TINNITUS: MECHANISMS AND MANAGEMENT

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ABSTRACT

Tinnitus is the perception of sound in the absence of external auditory stimuli. Patients experiencing tinnitus often describe it as a buzzing, hissing, whistling, chirping, or roaring sound. Tinnitus is a most accurately described as a symptom, not a disease. Tinnitus is often benign; however it can cause psychological distress in some patients or be a symptom of an underlying pathology. Tinnitus is estimated to affect somewhere between 40 to 50 million Americans. Of the approximately 40 million Americans that experience tinnitus it is estimated that around 10 million people seek medical intervention because their tinnitus is so severe or troubling that it is negatively impacting the productivity and quality of their lives. Popular belief holds that tinnitus cannot be cured, but rather the underlying pathology can be treated or people can be educated and given tools to live more comfortably with their tinnitus. This paper will present a thorough review of the processes underlying tinnitus and the audiologic management of tinnitus.

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CHAPTER 1

Introduction

Tinnitus is loosely defined as the internal perception of sound in the absence of external auditory stimuli. The majority of people will perceive sound in the absence of external stimuli, but it is worth noting as a general theme, this is not considered pathologic tinnitus. As Kiang, Moxon, and Levine (1970) explain, most people experience "transient ear noises". These noises are often described as a high pitched whistling and may be accompanied by a sudden, temporary hearing loss. These episodes can occur suddenly and unexpectedly and resolve just as suddenly. They are sporadic and short lived in nature. These "transient" episodes are considered "normal" ear noises and are different in nature from pathological tinnitus; however there are no published criteria to separate "transient" and "normal" ear noises from chronic pathological tinnitus (Henry, Dennis, & Schechter, 2005). Henry et al., 2005 reviews literature that presents that pathological tinnitus lasts at least five minutes in duration. For the purpose of this review, transient ear noises will be considered in a different category than pathological tinnitus. Pathological tinnitus is permanent in nature, can be intermittent or constant, and causes variable levels of disturbance to the patient. As Davis and Refaie (2000) explain, there is a tinnitus expression, etiology, and its effect on patients lives.

Classifications of Tinnitus

Patients experiencing tinnitus often describe it as a buzzing, hissing, whistling, chirping, squealing, or roaring sensation inside the ears or head. Tinnitus can vary in loudness from a quiet background noise, to one that appears to mask external sounds (Han, Lee, Kim, Lim, & Shin, 2009). Classifications of tinnitus can help to play in role in research and treatment (Davis & Refaie, 2000). However it can be difficult to classify tinnitus based on its subjective nature and unclear mechanism of generation (Davis & Refaie, 2000). For the most part, tinnitus can be classified by the way it is experienced by the patient. For example, tinnitus can be unilateral or bilateral. Acute tinnitus can last for days to weeks, while chronic tinnitus is more persistent, lasting for greater than six months (Folmer, Martin, & Youngbin, 2004).

Additionally, tinnitus can be classified as either vibratory or nonvibratory, further categorized as objective or subjective, and further categorized by the hypothesized site of lesion. Vibratory tinnitus, which follows a rhythm, occurs because the patient is able to hear one's own muscle contractions, eustachian tube movements, blood flow within the vascular system, and other activities near the ear. Nonvibratory tinnitus is attributed to a more central neural activity (Noell & Meyerhoff, 2003). Nonvibratory tinnitus is not rhythmic, but rather random. Central etiologies are hypothesized to arise from the temporal lobe, auditory nerve, or brainstem, whereas peripheral etiologies are believed to arise from the external auditory canal, middle ear, or cochlea (Noell & Meyerhoff, 2003). Most nonvibratory tinnitus is associated with hearing loss at the peripheral or cochlear level (Noell & Meyerhoff, 2003).

If others can also hear the sounds experienced by the patient, the tinnitus is classified as objective. Objective tinnitus is far less common in all age groups, with an incidence of less than one percent (Daugherty, 2007). However, it is worth pointing out that some disagree with the labeling of tinnitus as objective. As objective tinnitus can be heard by others, they argue that this goes against the definition of tinnitus, which as stated is the perception of sound in the absence of auditory stimuli. Since objective tinnitus is heard by both the patient and those near the patient, it is traced to a disorder that produces a real noise. As Hazell (1995) suggests, tinnitus should, by definition, be subjective. Hazell (1995) distinguishes between neurophysiologic and somatic tinnitus. As Hazell (1995) suggests, somatic tinnitus has an underlying vascular, muscular, respiratory, or temporomandibular origin and requires medical evaluation as these are possible medically corrected conditions.

Subjective tinnitus is heard only by the patient. Noell and Meyerhoff (2003) report subjective tinnitus can be vibratory or rhythmic in nature, but at a sound level that it is only experienced by the listener. Yet, subjective tinnitus is usually nonvibratory or irregular in pattern. Nonvibratory tinnitus can further be subcategorized to central or peripheral etiologies which will be further discussed.

Prevalence of Tinnitus

Epidemiologic studies across countries indicate that tinnitus affects approximately 10 to 15% of the adult population (Davis & Refaie, 2000). In the United States alone, the perception of tinnitus poses a significant clinical problem for millions of Americans. Tinnitus is estimated to affect somewhere between 40 to 50 million

Americans (Noell & Meyerhoff, 2003; American Tinnitus Association, 2007). Davis (1995) finds that the incidence of tinnitus is approximately 10.2% in the adult population and rises after the age of 50 years. Schwaber (2003) corroborates that the majority of people report tinnitus between the ages of 40 and 70 years; while approximately 30% of the population over 65 years has chronic tinnitus. Approximately 50% of people with hearing loss also experience tinnitus (Davis, 1998).

