

# Evaluation of Tinnitus in the Emergency Department

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## 1. Introduction

Tinnitus is defined as the perception of abnormal noise by the patient in the absence of an external acoustic source. It may be described as a ringing, whistling, buzzing, roaring, or clicking sound, although any type of sound may be reported. Tinnitus may be unilateral or bilateral, and can be described as occurring within the head or as a distant sound <sup>1</sup>. It may occur constantly or intermittently. Tinnitus severity may vary considerably, ranging from a minor annoyance for the patient to distressing enough for certain patients to consider suicide <sup>2</sup>.

Tinnitus appears to be a very common symptom, with a reported prevalence ranging from 10 - 25 % <sup>3,4</sup>. Of the 35 - 50 million adults with this symptom, approximately 12 million visit a physician; 2 - 3 million of these patients are severely impacted by tinnitus <sup>5</sup>. Furthermore, these patients often have numerous associated comorbidities including anxiety, depression, and reduced general health <sup>6-9</sup>. Tinnitus occurs more frequently in Caucasians, men, and older age groups <sup>10</sup>. Other reported risk factors include exposure to loud noise, hearing loss, smoking, and hypertension <sup>11,12</sup>. Tinnitus has also been reported in approximately 36% of children, but often goes undocumented due to the infrequency of their spontaneously reporting it <sup>13</sup>.

Numerous hypotheses have been developed for the pathophysiology of tinnitus; however, a precise mechanism has not been established <sup>14</sup>. Tinnitus may arise from any abnormality of the neural pathway from the cochlear neural axis to the auditory cortex. Proposed etiologies include damage to hair cells with accompanying excess stimulation of auditory nerves, increased activity in the auditory complex, and excessively active auditory nerves <sup>3,15</sup>. Multiple mechanisms likely account for tinnitus because of the complexity of the hearing pathways, and thus this symptom is non-specific.

Several classification types have been used to describe tinnitus. One such method classifies tinnitus as objective or subjective. Objective tinnitus indicates that the sound may be heard by the physician by auscultation with a stethoscope over the head and neck adjacent to the patient's ear <sup>3</sup>. Subjective tinnitus is considerably more common, and is only perceived by the patient. This chapter will review the differential diagnosis of objective and subjective tinnitus as well as the evaluation and management of these patients.

## 2. Objective tinnitus

Objective tinnitus, occasionally referred to as somatosounds, is audible to the physician by use of a stethoscope or Doppler <sup>15</sup>. The tinnitus is often characterized by the patient as a clicking or pulsing sound. The cause is often due to a vascular abnormality, which may be arterial or venous in etiology <sup>1</sup>. Additional causes include neurologic lesions or eustachian tube dysfunction <sup>15</sup>.

### 2.1 Vascular causes

*i. Arterial sources.* Arteriovenous shunts include both arteriovenous malformations and arteriovenous fistulas, and represent an important cause of tinnitus that is essential to recognize in the emergency department <sup>1</sup>. Congenital arteriovenous malformations are generally asymptomatic and are uncommon causes of tinnitus, whereas acquired arteriovenous shunts are more likely to be symptomatic. Approximately 10 -15 % of intracranial arteriovenous fistula are dural, but are more often responsible for tinnitus than neck or cerebral arteriovenous shunts <sup>16</sup>. Dural arteriovenous fistulas likely arise due to dural venous sinus thrombosis that is most often due to trauma, but may also be secondary to infections, neoplasms, or surgery <sup>17</sup>. Mortality from hemorrhage of a dural arteriovenous fistula ranges from 10 - 20% <sup>17</sup>; thus, appropriate diagnosis by emergency physicians is essential.

A second significant cause of acquired arteriovenous shunt that may cause tinnitus is a paraganglioma in the temporal bone, classified as either a glomus jugulare or glomus tympanicum tumor <sup>15</sup>. Such tumors often cause the patient to perceive a constant blowing sound. Additional symptoms that may occur as the tumor enlarges include hearing loss, and deficits in cranial nerves VII - XII. These tumors are occasionally visualized as a vascular mass behind the tympanic membrane <sup>15</sup>.

