Differentiating and Treating Various Types of Tinnitus

- Dave Siever, February 2021

INTRODUCTION

Tinnitus is the perception of sound, usually a steady tone or ringing in the absence of an external auditory stimulus being present in the environment. Several factors contribute to the perception of tinnitus and various professions have their own perspective as to what causes tinnitus. Medical doctors might see tinnitus as referred sound emanating from arteries or other mechanical structures in which the sounds they make are perceived by the ear. Dentists see tinnitus from the perspective of muscle tension resulting from malocclusion. Psychologists see it as a secondary symptom stemming from stress, anxiety and insomnia. Neuroscientists view tinnitus from the perspective of neuroplasticity and neurological issues resulting in poor noise-cancelling and/or attentional circuits and abnormal brainwave rhythms in seemingly unrelated regions of the brain. For example, studies using electromyographic (EMG) biofeedback found it ineffective for the treatment of tinnitus (Haralambous et al., 1987; Borton & Clark, 1988), but an EMG biofeedback/stomatognathic (splint) treatment study which focussed on those with occlusal interferences, bruxism and jaw-clenching behaviors in relation to their tinnitus, found relatively good results (Erlandsson et al., 1991). Another study by Ogata et al., (1993), for tinnitus which was supposed to be reinforced by mental distress and muscle tension showed favorable results when the biofeedback was successful. In conclusion, in order to effectively treat tinnitus, it is important to understand the various types, as well as the etiologies involved.

TYPES OF TINNITUS

1) Objective or somatosound tinnitus - Where a real (objective) sound exists that is audible to another person such as when placing a stethoscope over the ear canal.

2) Muscle tension (myogenic) tinnitus - Resulting from (a) malocclusion (an unbalanced bite) or (b) stress and anxiety. TMJ-related tinnitus in which jaw-muscle tension places excessive forces on surrounding muscles, nerves and arteries of the ear. This form of tinnitus is often resolved by dental means or the reduction of stress and anxiety in which reduced bruxism and clenching leads to cessation of the tinnitus.

3) Neuroplastic changes from cochlear dropout (lost frequencies from cochlea problems) or deafferentation (nerve issues where the signal doesn't get through to the auditory cortex).

4) Neurological/noise cancelling circuits - This type is subjective tinnitus where the sounds are independent of an acoustic etiology, audible only to the person and is thought to largely be top-down neuronal dysregulation.

5) Neurological perspectives on tinnitus

1) Objective or Somatosound Tinnitus

Objective tinnitus (OT) is a perceived sensation of sound that occurs in the absence of external acoustic stimulation. But by placing a stethoscope over the external auditory canal, the tinnitus sounds can be heard by the examiner and therefore has an actual somatic source. OT is much less common than subjective tinnitus, but because it has an identifiable (somatic) cause, it is often treatable; whereas subjective tinnitus is often idiopathic and difficult to treat.

OT can occur either from perception of an abnormal body sound or abnormal perception of a normal body (Hertzano et al., 2016). Those with OT report that the frequency or intensity is altered by body movements such as clenching the jaw, turning the eyes, or applying pressure to the head and neck, which is why it is often referred to as somatosound.

OT may have vascular or mechanical origins, such as a referred sound from stenosis in the carotid or vertebrobasilar system. OT in the form of a series of sharp, regular clicks can originate from abnormal muscular contraction of the nasopharynx or middle ear and from palatal myoclonus (Lanska, 2020). Pulsatile tinnitus can also manifest subjectively as an increased awareness of blood flow in the ear (Han et al., 2009). Pulsatile tinnitus superimposed on steady tinnitus could result from the pulsation of blow flow with the spiral capillary of the basilar membrane (Lanska, 2020).

OT may be associated with a variety of vascular noises arising from disruption of the otherwise smooth laminar flow into turbulent eddies within the internal jugular vein or jugular bulb, arteriovenous malformations or fistulas, cavernous hemangiomas, aneurysms, vascular stenoses (particularly of the carotid arteries), vascular tumors, and intracranial hypertension (Lanska, 2020).

OT may also be associated with a patulous eustachian tube. The perceived sounds are synchronous with nasal breathing, which is usually more marked in the upright position and gradually disappears when lying down, or temporarily subsides with sniffing, snorting, or a valsalva maneuver (Lanska, 2020).

2) Muscle Tension (Myogenic) Tinnitus

Muscle tension tinnitus involves tense jaw-closing muscles such as the masseter and temporalis muscles. Tension within these muscles is easy to test with palpation. If pressing and slowly rubbing the masseter and/or temporalis muscles results in pain, then the muscles are likely fatigued. Typically, the mandible posteriorizes from the tension and the condyle (ball on the mandible) puts pressure on the auricular and tympanic nerves and arteries, which in turn causes tinnitus. Either long-term muscle tension or a physical hit to the jaw can tear the inter-articular disk, the cartilage within the temporo-mandibular joint (TMD) that separates and lubricates the lower jaw from the upper jaw. Once this cartilage has been damaged, the mandible will change position, thus straining the muscles and putting pressure on the tympanic and auricular nerves and arteries that involve the ear and hearing.

