic

structure may become the matrix for the subsequent development of a multiple brain tumour[62]. Tsukamoto, Hikita and Takaki[58] hypothesized that meningioma and neuroma association may be determined genetically, given that the genes responsible for these tumours are both found in chromosome 22. In the case described above, the combination of the two tumours most probably occurred incidentally. Nevertheless, the influence of some factor in tumour growth, which forces the second tumour to grow, is theoretically possible since tumours were near each other in the same anatomical region. Further studies are necessary for a better understanding of the mechanisms that cause multiple tumours to grow[60,61].

All patients with unilateral tinnitus should have, as soon as possible, a hearing test and an MRI, with particular attention paid to the cerebellopontine angle and the internal auditory canal, and even more so if an asymmetric hearing loss is present, in order to exclude a vestibular neuroma. Although a benign tumour, the latter may represent an emergency in some cases, determining adverse occurrences such as significant haemorrhages[63].

Endolymphatic sac tumours, which originate from the ductal epithelium and endolymphatic sac, are slow-growing, locally aggressive, low-grade malignancy. They often arise with tinnitus, sensorineural hearing loss, dizziness, mimicking Meniere's disease. Large tumours may be accompanied by neuropathies of the cranial nerves. These tumours may be associated to von Hippel-Lindau syndrome. Therapy consists of excision microsurgery. Radiation therapy has a limited role, in recurrences and tumours that cannot be treated surgically. An early diagnosis can help preserve hearing[64].

Symptoms such as vertigo, fullness, feeling of aural pressure, hearing loss and tinnitus, probably caused by the presence of endolymphatic hydrops and osteitis of the otic capsule, are typical of Ménière's disease[65,66] and Cogan's disease[67], which has similar symptoms to Ménière's disease, and needs to be distinguished by careful diagnosis. Some authors believe that it is important to monitor these patients, since they may have fluctuating symptoms over time, which could compromise their quality of life[68]. In some patients (6%), congenital or acquired syphilis may cause Ménière's disease[69], with initial symptoms similar to those of other etiologies. Consequently, failure to establish the specific etiologic diagnosis could result in unnecessary surgical treatment or inappropriate medical therapy. In certain cases, if steroids are not given promptly, rapid and permanent hearing loss will result. Etiology may be confirmed in these cases by a fluorescent treponema antibody absorption (FTA-abs) test[69]. The clinical features and history of the disease, in these patients, are unique: symptoms begin in the fifth decade of life, first in one ear and then, after a few years, in the other; caloric vestibular responses are reduced or absent. Prompt administration of steroids will be necessary, particularly in a medical emergency with severe and sudden hearing loss, at times together with an endolymphatic subarachnoid shunt operation. Long-term use of steroids seems to maintain effective hearing for more than 20 years[69].

A petrous apex cholesterol granuloma is the most common lesion in this anatomical region. Patients have various symptoms such as tinnitus, hearing loss, headache, facial spasms and diplopia. Kim et al[70] analyse the case of a 32-year-old man, who was first diagnosed with Ménière's disease. Based on this diagnosis, he was placed for several months on a low-salt diet and had relative pharmacological treatment. However, the symptoms persisted and grew worse. The patient subsequently came to the emergency department complaining of unilateral facial twitching and numbness. An MRI was performed to rule out a central neurological lesion. The test found a lesion at the petrous apex. Surgical excision was infralabyrinthine, with a middle cranial fossa approach, using a computerized surgical device (Brain Lab). After the operation, hearing loss and vertigo were no longer present. It is likely that the patient's Ménière disease-like symptoms were due to compression of the endolymphatic sac by part of the granuloma.

For diagnostic purposes, CT and MRI scans on the petrous temporal bone are of great help, particularly in clinical manifestations. The location and size of the granuloma are decisive with regard to disease-specific symptoms or signs present. Consequently, the possibility of a petrous apex lesion should be considered in patients who have the clinical characteristics of aggravated endolymphatic hydrops[69].

