

Tinnitus Management in Lateral Skull Base Lesions

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Abstract

Tinnitus, the phantom perception of sound in the absence of a physical sound source, is a complex problem with multiple etiologies. While most commonly presenting in a subjective fashion caused by measurable hearing loss, other etiologies including lateral skull base tumors that encroach on middle and inner ear structures can lead to phantom sound perception as well. In addition to discussing the basic background of tinnitus, here we also review current theories of etiology that include central auditory and nonauditory neural mechanisms and potential treatments that range from sound therapy to medications to cognitive and behavioral therapies and cranial nerve and brain stimulation. One main purpose of this article is to relate tinnitus causes to skull base tumors, surgical removal, and resultant sequelae, including damage to cranial nerves resulting in audio-vestibular dysfunction. We also discuss the utility of microvascular decompression for both tumor and nontumor-associated tinnitus and the current literature regarding hearing preservation rates and tinnitus perception, where documented, with the three common treatment modalities employed for most lateral skull base tumors that includes watchful waiting with serial imaging, stereotactic radiosurgery and primary surgical resection using hearing preservation and hearing ablative approaches. The management of skull base tumors is a complex process that depending upon the approach and sequelae, may lead to manageable or worsening phantom sound perception that must be considered when discussing the multiple treatment options with patients.

Keywords

- ▶ hearing loss
- ▶ meningioma
- ▶ skull base surgery
- ▶ stereotactic radiosurgery
- ▶ tinnitus
- ▶ vestibular schwannoma

Introduction

Tinnitus is the phantom perception of sound in the absence of a bona fide sound stimulus and is not akin to auditory hallucinations (e.g., voices) which are typically associated with mental illness. Tinnitus (e.g., ringing, buzzing, hissing, etc.), can be perceived in the head (tinnitus aurium), or in either one (unilateral), or both (bilateral) ears. Sound perception can be pulsatile (e.g., hearing the heartbeat in the ears), nonpulsatile, subjective (most common form; only the patient perceives) or is objective (heard by the examiner, known as somatosound, e.g., turbulent blood flow at the cranial base, stapedial myoclonus, etc.).

Tinnitus is highly prevalent with an estimated 10 to 15% of the U.S. adults being affected.¹ Military personnel are particularly at risk and tinnitus is the number one service related

disability, with nearly 750,000 veterans receiving associated compensation at a cost of over \$2 billion in 2014.¹ Hearing impairment, advancing age, noise-exposure, and being male (particularly in military and combat zones) have been identified as the most relevant risk factors for tinnitus.² Tinnitus is a global problem with many at risk populations. Due to the vast heterogeneity in symptom presentation and onset, resultant variability in tinnitus perceptions often leads to considerable gradations in patient frustration, hypersensitivity to sound (hyperacusis), sleep disruption, and even manifestations of clinical depression and/or anxiety.³

Tinnitus Etiology (Non-Skull Base Tumor Causes)

The underlying etiology of tinnitus is not well-defined, yet has been typically associated with peripheral ear pathology

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or disturbances leading to aberrant neural activity within central auditory and even nonauditory brain stations and pathways.⁴ Most tinnitus cases (non-skull base tumor related) result secondary to peripheral auditory lesions/pathology (e.g., cochlear/auditory nerve) and include, but are not limited to, sudden sensorineural hearing loss, noise exposure or noise-induced hearing loss (most common cause), head or neck trauma, chronic neck or jaw problems, systemic ototoxic therapies, acute or chronic otologic infection, or iatrogenic (surgery) causes. Alternatively, damage or compression of the auditory nerve (e.g., microvascular compression from skull base mass; e.g., vestibular schwannoma) can also lead to tinnitus perception.