Psychophysiologic derived TMJ and associated tinnitus may have emotional origins. It has been shown that all people (controls and TMJ Dysfunction) show high levels of masseter tension during initial exposures to a stimulus-response task (Yemm, 1971). Further, it has been shown that masseter muscle

activity increases during challenging tasks, primarily when the subjects made errors (Yemm, 1969). The Yemm study implies a direct relationship between self-critical thoughts and tension. However, controls show a trend towards relaxation with repeated exposures to the task, whereas those suffering with TMJ Dysfunction show an initial relaxation phase during the first few exposures followed by a marked increase in masseter muscle tension with repeated exposures to stimulus-response tasks. This performance anxiety was termed "TMJ Personality" by Yemm. Anxiety, stress and the consequent impact on state and trait arousal are a major part of a variety of dental disorders, which often include tinnitus as a symptom. (Spielberger et al., 1970; Yemm, 1971; Weinstein et al., 1971; Rugh & Solberg, 1975).

Tension-caused tinnitus will typically show soreness on palpation of the masseter, temporalis, and lateral-pterygoid muscles, often accompanied with soreness of the TM joint. A damaged TM joint can cause a locking jaw or clicking and popping sounds when opening and closing the jaw. Damage to the TM joint and the resulting discomfort and pain is termed Temporo-Mandibular Dysfunction (TMD). In my TMD research days at the University of Alberta, we treated hundreds of cases of tinnitus simply by anteriorizing the mandible by a few mm with a splint.

The stress-caused type of tinnitus also involves the jaw-closing muscles such as the masseter and temporalis muscles but also may include the tensor veli palatini (TVP) muscles. These are two small muscles with each one venting the eustachian tube opening. Their job is to vent the eustachian tubes during swallowing. You may have noticed that while changing elevation, your ears become plugged, but when you swallow, there is a click or popping sound and you can suddenly hear again. That is from the TVP muscles venting the middle ear during a swallow. Under stress, these muscles often go into spasm and not provide venting during swallowing and therefore the air in the middle ear becomes stale, builds up pressure and causes tinnitus to occur (Ellenstein et al., 2013). Sometimes, during a cold or flu, viral damage and/or mucous can plug the opening and cause tinnitus (Cohen et al., 2014) as well as viral vaccinations (Kolarov et al., 2019).

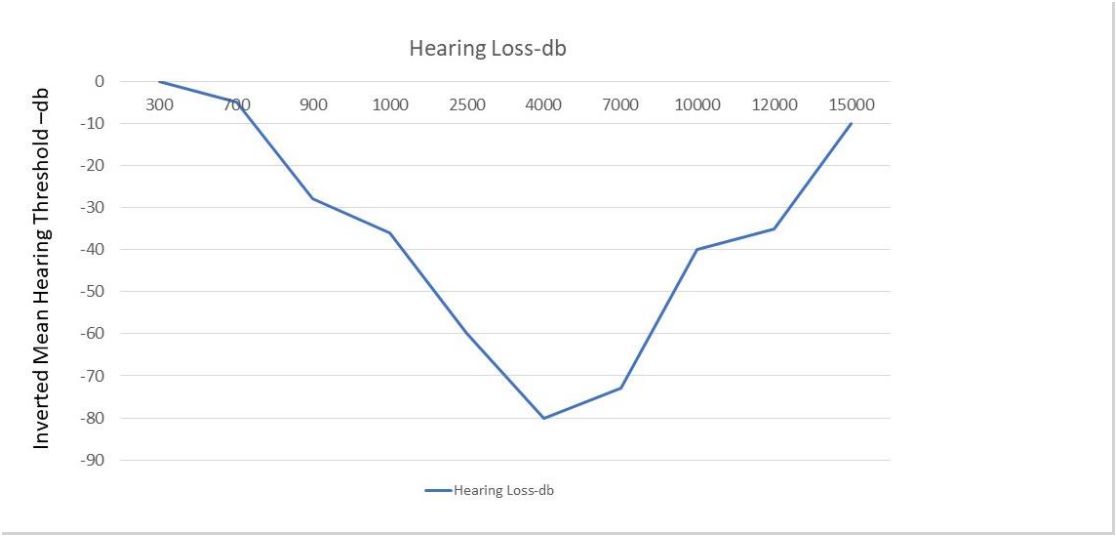
3) Cochlear Dropout or Deafferentation

Cochlear dropout (CD) type of tinnitus involves a loss of a frequency range from cochlear damage or damage to the nerves feeding up to the pons or medial geniculate. Because the frequency range is now lost, the brain recruits more neurons to reclaim the lost audio and, in the process, this produces ringing in the ears. Damage to the hearing system in which the auditory signals don't reach the brain, is known as deafferentation. Neuroplastic brain changes in response to deafferentation comprise both auditory circuits and hippocampal (learning) circuits, depending on the severity of deafferentation (Vanneste & De Ridder, 2016).

