

Science Notes

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Tinnitus: Causes and Treatment



The high-pitched sound at the beginning of the TV show Outer Limits is very similar to the sounds heard by many tinnitus sufferers.

In the United States, up to 15% of the population suffers from tinnitus, known colloquially as "ringing in the ears." Tinnitus is not just unwanted noise; it is extremely unpleasant and often interferes with enjoyment of music. It can make verbal communication impossible and can cause depression.

Conventional medical thinking used to be that tinnitus arises from injury to the middle ear or the vestibulocochlear nerve (also known as the acoustic nerve, auditory nerve, or cranial nerve VIII), and is therefore difficult or impossible to treat. However, it is now recognized that this is not always the case. Researchers now realize that rewiring of an area in the brainstem called the dorsal cochlear nucleus plays an important role in tinnitus. This new understanding of its causes may result in new treatments for many patients. Indeed, recent breakthroughs based on this theory are already leading to effective forms of treatment.

However, many physicians are unaware of the causes of tinnitus and the problems endured by tinnitus sufferers, and they will often tell the patient that the problem is imaginary or unimportant. This often causes the patient to abandon attempts to get treatment. This is unfortunate, because recent research suggests that tinnitus is easier to cure when treatment is given early. In this article, I will discuss what is known about tinnitus and what tinnitus sufferers can do about their affliction. The information on this page is not medical advice, but is presented for informational purposes.

Types of tinnitus

One way tinnitus can be classified is by the type of sound. By this criterion, there are three main types of tinnitus:

Continuous ringing

In this type of tinnitus, the patient hears a continuous, high-pitched ringing or hissing sound that is unaffected by body movement. Often, tinnitus is accompanied by a partial loss of hearing. Usually, only higher frequencies are lost.

Researchers believe it is no coincidence that tinnitus sufferers most often report hearing high-pitched sounds.

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High frequencies (around 4kHz) are usually the first to be lost after noise trauma (which is a major cause of sensorineural hearing loss), as well as in presbycusis (age-dependent hearing loss). As many as 80% of tinnitus sufferers also have some form of hearing impairment. It is believed that the nervous system adapts to this loss of acoustic stimuli by creating artificial phantom sounds. This is analogous to phantom limb syndrome, where constant pain is felt in a limb after it has been amputated. Thus, patients with a history of exposure to loud noise are most likely to report hearing high pitched ringing sounds.

The pitch of tinnitus often coincides with the frequency region in which the audiogram starts to show a steep decline. This suggests that tinnitus might result from an "edge effect" caused by the brain's attempts to equalize different parts of the acoustic spectrum [16].

There is some relationship between pitch and cause of tinnitus. In the ear, higher-pitched sounds are detected in the outermost portion of the cochlea. Thus, the frequency may depend on the exact part of the middle ear where the original injury occurred. Tinnitus caused by sensorineural hearing loss is usually high pitched. The tinnitus that occurs in Ménière's disease, a disease of the inner ear, is typically of a much lower pitch.

Sound Frequencies in Tinnitus

| Cause | Median frequency, Hz | Frequency range |
|--|----------------------|-----------------|
| Sensorineural hearing loss or Otosclerosis | 3900 | 545-7500 |
| Conductive hearing loss | 490 | 90-1450 |
| Ménière's Disease | 320 | 90-900 |

Source: [15]

Ringings affected by body position

When the ringing sound is made better or worse by changes in body or neck position, it is called somatic tinnitus. This is the most common type of tinnitus.

Non-ringing

When the sound is not a ringing, but a rushing, clicking, thumping, or other atonal sound, it usually represents some mechanical process in or near the ear. Often these sounds can be heard by another person using a stethoscope, and therefore they are called "objective" tinnitus. A common example is a pulsatile bruit caused by turbulent flow through blood vessels in the neck.

Clicking sounds are often associated with temporomandibular joint (TMJ) syndrome. Clicking sounds can also be caused by intermittent contraction of various muscles in the middle ear, including the tensor tympani or stapedius muscles. This condition is called middle ear myoclonus. Although thought to be rare, it may actually just be widely undiagnosed. A related condition is palatal myoclonus. A physician may prescribe muscle relaxants such as orphenadrine, or in severe cases, may inject botulinum toxin to stop the muscle from contracting.

Another way to classify tinnitus is between bilateral and unilateral tinnitus. When the same sound is heard in both ears, it usually means the problem is not in the ears, but in the nervous system. Treatment is different for each type. For example, microvascular decompression (see below) is usually ineffective against bilateral tinnitus.

Causes of tinnitus

In general, tinnitus usually starts with some injury to the ear--either a noise trauma, a blow to the head, or some disease-induced injury. According to the current theory, a part of the brainstem called the dorsal cochlear nucleus tries to adapt to the injury, but in so doing creates phantom sounds. Some of the causes of tinnitus can be very serious, and indicative of some underlying pathology, while the majority of cases are much less serious.

Pathological causes

Pathological causes of tinnitus include head injury; disorders affecting the CNS such as stroke, meningitis, and encephalitis; cardiovascular disorders such as intracranial hypertension, aneurysm, aortic stenosis, or carotid artery stenosis; ear infections, cancer, and surgery-induced injury.