Medical/Systemic Causes

Phantom sound perception secondary to hearing loss is a common clinical scenario, yet this cause and effect relationship is not clearly delineated as many patients who suffer from debilitating and profound levels of hearing loss do not endorse tinnitus percepts. Conversely, many who suffer with tinnitus do not show detectable objective levels of hearing loss per traditional audiometric booth testing.⁵ While this observation may not account for the possibility of occult (e.g., hidden hearing loss) or subtle hearing loss, it highlights and suggests other nonhearing loss etiologies of tinnitus. Objective forms of tinnitus typically present as pulsations akin to heart beats appreciated in the ear that are often unrelenting and typically caused by increased turbulent blood flow within the cranial base vasculature, idiopathic intracranial hypertension (pseudotumor cerebri), vascular abnormalities (e.g., arteriovenous malformations), skull base carotid artery bony dehiscence, sigmoid sinus dehiscence or diverticula, carotid artery dissection, or stenosis. Other identified causes of objective tinnitus include middle-ear myoclonus (stapedial or tensor tympani), otoacoustic emissions and superior semicircular canal dehiscence syndrome (SSCD).

Neural Mechanisms of Tinnitus

The neuropathophysiology of tinnitus is not completely understood. A main theory is that tinnitus perception is the result of peripheral otologic insults that result in increased gain of function within central auditory circuits.⁶⁻⁸ This gain increase is thought to occur in both auditory and nonauditory brain centers that leads to conscious perception of phantom sound. This hypothesis has been reinforced by observational studies whereby long-standing tinnitus percepts exist following auditory nerve surgical transection⁹ underscoring the critical involvement of central auditory mechanisms in tinnitus generation. This notion of homeostatic plasticity in the form of increased central gain has been validated in animal models and extends from the brainstem cochlear nuclei through the mid-brain and thalamus up to and including the primary auditory cortex (A1) to compensate for reduced peripheral input.⁶ Within A1, decreased peripheral inputs lead to reduced inhibition in de-energized areas with subsequent encroachment of nearby intact regions into the void cortical regions. This concept of A1 tonotopic reorganization may contribute to

tinnitus generation.¹⁰ Interestingly, tinnitus-related activity changes/neurophysiologic correlates (increased neuron spontaneous firing rates and increased neural synchrony) in the central nervous system (CNS) are not restricted to auditory pathways.^{1,11} The involvement of nonauditory brain including the insula, cingulate, and thalamus explain, in part, the potential etiology for conscious auditory perception of phantom sound. Both auditory and nonauditory neural changes may be modulated by numerous underlying neurotransmitter-mediated mechanisms that include but are not limited to acetylcholine, glutamate, gamma-Aminobutyric acid (GABA), serotonin, and glycine.¹⁰

Somatic Tinnitus

Somatic or somatoform tinnitus is an emerging and alternative cause for tinnitus perception in the absence of an objectified hearing loss or hidden hearing loss. This stand-alone etiology is based on growing evidence that strongly demonstrates somatosensory inputs from jaw musculature, the face and neck that can directly influence central auditory neural pathways.² This is clinically evident in those who suffer from temporomandibular joint (TMJ) disorders, cervical spine maladies including arthritis, cervical spine joint and intervertebral disk degeneration, fibromyalgia, and whiplash injuries that have been linked with tinnitus perception.¹² The specific neural mechanisms include specific trigeminal nerve and cervical inputs (from C2 level) to the brainstem dorsal cochlear nucleus.^{2,13} Evidence also suggests that these inputs can influence A1 neurons as well.¹⁴ Consequently, many patients have the on-demand ability to modulate tinnitus pitch and perception with head and neck maneuvers that include any head movement, jaw thrust, rubbing of the face, and changes in temperatures applied to the facial skin. These percepts may also be modulated by stress and emotional factors highlighting the multifactorial etiology that may be separate from peripheral auditory deprivation and rather a direct result of altered somatosensory inputs leading to abnormal central auditory activity.² When evaluating patients with tinnitus all relevant head and neck, medical, surgical, auditory, and somatic factors must therefore be considered.