Our acoustic environment is filled with amplified audio sources (e.g. MP3 players, video gaming stations, and sports/entertainment venues). Excessive audio levels are causing mechanical damage to hair cells, with the most vulnerable structures being the stereocilia. Damage to these structures can cause permanent loss of hearing in certain frequency bands (Harrison, 2012). Damage to the auditory nerve and associated circuits can also contribute to the deafferented type of tinnitus. Figure 1 is an inverted

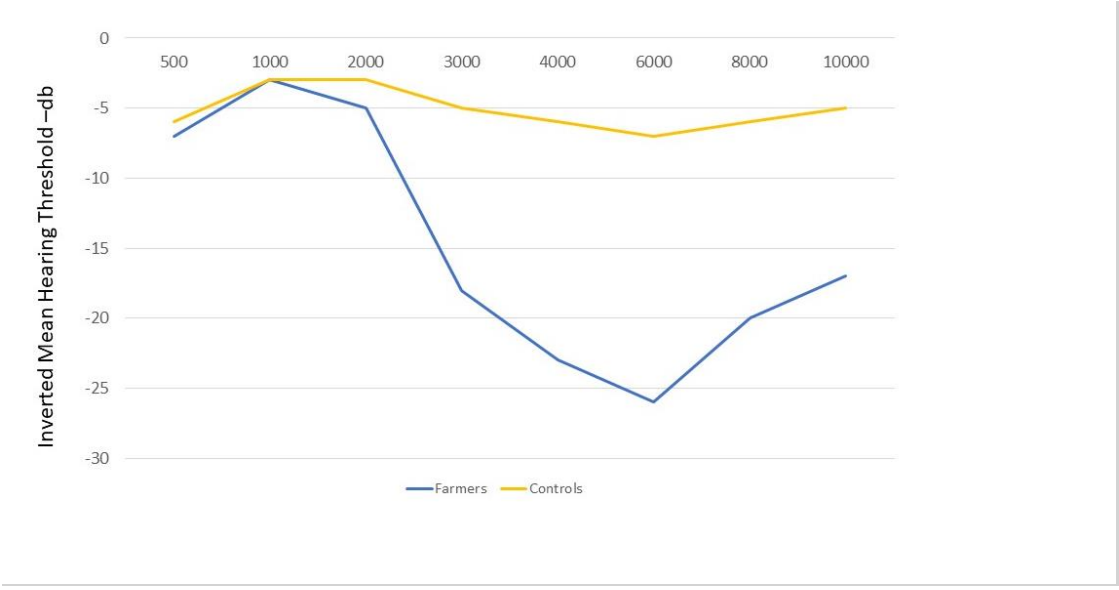
graph of hearing threshold (showing hearing loss) in a cat two months following exposure to a 3 KHz tone at 115 dB for 2 hours (adapted from Harrison, 2012).

Figure 1. Hearing Loss in a Cat Two Months Following an Exposure to a 3 KHz Tone for Two Hours



In days past, farmers and rock musicians were the demographics most prone to CD. The CD in farmers came from exposure to droning tractor engines and machines (Plakke & Dare, 1992). More recently, modern sound-proofed farm equipment and improved preventative behavior has greatly reduced CD for farmers. Figure 2 shows hearing in farmers in their 40s and 50s correlated with increased hearing impairments. As age increases, hearing impairments in farmers further increases in comparison to the control group.

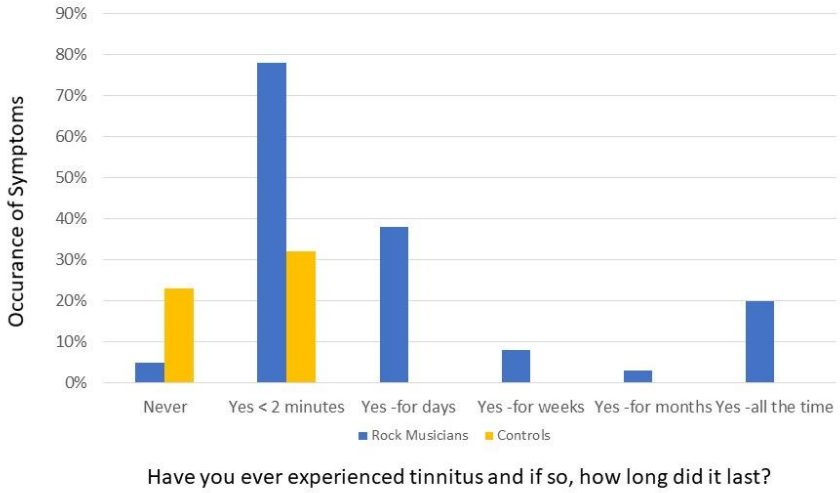
Figure 2. Hearing Loss in 40- to 50-Year-Old Farmers as Compared to Controls