Kuhn et al[71] describe, in a prospective case study over a one-year period in an emergency department, and through a review of the literature, the clinical characteristics (migraine, pain in the distribution of the occipital nerve, Tinel's sign, and partial or complete relief from pain after lo-

cal anaesthetic injection) that can aid in diagnosis of the occipital neuralgia syndrome. The latter is often accompanied by tinnitus, vertigo, nausea, visual disturbances and scalp paraesthesia. Occipital neuralgia is the benign extracranial cause of headache. After local anaesthetic injection, further aiding diagnosis, patients had more relief from both pain and other symptoms[71].

Desai et al[72] report the case of a 29-year-old who, after a lumbar puncture, presented classic symptoms such as postural headache, nausea, vomiting and ocular disturbances. These complications are well known and may be due to multiple factors: needle size, type and needle bevel orientation. In order to clear up the symptoms, conservative treatment is usually sufficient; resting in bed, intravenous hydration, caffeine and analgesics. An epidural blood patch may be necessary in extreme cases.

Doganay et al[73] describe the case of a 24-year-old patient with a normal clinical history and normal laboratory tests, who developed a subdural haematoma after spinal anaesthesia at L4-L5 level. This severe and rare complication can be fatal if not treated.

Puncture of the dura mater can cause cerebrospinal fluid losses and strained and ruptured meningeal blood vessels with relative bleeding. The first day after spinal anaesthesia, the patient began to have a headache. A post-spinal headache was diagnosed and conservative treatment undertaken. Since the pain behind the left ear persisted, associated with tinnitus, emergency CT scan was performed and showed an acute fronto-temporo-parietal subdural haematoma. Thus, a continuous atypical headache, with tinnitus, after spinal anaesthesia, may indicate a possible underlying subdural haematoma. In these cases, an urgent diagnosis needs to be made, based on the patient's history and imaging techniques.

Sahin et al[74] described the case of a 50 year old woman who developed sudden bilateral hearing loss with tinnitus after spinal anesthesia performed for hallux valgus surgery. A sudden hearing loss with bilateral tinnitus after spinal anesthesia has been reported only in a few cases. The patient's hearing improved almost fully in the morning of the 3rd day after surgery. No recurrence of hearing loss, tinnitus or vertigo have been reported during the follow-up of six months. Auditory complications after spinal anesthesia should be carefully evaluated by ENT specialist for an early diagnosis and its treatment.

Achache et al[75] presented a case of retarded pneumolabyrinth subsequent to undiagnosed traumatic perilymphatic fistula. From a review of the literature, the authors indicated the systematic management procedure to be followed in order to prevent further complications. Labyrinth involvement is rare but there is a risk of perilymphatic rupture that is often underestimated on initial clinical examination due to the predominance of neurological and/or somatic symptoms.

Idiopathic sudden sensorineural hearing loss (ISSNHL), often associated with the onset of unilateral tinnitus, should be considered a proper ontological emergency. It is a clinical condition characterized by the sudden onset of hearing loss, mainly unilateral[76-79]. When ISSNHL is suspected, an appointment with a specialist should be made as quickly as possible, together with a hearing test, since the percentage recovery and prognosis can be improved by prompt diagnosis and subsequent therapy[80-82].

Diao et al[83] referred the case of a 31 years old patient presenting with tinnitus and unilateral sensorineural sudden hearing loss as the first manifestation of chronic myelogenous leukemia, a rare event in patients with leukemia. Pure-tone tone audiometry revealed a pantonal profound hearing loss in the left ear. Subsequent investigations led to a diagnosis of chronic myelogenous leukemia. The cases of sudden hearing loss should, therefore, be carefully evaluated in consideration of the possible coexistence of any haematological disorders.

In the last few years, sudden deafness has been frequently described in association with anterior inferior cerebellar artery (AICA) infarction, generally together with other brainstem and cerebellar signs such as ataxia, dysmetria and peripheral facial palsy[78]. Martines et al[78] describe the case of a 53-year-old man who suddenly developed hearing loss and tinnitus without any brainstem or cerebellar signs. CT of his ear and temporal bones was normal, and the lesion visible only by MRI test. This case represents the fifth described in the literature to date and confirms the importance of neuroimaging in emergency assessments, in order to limit the extent of the lesion with immediate therapy[78]. Moreover, early hearing recovery, in the first four weeks after the onset of hearing loss, is directly correlated with tinnitus handicap[84].