Rarely, tinnitus may be caused by dissecting aneurysms of the of the internal auditory canal or the vertebral artery <sup>17</sup>. Additional symptoms that may occur are pain, Horner's syndrome, cranial nerve deficits, subarachnoid hemorrhage, and transient ischemic attacks. Vasculopathies that predispose individuals to this condition include Marfan syndrome, osteogenesis imperfecta, and fibromuscular dysplasia <sup>17</sup>.

Tinnitus may occasionally be the presenting symptom of atherosclerosis in the carotid artery <sup>17,18</sup>. The source of the somatosounds is stenosis in regions of the carotid artery that result in blood flow turbulence. Associated risk factors are those typical for atherosclerosis and include older age, smoking, diabetes, hyperlipidemia, and hypertension. Carotid bruits have been reported in patients with this source of tinnitus <sup>18</sup>. Tinnitus may also result from arterial bruits in other vessels in the temporal bone including branches of the external carotid, basilar, vertebral arteries, and vascular abnormalities located in the auditory canal <sup>15,19</sup>. These patients generally lack other otologic symptoms such as vertigo, hearing loss, otalgia, and symptoms of aural fullness <sup>15</sup>.

*ii. Venous sources.* Tinnitus may also arise from venous sources. A venous origin of tinnitus may be differentiated from an arterial source by application of pressure on the ipsilateral jugular vein; this maneuver results in cessation of the tinnitus from venous sources <sup>17</sup>.

The most important etiology of venous causes of tinnitus is pseudotumor cerebri, also called idiopathic intracranial hypertension. Pseudotumor cerebri classically occurs in young, obese, women. Pulsatile tinnitus in one series was reported to occur in 60% of patients;

accompanying symptoms include headache, visual disturbances, and retrobulbar pain <sup>20</sup>. Indeed, the combination of headache with pulsatile tinnitus is fairly specific for the diagnosis of pseudotumor cerebri <sup>20-22</sup>. The most common signs on physical exam include papilledema, loss of visual fields, and cranial nerve VI palsy. Occasionally, pseudotumor cerebri has been reported in the absence of papilledema <sup>22-24</sup>. Untreated pseudotumor cerebri may lead to permanent vision loss <sup>25</sup>.

Venous hums are another source of tinnitus. They may be heard in patients with hypertension, which may be systemic or intracranial <sup>15</sup>. Another cause of venous hum tinnitus is a dehiscent jugular bulb, an aberrantly high location of the jugular bulb that extends into the middle ear space <sup>15</sup>. Dehiscent jugular bulb tinnitus results in a low-pitched, soft hum that decreases with activity, movement of the head, or application of jugular vein pressure. Hearing loss has been reported secondary to a dehiscent jugular bulb <sup>26</sup>. A dehiscent jugular bulb may be visualized behind the tympanic membrane, and must be differentiated from a glomus tumor <sup>15</sup>.

## 2.2 Non-vascular causes

Palatal myoclonus and stapedial muscle spasm are two neurologic disorders that may cause objective tinnitus. Palatal myoclonus is caused by inappropriate contractions of the superior constrictor muscles, the salpingopharyngeus, the tensor veli palatini, and the levator veli palatini muscles <sup>27</sup>. The muscular contractions occur 10 - 240 times per minute, and occur intermittently; the objective tinnitus results from abrupt closure of the eustachian tube <sup>28</sup>. This condition may occur in any age group, and may be accompanied by temporomandibular joint pain or occipital headaches; other reported symptoms include hearing loss, alteration of sounds, and the sensation of aural pressure <sup>15</sup>. The diagnosis can be confirmed by viewing palatal myoclonic jerks or by listening with a Toynbee tube. This condition may occur secondary to other neurologic disorders such as cerebrovascular disease, central nervous system tumors, and multiple sclerosis <sup>27</sup>.