Hearing loss and tinnitus is prevalent amongst rock musicians. Rock musicians experience a broad range of hearing loss which increases with time playing music. Bass and guitar players showed greater hearing loss than vocalists. Data shows that wearing hearing protection does reduce hearing loss as shown in Figure 3. There is a strong correlation between rock musicians vs normal for the development of tinnitus as seen in Figure 4 (Lein Størmer et al., 2015).

Figure 3. Hearing Loss in Rock Musicians with and without Hearing Protection

Figure 4. Prevalence of Tinnitus in Rock Musicians vs Normals



In 2005, Helfer et al. found that soldiers deployed to battle zones were 52.5 times more likely to develop auditory damage than non-deployed soldiers. Presently, non-combat type of CD-type of tinnitus is prominent in children, and caused by recreational music, which is much too loud, resulting in damage to

the cilia in the cochlea (Harrison, 2008). Given that hearing is most acute at 1 KHz and a lot of music (guitars, vocals, and other musical instruments) are rich in frequencies around 500 Hz to 4 KHz, tinnitus is generally produced in around that frequency range. (To get an idea as to how annoying 1 to 2 KHz sounds are, there are plenty of sound-generating apps for iPhones and Androids, which allow a person to experience various tone pitches). There can be other causes of damage to the cochlea, auditory nerves, and brain stemming from physical trauma, viral infections, ageing, and so on. Both CD and deafferentation will show a loss of hearing at certain frequencies during an audiology exam.

Weisz et al. (2007) tied deafferentation and CD with brainwave activity and concluded that: "Based on present and previous results, we have concluded that cochlear damage, or similar types of deafferentation from peripheral input, triggers reorganization in the central auditory system. This produces permanent alterations in the ongoing oscillatory dynamics at the higher layers of the auditory hierarchical stream. The change results in enhanced slow-wave activity reflecting altered corticothalamic and corticolimbic interplay. Such enhancement facilitates and sustains gamma activity as a neural code of phantom perception, in this case auditory."

This "reorganization" is mediated by a shift of alpha activity towards high-frequency gamma band oscillations (Joliot et al., 1994). In a deafferented state, neural activity is shifted towards theta band activity (Steriade, 2006) which in turn leads to a decreased lateral inhibition mediated by γ-amino butyric acid, or GABA (Llinás et al., 2005) and results in a persistent and thus pathological gamma activity of the neighboring neurons, also known as the 'edge effect' (Llinás et al., 1999; Llinás et al., 2005). This sustained gamma band activity present in temporal areas is related to tinnitus as observed by quantitative electroencephalography (qEEG) (Ashton et al., 2007) and magnetoelectroencephalography (MEG) (Weisz et al., 2007; Llinás et al., 1999; Llinás et al., 2005). The coupled presence of theta and gamma activity in tinnitus has also been demonstrated by recordings from an implanted electrode overlying the auditory cortex in a tinnitus patient (De Ridder et al., 2011). Furthermore, this theta-gamma coupled activity is maximal at the area of blood oxygenation level dependent (BOLD) activation evoked by tinnitus-matched sound presentation in the magnetic resonance imaging (MRI) scanner (De Ridder et al., 2011), suggesting that the BOLD signal can demonstrate pathological tinnitus-related activity in the auditory cortex (Smits et al., 2007). Moreover, a positive correlation has been demonstrated between the amount of gamma band activity in the auditory cortex and the perceived tinnitus loudness in the contralateral auditory cortex (van der Loo et al., 2009).

4) Neurological/Noise Cancelling Circuits

The following description by Rauschecker et al. (2010), is the best description that I have come across to date. Having taken a few liberties with some editing, it basically goes as follows:

The initial tinnitus creating process results from peripheral deafferentation or "dropout" in a frequency range from the loss of hair cells through injury, aging, or loud-noise exposure that begin to induce a process of plastic reorganization, which leads to a perceptual filling-in of the deafferented frequency range but also generates hyperactivity (and thus an initial tinnitus signal) in the ascending auditory pathways. Normally, this unwanted noise signal is identified by the limbic system and eliminated "tuned

out" from perception by feeding this noise back to the (inhibitory) Thalamic Reticular Nucleus (TRN), which subtracts it from the afferent (incoming) auditory signal. Thus, this circuit serves as an active neural "noise-cancellation" system.

