Chapter 13
Medical Therapy

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There are a multitude of papers detailing surgical treatments for superior semicircular canal dehiscence (SSCD) and other third mobile window disorders (TMWD). However, there is scant mention of medical or non-surgical treatment options, let alone papers dedicated to options/outcomes for medical treatment. There are obvious benefits for successful medical management of TMWD. These include reduced risk, reduced discomfort, and reduced cost. Another potential benefit would be better patient outcomes for some of the symptoms not so well controlled with surgery.

SSCD existed prior to its first report in 1998 [1]. In the pre-1998 days, SSCD patients were typically diagnosed with other otologic conditions. Among these were Ménière's disease, atypical Ménière's disease (vestibular hydrops/cochlear hydrops), perilymph fistula, and vestibular migraine.

One of the first SSCD surgeries in 1998 we performed was a patient with a Ménière's presentation [2]. Two years prior to his presentation, he had been treated with a vestibular nerve section for left-sided aural fullness, hearing fluctuation and episodic vertigo, after a prior unsuccessful endolymphatic sac procedure. He presented to us with similar symptoms on the right side. A CT scan at the time demonstrated bilateral SSCD. A middle fossa craniotomy with repair of the right SSCD resolved his vertigo, right-sided fullness, and hearing fluctuation. Unfortunately for the patient, the left side that had undergone the vestibular nerve section had persistence of hearing fluctuation and fullness.

Similarly, we have witnessed many patients over the past 24 years who had SSCD and had been previously treated for a presumed diagnosis of Ménière's disease. Unlike our patient in 1998, some of these patients did extremely well being managed with conventional Ménière's disease treatment strategies. In 2015, a 75-year-old man presented to us with a 2-year history of right-sided fullness,

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hearing fluctuation, and episodic vertigo lasting hours per spell—often precipitated by straining. He had a similar presentation 25 years earlier and was treated with right endolymphatic sac surgery. He requested that we simply "do that same surgery that worked so well 25 years ago." CT scan demonstrated bilateral SSCD and his Valsalva, Fistula, and Tullio tests were abnormal in the right ear. We performed a right SSCD repair via a combined transmastoid-middle fossa approach. He has been free of vertigo since SSCD repair, but he was vertigo free for 23 years after his right endolymphatic sac surgery in 1990. While these examples involve surgery, we have witnessed those who have done extremely well with medical management techniques employed for Ménière's disease as well.

In the management of perilymph fistula (PLF) patients, traditional medical management often involves a period of bedrest and restriction from straining. Again, a considerable number of PLF patients will have had success with such conservative measures. It is interesting to note that many patients previously thought to have PLF, have SSCD and other TMWD.

Many SSCD patients who have successful relief of vertigo and autophony will continue to have persistent symptoms after successful surgical treatment. Of note is the symptom of aural fullness which we and others have found to not be reliably relieved by surgical intervention [3]. An interesting study by Ray et al. [4] looked at 33 ears in 24 patients with SSCD. These patients underwent a 4-h delayed intravenous Gd-enhanced 3D-FLAIR MRI using a compartmental endolymphatic hydrops grading system. They found 27.3% had MRI findings of endolymphatic hydrops. There was no correlation to cVEMP or oVEMP testing, but they did find a greater degree of sensorineural hearing loss in the hydropic patients. Similarly, other MRI studies of SSCD patients have reported 23–80% prevalence of endolymphatic hydrops [4–6]. While it is certainly possible that these may be concomitant Ménière's along with SSCD, this does raise the question as to whether endolymphatic hydrops is part of, or sequela of, SSCD pathophysiology. The corollary is the question of whether traditional medical management of Ménière's would be effective medical management of SSCD.

TMWD is a pressure problem resulting from increased compliance in the inner ear resulting in abnormal cochlear and vestibular stimulation. We propose that anything that reduces the pressure exerted on the inner ear will tend to improve the resulting symptoms from abnormal stimulation. These abnormal pressure influences result from both internal (intracranial) and/or external (middle ear) pressure waves. Surgical repair results in significant reduction in these pressure waves by reducing the inner ear compliance. Medical therapy does not change the inner ear compliance but is aimed at reducing the pressure waves.

These patient experiences led us to reconsider the concept of medical management for SSCD and TMWD in general. We now employ some of these measures before considering surgery in SSCD or TMWD patients. This chapter will detail specifics on medical management of TMWD. We estimate that over half of our patients who would have been considered surgical candidates, respond to a combination of medical measures we describe in this chapter.