The prevalence rate of tinnitus in children with normal hearing thresholds has been reported to range from 6 to 36% (Holgers & Juul, 2006). This prevalence rate has been reported as high as 76% for children with a documented hearing loss (Holgers & Juul, 2006). Clearly there is a large variability in the estimated prevalence rates of tinnitus in children. There are several hypothesizes as to how to explain this large variability in reported tinnitus rates. Many of these hypothesizes also help explain why it can be difficult to study tinnitus in children. One possible explanation for this large variability in prevalence rates is due to the difficulty involved in interviewing children. It has been suggested that children rarely complain or talk about tinnitus when they experience it (Savastano, 2007). Some possible explanations for this include: children do not see tinnitus as a problem and have learned to live with it, children are scared to talk about tinnitus, children do not have the vocabulary to discuss tinnitus, or children may not be concerned about tinnitus when they do experience it. Savastano (2007) reports that children rarely spontaneously complain about tinnitus, but will talk about tinnitus when probed. Savastano (2007) reports a nearly 30% increase in tinnitus reports when the issue is specifically addressed to children. This is an important figure to remember as

it points to the importance of probing children about tinnitus during the case history or audiologic examination.

While some people experience tinnitus constantly, many people only experience tinnitus when in a quite environment. People who only experience tinnitus in a quiet environment may be less likely to seek medical intervention for the condition as they do not find it to hamper their quality of life. However, of the approximately 40 to 50 million Americans that experience tinnitus, it is estimated that around 10 million people seek medical intervention because their tinnitus is so severe or troubling that it is negatively impacting the productivity and quality of their lives (Noell & Meyerhoff, 2003).

Effects of Tinnitus

Reactions to tinnitus can vary in severity from mild irritation to suicidal ideation (Noell & Meyerhoff, 2003). As Han et al., (2009) described, the most common reactions to tinnitus include annoyance, concentration difficulties, sleep disturbance, and decreased speech clarity. It is estimated that approximately 20 to 30% of patients with tinnitus find their tinnitus disruptive to everyday activities, mood, and sleep (Andersson, Baguley, McKenna, & McFerran, 2005). Both anxiety and depression are commonly reported by tinnitus patients (Anderson, 2002). Other psychological distresses associated with tinnitus include anxiety, depression, irritability, anger, and insomnia (Wilson, Henry, Bowen, & Haralambous, 1991). Herbert and Carrier (2007) report that the majority of studies show that sleep difficulties are associated with tinnitus and these difficulties increase as the reported severity of tinnitus increases.

Children with tinnitus may demonstrate additional problems to those seen in adults. Savastano (2007) indicates that tinnitus can cause nervousness, depression, and irritability in children. Additionally, tinnitus may make it difficult for children to pay attention, sit still, and sleep (Savastano, 2007). These characteristics may be very similar to those seen in children with attention deficit disorders. Tinnitus can also have a negative impact on a child's academic development. Savastano (2007) indicated that tinnitus can cause learning and writing difficulties as well as a decrease in lingual capacity, and failing grades. These academic concerns emphasize the need for prompt and appropriate diagnosis and intervention. Although there is little research into the severity of tinnitus in children, Baguley (2002) indicates that is reasonable to believe that psychological attributes such as depression and concentration can be attributed more to tinnitus severity than audiometric thresholds across all populations.

CHAPTER 2

Processes Related to Tinnitus

The sensation of hearing occurs after sound waves travel through the outer, middle, and inner ear to the cochlea, up the auditory nerve, to the brainstem, and into the auditory cortex of the temporal lobe. It is important to have a basic understanding of this process because theories of tinnitus relate to breakdowns along any one of these routes of transmission. When no sounds are present, random activity occurs in the neurons of the auditory pathway. The brain prioritizes all the sound information it receives, so the nervous system generally filters out this random neural activity thus no sound is perceived (Daugherty, 2007). In a normal auditory system, external noise overrides any internal or random noise. However, tinnitus occurs when internal noise is given higher priority and thus dominates or accompanies external noise (Baguley, 2002). Regardless of the underlying pathology or pathophysiology behind tinnitus, the internal signal is processed by the central auditory nervous system and perceived as sound in the auditory cortex (Henry, Dennis, & Schechter, 2005). Neuromonics (2010) explains tinnitus is the result of neurological changes within the auditory system and within parts of the brain that influence attention and emotions.

In dealing with tinnitus, it is important to realize that the exact cause is not known but rather theorized. Much research on the topic still needs to be completed. As Henry et al., (2005) indicate, knowledge of the pathologic mechanisms and cellular events that surround tinnitus are insufficient to enable identification even in cases where the cause seems evident. Tinnitus in and of itself is often not classified as a disease, but rather as a symptom of an underlying problem (Han et al., 2009). This section will investigate the theorized mechanisms of pathophysiology and factors associated with tinnitus.

Classifications of the Pathophysiology of Tinnitus

Since tinnitus is described by patients in terms qualitatively similar to external sounds, such as buzzing or ringing, it can be suggested that the pathways responsible for tinnitus are the same as those that process external sounds (Henry, Dennis, & Schechter, 2005). Zenner and Pfister (1999) proposed three broad classes of tinnitus based on anatomical and functional divisions of the auditory system. These classes are comprised of conductive tinnitus, sensorineural tinnitus, and central tinnitus. These classifications compromise all divisions of the auditory system and highlight the idea that tinnitus generation can originate from the peripheral auditory system up to the level of the central auditory system or temporal lobe. As Henry et al., (2005) explain, conductive tinnitus would be caused by some pathology at the level of the middle ear, such as otitis media or otosclerosis. This would be the rarest of all classes of tinnitus. Sensorineural tinnitus contains four subclasses. These subclasses include: 1) tinnitus related to the outer hair cells also referred to as motor tinnitus, 2) tinnitus related to the inner hair cells also

referred to a transduction tinnitus, 3) tinnitus related to the auditory nerve also referred to as transformation tinnitus, and 4) tinnitus related to the "extrasensory" sources also referred to as objective tinnitus (Zenner & Pfister, 1999). Central tinnitus relates to tinnitus originating anywhere in the central auditory pathways (Zenner & Pfister, 1999). Although these classes of tinnitus are not necessarily universal, they do cover all the possible cites of origin of tinnitus (Henry et al,, 2005). It is believed that all levels of the nervous system are involved in the perception of tinnitus to some degree (P.J. Jastreboff, 1990).