Treatments

Given the multifactorial nature of tinnitus etiology, touted treatments ideally target the underlying cause, if they are identified. While there is no definitive treatment or cure for subjective tinnitus, a broad range of therapeutic approaches exists from typical masking with sound therapy using broadband or white noise or via traditional amplification with hearing aids,¹⁵ to cochlear implantation,¹⁶ psychological counseling, cognitive behavioral therapy, pharmacologic management using off-label medications, and various types of brain and cranial nerve stimulation strategies.¹⁷ The paucity of data regarding the efficacy of many treatment strategies reflects the multifactorial nature of this disease and often highlights the placebo and idiosyncratic effect of treatments in this often, desperate patient population and/or reflects inadequate clinical research data.¹⁸

Sound Therapy (Hearing Aids and Cochlear Implants)

A traditional and broadly used treatment approach to manage subjective tinnitus is the use of environmental white noise or directed sound to mask phantom percepts. The use of relaxation and masking is designed to create an environment that is less disruptive than the actual tinnitus percept. Traditional amplification with hearing aids provides broadband sound stimulation that can completely or partially mask phantom sound perception. These are ideal options for many patients as the bulk of tinnitus sufferers also contend with sensorineural hearing loss that is amenable to amplification. The limits of amplification are usually to the higher frequency ranges (typically tinnitus frequency range < 6 kHz)¹⁵ and therefore this treatment option is unable to rehabilitate pan-frequency sensorineural hearing loss. In those patients with profound sensorineural hearing loss and tinnitus, traditional amplification often does not provide meaningful benefit or effective tinnitus suppression. In those instances, cochlear implantation¹⁶ has been utilized to rehabilitate hearing loss and dampen tinnitus perception for both bilateral and unilateral instances. While effective this treatment strategy is not ubiquitous and patients should be counseled about realistic expectations about symptom control.

Tinnitus Retraining Therapy

Tinnitus retraining therapy (TRT) is a therapeutic approach that utilizes a combination of psychological counseling and focused sound therapy to improve symptom control. The rationale for TRT is based on the hypothesis that tinnitus etiology is the result of abnormal, neural activity within and between both auditory and nonauditory brain pathways.¹⁹ Therefore, TRT attempts to habituate tinnitus symptoms through counseling to reclassify the percept from an unnatural sound disturbance to neutral stimuli. The benefit of adding sound therapy to this approach is designed to reduce phantom sound perception strength. Limited randomized controlled trials with TRT have led to mixed overall efficacy outcomes leaving providers and patients with limited conclusions.¹⁹ That coupled with the high cost of TRT, have limited its use.

Pharmacology

While a significant number of pharmacologic agents have been tried to treat the symptoms of tinnitus, no current drug has been approved by the Food and Drug Administration (FDA). Off-label uses of many medications have been employed including the anesthetic lidocaine that has been shown to transiently suppress tinnitus perception.²⁰ This data suggests that neural sodium channels may potentially serve as therapeutic targets for treatment. However, like many other off-label medications that have been trialed for tinnitus treatments, transient improvements in phantom sound perception suggest that multiple mechanisms or targets may contribute to the underlying etiology. Other drug classes including anxiolytics, antidepressants, and anti-seizure medications have had limited²¹ effect on tinnitus perception. However, these medications have been shown to have potential benefit in managing the psychological and emotional effects of persistent tinnitus. Other medications

including neural pain medications gabapentin and lamotrigine have shown some evidence of symptom control.²² Benzodiazepines as a class of medications have also demonstrated some positive benefits in that they allow patients to tolerate phantom sound perception rather than alleviating the actual perception itself.²³ Caution should be exercised with all medications, particularly those like the benzodiazepines as they have potential for dependence, abuse, and the need to taper off when stopping usage.