Stapedial muscle spasm is an idiopathic condition that is described as a rumbling sensation in the ear <sup>15</sup>. It is often exacerbated by other noises such as speech. The diagnosis is made by visualizing contractions of the tympanic membrane that coincide with the sensation experienced by the patient. Stapedial muscle spasm is considered a benign, self-limited condition <sup>15</sup>.

Finally, dysfunction of the eustachian tube may result in objective tinnitus <sup>15</sup>. This type of tinnitus is often described as a roaring sound that coincides with breathing. Patients may additionally report autophony and reverberation. The symptoms typically improve with lying down and recur after rising. It may be diagnosed by visualizing a fluttering of the tympanic membrane when the patient strongly inhales through the nose. This condition typically develops after a large weight loss of any type <sup>15</sup>.

## 3. Subjective tinnitus

Subjective tinnitus refers to the perception of a sound that is not audible to the examiner. Patients describe the perceived sounds as a ringing, buzzing or clicking <sup>29</sup>. The causes for subjective tinnitus generally stem from hearing loss from damage to the auditory pathway anywhere from the external auditory canal to the auditory nerve.

External causes for subjective tinnitus include cerumen impaction as well as cerumen removal procedures <sup>30</sup>, otitis externa <sup>31</sup> and temporomandibular disorders <sup>32</sup>. The presence of

swelling in the external auditory canal may amplify tinnitus and must be ruled out with a thorough examination of the ears.

Within the middle ear, diseases of the ossicles may result in conductive hearing loss that results in a subjective tinnitus<sup>33</sup>. Patients with otosclerosis may describe a hearing loss which appears to improve in noisy environments. Otosclerosis may present in young or middle-aged adults and may be inherited in an autosomal dominant manner<sup>34</sup>.

Damage to cochlear hair cells encompasses some of the most common causes of subjective tinnitus. Noise-induced hearing loss involves damage to cochlear hair cell from exposure to loud sounds in the environment, ranging from close proximity to explosions to overuse of headphones playing music at a high volume, and the severity of the tinnitus has been found to be associated with the degree of hearing loss<sup>35</sup>. These noise-induced insults may occur in children and young adults. In contrast, age-related hearing loss involves degeneration of cochlear hair cells, especially those in the higher frequency ranges, and may result in tinnitus corresponding to the frequencies of lost hearing<sup>36</sup>.

Other components of the cochlea may be affected in addition to the hair cells that can result in hearing loss and subjective tinnitus. In Ménière's disease, there is an excessive accumulation of endolymph in the membranous labyrinth of the cochlea that leads to episodes of tinnitus, vertigo and progressive hearing loss<sup>37</sup>. Tinnitus in these subjects may vary over time, and reported handicap resulting from tinnitus has been found to associate with the stage of Ménière's disease<sup>38</sup>.

Compression of the auditory nerve itself may result in increased firing of afferent neurons to the auditory cortex, leading to a gradual or abrupt onset of subjective tinnitus. Tumors within the internal auditory canal may lead to tinnitus and hearing loss<sup>39,40</sup>. Vestibular schwannomas, or acoustic neuromas, arise from the Schwann cells surrounding the eighth cranial nerve, resulting in both hearing loss and tinnitus as well as vertigo and disturbances in balance<sup>41</sup>. Patients with a strong suspicion for acoustic neuroma should undergo contrast-enhanced imaging to both make a diagnosis as well as to monitor the growth of the tumor, which tends to be slow<sup>42</sup>. The surgical resection of acoustic neuromas may also result in hearing loss and tinnitus from direct damage to the auditory nerve<sup>43</sup>.

Involvement of the cerebral cortex and brainstem through tumors and infarctions may result in subjective tinnitus. Tumors of the inferior colliculus<sup>44</sup> and within the cerebellopontine angle<sup>45</sup> may cause tinnitus with auditory symptoms. Infarctions of the inferior colliculus<sup>46</sup>, cerebellum<sup>47</sup>, and the basal ganglia, thalamus and pons of the cerebral cortex<sup>48</sup> have been associated with subjective tinnitus.