One of the biggest challenges facing treatment based on symptoms of TWMD is the variability of symptoms—and their overlap with other disease processes. For

some patients, aural fullness or pseudo-conductive hyperacusis is most concerning, whereas others are most bothered by autophony, pulsatile tinnitus, imbalance, or vertigo provoked by sound or pressure. The finding of SSCD on CT scan does not imply causation of symptoms. For instance, we all have seen patients with CT evidence of SSCD and aural fullness, found to have bruxism and temporomandibular joint disorder, which when treated, resolved the aural fullness. Patients also exhibit symptoms related to damage associated with these abnormal pressure waves. These symptoms are treated with supportive care such as antinausea medications and vestibular rehabilitation aimed at expediting central vestibular compensation and treatment of concomitant BPPV. Awareness of these scenarios must also be considered in medical treatment strategies.

Avoidance of Triggers

In a classic scene from a Marx Brothers movie, the patient, as he lifts his arm, says to the doctor, "Doctor, Doctor! It hurts when I do this!" Groucho Marx, who plays the doctor, replies, "Then don't do that." We have all practiced some variation of "Groucho Marx" medicine throughout our careers, and it certainly applies to TMWD. One of the characteristic symptoms of TMWD is strain-induced vertigo/dizziness. Elimination of straining will eliminate many episodes of vertigo for TMWD patients. Many patients know this from prior experience and will avidly avoid such triggers. However, there is a sizeable number of patients who do not understand that this is one of the triggers for their symptoms until it is pointed out by the clinician. While we explain this to patients, we also give them a handout describing things to avoid (Fig. 13.1). We recommend this be strictly followed for six weeks, while other additional medical measures take effect. This is how we have treated PLF patients in the past and is often at least partially successful in resolving the patients' symptoms. Some of the more obvious factors to clinicians, but less so for patients, are weightlifting and other resistance-type exercises. We are assiduous in discussing these restrictions but still find patients who did not understand that abdominal "crunches" (or other core-muscle exercises) will continue to aggravate their condition.

Similarly, we advocate avoidance of activities where the abdomen/chest/head are subjected to major pressure altering conditions. Among these is childbirth by vaginal delivery. We have witnessed many SSCD patients describe their symptoms to occur or worsen after a vaginal delivery. We discuss cesarean section deliveries for our pregnant patients, if medically reasonable. Although anecdotal, we have not seen any exacerbation of TMWD with c-section births.

Another common complaint we have encountered is onset or worsening of TMWD after a non-otologic surgical procedure performed under general anesthesia. When undergoing general anesthesia there are a number of factors that cause large intracranial pressure changes. Patients are pre-hydrated with IV fluids to treat dehydration from an NPO status and to avoid a drop in blood pressure during induction. This fluid loading will cause a rise in intracranial pressure to some degree. After surgery, the patients are often not extubated until fully awake and coughing

LABYRINTHINE DEHISCENCE-FISTULA PRECAUTIONS

A labyrinthine dehiscence-fistula is an abnormal opening somewhere around the inner ear. In this condition, the fluid can shift out of the inner ear into another space, such as the middle ear. When this happens, this can tear delicate membranes in the inner ear and cause vertigo/dizziness, tinnitus (ringing in the ear) and hearing loss. Initial treatment of this is geared toward reducing any pressure directed toward the ear. Often, we use dietary methods such as restricting caffeine or salt in the diet to reduce the pressure on the ear. Sometimes a diuretic or fluid pill will be used to reduce fluid pressure as well. In addition, there are a number of activities that you should refrain from in order to reduce pressure on the ear and reduce this from being a problem. Among these are the following:

1.) Avoidance of nose blowing or sneezing through the nose. Sniffing is okay and does not seem to affect the pressure in the inner ear. If you have to sneeze, open your mouth to sneeze. Please do not stifle a sneeze since this is worse than actually sneezing through the nose.

2.) Avoid any strenuous activity. In particular, any activity that increases the pressure in your abdomen or chest will get transmitted to the head and consequently to your inner ears. In general, any activity that requires more effort than lifting 10 pounds should be avoided.

3.) Avoid bending over at the waist. If you need to pick up something off the ground, bend at the knees.

4.) Sexual activity is also restricted since this will place significant pressure on the inner ears as well.

5.) Straining can occur during times of constipation and should be avoided when having a bowel movement. Also, because of this, we recommend a stool softener.