Autoimmune vestibulo-cochlear disorders (AVCD) represent a group of syndromes probably caused by an autoimmune mechanism with

overlapping clinical features, manifesting as sensorineural hearing loss, often associated with vertigo, tinnitus and fullness[84,85]. Definitive evidence of a classic autoimmune process is still lacking, but inner ear inflammation progresses to severe, irreversible damage within three months of onset (and often much more quickly). Thus, patients with rapidly progressive AVCD must be treated urgently, since prompt treatment with corticosteroids and other antirheumatic/immunosuppressive agents can preserve hearing and vestibular functions.

Among other pathologies, of probable autoimmune origin, that present tinnitus is the Vogt-Koyanagi-Harada syndrome[86]. Another condition to be considered is tinnitus associated with multiple sclerosis. Rarely, cases of hearing loss, with or without tinnitus, occur in adults, particularly during disease exacerbation, rather than as an isolated feature of the disease.

Rodriguez-Casero et al[87], on the contrary, describe the case of an 11-year-old girl in whom persistent tinnitus and reversible hearing loss were the sole manifestation of multiple sclerosis in the initial symptoms.

Another case in the literature of reversible hearing loss is that reported by Granata et al[88], that observe the reversible posterior encephalopathy syndrome, a rare radiographic clinical entity, characterized by typical findings in the occipital and parietal lobes, caused by a subcortical vasogenic edema. The etiopathogenesis is not clear, although it is known that it is a endotheliopathy the posterior cerebral circulation, leading to failure of the cerebral autoregulation, posterior edema and encephalopathy. Of note, a possible pathological activation of the immune system has been suggested. Most common clinical manifestations are headache, seizures and blurred vision. Also frequent are acute tinnitus and vertigo. The symptoms can be reversible, but hemorrhage or cerebral ischemia may occur. Diagnosis is based on MRI, in the presence of a development of acute neurological symptoms and clinical signs, high blood pressure and/or conditions associated with toxic effects on the endothelium.

We will review other rare cases of acute tinnitus, associated with other symptoms, which may represent a genuine emergency.

Cooper[89] describes cases where people are struck by lightning or have electrical injuries. Lightning injuries affect 800 to 1000 persons per year. High-voltage electrical injuries may be devastating, with extensive burns, cardiac arrest, amputations and long, complicated hospitalization. Low-voltage injuries tend to be benign, although they may have significant long-term morbidity, including chronic pain syndromes. The main cause of death is cardiac arrest. Apart from burns, patients may have tinnitus, blindness, confusion, amnesia, cardiac arrhythmias and vascular instability. Other long-term problems are sleep disturbances, anxiety attacks, pain syndromes, damage to the peripheral nervous system, and diffuse neurologic and neuropsychologic damage[89]. Other sequelae such as seizures or severe brain damage from hypoxia, during cardiac arrest, and spinal artery syndrome from vascular spasms are indirect results of electrical and lightning injury. Desai et al[90] describe the case of a young woman struck by lightning as she was playing soccer, resulting in tinnitus, loss of consciousness, paraesthesias, muscle spasms and the onset of a speech impediment.

Hatten et al[91] present the case of a healthy woman, who had no apparent risk factors for infestation, affected by myiasis in the middle and external ear, requiring surgical intervention. Myiasis is an otolaryngological disease that is rare in developing countries. It is linked with environmental conditions and typically seen in disabled patients living in poor hygienic conditions. Prognosis is usually good. The patient's symptoms of tinnitus, otalgia and otorrhea were resolved after various attempts at extraction of the larvae, with their complete eradication. Tympanoplasty was subsequently necessary to reconstruct the damaged external and middle ear. Another case described by González Poggioli et al[92] is of a 65-year-old woman with pain and tinnitus, which had lasted for a week, in her left ear. An otoscopy in the emergency department showed numerous dipteran larvae totally occupying the external auditory canal. The larvae were removed and the patient was discharged after 24 hours of medical observation. Diagnosis of otic myiasis is through direct visualization of the larvae in the ear cavities. Treatment is by removal of larvae from the ear. Otic myiasis should always be suspected clinically in patients who travel widely and have an atypical presentation of acute otalgia associated with otorrhea.