Tinnitus and the Autonomic Nervous System

The auditory system is designed to hear and interpret sound. As such, the ears are constantly searching for meaningful sounds in the environment. Sounds that carry little meaning are quickly habituated to and ignored by the central nervous system (Henry, Dennis, & Schechter, 2005). For example, when driving in a car, one will pay attention to the radio and not the road noise. Since tinnitus is a sound that carries no meaning, habituation should be the norm (Hallman, Rachman, & Hinchcliffe, 1984). Habituation is in fact the norm for more than 80% of those who experience tinnitus (Han et al,, 2009). However, in patients who seek treatment for tinnitus, habituation does not occur. Rather, tinnitus becomes the focus. As tinnitus becomes the focus, it becomes bothersome, disturbing, and intrusive (Henry et al,, 2005). Even if the condition underlying tinnitus (e.g. hearing loss) has been present for months or years, the patient begins to attend to tinnitus essentially making it more audible. As the patient begins to attend to the tinnitus more and more, they may begin to fear that they are going deaf or they have a serious

underlying medical condition. Slowly they begin to associate anxiety, fear, and distress with tinnitus, thus paying more attention to the condition. The more the patient attends to the tinnitus, the stronger the cycle of worry and depression becomes. Psychological reactions and negative associations begin to amplify and exacerbate the perception of tinnitus (Henry et al., 2005).

P. J. Jastreboff (2007) believes that there are no known differences in psychoacoustical characteristics of tinnitus, such as pitch, loudness, and masking level, for those who experience tinnitus versus those who suffer from tinnitus. Therefore, tinnitus must be more than just an auditory system dysfunction. If tinnitus were related strictly to the auditory system, one would expect perceived psychoacoustical characteristics to be more intense for those with more bothersome tinnitus. However, since this is not the case, it is theorized that the level of annoyance from tinnitus is related to the degree of activation of the limbic and autonomic nervous systems (P. J. Jastreboff & Hazel, 2004). Once the perception of tinnitus begins to produce annoyance and anxiety, it begins to become associated with annoyance and anxiety, and therefore tinnitus begins to lead to annoyance and anxiety. The processes underlying tinnitus are often described as a vicious cycle. The perception of the tinnitus begins to increase as does the annoyance and anxiety thus resulting in enhanced activity of the limbic and autonomic nervous system (P. J. Jastreboff & Hazel, 2004). It is in fact the limbic and sympathetic nervous system that becomes responsible for the negative reaction to tinnitus (P. J. Jastreboff & Hazell, 2004). P. J. Jastreboff (2007) presents the development of tinnitus suffering: tinnitus is caused by abnormal neural activity which is interpreted and

perceived in the central nervous system and leads to the activation of the autonomic nervous system and limbic system which are areas in the brain that are not auditory in nature.

Peripheral/Sensorineural Tinnitus

As mentioned, the site of generation of tinnitus can arise anywhere from the middle ear to the central hearing system, yet sensorineural tinnitus is believed to be the most common (Henry, Dennis, & Schechter, 2005). Sensorineural tinnitus would fall under the classification of peripheral tinnitus, arising in the inner ear. The idea that tinnitus originates in the inner ear has been supported by the idea that patients often localize tinnitus to one ear versus the other (P. J. Jastreboff, 1990). Generally, factors that are associated with hearing loss are believed to be associated with tinnitus. It serves that as sensorineural hearing loss is the most common form of hearing loss, and hearing loss is so highly correlated with tinnitus, sensorineural tinnitus would be the most common form of tinnitus.

It has been established that tinnitus is highly correlated with hearing loss. In fact, populations that have a higher prevalence of hearing loss also have a higher prevalence of tinnitus and the majority of tinnitus patients also have hearing loss (Blackwell, Collins, & Coles, 2002; Davis & Refaie, 2000). Yet, Perry and Gantz (2000) cautions that other medical factors also increase with age and could contribute to tinnitus. These factors would include vascular disease, middle ear pathologies, diabetes, hypertension, autoimmune disorders, and neural disorders. Likewise, medical conditions

may lead to the increase use of medications which could also be linked to tinnitus. Thus it is always important to stress that correlation does not imply causality.

Noise exposure is commonly correlated to the onset of tinnitus. Noise induce tinnitus can be either acute or chronic, lasting for several minutes after noise exposure or lasting for extended lengths of time (Han et al.,, 2009). In the case of noise induced hearing loss, the damage to the auditory system is cochlear and thus one would surmise that as the hearing loss and tinnitus occurred at the same time, the tinnitus is also cochlear in nature (Henry, Dennis, & Schechter, 2005). This type of tinnitus is most commonly described as a high piched tonal or hisslike sound (Sweetow, 1996).

Schaette and Kempter (2009) concur that there is much evidence that tinnitus is related to hearing loss, yet propose that there is little known on how hearing loss leads to tinnitus. They suggest animal studies show that tinnitus is correlated to increased spontaneous firing rate of central auditory neurons. Schaette and Kempter (2009) present an experimental design that reproduced tinnitus related hyperactivity and predicted tinnitus pitch from audiograms of tinnitus patients with noise induced hearing loss and tone- like tinnitus. They theorize that decreased auditory activity due to hearing loss is counteracted by an increase in neural response gain. The increased neural response gain in effect restores the mean firing rate, but also leads to hyperactivity in the central auditory neurons. They show that hyperactivity patterns are strongest at frequencies close to perceived tinnitus pitch which also correlates to patient's audiogram in terms of hearing loss. Thus they suggest tinnitus can be caused by cochlear damage.

Tinnitus and Hair Cells

Many peripheral theories of tinnitus focus on the role of inner and outer hair cells in the perception of tinnitus. Inner and outer hair cells line the cochlear basilar membrane. Outer hair cells act as cochlear amplifiers providing 50- 60 dB of gain, while inner hair cells are the receptor cells that transduce hydraulic vibration in the cochlea to neural code enabling sound perception (P. J. Jastreboff, 2007). Due to the anatomy of the auditory system, hearing loss is often first triggered by the loss of outer hair cells. The discordant dysfunction theory holds that on each area of the basilar membrane where outer hair cells are dysfunctional and inner hair cells are more functional, the imbalance in function causes imbalance in the dorsal cochlear nucleus DCN (P. J. Jastreboff & Hazell, 2004). Specifically the hair cell dysfunction causes disinhibition in the DCN. This may cause an increase in spontaneous activity which is perceived as tinnitus. This theory is used to explain why some people with normal hearing also experience tinnitus: dysfunctional outer hair cells. It is also used to explain why some people with profound hearing loss do not experience tinnitus; both inner and outer hair cells have similar levels of dysfunction. Given this theory, temporary tinnitus following noise exposure can also be explained; noise creates temporary damage to the outer hair cells and does little to the inner hair cells (P. J. Jastreboff, 2007).