There are a number of systemic illnesses that are known to cause subjective tinnitus. Anemia may result in a cerebral hypoxia that can cause symptoms of tinnitus, vertigo and headache, as in the setting of cancer-related anemia<sup>49</sup>. Hyperlipidemia may cause or worsen tinnitus, and lowering blood cholesterol levels has been found to improve subjective tinnitus<sup>50</sup>. Patients with low thyroid function may report some degree of hearing loss with tinnitus<sup>51</sup>. Multiple sclerosis may manifest with hearing impairment with tinnitus<sup>52,53</sup>. Syphilis may manifest in otologic symptoms in both early and late stages of the disease. The presence of otosyphilis may be characterized by hearing loss or hyperacusis with tinnitus along with vestibular disturbances<sup>54</sup>.

Medications from nearly every major category may result in ototoxicity and tinnitus. The use of salicylates, such as aspirin, may result in damage to the cochlea spiral ganglion neurons<sup>55</sup> and changes in cochlear NMDA receptor currents<sup>56</sup>. Aminoglycoside antibiotics, such as gentamicin, are well known to cause hearing loss and vestibular damage<sup>57</sup>. Loop

diuretics, such as furosemide, may result in transient or permanent ototoxicity, and these effects may be minimized by delivery with slow infusion rather than bolus injection or using divided oral doses<sup>58</sup>. Additionally, many chemotherapeutic agents<sup>59</sup>, heavy metals<sup>60,61</sup> and anti-malarial drugs<sup>62,63</sup> may contribute to hearing loss and tinnitus.

Finally, psychiatric stressors may worsen the handicap resulting from subjective tinnitus. Depression<sup>64</sup> and fibromyalgia<sup>65</sup> have been found to associate with and exacerbate chronic tinnitus. It should be noted that subjective tinnitus generally differs from auditory hallucinations observed in psychotic disorders by the nature of the perceived sound; tinnitus generally manifests as a more simple ringing or humming, whereas auditory hallucinations tend to involve more complex sounds or speech<sup>66</sup>.

#### 4. Diagnostic evaluation of tinnitus

The main goal of the evaluation of tinnitus in the emergency department is to identify life-threatening causes, preserve hearing, identify causes that are treatable, and provide the appropriate referral and symptomatic treatment. The initial evaluation of tinnitus begins with a complete history, including the onset, location, characteristics, associated symptoms, pattern, alleviating/exacerbating factors, past medical history and surgeries, and medication use. The onset of tinnitus should be characterized as sudden versus gradual. A sudden onset of tinnitus is concerning, and may indicate a vascular or traumatic etiology. Questions regarding the pattern of tinnitus should attempt to differentiate pulsatile from continuous or episodic tinnitus. Pulsatile tinnitus is frequently due to a vascular source whereas Ménière's disease tends to be episodic. Specific associated symptoms to inquire about include hearing loss, vertigo, and aural fullness. The impact of patient positioning on the tinnitus should be asked; specifically, eustachian tube dysfunction is often alleviated by lying down. A past medical history of hyperlipidemia or diabetes may indicate carotid artery atherosclerosis, whereas a thyroid disorder or anemia may suggest a high output cause. Finally, a number of medications are known to cause tinnitus.

A thorough head and neck exam should be performed on all patients presenting with tinnitus. A search for an objective source of tinnitus should be performed by auscultation of the auricular region, the mastoid, and the carotid arteries. Objective tinnitus secondary to a venous etiology is identified by disappearance of the sound when the ipsilateral jugular vein is compressed. Careful otoscopy should be performed to evaluate for middle-ear infection, cerumen impaction, a dehiscent jugular bulb, or glomus tumor. The oral cavity should be examined for contractions of the palatal muscles. The cranial nerves should be evaluated for evidence of hearing loss or brainstem dysfunction. Finally, a fundoscopic exam should be performed to look for papilledema in suspected cases of pseudotumor cerebri.