6.) If at all possible, we recommend the period of fistula precautions should begin with 5 days of bed rest with the head elevated above the heart. During this time period you should only get out of bed to go to the bathroom.

7.) If you have a chronic cough or problems with nasal congestion/chronic sneezing, you should contact your physician for aggressive treatment of this since this will make your situation much worse.

8.) Do not use earplugs. You can use cotton with Vaseline to prevent water from getting in the ear.

9.) It is also recommended that you not have any dental work, massages, chiropractic work or physical therapy, during this 6-week period.

The above-mentioned restrictions generally apply for a 6-week period. If you develop any episodes of vertigo during this time period, please note which activities seem to provoke the spell and record them for later consultation. Many patients with perilymphatic fistulas find that this will significantly improve their symptoms. However, if it does not, surgical repair may be required in order to treat your perilymphatic fistula.

NOTE: NOTIFY DOCTOR IMMEDIATELY IF YOU EXPERIENCE LEG PAIN OR SHORTNESS OF BREATH.

Fig. 13.1 Patient handout describing the physical restrictions employed as an initial means to control vertigo triggers. This handout is routinely given to our TMWD patients and is rigidly enforced for the first six weeks of medical therapy, while other medical measures are begun to reduce intracranial pressure (diet and carbonic anhydrase inhibitors or diuretics). After six weeks, the physical restrictions are relaxed but the principle of avoidance of extreme straining remains

with the endotracheal tube in place. This coughing against the endotracheal tube will further cause transient significant increases in intracranial pressure (ICP). Lastly, postoperative nausea and vomiting will add to the pressure increases from above. To minimize the risk to the TMW defect, we make recommendations to the anesthesiologists in Fig. 13.2. Since instituting this strategy, we have not seen any patient with worsening of TMWD after surgery with general anesthesia.

Anesthesia Considerations for Patients with Inner Ear Pathology Undergoing Non-ear Surgery

- Limit the amount of I.V. hydration
- Hyperventilation if possible (CO_2: 26-30)
- Avoid significant intra-thoracic, intra-abdominal or intra-cranial pressure changes
- Consider using an LMA (laryngeal mask airway) if feasible
- Deep extubation if possible (i.e., avoid straining/coughing on endotracheal tube)
- Anti-emetics as indicated – Please consider using high dose Zofran (12 mg IV) preop.

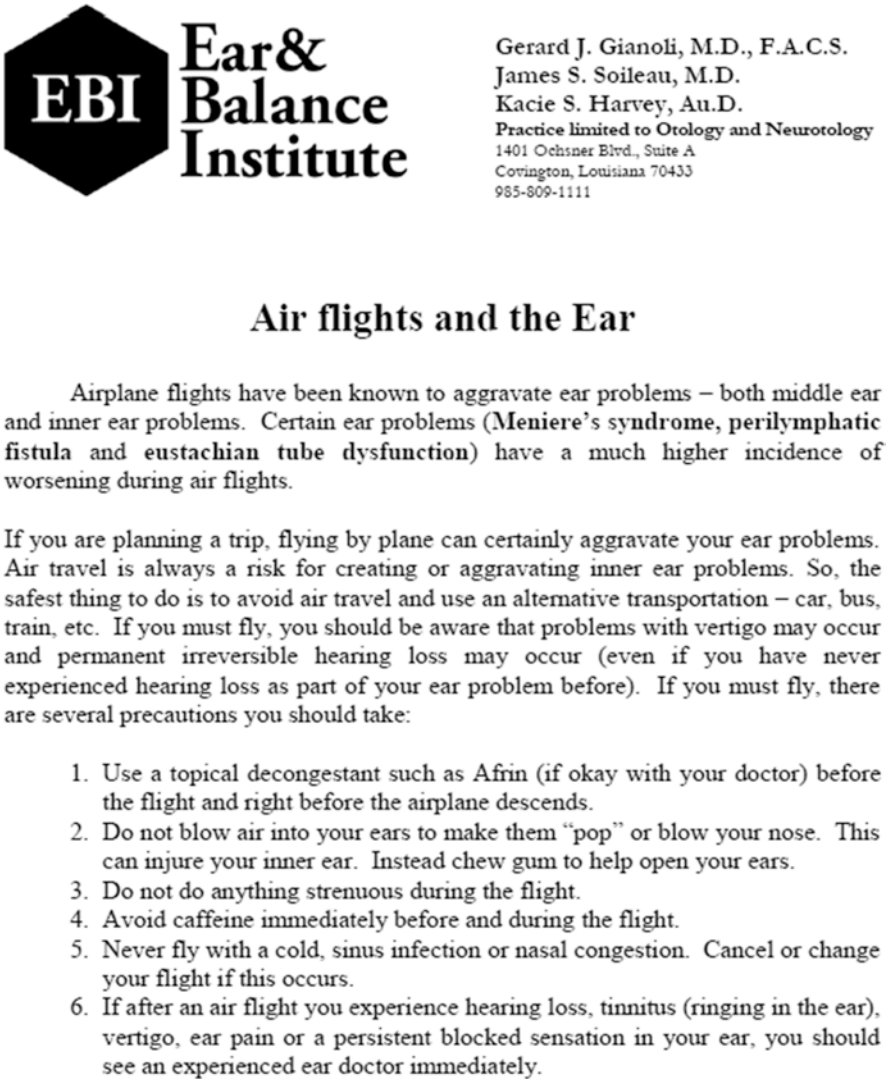
Fig. 13.2 These are the recommendations for anesthesia care of the TMWD patient who is undergoing general anesthesia. The goal is to avoid large changes in intracranial pressure to prevent subsequent exacerbation or recurrence of vestibular symptoms