Central Tinnitus

As the earliest theories regarding the site of tinnitus generation pointed to the cochlea, over time the idea that tinnitus was solely confined to the inner ear began to come into question (Henry, Davis, & Schechter, 2005). In cases of severe tinnitus, some

patients underwent surgical sectioning of the auditory nerve. As these surgeries did not always eliminate the perception of tinnitus, it was theorized that tinnitus is generated by the central nervous system as triggered by cochlear damage (Henry et al,, 2005).

Møeller (2003) suggests that abnormal auditory signals activate neural plasticity within central structures which is perceived as tinnitus. In other words, pathologies in the ear or auditory nerve result in abnormal input which leads to change in more central structures. This change is then perceived to the patient as tinnitus. So, damage to the cochlea, enhances neural activity in the central auditory system. This is referred to as the auditory plasticity theory of tinnitus (Han et al., 2009).

The DNC has also been implicated as a possible site of generation for tinnitus (Kaltenbach, Zhang, & Finlayson, 2005). Following ototoxic drug treatments, such as cisplatin, or extreme noise exposure, the DNC can become hyperactive (Kaltenbach, Zhang, & Finlayson, 2005). It is hypothesized that a reduction in auditory nerve input lead to disinhibition of the DCN and an increase in spontaneous activity in the central auditory system which is perceived as tinnitus (Levine, 1999).

Neurophysiologic Model of Tinnitus

The neurophysiologic model of tinnitus was first described by P. J. Jastreboff (1990). The neurophysiologic model of tinnitus does not identify the mechanisms responsible for generating the neural activity that results in tinnitus, but rather the results of this neural activity (P. J. Jastreboff, 2007). The model presents three main postulates regarding clinically significant tinnitus. The first postulate holds that other systems in the brain aside from the auditory system have to be involved in the perception of tinnitus (P.

J. Jastreboff., 2007). As was previously discussed, P. J. Jastreboff and Hazell (2004) believe this other system is the autonomic nervous system which assigns negative reactions to tinnitus.

The second postulate is that tinnitus is phantom perception (P. J. Jastreboff, 1990). P. J. Jastreboff, (2007) reported that tinnitus perception results from the detection and perception of neural activity within the auditory pathway, despite the absence of external sound. There is no "vibratory activity" in the cochlea that is causing tinnitus. P. J. Jastreboff, (2007) used this postulate to explain why tinnitus can often not be masked or suppressed by external sounds. The reason being, tinnitus has no connection to external stimuli.

The final postulate stresses the difference between tinnitus perception and tinnitus suffering (P. J. Jastreboff, 2007). As P. J. Jastreboff (2007) explains, the mechanisms responsible for the generation of tinnitus neuronal activity are the same in people who experience tinnitus and those who suffer from tinnitus. He cites the lack of difference between pyschoacoustical characteristics of tinnitus, such as pitch and loudness, as support for this postulate. The difference between those who merely experience tinnitus and those who suffer from tinnitus lies in the mechanisms responsible for spreading tinnitus related neural activity throughout the brain. This abnormal spread of neural activity is present only in those whom suffer from tinnitus.

Medical Conditions Associated with Tinnitus

Tinnitus is often associated with the same otologic disorders that cause conductive and sensorineural hearing loss (Crummer, 2004). Causes of conductive

hearing loss that may lead to tinnitus include cerumen impaction, otitis media, eustachian tube dysfunction, otosclerosis, and other diseases of the ossicles (Daugherty, 2007). Cerumen impaction is reported by many produce a low pitched, intermittent non-vibratory sound that is resolved with the removal of the cerumen impaction (Noell & Meyerhoff, 2003). Although not necessarily conductive in nature in the sense of originating from the middle ear, some dental disorder and temporomandibular-joint (TMJ) dysfunction have also been documented to be associated with tinnitus (Han et al., 2009).

As was previously mentioned, in less than one percent of all tinnitus cases, the tinnitus can be heard by both the patient and others. This form of tinnitus, although controversial as it is experienced by others and not just the patient, is labeled objective tinnitus (Daugherty, 2007). Objective tinnitus is often caused by disorders affecting the middle ear. Occasionally, objective tinnitus is experienced as a heartbeat or pulsatile sensation. Pulsatile tinnitus is often associated with vascular disorders. Glomus tumors also produce tinnitus that is pulsatile in nature. Glomus tumors are slow growing vascular tumors which can grow in the temporal bone or middle ear space (Gombos & Zhang, 2008). Muscular disorders of the middle ear, such a spasm of the stapedial muscle, have also been associated with objective tinnitus (Daugherty, 2007). Finally, eustachian tube dysfunction and patulous eustachian tubes have been linked to objective tinnitus (Folmer, Martin, & Yongbing, 2004).

Other comorbid conditions provide insight into tinnitus. Tinnitus is often reported following or in association with the use of drugs. Asprin, quinine and cisplatin

are the most common drugs known to induce tinnitus (P. J. Jastreboff, 2007). Tinnitus associated with ototoxic drugs is often reported as a hig-frequecny ringing (Noell & Meyerhoff, 2003). Alcohol, caffeine, and tobacco have also been linked to an increase in tinnitus symptoms (Daugherty, 2007). Likewise, low-pitched roaring tinnitus is often a symptom of Ménière's disease (Sweetow, 1996). Ménière's disease is often referred to as endolymphatic hydrops. Ménière's disease is believed to be related to hair cell damage due to too much endolymph in the cochlea (Henry, Dennis, & Schechter, 2005). Finally, patients with acoustic neuromas often report unilateral tinnitus (Noell & Meyerhoff, 2003).