Diagnostic testing should be guided by the results of the history and physical examination. A complete blood count and thyroid function tests may reveal conditions that cause increased cardiac output and cerebral blood flow that can result in tinnitus. Contrast enhanced computed tomography (CT) should be performed on patients with a tympanic mass visible on otoscopy, which may reveal jugular bulb abnormalities, glomus tumors, and vascular abnormalities. CT or MR angiography may be needed to diagnose dissecting aneurysms and arteriovenous fistulas. Carotid ultrasonography may confirm suspected carotid atherosclerotic artery disease. A lumbar puncture should be performed in patients who are being considered for a diagnosis of pseudotumor cerebri. The suggested approach to patients with tinnitus is depicted in Figures 1 and 2.

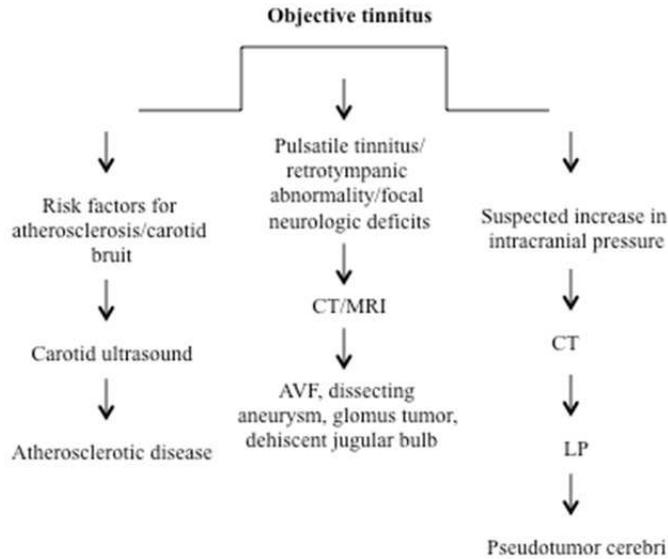


Fig. 1. Suggested approach to objective tinnitus in the emergency department.

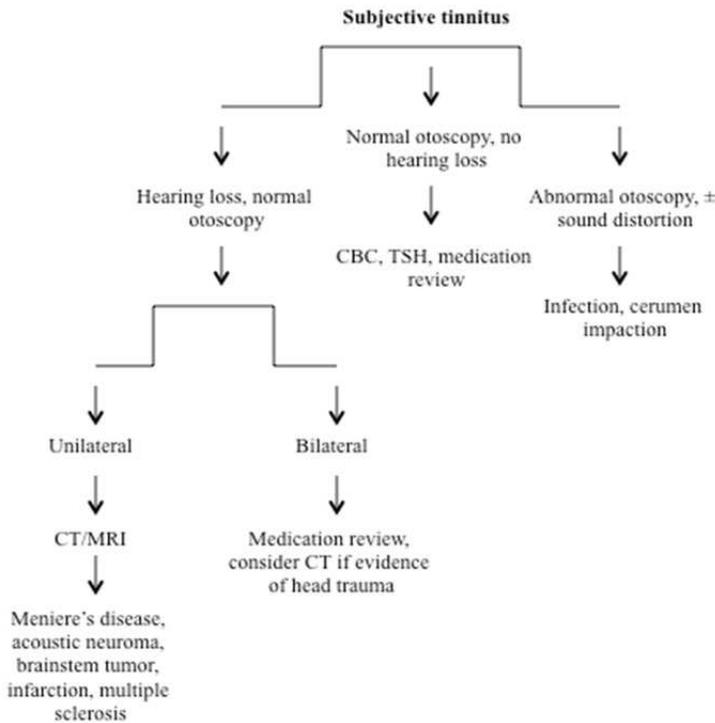


Fig. 2. Suggested approach to subjective tinnitus in the emergency department.