Tinnitus is always present in a type of cancer known as vestibular Schwannoma, and is usually present in acoustic neuroma. Tinnitus, along with vestibular (balance) problems, is always present in Ménière's disease, a serious disorder of the fluid balance mechanism of the inner ear. Other possible causes are vascular tumors or large arteriovenous malformations.

Injury to the auditory nerve (8th cranial nerve) produced by certain types of surgery produces gaze-induced tinnitus, in which the intensity of the sound changes when the patient changes the angle of their gaze.

Hyperthyroidism, as in conditions like Graves' disease, is another possible cause of tinnitus. The opposite condition, hypothyroidism, frequently causes tinnitus as well as hearing loss. Thyroid patients need to have their TSH and T3 levels monitored periodically to avoid losing their hearing.

Another cause of hearing loss and tinnitus is Paget's disease of bone, which is a disorder caused by increased bone turnover and enlarged areas at discrete areas of the skeleton. Paget's disease has a genetic (familial) component and occurs more frequently in older people. In one study, 61% of patients with Paget's disease reported tinnitus, compared to 36% of healthy patients in the same age group [23]. Paget's disease is not as rare as many people think.

Non-pathological causes

A variety of non-pathological problems can also cause tinnitus. Although the initiating factor can vary, the process usually starts with some initial injury in or around the ear, which may or may not by itself produce tinnitus. Later, the brainstem becomes involved, and tinnitus is produced. Even though the sound and physical sensations appear to be emanating from the ear, researchers believe that the nerve impulses are generated not in the ear itself, but in the dorsal cochlear nucleus (DCN), which is the relay point in the brainstem for acoustic signals. Different types of tinnitus feel and sound distinctively different to the patient. A patient may even experience one type of tinnitus in one ear and another type in the other ear simultaneously. Some of the non-pathological causes of tinnitus are:

1. Toxic substances

Salicylate, aspirin, antibiotics, cisplatin, quinine, furosemide, hydroxychloroquine, ethacrynic acid, bumetanide, amphotericin B, heavy metals such as mercury, antidepressants such as Wellbutrin (Zyban), and possibly caffeine can cause tinnitus. The inclusion of caffeine in this list is controversial. [This site](#) has a list of other drugs that can produce tinnitus.

2. Noise-induced hearing loss

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Noise-induced hearing loss is often accompanied by hyperacusis, which is a lowered tolerance to elevated levels of sound. For example, louder sounds may sound harsh, unpleasant, or screeching. Mild acute noise-induced hearing loss is also accompanied by a feeling of ear fullness. High levels of noise (acoustic trauma) not only kills hair cells in the cochlea, but also kills neurons in the acoustic nerve and the dorsal cochlear nucleus. The automatic repair process in these nerves is believed to be the main cause of chronic tinnitus (see *Neural Pathways* below).

Although conventional audiology tests are still very important in diagnosing noise-induced hearing loss, the otoacoustic emissions test is now considered the definitive test for noise-induced hearing loss. It is particularly valuable for testing the hearing of infants [3].

3. **Aging**

Aging causes loss of cells that secrete the inhibitory neurotransmitter glycine. Glycine carries the "off" or inhibitory signal from one neuron to another. When glycine-carrying nerve fibers are lost from old age or other factors, the off signal is also lost and certain other neurons begin to fire spontaneously, producing tinnitus. The characteristics of hearing loss caused by aging and by noise trauma are virtually identical. However, the hearing loss caused by noise trauma tends to be maximal around 4 kHz, while hearing loss caused by age begins at the highest frequencies and progresses over time to lower frequencies.

4. **Temporomandibular joint (TMJ) syndrome**

Although TMJ syndrome is usually associated with clicking and popping noises, the ringing form of tinnitus is also a common occurrence. Whiplash can also cause tinnitus. About 10% of whiplash victims develop some ear problem such as tinnitus, deafness, or vertigo [25]. This is generally thought to be secondary to TMJ involvement [26,27].

TMJ syndrome is almost always accompanied by pain or limitation of jaw movement. It's caused by grinding or clenching the teeth by excessive gum chewing, arthritis, or teeth malocclusion. Usually there is a clicking sound when the jaw is opened or closed. It is not unusual for TMJ syndrome to cause muscle spasms that produce tinnitus. Most of the muscle sensory nerves pass close to the nerves that relay sound from your acoustic nerve, so it would not be surprising that pressure on the muscles on the head can trigger tinnitus. It is also very common for movement or pressure on the TMJ to trigger tinnitus. Most experts say that successful treatment of TMJ syndrome will eliminate the tinnitus. However, this does not always happen and may take as long as two years.

According to R.A. Levine, TMJ syndrome is commonly associated with somatic tinnitus. It is generally believed that the ringing noise is caused by the contraction of the jaw muscles and not by the joint itself. If so, the most important thing would be to avoid grinding and clenching the teeth and practice relaxing the affected muscles.