To expand on this, Rauschecker et al. have theorized that the nucleus accumbens (NAc) and its associated paralimbic networks in the ventromedial prefrontal cortex (vmPFC) are involved in long-term habituation to continuous unpleasant sounds. Sound-evoked neural activity is relayed from the auditory periphery via the brainstem and thalamus via the Medial Geniculate Nucleus (MGN) to the auditory cortex for conscious perception. The same auditory signal is also directed in parallel to the amygdala and the subcallosal area (which includes the Nucleus Accumbens (NAc) region of the ventral, "limbic" striatum and the ventral medial Pre-Frontal Cortex (vmPFC) for evaluation of the sound's emotional content.

From the subcallosal area, an excitatory projection feeds back to the TRN, which in turn selectively inhibits the sections of the MGN corresponding to the unpleasant sound frequencies. This gain-control mechanism leads to a highly specific filtering (tuning out) of repetitive unwanted noises, which in turn, no longer reach conscious perception in the auditory cortex.

So long as the NAc-system is intact, the tinnitus signal is filtered out and will not be relayed to the auditory cortex. If, however, the NAc-system becomes compromised, noise cancellation of the tinnitus signal at the thalamic level (via the MGN) stops and tinnitus perception results. Long-term reorganization of the auditory cortex develops and the tinnitus becomes chronic.

The raphe nuclei, which control serotonin levels and sleep cycles, provide a major input to the NAc and may thus contribute to the correlation between tinnitus strength and insomnia.

5) Neurological and Emotional Perspectives on Tinnitus

Neurological tinnitus has been hypothesized to be the expression of a thalamocortical dysrhythmia (TCD), in which there is a constant (pathologic) coupled theta-gamma band activity (theta 4–7 Hz, gamma> 30 Hz) due to hyperpolarization of specific thalamic nuclei (Llinás et al., 1999). TCD has been implicated in a variety of other disorders, including visual snow (VS) a kind of visual noise, similar to the experience of watching an old-fashioned analog TV with poor reception (Lauschke et al., 2016), neurogenic pain, tinnitus, Parkinson's disease, and depression (Llinás et al., 1999). TCD is associated with schizophrenia, obsessive-compulsive disorder (OCD), and depression (Schulman et al., 2011). Given that these conditions also often involve TCD in the same brain regions as tinnitus, it is important to determine if the tinnitus preceded these maladies or is a secondary symptom of these maladies.

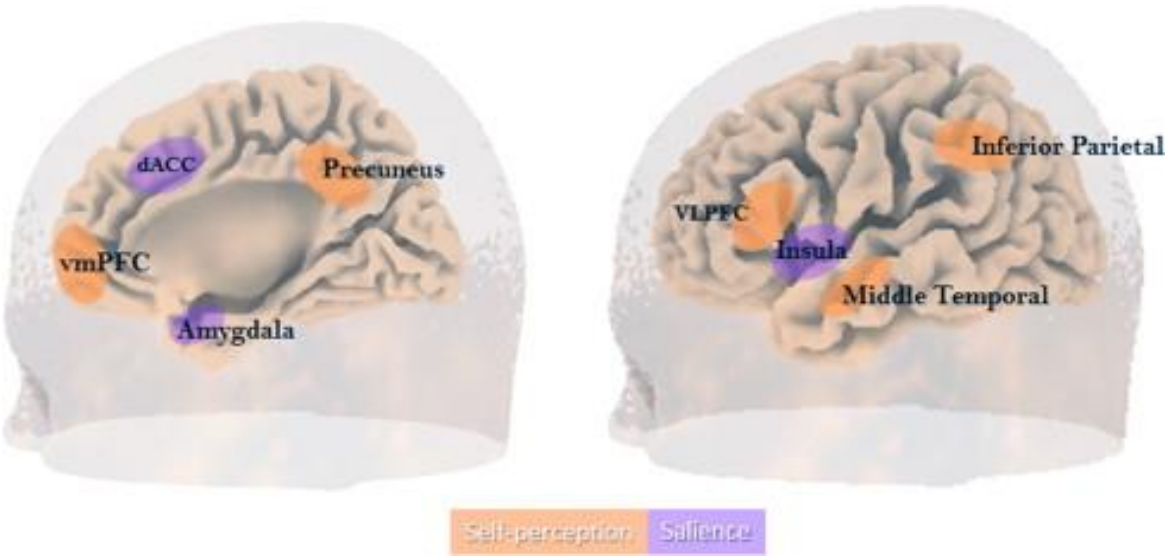
With hearing, in normal circumstances, incoming auditory stimuli induce a transient gamma band activity (Joliot et al., 1994) in a restricted area (von Stein & Sarnthein, 2000) which binds by nesting on theta activity (Canolty et al., 2006; Lakatos et al., 2005) that is a transient coupling between a high and low frequency band of ongoing electrical activity in the human brain (Canolty et al., 2006).