Wolf et al[93] observe two cases of acute quinine toxicity, one from self-poisoning and the other from an unidentified source. Both patients had acute bilateral blindness, associated with the classic symptoms of cinchonism, including tinnitus, nausea, vomiting and prolongation of the QT

interval. Another case reported in the literature by Bodenhamer et al[94] describes delayed cardiotoxicity (cardiac conduction defects, dysrhythmias and cardiovascular collapse) eight hours after quinine ingestion. Smilkstein et al[95] refer two cases of acute blindness caused by quinine poisoning, with an initial unsuspected diagnosis. One patient presented tinnitus, hearing loss, vomiting, abdominal pain and mental confusion, while the other complained of hearing loss, headache, confusion, tachycardia, first-degree atrioventricular block and subsequent bradycardia. In both cases, the onset of blindness was delayed for more than 12 hours after ingestion. Afterwards, one patient regained normal visual acuity while the other developed marked constriction of visual fields and decreased visual acuity.

Jammehdiabadi et al[96] carried out a retrospective review of 150 cases to determine the diagnosis of overdose patients who took salicylates, tricyclic antidepressants and benzodiazepine. A positive history of salicylate ingestion, tinnitus and hyperventilation were the best predictive indicators of salicylate ingestion. Considering the salicylate abuse in the United States of America, Chyka et al[97] established guidelines for outpatient management, which could potentially optimize outcomes, avoid unnecessary emergency department visits, reduce health care costs and reduce life disruption for patients and caregivers. The presence of typical symptoms of salicylate toxicity such as tinnitus, hearing loss, hematemesis, tachypnea, hyperpnoea, dyspnoea, lethargy, seizures and mental confusion indicate chronic salicylate toxicity and are useful and sufficient elements for referral of the patient to an emergency department.

Van Donselaar-Van der Pant et al[98] observed four rare cases of acute intoxication from systemic toxicity after local use of lidocaine. Symptoms included tinnitus, dizziness, paraesthesia of the tongue, visual disturbances, muscle spasms, convulsions, reduced consciousness, coma and respiratory arrest.

Weaver stated that about 50,000 people in the United States of America each year are admitted to emergency departments for carbon monoxide (CO) poisoning, which can occur either from brief exposures to high levels of CO or from longer exposures to lower levels. Symptoms include headaches, nausea, vomiting, dizziness, general malaise, blunting, chest pain and dyspnoea. Neurological problems may often also be associated, such as tinnitus, hearing loss, vestibular disorders, cognitive sequelae, anxiety, depression, headaches, insomnia, fatigue, visual acuity disturbance, peripheral neuropathies and Parkinsonian-like syndrome. While breathing oxygen hastens the removal of carboxyhaemoglobin (COHb), hyperbaric oxygen (HBO_2) hastens CO-Hb elimination and favourably modulates the inflammatory processes caused by CO poisoning, improving the mitochondrial function, transiently inhibiting lipid peroxidation, impairing leukocyte adhesion to injured microvasculature, and reducing cerebral inflammation caused by CO-induced formation of myelin basic protein[99].

Culić et al[100] suggested a possible linkage between different myocardial infarction sites and specific symptoms. Anterior infarctions, with respect to inferior and lateral ones, presented tinnitus in association with headaches, asthenia, dyspnoea, coughs and dizziness.

An acute acoustic trauma (AAT) may be treated as an otological emergency. Vavrina et al[101] presented a retrospective study on the therapeutic effect of hyperbaric oxygenation in patients with unilateral or bilateral acute acoustic trauma. Two groups of patients were treated in the same pharmacological way (dextran [Rheomacodrex]), ginkgo extracts (Tebonin) and prednisone, but only one group underwent additional hyperbaric oxygenation at a pressure of 2 atmospheres absolute for 60 minutes, once a day. Both treatment groups were comparable as far as age, gender, initial hearing loss and prednisone dose are concerned. The delay at the beginning of therapy was 15 hours in both groups and treatment was started within 72 hours in all cases. Control audiometry, performed after about one week of therapy, showed a significant hearing gain in the group exposed to hyperbaric oxygenation. Ylikoski et al[102] further indicated how, in acoustic trauma cases, hyperbaric oxygen therapy has a positive effect on both loss of hearing and tinnitus. The therapeutic rationale of administering positive pressure oxygen is based on experimental studies showing how noise exposure results in cochlear hypoxia. Hyperbaric oxygenation is the only method of increasing concentration of oxygen in the inner ear fluids, which thus facilitates the cellular regeneration processes[103].