Cranial Nerve and Brain Stimulation

Transcranial magnetic stimulation (TMS) of the brain and select cranial nerves is a therapeutic approach that employs periodic magnetic pulses transmitted through the scalp to modulate cerebral cortical activity. The rationale behind this touted therapeutic modality is modulation or a resetting of aberrant central neural circuits in hopes of abolishing cellular and system pathways that are contributing to tinnitus generation. TMS has shown some positive results that are unfortunately not sustained for the long-term.^{17,24} The primary criticisms of these studies include minimal effects sizes, large individual variability, and a lack of long-term improvement in perception control. Evidence of tinnitus control using deep-brain stimulation to portions of the subcortical striatum (caudate nucleus) suggest alternative noncortical neural targets also exist that may be of clinical benefit.²⁵ Less invasive approaches are emerging that utilize vagal nerve stimulation with paired acoustic stimulation or devices that provide stimulation to the trigeminal nerve with acoustic pairing have shown preliminary evidence of tinnitus control.^{26,27}

Skull Base Tumor-Associated Tinnitus

Any tumor located in the cerebellopontine angle (CPA) may cause tinnitus due to mass effect on the vestibulocochlear nerve. Although uncertain, the pathophysiology appears to be like other more common types of tinnitus in which peripheral hearing loss leads to central auditory plasticity and resultant phantom sound perception.²⁸ Other symptoms that commonly accompany CPA lesions largely result from tumor mass effect on cranial nerves (e.g., hearing loss, generalized and persistent disequilibrium, facial weakness and numbness), the cerebellum (e.g., ataxia), and on the cerebrum (e.g., headache and obstructive hydrocephalus).²⁹ Vestibular schwannomas (also known as acoustic neuromas) are most common, accounting for 80 to 90% of CPA mass lesions^{30,31} followed by meningiomas (5–10%), epidermoid tumors (5–9%), and rare lesions (1–5%). Tinnitus, hearing loss, and disequilibrium are a classic triad of vestibular schwannoma (VS) clinical presentation; however, the triad only has a specificity of 10% for VS.³² Different tumors present with varying patterns of symptomatology and radiographic characteristics can provide helpful clinical and diagnostic cues to the underlying pathology.

As discussed, if the root cause of tinnitus perception can be identified there is a higher likelihood of symptom control. Regarding lateral skull base tumors, if the anatomy, pathology, and existing audiometric parameters are favorable, the goals of

complete tumor removal with concurrent hearing preservation may ameliorate and even cure phantom sound perception. As such, the ideal conditions may not always be available and all treatment options including the risks and benefits of each needs to be thoroughly discussed with each patient. Treatment choices for these lesions typically includes watchful waiting with serial surveillance imaging and audiometric testing, microsurgical resection through either a hearing preservation or ablative approach, and/or stereotactic radiosurgery/therapy. Often, these tumors are either slow growing or do not grow at all. For this reason, the first step in treatment is often to observe with serial imaging to establish the natural growth rate of the tumor assuming the tumor is not causing debilitating symptoms at the time of presentation/diagnosis.

Vestibular Schwannomas

Epidemiology and Clinical Presentation

Over the past decade, the incidence of VS has increased from 1.5/100,000 person-years in the 1960s to 4.2/100,000 person-years from 2006 to 2016 (Rochester Epidemiology Project, Mayo Clinic, Rochester, MN).³³ There is no difference in incidence between men and women and incidence increases with age (median age is 62).³³ The most common symptoms at time of diagnosis are hearing loss (90% all over; 62% experience gradual loss), tonal tinnitus in the affected ear (59–70%), trigeminal nerve dysfunction (33–71%), generalized imbalance (20–45%), and aural fullness (16%).^{29,33} Although not consistent across all studies, some have found tinnitus at presentation may be a predictor of poorer overall outcomes, with one study finding an increase of 2.9 (confidence interval [CI]: 1.1–7.6) in the odds ratio (OR) of tumor growth.^{34,35} As a result, unilateral tinnitus in the absence of a bona fide history or event should always be evaluated with an inner ear protocol MRI (magnetic resonance imaging) to rule out inner ear or retrocochlear pathology to include but not be limited to a vestibular schwannoma.