Recent models of tinnitus (Figure 5) were proposed according to which frontal brain areas, including the ventromedial, the ventrolateral, and the dorsolateral prefrontal cortices (PFC), play a crucial role in the generation and perception of tinnitus (De Ridder et al., 2014; Elgoyhen et al., 2015; Leaver et al., 2011; Rauschecker et al., 2010). A reduction in the gray matter volume has been observed in the ventromedial PFC (vmPFC) in tinnitus patients (Leaver et al., 2011; Seydell-Greenwald et al., 2012) and was associated with a higher reactivity of the nucleus accumbens during auditory stimulation at the tinnitus frequency using functional magnetic resonance imaging (fMRI) (Leaver et al., 2011). Whereas the activity in the dorsolateral PFC (dlPFC) was strongly and specifically correlated with tinnitus loudness (Seydell-Greenwald et al., 2012). Transcranial magnetic stimulation (TMS) applied to the left ventrolateral PFC (vlPFC) modulated the loudness of tinnitus (Vanneste & De Ridder, 2016). Anodal transcranial DC stimulation (tDCS) over the right dlPFC with the cathode over the left dlPFC reduced tinnitus loudness in 30% of patients (Vanneste & De Ridder, 2016).

Using two different behavioural tasks, it was found that top-down cognitive control was specifically altered in tinnitus patients (Heeren et al., 2014). Those with tinnitus were found to be slower and less accurate than controls during both auditory and visual spatial Stroop tasks, but auditory reaction speed and stimulus processing speed were affected only in tinnitus sufferers. Tinnitus is associated with both deficits along the auditory processing system and an impairment of cognitive control mechanisms that are involved both in vision and audition (Araneda et al., 2015a). Araneda went on to assess the prefrontal cortex in the maintenance of chronic tinnitus using fMRI during a Stroop task (Araneda et al., 2018).

It was also observed that their inhibitory control was altered in a “go-no go” task. Tinnitus patients were abnormally sensitive to cross-modal interference, revealing further their difficulties to ignore irrelevant stimuli (Araneda et al., 2015b). This led to the conclusion that a deficit in executive functions in the prefrontal cortex could be a key factor for tinnitus to develop and become chronic.

Figure 5. Regions Involved in the Neurological Perception of Tinnitus (De Ridder et al., 2014)



Treating TMJ or Malocclusion-Caused Tinnitus

Within the brain is a circuit called an occlusal generator (OG). The OG expects to "see" an even, straight bite with many occlusal contacts when the teeth come together during a bite. A poorly fitting bite may develop into muscle tension and TMJ issues associated with the poor bite and associated muscle tension. Dentists lacking an understanding of the underlying neurological systems involved in mastication (chewing) may not realize that TMD can be quite easily triggered from a poor fitting filling, crown, or cap (Kaur & Datta, 2013).

If you experience jaw tension and pain shortly after having some dental work done, it is recommended to return to your dentist as soon as possible and get that dental work fixed. A dentist typically rectifies dental-caused issues by placing articulating paper in between the teeth and having the patient grind back and forth/left and right. The spots of color left by the articulating paper on the teeth will indicate which surfaces of the teeth are hitting early (pre-occluding) or are crooked. The problem that occurs when dentists do this, is that they typically have the patient lying down as it is convenient for the dentist. However, the weight of the jaw may cause the patient to bite in the wrong (posterior) position and this new position will stress the joint and the muscles. It is important that the patient be sitting upright when the dentist inserts the articulating paper to look for these "hot" spots. By sitting upright, the jaw (mandible) will be a position that is better suited to where the muscles are balanced and "happy" with the position. This is termed a myocentric position (Jankelson, 1979; 1984), with muscles becoming relaxed in this position.