Foreign bodies in the external auditory canal can cause unilateral tinnitus. Auricular foreign bodies can be easily identified at an emergency department. Ness et al[104] report the case of a 16-year-old patient, who came to the emergency de-

partment complaining of unilateral tinnitus and dysgeusia. The only history of injury was an incident that had occurred in the past few days. A wooden foreign body was found by otoscopy in the auditory canal. A CT scan revealed it to be about 3 cm. It had pierced the middle ear, disrupting the ossicles. Subsequent otomicroscopy investigation in the operating theatre showed a severed chorda tympani nerve with ossicle rupture. This is certainly an unusual presentation for an aural foreign body but, in consideration of possible lesions to the middle and inner ear, it is important for a careful clinical evaluation to be carried out before and after removal of foreign bodies.

Fang et al[105] described the case of a 48-year-old woman who had worn a hearing aid in her left ear for many years and who came to the emergency department complaining of severe otalgia, with otorrhagia, that had lasted for one day. An otomicroscopy revealed a fruit-fly larva moving in the external auditory canal. The skin over the floor of the canal, close to the eardrum, was eroded. The larva was removed and the patient received topical antibiotic treatment with ofloxacin, which resolved the otalgia immediately. Two weeks later, the erosion was completely healed.

Ear trauma is a common problem in emergency medicine. Although injuries of the ear are not usually life threatening, they can account for significant morbidity. Patients may experience tinnitus, hearing loss, vertigo and pain. Injuries to the ear may occur because of penetrating or blunt traumas, loud noises, chemical exposure, explosions and thermal injury[106]. Chukuezi et al[107] analyse ear trauma with regard to etiology, presentation and influencing factors. In the majority of cases, the trauma affects the tympanic membrane because of the sudden increase in pressure to which is it exposed. Slaps are the most common aetiology, while bleeding from the ear, hearing loss and tympanic membrane perforation are the most frequent presentations.

Reitsma et al[108] observe a rare case of a nine years old patient with idiopathic intracranial hypertension, which presented tinnitus, bilateral progressive sensorineural hearing loss, headache, ear pain and dizziness. This is the second case described in the literature of pediatric idiopathic intracranial hypertension that occurs with hearing loss. The patient underwent physical examination, audiometry, MRI, CT scans, lumbar puncture. Fundus examination and imaging techniques showed no particular anomalies. At first observation pure-tone audiometry revealed a hearing threshold of 30 dB HL in both sides. Two months later the threshold lowered to 35 dB HL. Lumbar puncture revealed an increased intracranial pressure: an idiopathic intracranial hypertension was therefore diagnosed. After the lumbar puncture problems were gradually solved and hearing returned to normal.

Syed et al[109] observe two cases of primary epidermoid cysts of the mastoid. One presented with headache and progressive imbalance, the other with unilateral hearing loss and tinnitus. Epidermoid cyst of the temporal bone are extremely rare: such lesions that arise in isolation within the mastoid have never been reported previously in the literature. In both cases surgical treatment was necessary. The diagnosis of an epidermoid cyst is based on clinical history, physical examination the radiological, histological and intraoperative features. Complete removal of the lesion with its capsule is recommended to prevent recurrence and to enable a good long-term prognosis.

Tinnitus and emergency psychiatric comorbidity

Tinnitus is a clinical symptom of disorders in the auditory system. It often does not have an organic origin and is correlated with stress. Exposure to noise can be, in these cases, a triggering factor[110]. Severe tinnitus can have a significant impact on the quality of life of the affected person[111]. Approximately half of adults and one-third of children affected with tinnitus and seeking help suffer from anxiety and/or depressive disorders, with epidemiological peaks reaching 77%[21]. Consequently, it is important to identify these disorders promptly and refer suitable treatment[112]. In the literature and in the clinical environment, the comorbid disorders most frequently present in people needing specialist assistance for tinnitus are those of a psychological nature. The definition itself of "decompensated tinnitus"[13] refers to concomitant emotional conditions and stress. The tinnitus "discomfort" level seems to be correlated mainly with the compresence of a psychological disorder, rather than with the audiologic characteristics of the symptom[113].