Treatment

Observation is the most common treatment choice, with 58% of patients and providers choosing this approach.³³ No baseline parameters have been found to be predictive of growth; however, growth in the 1st year is predictive of future growth.^{28,35} One half to two-thirds have not grown at 5-year follow-up.^{28,35} Based on data for all diagnosed VS in Denmark since 1976, if the tumor had not grown after 5 years, it did not grow.³⁶ Of the patients that had American Academy of Otolaryngology (AAO) class A hearing (good and preserved hearing, pure tone average less than 30 dB and speech discrimination > 70%), 26% had lost it after 1 year, 45% after 5 years, and 54% after 10 years.³⁶ Male gender and hearing impairment are predictors of preoperative development of tinnitus in patients with unilateral sporadic VS, whereas complete hearing loss is a negative predictor for the development of tinnitus.³⁷ Although there are no guidelines, a common protocol is to repeat MRI imaging at 6 months. If the tumor has grown more than 2 mm and the patient wishes to continue with observation, repeat MRI

imaging in 6 months. If the tumor growth did not meet the 2 mm threshold then repeat MRI once per year for 5 years.³⁸

If the decision is made to proceed with surgery, the three main approaches are the retrosigmoid (RS), translabyrinthine (TL), and middle cranial fossa (MCF). The factors influencing the choice in approach include size and extension of the tumor, presence of preoperative hearing level and expected ability to preserve based on tumor size, location, patient's age, auditory brainstem response waveform morphology, and preference of surgeon and patient.²⁹ There can be no hearing preservation following TL; however, some providers perform simultaneous cochlear implantation, usually in cases of bilateral VS or VS in the only serviceable ear. At least one case report, and more recently a small number of case series, has also demonstrated benefit of doing so in sporadic VS with normal contralateral hearing, including improvements in sound localization, hearing, and tinnitus.³⁹ Postoperative tinnitus is not specifically related to the surgical approach and should not be used as the sole reason for performing surgery.⁴⁰ It should be made clear that given the strong link between hearing loss and tinnitus perception, patients need to be aware that if they either lose hearing with a hearing preservation microsurgical approach or if they expectedly lose hearing with an ablative approach that phantom perception after surgery could be worse than preoperative levels. Appropriate alternative treatments (previously discussed above) will have to then be considered.

Since the literature on CPA tumors largely focuses on hearing loss/preservation and facial nerve dysfunction, more definitive data needs to be collected on outcomes regarding tinnitus both pre- and posttreatment regardless of modality (observation, surgery, or radiation). These data should be collected and considered based on the known close association between hearing loss and tinnitus generation. As one could hypothesize, greater rates of hearing loss (either directly or indirectly related to CPA tumors) will lead to greater rates of tinnitus presentation and heightened symptoms severity. In a systematic review, Ansari et al analyzed postoperative outcomes of the three different approaches while accounting for tumor size.⁴¹ They concluded that for tumors less than 1.5 cm, the MCF approach led to decreased levels of hearing loss compared with RS (43.6 vs. 64.3%), with no statistical difference in facial nerve outcomes between any of the three techniques.⁴¹ For tumors between 1.5 and 3 cm, there was no difference in hearing outcomes between MCF and RS but facial nerve dysfunction was lower for RS than both TL and MCF (6.1 vs. 15.8% and 17.3%, respectively).⁴¹ Tumors larger than 3 cm, RS resulted in lower facial nerve dysfunction than TL (30.2 vs. 42.5%).⁴¹ When looking at all intracanalicular tumors, there was no difference in hearing outcomes between MCF and RS but facial nerve dysfunction rates were higher for MCF (16.7%) than TL or RS (0 and 4%, respectively, no statistical difference).⁴¹

Stereotactic radiosurgery (SRS) is an alternative to microsurgical removal with similar outcomes in selected cases. No conclusive difference in tumor control, hearing outcomes, or complications has been found between single-dose and

fractionated SRS.^{29,42,43} In a review that included data from 4,234 patients, Yang et al (2010) found that compared with patients receiving doses greater than 13-Gy, using fewer than 13-Gy dose resulted in statistically significant higher hearing preservation rate (60.5 vs. 50.4%) but lower tumor control rate (90 vs. 94%).⁴⁴ In a recent study, serviceable hearing was preserved in 72% of patients who received primary RT and local control in 94.1% after 10 years.⁴³ In one study, tinnitus disappeared in 20% of those who had it prior to treatment.⁴³ Other studies have found overall no statistical change in the rate of tinnitus with either single-dose or fractionated SRS.^{42,45,46}