A very characteristic symptom of TMWD is sound-induced dizziness or Tullio phenomenon. Avoidance of very noisy environments may be possible for some patients but not for others. Where avoidance of noise is not possible, noise-cancelling devices (NCD) offer help. While barrier ear plugs or earmuffs are capable of attenuating high frequency noise (>1 kHz), they are not very useful for attenuating low frequency noise (<1 kHz). Unfortunately, low frequencies are the most inciting sounds for TMWD. Noise-cancelling devices with active sound reduction are aimed at reducing low frequency noise. Noise-cancelling devices employ microphones to measure incoming low frequency sound and have an active output of low frequency sound in the opposite phase (anti-phase) of the incoming sound. This results in the "cancellation" effect [7]. Theoretically, NCDs would significantly reduce both low and high frequency sound-induced vestibular stimulation in sound-sensitive patients who wear them.

Feinberg et al. recently published the "Inverse Tullio Effect" [8]. In this paper, they reported the use of NCDs in TMWD patients resulting in significant resolution of many of their symptoms. However, the most interesting finding was that 40% of the patients treated with NCDs were not aware of any sound sensitivity prior to NCD use. We are bathed in sound and never outside of sound. Even inside a sound-proof booth, noise is present. ANSI (American National Standards Institute) maximum permissible sound levels in audiology sound-proof booths range from 19.5 to 47.5 dB SPL, depending on frequency [9]. It reasons that these patients did not complain of noise sensitivity because they were always exposed to everyday, ambient noise until they went through a trial with an NCD.

Otic barotrauma with eustachian tube dysfunction is a common entity, especially with air travel and scuba diving. Usually this results in nothing worse than otalgia during airplane descent. However, in TMWD patients, otic barotrauma can cause significant exacerbations in vertigo/dizziness and hearing loss [10]. Of course, not

all TMWD will have ETD, but for those who do, we advocate proactive measures to prevent otic barotrauma. These measures include nasal decongestants, "Earplanes" and, if these are unsuccessful, myringotomy or ventilation tube placement. Figure 13.3 is the handout we give to patients with TMWD who plan on air travel.



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Air flights and the Ear

Airplane flights have been known to aggravate ear problems – both middle ear and inner ear problems. Certain ear problems (**Meniere's syndrome, perilymphatic fistula** and **eustachian tube dysfunction**) have a much higher incidence of worsening during air flights.

If you are planning a trip, flying by plane can certainly aggravate your ear problems. Air travel is always a risk for creating or aggravating inner ear problems. So, the safest thing to do is to avoid air travel and use an alternative transportation – car, bus, train, etc. If you must fly, you should be aware that problems with vertigo may occur and permanent irreversible hearing loss may occur (even if you have never experienced hearing loss as part of your ear problem before). If you must fly, there are several precautions you should take:

1. Use a topical decongestant such as Afrin (if okay with your doctor) before the flight and right before the airplane descends.
2. Do not blow air into your ears to make them "pop" or blow your nose. This can injure your inner ear. Instead chew gum to help open your ears.
3. Do not do anything strenuous during the flight.
4. Avoid caffeine immediately before and during the flight.
5. Never fly with a cold, sinus infection or nasal congestion. Cancel or change your flight if this occurs.
6. If after an air flight you experience hearing loss, tinnitus (ringing in the ear), vertigo, ear pain or a persistent blocked sensation in your ear, you should see an experienced ear doctor immediately.