There are several other theories about how TMJ produces tinnitus, including blockage of the Eustachian tube, clonus of the tensor tympani muscle, problems caused by an extra ligament left over from childhood, or impairment of middle ear conduction. The conduction theory is favored by Chole and Parker, and would make sense in cases where TMD is causing a measurable hearing impairment.

5. **Neck injury and electrocution**

Direct blunt neck trauma can cause hearing impairment that resembles noise-induced hearing loss. About 55% of one group of patients who complained of hearing loss after neck trauma also reported tinnitus [28]. There are also reports of hearing loss and tinnitus after high-voltage electric shock and in patients who were struck by lightning. These cases are rare and are poorly understood.

6. **Ear-related causes**

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Although obstruction of the ear canal by wax (cerumen) is an obvious possible cause of tinnitus, some researchers believe that even small amounts of ear wax may contribute to tinnitus. Conductive deafness caused by perforation of the eardrum is another obvious (and easy to diagnose) factor.

7. Dental problems

Dental problems are another frequent cause of tinnitus. Tooth abscesses or impacted wisdom teeth can cause tinnitus. In such cases, further dental work will often cure the problem. Injury of the nerves during extraction of a wisdom tooth has also been known to cause tinnitus.

8. Muscle spasm

Muscle spasm in head or neck is probably the most common cause of tinnitus, accounting for 80% of patients [8]. When the noise is made better or worse by changes in bodily posture, or arm or neck movements, the patient has "somatic tinnitus." Somatic tinnitus is usually unilateral. In its earliest stages, it may be caused by hearing trauma, an injury, or a muscle contraction (such as by grinding one's teeth) that compresses some part of the auditory system. Later, cross-talk occurs between the signals the muscles send to the brain and the signals from the ear. For these patients, relaxation therapy and biofeedback, which help the patient establish voluntary control over the muscle, can be helpful. Once alerted to the possibility that stretching and muscle relaxing exercises may be able to alleviate their tinnitus, patients may be able to devise an effective treatment on their own. In extreme cases, botulinum toxin to temporarily paralyze the muscle, or even surgery, can be performed (see *Treatment* below).

For some patients, anything that causes torsion of the neck, such as having bad posture, using a pillow at night, or bending the neck to look through a microscope, even for a few seconds, causes a muscle spasm that invariably produces tinnitus. This muscle spasm and the resulting tinnitus may not occur immediately, but can start several hours after the neck is twisted, making the cause difficult to identify.

Many leading researchers on somatic tinnitus, such as Robert A. Levine [2], believe that somatic tinnitus resulting from muscle spasms is not produced by compression of nerves or blood vessels in the ear, but rather results solely from the convergence in the dorsal cochlear nucleus of sensory signals from muscle spindles in the head and neck with sound signals from the cochlea. This may explain how somatic tinnitus can even occur in deaf people [4]. The characteristics of somatic tinnitus are [5]:

1. The muscle and tinnitus are ipsilateral (same side).
2. There are no vestibular or neurological abnormalities.
3. Tone and speech audiometry is always symmetric and within normal limits.

Therefore, the first step in identifying the cause of tinnitus is to get a hearing test. If hearing is normal, and pathological causes can be ruled out, it points to a possible muscle, dental, or TMJ problem.

The sternocleidomastoid muscle appears to be the culprit in most cases. Palpation of this muscle, which is the large muscle under the ear on both sides of the neck that acts to rotate the head, aggravates tinnitus in many patients. Other muscles that are commonly involved include the masseter, splenius capitis, and even the middle and upper trapezius and temporalis muscles. The lateral pterygoid muscle may also serve as a trigger point. The current thinking is that these effects are mediated by their excitatory connections to the DCN. Palpation or pressure at other points on these same muscles can sometimes reduce tinnitus.

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The sources of the muscle sensory signals are proprioceptive (location) and cutaneous (touch) projections. Nociceptive (pain) signals are not involved in tinnitus [6].

9. Middle ear infection

A recent Polish study found that over two thirds of the cases of tinnitus in patients below the age of 35 are caused by infection [18]. Infections of the middle ear, which are common in children, are called *otitis media*. This can be a viral or bacterial infection. The symptoms are otalgia (earache) and temporary sensorineural hearing loss. These patients should avoid swimming activities.

Untreated syphilis can also cause tinnitus and fluctuating hearing loss. About 50% of these otosyphilis patients also experience vertigo (dizziness), which indicates inner ear involvement. Treatment with penicillin and steroids restores hearing only about 25% of the time.

A rare condition known as Ramsay Hunt syndrome can also cause tinnitus. This disease is caused by the varicella zoster virus (VZV), which causes chickenpox and shingles. VZV remains dormant in the nervous system after a patient recovers from chickenpox. Sometimes VZV becomes reactivated and produces a skin rash, which is called herpes zoster. When the virus affects the 8th cranial nerve and the geniculate ganglion of the facial nerve, it causes Ramsay Hunt, which is characterized by severe pain behind the ear, tinnitus, facial nerve palsy (sudden paralysis of the face muscles on one side), and a rash on the ear or in the mouth. Ramsay Hunt is similar to Bell's palsy, which is caused by herpes simplex virus. Any paralysis in the face is potentially serious and should be treated as a medical emergency.