CHAPTER 3

Management of Tinnitus

It is worth reiterating that of the estimated 50 million Americans with tinnitus, only 12 million seek treatment and only 2 million consider themselves debilitated by their tinnitus (American Tinnitus Association, 2007). Therefore, the course of treatment for each tinnitus patient will be varied and unique patient characteristics should be carefully considered. Tinnitus treatment is even further complicated by the idea that the exact causes of tinnitus have not been identified. As described Henry, Dennis, and Schechter (2005) identifying the mechanisms of tinnitus is of utmost importance as treatment could be directed at the cause of tinnitus rather just the consequences of tinnitus. It is noted in much tinnitus literature that since tinnitus is a subjective symptom, it is difficult to do epidemiologic and efficacy studies for treatment.

Currently tinnitus management can be broken down into several main categories. Noble (2008) identified four major classes: pharmacological, acoustic, psychological, and any combination of the aforementioned. Medical or surgical treatments and alternative medical treatments would also be worth adding to these major classes. It is necessary to gather information on both the auditory characteristics and perceived intrusion tinnitus is imposing on the patient in order to form a treatment plan.

Medical/Surgical Treatment

If an underlying medical or otologic condition is determined to be correlated with the tinnitus, surgical intervention may provide relief. In fact, De Ridder, Menovsky, and Vande Heynig (2007) caution that providers immediately opt for non-surgical intervention forgetting that surgical intervention may be an option. They emphasize a thorough investigation of possible surgical solutions for those with severely troubling tinnitus. De Ridder et al., (2007) summarize the most relevant pathologies that may be treatable with surgical intervention including; vestibular schwannomas and other cerebellopontine angle lesions, arachnoid cysts, Ménière's disease, otosclerosis, brain tumours along the auditory pathways, Chiari malformations, microvascular compressions of the vestibulocochlear nerve, benign intracranial hypertension, arteria carotid stenosis, glomus tumours, vascular lesions of the petrous bone and skull base, ateriovenous malformations, aneurysms, and vascular loops inside the internal auditory canal.

Dental treatment for temporomandibular joint (TMJ) problems may help patient who have tinnitus associated with their TMJ (American Tinnitus Association, 2007). Inconsistent results have been obtained regarding stapendectomy surgery for patients whose tinnitus is associated with otosclerosis Some patients report tinnitus symptoms improve greatly while others notice no difference. If an acoustic neuroma or glomus tumor is found, surgical removal may help alleviate tinnitus. In severe cases of Meniere's disease surgery can help improve both tinnitus and vertigo symptoms (Noell & Meyerhoff, 2003).

Diet/Lifestyle

Patients with tinnitus are often told to avoid certain foods, medications, and stimulants that have been linked to tinnitus (Noell & Meyerhoff, 2003). Certain drugs, such as aspirin and NSAIDs, are also known to exacerbate or cause tinnitus in some patients. Patients should limit or avoid these medications if they can be linked to tinnitus onset. People with Ménière's disease are often told to avoid salt as low sodium diets are linked with an improvement in symptomology (Noell & Meyerhoff, 2003). As Ménière's disease is often related to a fluid imbalance in the inner ear, reduction in sodium can help reduce fluid fluctuations in the body. Since noise exposure is highly correlated with tinnitus, tinnitus patients should be cautious regarding exposure to damaging intensity levels. Limiting noise exposure is an attempt to prevent the cochlear damage that is theorized to cause tinnitus. Stimulants such as caffeine contained in coffee, tea, and pop has been popularly linked to an increase in tinnitus symptoms and patients with tinnitus are often told to avoid ingesting large amounts of caffeine. However, St. Claire, Stothart, McKenna, and Rogers (2010) contend that although popular opinion reports that caffeine may affect tinnitus, there has been no scientifically documented link between dietary consumption of caffeine and tinnitus. In fact, they argue that encouraging patients to eliminated caffeine from their diets may in fact have adverse effects. They note that studies have been established that can replicate symptoms of caffeine withdrawal which include but are not limited to decreased alertness, irritability, and difficulty concentrating St. Claire, Stothart, McKenna, and Rogers (2010) completed a double blind study which concluded there was no evidence that abstaining from caffeine lessened tinnitus and in the short term, caffeine withdrawal added to the burden of tinnitus in some patients. It is worth noting that the results of this study found that

withdrawal effects were short term, however, this study highlights the importance that clinicians must be cautions when and if encouraging patients to change their diet. The same is true for nicotine. However, unlike caffeine, the health benefits of giving up nicotine are not as questionable and favor the patient under all circumstances.

Pharmacology

Darlington and Smith (2007) explained that many of the drug treatments currently used for tinnitus treatment are aimed at either the cochlea or central nervous system. More recently, antidepressants have been used in an attempt to address the emotional aspects of tinnitus (Darlington & Smith, 2007). As Langguth, Salvi, and Elgoyhen (2009) reiterated, tinnitus has no "curative" treatments available but rather treatments exist to help patient cope more effectively with the negative impacts such as stress, agitation, and depression, that tinnitus can have on quality of life.

Intratympanic administration of gentamicin has been used successfully to treat Ménière's disease and the tinnitus associated with Ménière's disease (Darlington & Smith, 2007). The uses of aminoglycoside antibiotics, such as gentamicin, are believed to be successful as they are ototoxic and therefore can be used to reduce tinnitus activity in the affected ear (Darlington & Smith, 2007). As Ménière's disease is related to fluid imbalance, diuretics have also been successfully used in the treatment of Ménière's symptomology, including tinnitus (Darlington & Smith, 2007). Intravenous administration of lidocaine has also been used historically to treat tinnitus (Darlington & Smith, 2007). Lidocaine is both a local anesthetic and anti-arrhythmic agent that is believed to target the cochlea and the central auditory nervous system to suppress tinnitus (Trellakis, Lautermann, & Lehnerdt, 2007). Benzodiazepines, are also used to treat

tinnitus as they are believed to limit hyperactivity; however, no controlled clinical trials appear to corroborate this belief (Darlington & Smith, 2007). Anticovulsants in small doses have also been used to try to decrease the neural hyperactivity theorized to cause tinnitus (Darlington & Smith, 2007). Antidepressants are historically used to treat the psychological side effects of tinnitus. However after a thorough literature review, Robinson, Viirre, and Stein, (2007) concluded further trials are needed to replicate current findings on antidepressants for tinnitus patients.