Mortality of patients with decompensated tinnitus appears substantially linked to the risk of suicide: data in the literature estimate that the risk of suicide in tinnitus patients is ten times higher than the general population[23]. As regards morbidity, or the result of tinnitus handicap, concomitant psychiatric symptoms represent the

most reliable measure of compromised quality of life[114].

Somatic symptoms such as tinnitus are frequent in patients with distress from post-traumatic stress disorder (PTSD)[115]. Fagelson[116] has confirmed that 34% of 300 patients with tinnitus enrolled in his clinic carried a diagnosis of PTSD. In 2006, Hinton et al[117] reported that 50% of Cambodian refugee patients with a history of PTSD suffered from disturbing tinnitus. Somatic symptoms are correlated with greater PTSD severity. Tinnitus has also been considered to be a trigger of memories of the specific trauma. Hinton et al[117] describe the possibility that the memory of the traumatic episode could be connected and/or caused by tinnitus. Some authors maintain that flashbacks and intrusive memories contribute to exacerbation of the tinnitus itself. Henry et al[118] suggested that exacerbation of tinnitus in some patients, in the presence of particular environmental sounds, depended, at least in part, from the fact that the sound triggered a memory of past events, which produced a distressing emotional state. These associations would be particularly evident in those patients where the onset of tinnitus can be traced back to a specific episode of exposure to a sound or a traumatic event, like that which caused the PTSD.

Besides acute PTSD, psychiatric urgency is represented by the risk of suicide or by a psychiatric comorbidity, which makes the tinnitus so debilitating that urgent consultation is necessary. Conflicting reports emerge from the literature: some studies affirm that there is no direct correlation between tinnitus and suicide[119], while others uphold the contrary[120]. In both cases, comorbidity should be carefully assessed. Or rather, if some authors attribute the risk of suicide to the copresence of depressive disorders[117], the latter, besides being extremely frequent[121] are, in each case, directly correlated with the severity of the tinnitus itself[122]. Consequently it is reasonable to consider comorbidity therapy urgent, both with regard to the risk of suicide or attempted suicide and in terms of acute decompensation. In a 2006 study, Langguth et al[123], in support of these considerations, surmise a complex interaction between tinnitus and depression, based on a common physiopathology, or compromise of hippocampus neurogenesis. Moreover, from genetic research, tests show how a variant of the brain derived neutrophic factor (BDNF) can act as a common susceptibility factor in both disorders[123]. Various studies have also been undertaken on the serotonin transporter gene[124].

A 1992 study by Lewis et al[125] on a group of tinnitus sufferers suggest that the suicide risk was 118 per 100000 people a year (more than ten times the general rate). These estimates should be interpreted with care, given the methodological weaknesses present and the limited number of cases taken into consideration. In another study in 1994, Lewis et al[126] found 28 cases of suicide in tinnitus sufferers. These authors also identified a series of common suicide risk factors – male, elderly, socially isolated, history of psychiatric illness – particularly depression (70%), problems of alcohol abuse and previous history of attempted suicide. Although the sample evaluated is small, the study produced interesting results. In a study in 1996, Johnston et al[127] drew attention to the significance of concomitant depression and risk of suicide in the geriatric population. The elderly may often have atypical symptoms such as an impaired ability to communicate, feelings of helplessness and tinnitus.

Lewis et al[128], in a study over a short three-month period of 184 patients in South Glamorgan hospital in Wales, United Kingdom, evaluated the percentage of people who had attempted suicide by poison, pointing out that patients with tinnitus were under-represented (1.6%), with respect to the general population (7%). The results obtained should be interpreted bearing in mind that: (1) the three-month study period enabled only a somewhat limited sample to be considered; (2) only one hospital was studied, and (3) suicide modalities are numerous.