No randomized trial has been conducted comparing SRS versus surgery. In two prospective studies (patient decided treatment), Gamma knife radiosurgery (GKRS) was shown to be superior compared with surgical removal using the RS approach for both preservation of serviceable hearing and facial nerve outcomes.^{47,48} It should be noted that hearing preservation rates in both studies (0–5%) were lower than those reported in other large volume studies (40–80%).⁴¹ A retrospective study specifically documenting tinnitus outcomes found that patients who underwent TL had an improvement in both the tinnitus handicap inventory (THI) and visual analogue scale (VAS), while those who underwent GKRS worsened in both categories.⁴⁹

As discussed previously, tinnitus is not an indication for surgical treatment and results with radiotherapy are inconclusive. When a patient's main complaint is tinnitus, treatment modalities specifically aimed at tinnitus could be performed as part of the observation paradigm. As with treatments for other forms of subjective tinnitus (discussed above), it is important to counsel patients that phantom sound perception will likely not go away completely but instead there might be a reduction in the loudness and its negative effects on quality of life. If hearing loss is present on the affected side, the masking benefits of hearing aids can be explored. Ambient stimulation (e.g., sounds of music and nature), personal listening devices that don't occlude hearing and total masking therapy may be useful for those that don't benefit from hearing aids. Cochlear implants concurrently placed during surgical removal are also an option. Education, counseling and cognitive behavioral therapy are additional modalities that can be useful.

Neurofibromatosis 2

Neurofibromatosis 2 is an autosomal dominant disease of the *NF2* gene on chromosome 22 with a prevalence of 1 in 3,300 to 40,000 with no gender or ethnicity bias.⁵⁰ The defect in the gene leads to a tumor predisposition syndrome, with the hallmark of bilateral VS occurring in 90 to 95% of patients with the condition.⁵⁰ In cases with bilateral VS, the surgical removal should be staged with the second operation taking place at least 3 months after the first one. Early intervention may be crucial for hearing preservation.⁵¹ Given the inevitability of bilateral tumors, hearing preservation is paramount and considerations for cochlear implantation or auditory brainstem implants should be considered. Unfortunately, there is no specific data describing tinnitus rates and treatment efficacy in the *NF2* populations.

Meningiomas

Epidemiology and Clinical Presentation

Although audiovestibular symptoms occur with less frequency with meningiomas in the CPA compared with VS, for these lesions they remain the most common presenting symptoms. Hearing loss at presentation occurs in 50 to 80%, tinnitus in 15 to 60%, and generalized disequilibrium in 30 to 60%.^{31,32} Symptoms involving other cranial nerves can provide cues that the tumor is not a VS. At presentation, patients with meningiomas can experience facial pain and numbness up to 30% of the time and facial dysfunction is seen in 10 to 50%, compared with less than 5% in those with VS.^{29,31} Cerebellar signs (i.e., dysdiadochinesia, ataxia) occur in 30 to 90% of patients at presentation, a rare occurrence in those with VS.³¹ The clinician should also be aware that 20% of adolescents presenting with a meningioma in the CPA have *NF2* gene.³ Specific rates of tinnitus perception with skull base meningioma are not available and when considering the onset and management of phantom sound perception in these patients, a similar approach to counseling and treatments employed for VS should also be performed.

Treatment

Since meningiomas affect hearing less frequently, the standard approach is RS because it typically offers ideal exposure to the CPA and to these tumors that are typically located off center from the internal auditory canal. TL approach offers direct access to CPA and should be performed if there is extensive IAC involvement and hearing preservation does not seem viable through preoperative auditory compromise, tumor size, or other patient factors. The MCF approach can be performed to reach the lateral portion of IAC for hearing preservation but it is not typically indicated if the lesion extends more than 1 cm into CPA or if there is no evidence of a CSF cap lateral to the tumor at the cochlea or if radiographic evidence demonstrates that the tumor directly invades the cochlear canal and modiolar fibers.³¹ Most common complications of RS involve damage to cranial nerve (CN) V (8%), VII (8%), and VIII (12%).⁵² Most of these complications occurred in those that had tumor extension into the jugular foramen. Of those with normal hearing, 8% worsened (deaf) of those with hearing deficiency, 70% stayed the same or improved.⁵²