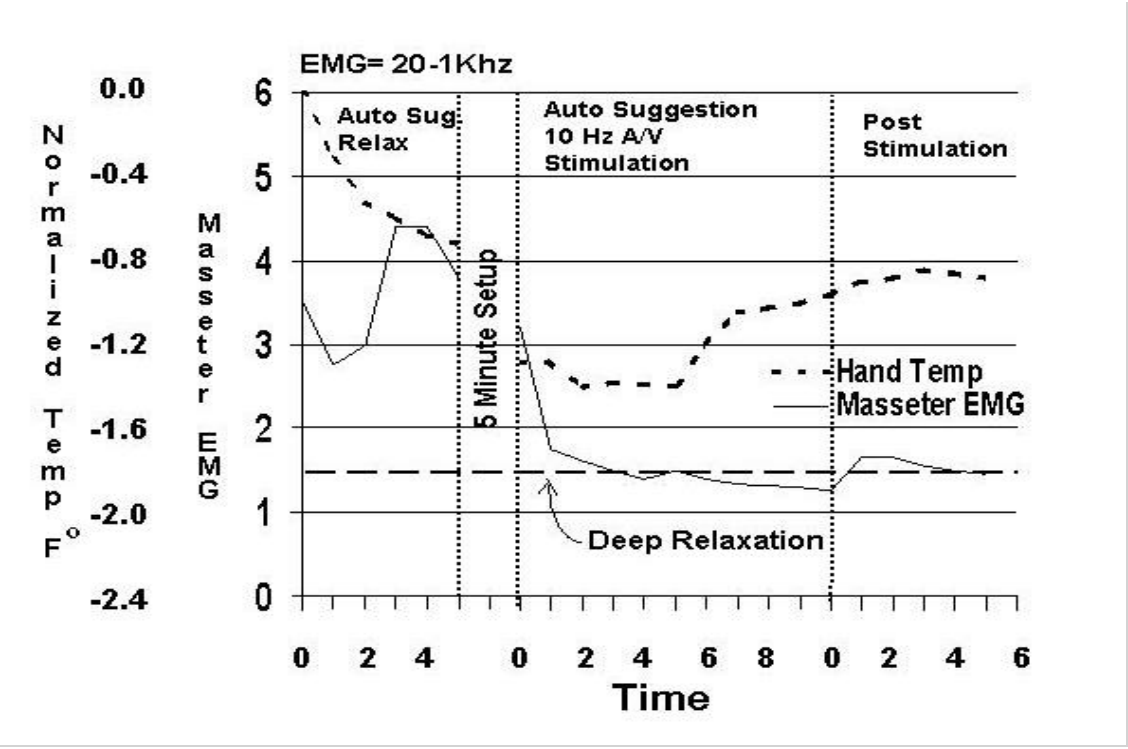
Treating Stress-Caused Tinnitus

Jaw tension, muscle soreness around the jaw, and subsequent TMJ issues are easily triggered by stress and anxiety. Any form of relaxation-enhancing/stress-reducing treatment will help alleviate this form of tinnitus. Techniques include biofeedback, heart-rate variability (HRV) breathing, meditation, and exercise. Although there are no published studies to date, Audio-Visual Entrainment (AVE) and Cranio-Electro Stimulation (CES) are quite effective for treating stress-induced tinnitus. Reducing stress, getting an occlusal splint or night guard from your dentist, having your dentist file down or reshape a poorly shaped filling or cap can also be very helpful. Auditory entrainment and audio-visual entrainment (AVE) have been shown to be quite effective at alleviating emotionally related TMJ issues (Manns et al., 1981; Thomas & Siever, 1989).

In Figure 6, we see an example of TMJ Personality (Thomas & Siever, 1989). In the first five minutes of the session, the participants (who have suffered jaw pain and headaches for at least 10 years) were given a guided imagery that had been shown to induce relaxation. Masseter EMG and finger temperature were measured. However, just before the imagery commenced, the participants were told that they were expected to be relaxed by the end of the imagery, which resulted in them all to tense up. We can see their jaw tension increasing and finger temperature dropping as a result of vasoconstriction – a reflection of sympathetic overactivation. In the last minute of the guided imagery, they relaxed a bit because they quit tensing up from "trying" to relax. There was a 5-minute set up period with the Digital Audio-Visual Integration Device (DAVID1), which did indeed produce some relaxation, but when the

session officially commenced at 10 Hz (alpha band), the relaxation was fast and profound. Masseter EMG dropped and finger temperature began increasing at 5 minutes. There was a mild increase in muscle tension during the removal of the DAVID1 eyesets and headphones. Most participants reported they were headache free for two weeks following this single session of AVE.

Figure 6. TMJ Dysfunction Patients During a Relaxation Task and During AVE at 10 Hz



Treating Deafferented and Neurological Tinnitus

Transcranial DC Stimulation

The best non-invasive technique for the remediation of neurological tinnitus utilizes tDCS. Several studies using tDCS have shown moderate to significant results. All studies found that anodal tDCS of the left temporoparietal area (LTA) near 10-20 EEG electrode placement position T3 was effective in reducing tinnitus, whereas cathodal tDCS worsened the perception of tinnitus (Ladeira et al., 2011). Most studies used a 35 cm^2 electrode with a current of 2 milliamps with the cathode on the contralateral shoulder.