Contrasting data emerge from the literature. A relationship between tinnitus and the risk of suicide has been shown, particularly in the elderly, and is frequently correlated with depression. Few studies[111] have been carried out to date on acute decompensated tinnitus, which is perceived as a chronic problem in the majority of cases. Based on our case series, there may be exacerbation of "chronic decompensation", in correlation with particularly stressful or significant events in a person's life, followed by adjustment disturbances, which require urgent consultation. It is important, therefore, to carry out early treatment to prevent decompensation[129]. In 2004, a study by D'Amelio et al[130] on patients with severe tinnitus showed the importance of early intervention in the acute stage of tinnitus, since the patient's immediate reaction to the manifestation of tinnitus could be of prognostic value for the level of emotional stress to be experienced with chronic tinnitus.

In accordance with data from the literature, we consider early intervention to be important to prevent decompensation, which influences the tinnitus handicap[131], particularly in terms of severity[132].

Results

Writings in the literature are based on small-sized samples or case reports, given the rarity of some conditions that are implicit in tinnitus in the acute stage, and the few available studies on the matter. In our opinion, this further highlights the importance for a clinician to have a "diagnostic instrument" available to assess the tinnitus patient, particularly when tinnitus is "decompensated". In light of the above, we have elaborated an algorithm that could be useful in the "management of tinnitus in the acute stage". Clinicians, to assess decompensation, lacking instrumental symptomatology that can digitalize and register the symptom with pathology and associated risk, may now take advantage of an instrument widely used in first assessment of the tinnitus handicap: the Tinnitus Handicap Inventory (THI). This questionnaire constitutes a first-level screening test to determine psychiatric comorbidity[114]. THI has gained widespread acceptance as a "self-report" measure of tinnitus handicap. At this point, it is used widely in the literature; it assesses the impact of tinnitus on the activities of daily life. THI consists of 25 multiple-choice questions, which assess various areas – functional, emotional and catastrophic. The test enables grading of the tinnitus handicap based on scores (very mild with score of 0-16, mild 18-36, moderate 38-56, severe 58-76, catastrophic 78-100). When the test score is greater than 36[133], psychiatric investigation is needed, to evaluate possible psychiatric comorbidities, by category (Diagnostic and statistical manual of mental disorders, fourth edition – DSM IV), and with probable further suitable psychotropic treatment, since there is a correlation between psychopathological conditions and THI score[133].

The algorithm we have presented, based on our experience and on cases in the literature, makes a first distinction between pulsatile and non-pulsatile tinnitus, leaving prominent space for an evaluation of the patient's psycho-emotional state.

The most probable underlying pathologies causing the symptom are listed in Figure 1. Based on the characteristics of the tinnitus, its onset, model, any associated symptoms, impact of the patient's attitude on the tinnitus, examination of the temporomandibular joint and the cervical spine, it will be possible to target any underlying pathology and diagnostic-therapeutic treatment.

Conclusions

To date, there are no clinical standards and guidelines for better management of patients with tinnitus in emergency situations[134]. What emerges from our work is the realization that a multidisciplinary approach is the most appropriate way of dealing with the "tinnitus problem" and avoiding decompensation. In management of tinnitus at the acute stage, it will be necessary in the first place to consider the medical urgency, since multiple, even severe, pathologies may underlie the tinnitus symptom, which will need immediate medical or surgical treatment. It will be important as well to recognize, assess and manage any concomitant psychiatric urgency, in order to rationalize therapeutic intervention.

At present, tinnitus is not regarded by the Italian Hospital Otology Association (Associazione Otorinolaringologi Ospedalieri Italiani [AOOI]) as an urgency[135], but as a symptom associated with internal medicine, surgery and otorhinolaryngology.

When we speak of tinnitus, a question arises spontaneously: "… are we faced with a simple symptom or a threat?" Tinnitus is too often an underestimated symptom, even when urgent. At present, we still see a somewhat superficial management of tinnitus patients on the part of doctors, where the clinical case is often underestimated. This cannot be justified, particularly in the case of sudden or rapid decompensation tinnitus. We could be faced with a real emergency. In fact, tinnitus could be the sign of an ominous pathological process, hiding even severe psychological effects that can lead to suicide. For this reason, clinical cases presenting tinnitus should be analysed with particular attention.