Recently, SRS has also been used to successfully treat CPA meningiomas smaller than 2.5 cm. Progression free survival was 98 to 99% at 1 year, 96 to 98% at 3 years, 93 to 95% at 5 years, and 77 to 87% at 10 years.^{53–55} Cranial nerve function was improved in 31 to 34%, unchanged in 49 to 58%, and worsened in 11 to 17%.^{54,55} The least likely symptoms to improve were hearing loss and tinnitus, with improvement in 31% of patients at 3-year follow-up.⁵⁴

Epidermoids

Epidemiology and Clinical Presentation

Epidermoids in the cerebellopontine angle arise from displaced ectodermal cells during early embryological development or from developing neurovasculature later in embryological

development.^{31,56} The most common presentation of epidermoids is headache (67%).⁵⁷ The most common cranial nerve deficit at presentation involve the vestibulocochlear nerve but these occur in a fewer percentage of patients compared with VS (50–80%).³² Trigeminal and facial nerve are involved in 30 to 50%.³¹

Treatment

The most common approach to removal of these is the RS approach, although a frontotemporal craniotomy with a subtemporal approach can also be performed.^{31,56} The goal of surgery is to decompress the cyst and remove the capsule completely.³¹ There has much debate over the aggressiveness with which to treat these typically nonmalignant tumors. A recent study by Schiefer and Link which reviewed 20 years of data from their institution concluded that morbidity and mortality is not increased in total resection versus subtotal resection if the tumor has not expanded significantly from the CPA. If the tumor has significantly expanded, there were no differences in rates of recurrence between total and subtotal removal, indicating that in those cases it would be reasonable not to pursue total resection.⁵⁶

Microvascular Decompression

Microvascular decompression (MVD) for the treatment of tinnitus is very controversial. The concept is to remove any potential inciting anatomic structure that could be stimulating the cochlear nerve leading to phantom sound perception. As such, it has been used for both nontumor (vascular only) and select tumor cases. The literature describes the concept of neurovascular conflicts (NVCs; e.g.; trigeminal neuralgia; hemifacial spasms) and when involving the cochleovestibular nerve could lead to tinnitus. A recent systematic review included 572 patients from 35 studies.⁵⁷ They reported that only 28% of patients with tinnitus had complete symptom of relief with MVD with more than one complication noted in 11% of patients. Interestingly, patients with both tinnitus and vertigo had a higher chance of success than in those with tinnitus alone. Due to low rates of success and substantial complications, they concluded that MVD cannot be included as a standard treatment method for tinnitus or vertigo. The controversial portion of MVD is such that it has greater efficacy and success rates in those patients who suffer from combined symptoms. When combined symptoms occur, it has been suggested that NVC is the underlying etiology and therefore MVD is therefore warranted. The authors; however, state that a lack of strong evidence in the included studies, mandate that MVD be utilized with caution.⁵⁷ Further studies and validation are required to determine if patients with single (tinnitus only) or combined symptoms are indeed better candidates for MVD.

Conclusions

Tinnitus is a common and often debilitating disorder with multiple etiologies and touted treatments. While there is no current cure for tinnitus, determining the underlying etiology is key to at least crafting a treatment plan that may ameliorate phantom sound perception. When the cause is related to a

lateral skull base tumor, a detailed discussion about tumor management including the ramifications of tinnitus should be carefully discussed with patients. Depending on treatment approach including watchful waiting, stereotactic radiosurgery or definitive microsurgical resection that may or may not be hearing preserving, clear expectations about tinnitus should be discussed. Alternative management options can be offered including sound therapy (hearing aids or a cochlear implant), medical management with or without psychological counseling, and even brain or CN stimulation can be discussed.

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None.

Conflicts of Interest

None.

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