Activities such as scuba diving and water skiing can cause traumatic injuries to the ear including eardrum perforation, which causes hearing loss, earache, tinnitus, and vertigo. Sinus infections and migraines are also occasionally associated with tinnitus.

Eustachian tube inflammation can also produce tinnitus, which would usually be accompanied by an earache (otitis) and a sensation of fullness in the ears. This is easily diagnosed by visual examination of the middle ear.

Tinnitus and headache

Many patients with tinnitus also report frequent headaches. Although there are not many articles in the research literature linking tinnitus with either ordinary headaches or migraines, one can imagine several ways the two disorders could be linked.

- **Head injuries** and muscle spasms resulting from stress can cause both tension headaches and somatic tinnitus.
- **Aspirin** and other NSAIDs taken for headache can cause toxic tinnitus.
- **Migraines** are sometimes associated with tinnitus and vertigo. This more often occurs during the aura phase that precedes the migraine than during the migraine attack itself. The association between tinnitus and migraine aura is probably underreported, because patients are understandably more focused on the intense pain than on the ringing sound. In a recent study, 45% of patients with basilar-type migraine experienced tinnitus during the aura phase [20].
- **Intracranial hypotension**, which is caused by a leak in the cerebrospinal fluid, causes tinnitus, hearing loss, neck stiffness, vomiting, vertigo and severe headaches that occur suddenly after standing or sitting (orthostatic headache). The symptoms sometimes resemble Ménière syndrome, but intracranial hypotension can also cause “brain sagging” that causes somnolence, impaired attention, and even coma [19]. This syndrome can also mimic frontotemporal dementia. It is usually caused by lumbar puncture, brain surgery (for instance, to remove a subdural hematoma), or by a fall,

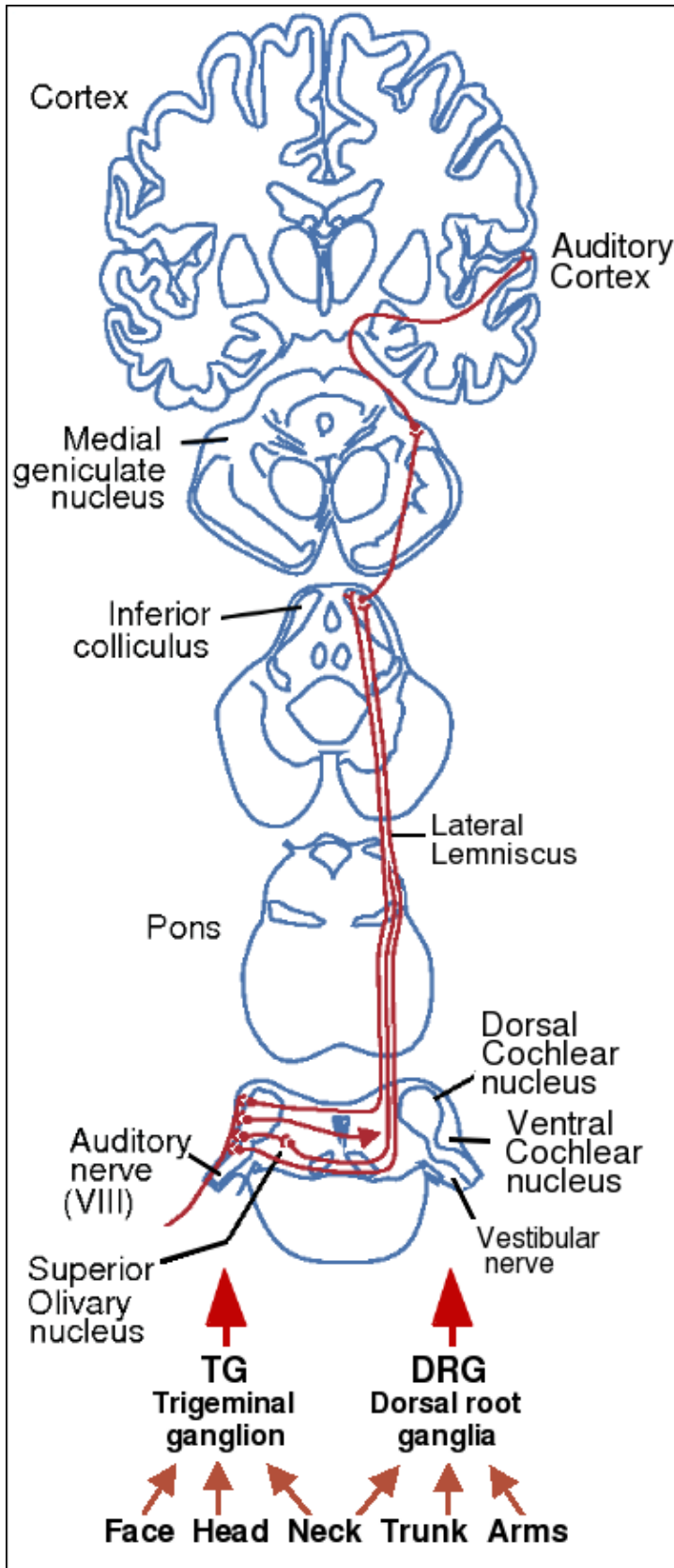
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or it can occur spontaneously. Intracranial hypotension of unknown etiology is called spontaneous intracranial hypotension or SIH.