As Darlington and Smith (2007) indicate, obstacles for finding effective drug treatments for tinnitus can be related to the obscurity of tinnitus itself. As there are many different pathologies surrounding tinnitus and the pathophysiology cannot be clearly identified, it is difficult to identify what drug therapies should target. Additionally, the literature indicates a lack of clinical trials to test even the most commonly used drugs in tinnitus therapy (Darlington & Smith). However, Langguth, Salvi, and Elgoyhen (2009) present that much research is being completed on the use of drug therapy with tinnitus patients. Yet they still acknowledge inconsistent methological rigor within existing clinical trials. Available drug trails do not appear to demonstrate long-term benefit to patients beyond the placebo effect and as such there are no FDA approved drugs for the treatment of tinnitus (Langguth, Salvi, & Elgoyhen, 2009).

Langguth, Salvi, and Elgoyhen (2009) recognize much off label drug use for tinnitus treatment. The use of complementary and alternative pharmacologics in tinnitus is mainly focused on vitamins and minteral, antioxidants, and herbal medications (Enrico, Sirca, & Mereu, 2007). Enrico et al.,, (2007) report vitamin A, B1, B3, B6,B9, B12, C, E, magnesium, calcium, potassium, manganese, selenium, and zinc as the most commonly used vitamins and minerals for

tinnitus management. In terms of antioxidants Enrico et al., (2007) site that evidence supporting their use is scarce, but admit that although antioxidants may not be a therapy for tinnitus, their use may be helpful as a supplemental treatment for patients undergoing therapy with ototoxic drugs. Ginko biloba seems to be the most commonly used herbal supplement for tinnitus management but Enrico et al., (2007) caution that despite popular belief, herbal supplements are drug and thus are able to exert both therapeutic and toxic effects so they should be used cautiously. Overall Enrico et al., (2007) caution that evidence regarding the efficacy of complementary and alternative medicine for tinnitus management is limited and often potential toxic effects are underestimated, thus caution should be used when attempting this type of tinnitus management.

Alternative Treatments

Repetitive transcranial magnetic stimulation (rTMS) is a promising new method of tinnitus treatment that induces electrical current into the brain through impulses of magnetic fields applied externally (Kleinjung, Steffens, Londero, & Langguth, 2007). Although the exact pathophysiology of tinnitus is unknown, rTMS is intended to target dysfunctional neuroplastic processes in the brain. As hyperexcitability or neurons in the auditory cortex is on possible pathophysiology of tinnitus, rTMS attempts to modulate the excitability of these neurons therefore causing a decrease in the perception of tinnitus (Kleinjung, Vielsmeier, Landgrebe, Hajak, & Langguth, 2008). Kleinjung et al., (2008) explain that rTMS can focally modulate cortical activity and as such, the assumption is made that application of rTMS to auditory cortical areas will relieve tinnitus. Current research on this treatment method is promising, but is limited

by small sample sizes and high variability. Much research is needed before rTMS becomes regular clinical practice.

Although acupuncture is commonly used to control pain, its use in tinnitus treatment has gained some recent popularity. Acupuncture employs needles to stimulate specific points on the human anatomy (Okada, Onishi, Chami, Borin, Cassola, & Guerreiro, 2006). Henry, Dennis, and Schechter (2005) explain that tinnitus and pain are theorized to share common mechanisms thus the recent trend toward managing tinnitus with acupuncture. However, they also report that studies seem to indicate little to no benefit in tinnitus relief for those who pursued acupuncture. Relaxation therapy, yoga, visualization, and hypnosis can also be used to help reduce the stress associated with tinnitus.

Amplification

Approximately 50% of those with hearing loss also experience tinnitus (Davis, 1998). Hearing aids can be an especially good option for patients with significant hearing loss. However, due to amplification limitations in the high frequencies, hearing aids are probably not an option for patients with hearing loss confined to the very high frequencies (Searchfield, 2006). Bo and Ambrosetti (2007) advocate the use of hearing aids in the treatment of tinnitus for two reasons. First, they suggest that hearing aids allow patients to become less aware or completely unaware of tinnitus. Second, they suggest that hearings aids removes the sensation that sounds are being "masked" by tinnitus. Henry, Dennis and Schechter (2005) explain this phenomenon by offering that hearing aids alone can sometime provide sufficient masking relief. As hearing aids amplify both speech and ambient noise, the speech amplification can act to divert attention

away from the tinnitus while the ambient noises can act to mask the tinnitus (Han, Lee, Kim, Lim, & Shin, 2009).

Bo and Ambrosetti (2007) propose that hearing loss reduces stimulation from external sounds which can lead to auditory pathways undergoing plasticity. This neural plasticity can present itself as hyperactivity in the auditory pathways which is proposed to result in tinnitus. They suggest the amplification provided through the hearing aid provides enough stimulation to the auditory pathway to evoke neural plasticity and even restore neural functioning. Bo and Ambrosetti (2007) present the rationale for hearing aids based on two neurophysiological assumptions. First, hearing aids increase the level of ambient noise, and in so doing, reduce or eliminate the contrast between the internal noise of tinnitus and the silence of hearing loss. And second, as hearing aids eliminate hearing deprivation, they can overcome the deleterious effects of tinnitus through neural plasticity. In other words, if tinnitus is caused by neural plasticity through hearing loss, hearing aids can reverse this neural plasticity with sound input.