The main objective, in assessing tinnitus in an emergency department, is to identify life-threatening causes, preserve hearing, identify curable causes and provide appropriate data and symptomatic treatment in order to avoid reaching a chronic condition or, even worse, decompensation of the symptom.

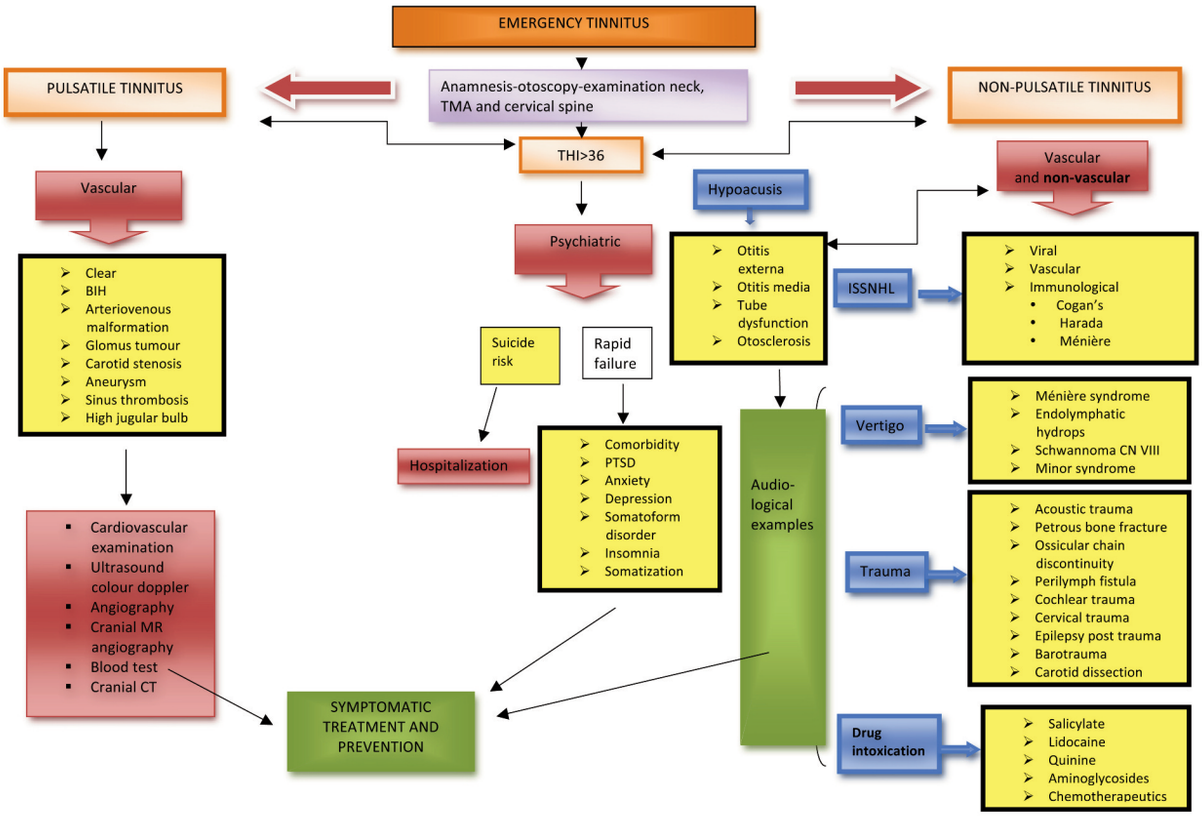

Figure I.

Other diagnostic-therapeutic algorithms have already been described in the literature but, in our opinion, the algorithm presented in our work has the merit of being simple and easy to use by all doctors, whether specialists or not. It also has the exclusive feature of allowing a primary assessment of the patient's psychological aspect through a questionnaire that can highlight possible psychiatric comorbidity, particularly when the underlying pathology is underestimated.

At present, since a positive universal and conclusive therapy for tinnitus is lacking, an in-depth study of the patient serves as an essential basis for subsequent more targeted and effective treatment.

Conflict of Interests

The Authors declare that they have no conflict of interests.

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