- **Superficial siderosis** A rare condition known as superficial siderosis, or iron in the outer layers of the brain caused by bleeding, causes ataxia and sensorineural hearing impairment which can manifest as tinnitus.
- **Subarachnoid hemorrhage** Some popular books claim that subarachnoid hemorrhage causes tinnitus. While this may be true, it is not a common cause of tinnitus. Any brain hemorrhage is a medical emergency, and in such cases ringing in the ears may be the least of the patient's problems. The classic symptom of a subarachnoid hemorrhage is a sudden violent headache, usually described as the worst headache of the patient's life.
- **Aneurysms** Brain aneurysms can also cause hearing loss and tinnitus [21]. Tinnitus resulting from an aneurysm is typically pulsatile with a “swishing” or roaring sound that coincides with the heartbeat [22]. If you experience this type of tinnitus, especially if you have symptoms of ischemia such as dizziness, you should have it checked, because it can be a sign of a weakened blood vessel wall that could hemorrhage without any warning, possibly causing a stroke. It could also be a sign of partial occlusion (stenosis) of the carotid artery, or it could be caused by a harmless malformation of the arteries. Your physician will perform angiography, ultrasound, or MRI to evaluate it.

In one study, 68% of the patients with pulsatile tinnitus were found to have some underlying pathology, the most common being a dural arteriovenous fistula or a carotid-cavernous sinus fistula [24]. However, in another study, so-called “benign” intracranial hypertension was the most frequent cause.

Neural pathways involved in tinnitus



Neural pathways involved in tinnitus. All

pathways and nuclei are bilaterally symmetric.

Although a variety of factors can cause tinnitus, the current theory is that they all produce tinnitus the same way. Therefore, it is important to understand the basic neuroanatomy of the auditory system to understand the

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various treatments that have been proposed.

Ventral cochlear nucleus

The ventral cochlear nucleus (VCN) is a relay point in the brainstem that receives data from muscle stretch sensors in the face, head, and neck. Sensors in muscles of the neck, trunk, and arms send signals to the VCN by way of the dorsal root ganglia and the trigeminal ganglion. These sensors tell the brain how strongly the muscle is contracted, and tell it the position of the body part affected by the muscle. The ventral and dorsal cochlear nuclei relay signals to each other and to the brain.

Inferior colliculus

The inferior colliculus is an important relay point in the midbrain. It receives data from lower brainstem nuclei such as the ventral and dorsal cochlear nuclei, and relays this information to the brain. Neurons in the inferior colliculus fire more rapidly when tinnitus is occurring. Signals from the tongue muscle and temporomandibular joint send signals to the dorsal cochlear nucleus by way of the inferior colliculus. This may explain why temporomandibular joint syndrome frequently causes tinnitus.

Dorsal cochlear nucleus

The dorsal cochlear nucleus (DCN) is the most important brainstem region for relaying auditory signals to the brain. It is also the region where researchers believe the phantom sounds heard by tinnitus sufferers are generated. As shown in the figure, the dorsal cochlear nucleus receives auditory signals from the hair cells in the cochlea, where sound is detected, as well as signals related to eye movements, mediated by the vestibular system. It also receives a variety of signals from muscle position sensors in the face, neck, head, trunk, arms, tongue, and temporomandibular joint, which are relayed to it via dorsal root ganglia and other nerve centers. The DCN acts as a relay and forwards all these signals to the appropriate place in the auditory cortex and elsewhere in the brain. As the signals travel from the DCN to the brain, left and right are switched. Therefore, sound entering the right ear and muscle signals on the right side of the head are processed in the left side of the brain, and vice versa. The feature to remember is that sensory data from muscles and acoustic data from the ears are both relayed to the brain at very nearly the same point in the brainstem.

Inhibitory signals

Auditory signals from the cochlea travel to the cochlear nerve, which combines with the vestibular nerve to form the vestibulocochlear nerve (acoustic nerve, cranial nerve VIII) on their way to the cochlear nuclei, which process them and relay them to the brain. However, these signals are not just 1's and 0's like in a computer. The DCN and VCN process both "on" (excitatory) and "off" (inhibitory) signals. The inhibitory signals perform a vital function: they prevent other neurons from getting stuck in a continuous "on" position. If the inhibitory signals are blocked due to injury or some other factor, the cochlear nuclei may "decide" that a sound is present when it is not. The patient would then hear a ringing or hissing sound.

These inhibitory signals are carried by the neurotransmitter glycine. This means that specific neurons, called glycine interneurons, transmit "off" signals to other neurons, which contain receptors for glycine. If a glycine neuron is injured and dies, the cells it is connected to must adapt to the loss. Each neuron "grieves" in its own way. Some neurons adapt by decreasing the number of glycine receptors, while other adapt by increasing the number of receptors. Thus, the entire network is affected and may start sending false signals. Some researchers believe that inhibitory synapses are more fragile than excitatory synapses. If so, that could explain why even small injuries to the head can cause lifelong tinnitus.