Sound Therapies

M. M. Jastreboff (2007), states that temporary tinnitus can be induced in almost anyone if they are placed in a quiet enough environment, however the sensations these people perceive are often similar in description to the experience tinnitus patients report when in a normal sound environment. Therefore, it has been hypothesized that the "tinnitus signal" exists in majority of people's "neural networks", but it is the strength of the signal varies. The strength of the signal is generally low, but when the background noise is sufficiently reduced, tinnitus is perceived. As such, sound therapies have been used to distract from or cover up tinnitus. More specifically, sound therapies are believed to increase the background neuronal activity in the auditory system

via sound stimulation, therefore distracting attention away from tinnitus. Ultimately, it is suggested that any treatment that can decrease the strength of tinnitus related neural activity can also act to lower the activation of the autonomic nervous system and limibic system (M. M. Jastreboff, 2007).

According to M. M. Jastreboff (2007), recommendations to avoid silent environments should be among the first advice given to any tinnitus patient regardless of any treatment. Sound therapies themselves may with goals ranging from masking tinnitus, distracting attention away from tinnitus, lowering stress associated with tinnitus, or lessening the tinnitus sound level (M. M. Jastreboff, 2007). Sound therapy can employ sounds ranging from a broadband sound to those associated with sounds encountered in the environment such as rain or music. Sound sources can range from table top devices to personal music devices (Han, Lee, Kim, Lim, & Shin, 2009). The use of ear level devices was popular in the early 1970s when masking therapy was first presented by Vernon, however, popular opinion seems to that completely masking the tinnitus signal may prevent long term habituation (Henry, Dennis, & Schechter, 2005; M. M. Jastreboff, 2007)

Han, Lee, Kim, Lim, and Shin (2009) explain that music therapy can be used as a desensitization method. Music can help reduce the stress and anxiety associated with tinnitus and put the patient into a more relaxed mindset. P.B. Davis (2006) explains that music directly affects the limbic system and can in fact bypass slower linguistic based processing in the auditory cortex. The idea of using music therapy with customized thresholds is the basis the Neuromonics.

Neuromonics

The Neuromonics Tinnitus Treatment program combines acoustic stimulation with a structured program of counseling and support administered by clinician trained in tinnitus rehabilitation (Davis, Paki, & Hanley, 2007). This form of treatment focuses on both the theorized cause of tinnitus, auditory deprivation resulting from hearing loss, and the role of the limbic system in creating a negative cycle of awareness and negative emotions surrounding the tinnitus. The Neuromonics Tinnitus Treatment is designed to stimulate the auditory system while positively engaging the limbic system.

Neuromonics is a six month or longer treatment plan that aims not to cure or eliminate tinnitus, but to habituate the patient to tinnitus (Davis, Paki, & Hanley, 2007). Treatment is based off of a general consensus as presented by Tyler (2005), that tinnitus treatment is most efficacious when done in a combination of counseling and acoustic therapy. Neuromonics employs a relaxing music signal imbedded with customized noise to allow the patient to momentarily and intermittently experience tinnitus while in a relaxed state (Davis, Paki, & Hanley, 2007). The music stimuli with noise is customized to the patients hearing and tinnitus characteristics so it can provide relief at a comfortable listening level while also providing stimulus across a wide range of frequencies. The patient is encouraged to use the device during key times of distress so that they can acquire a sense of control over tinnitus. Over time, the patient should become desensitized to tinnitus.

Davis, Paki, and Hanley (2007) site five main ways the acoustic stimulus used in Neuromonics promotes desensitization. First, it stimulates the auditory pathway across a wide frequency range including very high frequencies where hearing loss is often most prominent.

Second, it controls correlation between the right and left ear in an attempt to stimulate auditory integration pathways. Third, it presents the therapy signal in a musical format in an attempt to foster a relaxation response and to make therapy more pleasant. Fourth, as it is a self dosing therapy, it promotes a strong sense of relief and control over tinnitus. And finally, as the patient intermittently experiences tinnitus throughout the therapy through a relaxation inducing context, it promotes systematic desensitization.

Tinnitus Retraining Therapy

Popular belief holds that tinnitus cannot be cured, but rather the underlying pathology can be treated or people can be educated and given tools to live more comfortably with their tinnitus (Nelson & Chen, 2004). Treatments that help reduce stress associated with tinnitus, such as Tinnitus Retraining Therapy (TRT), have been suggested as effective tinnitus management tools. Tinnitus Retraining Therapy is based off of the neurophysiologic model of tinnitus and is a multi-step therapy developed by P. J. Jastreboff and his colleagues that involves counseling the patient about tinnitus by addressing fears and anxiety associated with tinnitus, while also initiating gradual habituation to tinnitus (Noell & Meyerhoff, 2003). The goal of TRT is to habituation the patient to the emotional reactions surrounding tinnitus, such as fear and anxiety, and in so doing habituate the patient to the perception of tinnitus (P. J. Jastreboff, 2007).

Specifically TRT consists of: (1) one –on-one directed educational counseling based on the neurophysiological model of tinnitus and (2) sound therapy as prescribed by a trained clinician (P. J. Jastreboff & M. M. Jastreboff, 2006). The overall goal of TRT is to train the brain to classify tinnitus as not significant so that habituation can occur (P. J. Jastreboff & M. M. Jastreboff). The purpose of the intensive one-on-one directive educational counseling is to

familiarize the patient with the physiologic processes underlying tinnitus. During this intensive one-on-one directive educational counseling, the patient is educated on processing of sensory information and the workings of the auditory system as well as the functioning of the limbic system. They are told that sounds become associated with experiences and in the case of their tinnitus, the brain has associated tinnitus with negative emotions. Following and supplementing the counseling, sound therapy is introduced. Sound therapy can be in the form of sound generators or environmental modifications. It is important to note it is never the goal to mask tinnitus. This is because as P. J. Jastreboff and M. M. Jastreboff (2006) theorize, a patient cannot habituate to a sound he or she cannot hear and ultimately the goal of TRT is to allow the patient to habituate to tinnitus and remove the negative associations of tinnitus.

Current research suggests that TRT is an effective treatment option for patients with tinnitus. However, this does not imply that every patient that enrolls in TRT will be successful. What studies do find is that for the majority of patients who enroll in TRT, a measurable benefit will be observed.