## 5. Management

The management of tinnitus first involves treating identified underlying causes. Tinnitus secondary to ototoxic medications may resolve after discontinuing the medication. Patients with arteriovenous fistula or dehiscent jugular bulb may be treated with vessel ligation or embolization. Those with glomus tumors can be referred for surgical resection or angiographic embolization. Carotid endarterectomy may benefit patients with tinnitus secondary to carotid artery atherosclerosis if the carotid artery stenosis is greater than 60%<sup>67-69</sup>. Patients with benign venous hums or arterial bruits may simply need reassurance, but may be referred for surgical ligation of the vessel if the tinnitus causes significant reduction in quality of life.

Patients with pseudotumor cerebri require intense follow-up with neurology and ophthalmology<sup>70</sup>. While lumbar punctures provide temporary relief, the benefit is short-term due to the rapid reformation of CSF and is not recommended as the primary therapy because of potential complications. Medical management involves treatment with carbonic anhydrase inhibitors, specifically acetazolamide at starting at doses of 500 mg twice daily<sup>71</sup>. Loop diuretics such as furosemide (20 - 40 mg/day in adults) are an adjunctive therapy<sup>72</sup>. Weight reduction also improves symptoms and is a critical component of management<sup>73-75</sup>. Patients with palatal myoclonus or eustachian tube dysfunction should be referred to an otolaryngologist for management. Injection of botulinum toxin into the palate has been successful for patients with tinnitus secondary to palatal muscle myoclonus<sup>76</sup>. Eustachian tube dysfunction may be managed by treatment with mucosal irritants such as tetracycline to the nose that cause disruption of the orifice of the eustachian tube<sup>15</sup>; alternatively, the nasopharyngeal orifice may be surgically closed<sup>77</sup> or silicone plugs placed through the middle ear<sup>78</sup>.

Unfortunately, there are very few effective treatments specifically for tinnitus. Gabapentin resulted in a significant improvement in tinnitus annoyance scores for patients with tinnitus secondary to trauma<sup>79</sup>, but does not appear to be effective in relieving idiopathic tinnitus<sup>79,80</sup>. Alprazolam was reported to decrease the loudness of tinnitus in one trial<sup>81</sup>, whereas a more recent study failed to find an effect on tinnitus loudness or the Tinnitus Handicap Inventory<sup>82</sup>. Clinical trials of the tricyclic antidepressant nortriptyline significantly reduced tinnitus, depression, and the resulting disability<sup>83,84</sup>. However, caution should be used with prescribing these drugs in the emergency department due to the dangers of overdose in suicidal patients.

Many experimental therapies warrant consideration in patients with tinnitus unresponsive to medications. The use of repetitive transcranial magnetic stimulation (rTMS) to stimulate regions in and around temporal auditory cortex has had some success in cases of chronic tinnitus<sup>85-87</sup>. Finally, behavioral based therapies such as tinnitus retraining therapy, masking devices, and biofeedback therapy have reported success<sup>10,88</sup>; consideration should be given to referring patients to providers who can provide these interventions.

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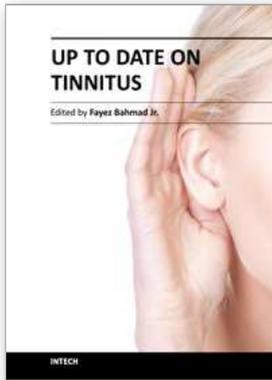
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### **Up to Date on Tinnitus**

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Up to Date on Tinnitus encompasses both theoretical background on the different forms of tinnitus and a detailed knowledge on state-of-the-art treatment for tinnitus, written for clinicians by clinicians and researchers. Realizing the complexity of tinnitus has highlighted the importance of interdisciplinary research. Therefore, all the authors contributing to the this book were chosen from many specialties of medicine including surgery, psychology, and neuroscience, and came from diverse areas of expertise, such as Neurology, Otolaryngology, Psychiatry, Clinical and Experimental Psychology and Dentistry.

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