Because the cochlear nuclei are so important in tinnitus, it is sometimes said that tinnitus is primarily a brain phenomenon. Proof of this is that tinnitus can still occur after the auditory nerve has been cut. About 50% of these patients, who are now completely deaf, still experience tinnitus. Thus, even those people so tormented by tinnitus that they would prefer permanent deafness cannot always obtain relief.

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However, calling tinnitus a "brain disorder" would be misleading. Tinnitus does not occur in the auditory cortex [7] although some researchers believe that tonotopic remapping (functional rewiring of the cortex) can occur on the auditory cortex. In this sense, tinnitus has some similarities to phantom limb syndrome. Tinnitus is not, however, a brain disease, and it is most definitely not a symptom of a psychiatric disorder or hysteria. In the absence of normal stimuli, the DCN undergoes synaptic plasticity, a form of neuronal reorganization similar to learning that, according to the current theory, results in tinnitus.

Synaptic plasticity

Trauma from loud noises not only injures the hair cells in the cochlea; it also causes neurodegeneration, or death of nerve cells, in the auditory nerve and brainstem. Researchers have found that in rodents, noise trauma causes neurodegeneration in the dorsal cochlear nucleus that continues to progress for up to two weeks, as well as neurodegeneration in the auditory nerve and ventral cochlear nucleus that can continue to progress for up to eight months after sound exposure. The exact length of time that neurodegeneration continues in humans is not known, but is probably similar. This neurodegeneration in turn triggers an automatic repair process that includes sprouting and regrowth of new axons and nerve terminals. Although they try to reconnect in exactly the same way as before, the new nerve terminals are not always able to do so; some rewiring, called synaptic plasticity, occurs.

As the neurons repair and regrow, two things can happen. First, since there are fewer inhibitory glycine neurons, the neurons may begin sending continuous signals to the brain. Secondly, signals from other, non-acoustic sources, such as the temporomandibular joint or muscle sensors in the head and neck, may get mixed with sound signals in such a way that movement of these muscles can trigger the generation of phantom sounds.

In some ways, this is bad news. We cannot administer drugs that interfere with synaptic plasticity, because it is essential for both learning and repair within the nervous system. Synaptic plasticity is an essential part of how we remember things. On the other hand, it is good news: it means these synapses may, in principle, be able to "learn" not to generate false signals. Researchers have found that this is indeed possible. White noise therapy (see below) can "teach" these neurons not to produce the false signals. If white noise is given in time, and for a long enough period, tinnitus can be prevented (see *Sound therapy* below).

Anxiety

Physicians used to believe that tinnitus results from anxiety or stress. We now know that this is false. Animal studies have conclusively shown that anxiety does not cause tinnitus. However, tinnitus often provokes anxiety and creates stress, because patients fear they may be losing their hearing or may have a serious underlying problem. Anxiety may exacerbate the tinnitus and the distress felt by patients. Patients with a "perfectionistic" personality type may be more likely to seek treatment, leading to the inaccurate perception among physicians that tinnitus has a psychological component. Tinnitus is also not a conditioned reflex, but different patients may be more or less disturbed by the same amount of tinnitus.

Treatment

Once mechanical problems such as impacted wax are eliminated as possible causes, a hearing test should be given to determine whether you have noise-induced hearing loss. Most treatments are still in the experimental stage. The most promising areas of research at the moment are sound therapy, neck exercises, and acupuncture. Some progress is also being made using drugs to inhibit the neuronal pathways.

Drugs

There are currently no drugs approved for treatment of tinnitus. The drugs listed below are in various stages of experimental testing.

Lidocaine The earliest treatment ever discovered was lidocaine (Xylocaine), which effectively suppresses tinnitus. Lidocaine is a local anesthetic which acts by blocking voltage-gated sodium channels in neurons, preventing neuronal transmission. Since it is only short-acting (hours) and must be injected by a physician, it is not considered to be useful as a treatment. High doses of lidocaine can also cause tinnitus. Intradermal lidocaine is a possibility, but it has not been studied thoroughly.

Glycine receptor agonists For tinnitus that is caused by overexcitation of the cochlear nucleus, it may be possible to develop glycine receptor agonists. These drugs would mimic the natural neurotransmitter glycine, and re-balance the excitation / inhibition pathways. Thus, the future for tinnitus sufferers is not as gloomy as is commonly believed. Research into glycine receptor agonists is underway.

An interesting fact is that the poisonous alkaloid strychnine is a glycine receptor antagonist, and acts by blocking the glycine receptor; thus glycine receptor agonists might also be beneficial as a treatment for strychnine poisoning.

GABA-A receptor agonists GABA is another inhibitory neurotransmitter, similar to glycine. A class of drugs called benzodiazepines already exists. These drugs are GABA-A receptor agonists, and might be expected to work on tinnitus by activating the inhibitory limb of the neural network. Some researchers have reported moderate success in treating tinnitus with GABA agonists such as baclofen, clonazepam, and diazepam. However, these drugs are also tranquilizers, and cause undesirable CNS side effects. If the current theory about tinnitus being generated in the brainstem is correct, topical application of these drugs is not likely to work, because the drug would be unable to reach the brainstem. Thus, a systemic drug that is targeted to the brainstem is needed.