Counseling Measures

It appears that the higher the level of tinnitus annoyance, the higher the level of psychological distress (Heinecke, Weise, & Rief, 2009). Heinecke, Weise, and Rief, (2009) emphasize that although the mechanisms of tinnitus are not fully understood, current knowledge holds that the emotional and cognitive factors hinder the habituation process. As Hallman, Rachman, and Hinchcliffe (1984) reported abnormal stress reactions can contribute to the onset and maintenance of tinnitus as it may lead to hyperactivity in the autonomic nervous system causing tinnitus symptoms to continue. As such, biofeedback might be an effective treatment as

it helps reduce hyperarousal therefore facilitating habituation (Heinecke, Weise, & Rief,). Biofeedback is a relaxation process that is thought to reduce the severity of tinnitus (Kirsch, Blanchard, & Parnes, 1987). In effect, patients are trained to use their own bodies to facilitate their treatment. With tinnitus patients, they may be trained to reduce muscle tension, especially in the head and neck area, to facilitate relaxation despite tinnitus (Heinecke, Weise, & Rief, 2009). It is based on the theory that tinnitus is induced or worsened by stress. As experiencing tinnitus can be stressful, tinnitus patients often find themselves in a cycle of stress, both the as tinnitus can cause stress and be exacerbated by stress. Biofeedback is thought to help reduce the stress associated with tinnitus and may also help the patient foster a sense of physiologic control over tinnitus therefore resulting in less focus on tinnitus perception (Kirsch, Blanchard, & Pames).

Cognitive behavioral therapy (CBT) aims to reduce the psychological distress associated with tinnitus. Cognitive behavioral therapy does not focus on reducing the patient's perception of tinnitus in any form (Daugherty, 2007). Instead, the therapy helps the patient eliminate some of the negative attitudes regarding tinnitus to improve their overall emotional state.

Sometimes the counseling required for tinnitus patients falls out of the audiologist's scope of practice. Counseling in regard to the emotional impact of tinnitus should be conducted by a trained professional. Train counselors, such as psychiatrists, may be a necessary referral to determine if a patient can benefit from medication to help improve areas of sleeping, anxiety, or depression associated with tinnitus.

Existing Therapy Plans

Henry, Zaugg, Myers, & Schechter (2007) created the Progressive Audiologic Tinnitus Management (PATM) to reflect a hierarchy of clinical services to address patient needs. Progressive Audiologic Tinnitus Management is designed for audiologists to uses as a comprehensive protocol for tinnitus management. It includes five levels of management; triage, audiologic evaluation, group education, tinnitus evaluation, and individualized management. During any level of the treatment, the patient can opt out is tinnitus becomes less bothersome. Triage involves the referral to any necessary medical personnel. This would include referrals for medical or psychological treatment. Audiologic evaluation includes a standard audiologic evaluation as well as the completion of tinnitus questionnaires. During this stage tinnitus treatment plans such as masking or hearing aids can be discussed. Group education has been shown to be effective in the management of tinnitus as it provides a social network of encouragement and support while also being time effective. Tinnitus evaluation involves looking further into comorbid factors that can interfere with tinnitus treatment. Finally, individualized management involves the use of sound management and counseling.

CHAPTER 4

Conclusions

As tinnitus is a very individual perception, the treatment should be individualized to each patient. Although there are no treatments available to cure tinnitus, options due exists that can alleviate discomfort and improve quality of life (Nelson & Chen, 2004). A detailed case history should be obtained from the patient. Information regarding the patient's hearing history should be thoroughly investigated. This includes any information that could provide clues to the cause of tinnitus including history of hearing loss, noise exposure, ear infections, and previous ear surgeries as well as the use of caffeine, nicotine, and ototoxic drugs (Daugherty, 2007). Detailed information should be gathered regarding the pitch, pattern, location, intensity, onset, progression, and duration of tinnitus (Noell & Meyerhoff, 2003). Audiologic evaluation should include immittance measures, air and bone conduction thresholds, speech and word recognition testing, and loudness discomfort levels, and extended high frequencies. During the tinnitus psychoacoustic assessment, an attempt should be made to match the loudness and pitch of tinnitus using the audiometer (Henry et al.,, 2005). Additionally, the audiologist should attempt to match the tinnitus to noise. This step of the evaluation involves finding minimum masking levels (MML) where broadband noise is raised until the patient's tinnitus becomes inaudible

(Henry, Dennis, & Schechter, 2005). Patients should also complete a tinnitus questionnaire (Henry, Dennis, & Schechter, 2005). All these questionnaires have the tinnitus patient subjectively evaluate how tinnitus impacts aspects related to quality of life, and tinnitus annoyance and disturbance. A referral to an otologist should be made to rule out any physical pathologies that could potentially be treated or cured, medically or surgically (Henry, Dennis, & Schechter, 2005). Appropriate referrals should be made for psychological counseling or intervention if deemed appropriate. Counselors may need to prescribe tranquilizers and antidepressants that treat the anxiety and depression associated with tinnitus. If tinnitus cannot be treated with medical intervention, clinicians should first take care to educate the patient about tinnitus. Patients with tinnitus should also be counseled on steps they can immediately take to help manage their tinnitus including avoiding foods with high salt content, limiting or eliminating caffeine in the diet, limiting the intake of medications such as aspirin and anti-inflammatory drugs (NSAIDS), and avoiding loud noises as these can all exacerbate tinnitus (Noell & Meyerhoff, 2003). Patients should also be counseled on environmental modifications they can make to try to alleviate their tinnitus. This includes using fans, ambient noise makers, etc. Patients should be given information on alternative treatments that may help lessen their tinnitus annoyance. These therapies include relaxation therapy and acupuncture. Also an audiologist can provide direct intervention other than counseling. This would include the prescription of hearing aids, tinnitus maskers, Tinnitus Retraining Therapy (TRT), and Neuromonics. With this patient population it is important to remember that one size does not fit all and significant effort may need to be given to the patient to ensure the most appropriate intervention is achieved. There is certainly a need for continued research in the area of tinnitus.

However the ultimate goal in any treatment is for the patient to report a better self assessed quality of life, decreases in anxiety and depression, and improved activities of daily life that were previously significantly impaired by tinnitus such as relaxation, concentration, sleep, and work.

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