One study found that dose-dependent current intensity of 2 milliamps into a 35 cm^2 electrode for 20 minutes was more effective than smaller currents or reduced times. Fifty-six percent of participants experienced transient suppression of tinnitus and 44% of participants experienced long-term improvement of symptoms (overnight—less annoyance, more relaxed, and better sleep) (Shekhawat et al., 2012). Another study found that anodal stimulation of either left or right auditory cortexes were equally effective in reducing tinnitus (Joos et al., 2014).

Now the big question is: why would anodal tDCS reduce tinnitus? Simple logic would suggest that anodal tDCS would increase tinnitus, as it excites the cortex. It is possible that Weisz et al. (2007) and Joos et al. (2014) have found the answer. Both groups point to theta activity increasing through the GABA neurotransmitter when tinnitus is involved. Anodal tDCS has been shown to reduce GABA influence on neural circuits (Stagg et al., 2009) plus reduce delta and theta EEG activity (Keeser et al., 2011).

Audio-Visual Entrainment

A study by Tönnies (2006) using a DAVID Pal by Mind Alive Inc., found remarkable improvements in tinnitus perception and emotion. The 30-minute protocol started at 10 Hz, lowered to 2 Hz and then back up to 10 Hz.

During each session of AVE, peripheral skin temperature increased by 2.1^{o} C, indicating a tipping of autonomic balance towards parasympathetic activation.

As a result of the 10 sessions of AVE over five weeks, emotional and cognitive impairments decreased significantly, psychological stress and anxiety decreased significantly, and the penetrance of the tinnitus became significantly less (all at a 5% level). In addition, there was a tendency to improve sleep disorders (10% level), so that in general the overall exposure to tinnitus decreased significantly (5% level) (Table 1), which was confirmed by the significant, less stressful classifications on visual analog scale (VAT) for general tinnitus impairment.

Table 1. Changes in Tinnitus Exposure Characteristics (according to TF)

TF Scale	Pre-test		Post-test		Error
	mean	SD	Mean	SD	Probability
Emotional Impairment	10.71	5.18	7.82	5.82	0.035a
Hearing Problems	5.18	3.21	4.76	3.07	0.795
Cognitive impairment	6.53	3.83	5.29	4.51	0.017a
Sleep disorders	3.47	2.50	2.76	2.31	0.056b
Penetrance of tinnitus	10.41	3.47	8.94	4.45	0.042a
Somatic discomfort	2.06	2.01	1.82	2.21	0.417
Mental impairment	17.24	8.47	13.12	10.12	0.016a
Total severity of tinnitus impairment	38.35	16.77	31.41	19.33	0.017a

[a]Sign.a.d.5% level
[b]Trend (10%)
[c]Wilcoxon test (2-page questionnaire)

Bimodal Auditory-Lingual Neuromodulation

Research by Conlon et al. (2020) showed that combining sound and electrical stimulation on the tongue can significantly reduce tinnitus. The study included 326 participants, providing evidence regarding the safety, efficacy, and patient tolerability of bimodal neuromodulation for the treatment of tinnitus.

The tinnitus treatment device used in the study, now branded as Lenire®, was developed by Neuromod Devices Limited (www.neuromoddevices.com). It consists of headphones that deliver sequences of audio tones layered with wideband noise to both ears, combined with electrical stimulation pulses delivered to 32 electrodes on the tip of the tongue by a proprietary device trademarked as Tonguetip®.

About 86 % of treatment-compliant participants reported an improvement in tinnitus symptom severity when evaluated after 12 weeks of treatment, with sustained benefits 12 months post-treatment.

Additional Treatments for All Types of Tinnitus

Bone-conducting headphones offer the convenience of playing a masking sound or music and still allowing full hearing because the ears are not blocked. Some headphones are placed on the mastoid process, while others are placed above the mandibular notch (the boney lump in front of the ear canal). A study of patients who experience mild-to-moderate tinnitus, shows that bone-conducted sound has the potential to relieve tinnitus in the same way as air-conducted sound (Holgers & Håkansson, 2002; Bahmad Jr. et al., 2018). Although these studies used bone-anchored hearing aids, headphones using simple over-bone placements have also worked out well. Some products of bone-conduction headphones include: AfterShokz, Vidonn, and LOKMAT.

CONCLUSION

There are multiple types of tinnitus. Objective tinnitus is one type where a real sound of various sorts can be perceived. Subjective tinnitus is a condition where a person experiences a continuous ringing or sound in the ear in the absence of an external stimulus. It appears that all forms of subjective tinnitus begin with issues that involve the cochlea in one form or another. Either way, subjective tinnitus is generally considered to be idiopathic and difficult to treat. Given there are numerous causes of tinnitus, spanning external somatic causes (dental, behavioral, and neurophysiological), it is critical that all these factors be considered in the development of an effective treatment plan. It would be beneficial for these various professions to form support groups where tinnitus symptomology may be shared, and a group diagnosis and treatment plan be made.

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