Anticonvulsants Because of the parallels between tinnitus and epilepsy (both of which result from overexcitation of neurons), low doses of anticonvulsants such as gabapentin and carbamazepine have been tried, but with little success so far. Although gabapentin acts on the GABA pathway, it is not a direct GABA agonist; it is believed to work by activating glutamic acid decarboxylase, the enzyme that converts glutamate to GABA. It therefore acts by causing the cell to produce more of the inhibitory neurotransmitter GABA.

Acamprosate Acamprosate, a drug used to treat alcoholism, acts as a glutamate antagonist and GABA agonist. It acts by increasing the number of GABA reuptake sites and increasing GABA transmission. One Brazilian group reported a modest but statistically significant benefit using acamprosate to treat tinnitus.

NMDA antagonists A class of drugs known as NMDA receptor antagonists has been shown to block salicylate-induced tinnitus in animals. These drugs interfere with the excitatory neurotransmitter glutamate. However, salicylate may produce tinnitus by a different mechanism than normal tinnitus, so NMDA antagonists may not be effective in patients. Since glutamate is the predominant neurotransmitter in the brain, these drugs would also act as general tranquilizers or anesthetics. Also, some NMDA antagonists are potent neurotoxins. NMDA antagonists such as caroverine have had moderate success, but unfortunately this class of drugs has very significant side effects, such as psychosis. The anti-Alzheimer drug memantine, and neramexane, which are both NMDA receptor antagonists, are currently being tested.

Neuromuscular-blocking drugs The only neuromuscular blocking drug currently used for tinnitus is botulinum toxin, which has been used to paralyze specific muscles. Other drugs, such as dantrolene, a muscle relaxant used to treat muscle spasticity, and drugs similar to tubocurarine, a powerful quaternary ammonium

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muscle relaxant, have not been tested. Some antihistamines, such as orphenadrine, which is used to treat muscle spasms, might also be useful. However, there are few reports of these drugs being tested. Injection of botulinum toxin into a muscle will paralyze it for 4-6 months; therefore, injecting it into one of the large muscles needed for chewing or supporting the head would be a *very bad idea*.

Antidepressants Tricyclic antidepressants such as nortriptyline and serotonin reuptake inhibitors such as paroxetine and sertraline have been reported to reduce tinnitus. These drugs may work because of similarities between tinnitus and neuropathic pain. However, clinical studies have shown conflicting results, with tinnitus sometimes being reduced in depressed patients but not non-depressed patients.

Several chapters on drug treatments can be found in reference [12].

Sound therapy

White noise The theory that tinnitus is created by the loss of inhibitory stimuli has resulted in the most promising new therapy for tinnitus so far: white noise therapy. White noise, such as that produced by a Marsona sound generator, has two effects: first, it masks the ringing sound; and second, it helps to stimulate the inhibitory pathways by producing a continuous background of sound. The extent to which these pathways can be rewired is still not entirely clear, but it is clear that white noise creates additional excitatory and inhibitory stimuli that appear to be able to influence the synaptic regrowth in the DCN, and prevent tinnitus from starting. (They are also great for preventing insanity caused by having a neighbor who is overly fond of rock music.)

According to the theory described above, noise-induced hearing loss produces tinnitus because it is, in effect, creating silence. Indeed, most normal people will experience tinnitus when placed in an anechoic chamber. Therefore, one could logically expect background noise to prevent it. However, this is difficult to prove, because noise also masks tinnitus.

Nevertheless, it is true--at least in animals. Noreña and Eggermont have found that if white or blue noise (5-20kHz) is presented immediately after noise trauma and maintained over a period of several weeks, the neuronal changes that cause tinnitus and hearing loss are abolished [9, 10]. This remarkable finding shows that it may be possible to prevent noise-induced tinnitus even in cases of acoustic trauma.

It goes without saying that white noise, or any other noise, above 85 decibels will cause hearing loss, especially if maintained for long periods of time. Moreover, there is some suggestion that white noise could be harmful for infants. Also, it stands to reason that sound therapy, or any other therapy, will not work unless whatever caused the tinnitus in the first place is stopped.

Stimulation of muscle stretch receptors

Certain neck exercises have also been highly effective at eliminating tinnitus in some patients. Specific exercises are sometimes recommended to stretch the sternocleidomastoid muscle. For example, the patient may be instructed to lower the shoulders, tilt the head up, then pull in the jaw to relax the muscle. This is an easy and safe therapy if done within reason. The theory predicts that relaxing the muscle will reduce the stretch receptor signal and thereby reduce the excitatory stimulation to the dorsal cochlear nucleus that creates the tinnitus. If the correct muscle is stretched, the tinnitus will often disappear within one or two minutes. If the patient is unable to stretch the correct muscle, but makes an effort to relax the muscles around the ear, it may take longer for the noise to disappear. Neck exercises need to be tailored to the individual.

Surgery

Microvascular decompression A surgical technique called microvascular decompression, which is an effective treatment for trigeminal neuralgia, is sometimes beneficial for tinnitus as well. More drastic treatments include cochlear implants that replace the missing neuronal stimulation with artificial electrical signals. Surgery of the ear or brain is extremely dangerous, and is considered a treatment of last resort.

Meniere's disease For Ménière's disease, an experimental treatment involves intratympanic injections of drugs such as gentamicin or steroids. In this procedure, the eardrum is anesthetized and the drug is injected through the eardrum. Since this procedure perforates the eardrum, it is difficult to evaluate its effectiveness for tinnitus. Because of the extreme invasiveness of this procedure, patients are understandably reluctant to undergo it. Patients might also falsely report improvement in order to avoid a second treatment.

Cochlear implants Because hearing loss produces tinnitus, anything that even partially restores hearing will often reduce the tinnitus. Many studies have shown that, if a cochlear implant restores some hearing, it usually reduces the tinnitus. However, because a cochlear implant completely and permanently destroys all remaining normal hearing, only patients with profound hearing loss are candidates for a cochlear implant. A neurologist should be the one to do a referral.

In a recent study, all the patients with tinnitus reported a partial (61%) or complete (39%) elimination of their tinnitus after receiving a cochlear implant. A cochlear implant produced tinnitus in 12% of the patients who did not have it before the operation [17].

Hearing aids A hearing aid may have the same effect, and would be much safer than an implant. There are special hearing aids that contain sound generators which stimulate the auditory pathway. In some patients, the combination of restored hearing and sound therapy can cause the neural pathways to reorganize in such a way that the generation of tinnitus is reduced. Unfortunately, as with implants, if hearing cannot be restored, sound therapy is unlikely to work.

Electrical brain stimulation Some researchers believe that another treatment called deep brain stimulation might be able to treat tinnitus. Magnetic stimulation, which is a non-invasive procedure, is used to find the precise location where tinnitus is being perceived on the auditory cortex. Then a permanent electrode is inserted at this location to prevent the patient from hearing the noise. This treatment will not restore hearing. It is still in the experimental stages and is not available for patients yet.

This treatment should not be confused with electrical stimulation of the ear, which was proposed as a treatment back in the 1980s. If this treatment worked at all, it was likely due to its effects on the muscle or by changing blood circulation.

Alternative medicines and nutritional supplements

Alternative medicine treatments, including ginkgo biloba, zinc, magnesium, and magnets, in general are believed by the medical establishment to have little benefit, but saying even this is controversial, because little solid research has been done on many of these treatments. However, there is no doubt that good general nutrition is important for recovery from any type of injury, including injury that produces tinnitus.

Magnesium is a very promising new treatment for noise-induced hearing loss [13, 14], which often causes tinnitus, but in such cases therapy must start within a few hours of the noise trauma to be effective.

Ginkgo biloba is supposed to act as a vasodilator, and may improve blood circulation in regions near the cochlea that have been stressed by noise trauma, but so far there has been more enthusiasm than rigorous

science in the field.

Dihuang (Rehmannia glutinosa) is a Chinese herb traditionally used for tinnitus and to protect against noise-induced hearing loss. Some evidence suggests it may induce GDNF (glial cell line-derived neurotrophic factor) or act as an antioxidant. It has not been rigorously tested.

Vinpocetine (Cavinton) Vinpocetine is an anti-inflammatory agent that inhibits the enzyme IKK. A recent report suggests it may be useful in treating chronic obstructive pulmonary disease. One Polish group in 1997 [29] reported improvement in hearing and tinnitus caused by acoustic trauma if given within the first week, when inflammation was presumably at a maximum. However, there have been no reports since then. There are anecdotal reports that it may also act as a vasodilator.

Zinc There is wide variability in the reported results with zinc. Some studies on the benefits of zinc have been hampered by researchers giving metallic elemental zinc instead of the active form, which is Zn^{2+} . There is also wide variation in epidemiological results on the prevalence of zinc deficiency, with estimates ranging from 2 to 69% of the population. As we scientists say, more research is needed.

Acupuncture has produced dramatic success in cases of somatic tinnitus that were not accompanied by hearing loss [11]. Other researchers have had less positive results. Because many Westerners seriously underestimate the complexity of acupuncture and the amount of skill necessary to make it work, it is necessary to find an acupuncturist experienced in tinnitus treatment.

Magnets appear to be useless, but magnets should not be confused with transcranial magnetic stimulation, which is an accepted technique for inducing electrical currents in the brain noninvasively. Electrical stimulation of the scalp and auricle has produced beneficial results in a few people.

Cognitive behavior therapy

Cognitive behavior therapy is the new term for psychotherapy, which has acquired very negative connotations. Since tinnitus is a physical problem, there is little reason to expect a benefit from cognitive behavior or "talk" therapy. The main goal seems to be to get the patients to ignore the sounds and live with it: to learn, in effect, to stop worrying and love their tinnitus. Relaxation therapy, however, may be of benefit in reducing muscle tension that contributes to tinnitus.

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