Fig. 13.3 Patient handout that includes tips on avoidance of problems with air travel. Most TMWD patients do not have significant eustachian tube dysfunction (ETD) and can fly without significant problems, but there is a sizeable portion of TMWD patients who do have ETD. Air travel in this group can provoke significant exacerbation of symptoms. For those with severe ETD, myringotomy and/or ventilation tube placement may be necessary

Upper respiratory tract infections (URI) and allergy flare-ups have been linked to vestibular disorders by multiple studies [11–13]. While multiple theories on the pathophysiology that links URI and allergy with vestibular disorders have been proposed, there is one aspect common to both that has been overlooked. Both URI and allergy are associated with frequent and often vigorous nose-blowing and coughing. Nose-blowing and coughing are known triggers for TMWD. We have employed proactive control of URI and allergy to prevent nose-blowing and coughing to improve the frequency and severity of TMWD symptoms in our patients. We caution our patients against nose-blowing and recommend sniffing, nasal lavage and judicious use of nasal decongestants and cough suppressants during URI. For allergy, we encourage aggressive treatment by their allergy specialists.

Diet

Given the finding of endolymphatic hydrops in SSCD patients mentioned above, it suggests looking at prior medical measures aimed at Ménière's that may be borrowed for use in TMWD. Dietary advice given to our patients include traditional Ménière's diet—avoidance of salt and caffeine [14]. We also discuss the Migraine diet and if there are food triggers for the patient, they are advised to avoid them [15]. We have found a subset of TMWD patients who are sensitive to dietary triggers while others who are not. For this subset, diet is an important aspect of medical therapy.

Medication

Carbonic anhydrase inhibitors have been a mainstay in our medical armamentarium [16]. Acetazolamide is the most prescribed medication of Idiopathic Intracranial Pressure (IIH), and we find it the most useful in TMWD. We suspect the mechanism is the same as in IIH—reduction in ICP. Reduction in ICP will reduce pressure transmission to the TMW and reduce abnormal vestibular stimulation. We find this to be extremely helpful in half of SSCD patients and a higher percent of non-SSCD TMWD patients. The major criticism of acetazolamide is the prevalence of side effects which most commonly include paresthesia, taste disturbance and fatigue. To avoid these side effects, we employ a titration strategy, starting with a low dose and gradually increasing the dose until there is either a resolution of symptoms or the patient cannot tolerate higher doses due to side effects. The range of dosing we have found successful has been very wide—62.5 mg/day to 4000 mg/day—but most patients take 500–1000 mg/day. To limit the side effect of fatigue, we like to use the extended-release version of acetazolamide and have the patient take it at night, prior to bedtime. Patients on carbonic anhydrase inhibitors must be tested initially and

monitored periodically with a complete metabolic panel. For patients unable to take acetazolamide due to renal problems or untoward side effects, methazolamide may be substituted.

While we favor carbonic anhydrase inhibitors as our medication of choice, there will be a sizeable portion of patients who cannot use them. For this group, we will employ standard diuretics as we have traditionally used to treat endolymphatic hydrops [16]. While there can certainly be differential dosing to control symptoms, compared to acetazolamide there is less leeway in dosing due to concern of dehydration, hypotension, and diminishing benefits with higher doses of diuretics.

Among medications used in PLF patients are stool softeners to prevent constipation and, hence straining. We find that most patients do not need this, but we discuss this with each patient since they may need this at some point in the future. We have also had a couple of patients with extreme constipation that was integral in the development of TMWD. In those patients, control of their constipation resulted in control of their TMWD symptoms.

Anxiety and panic attacks are found more frequently among patients with vestibular disorders [17] and among TMWD in particular [18]. Control of anxiety and panic attacks will not abate vertigo but can greatly improve the quality of life for these patients. We advocate the use of SSRI for this purpose and have found them helpful controlling these symptoms. We recommend avoidance of long-term (>2 weeks) use of benzodiazepines due to the problems with habituation. There has been a practice of placing patients on daily benzodiazepines—to suppress vestibular function and "prevent" anxiety/panic—which we believe should be highly discouraged. These medications do not resolve the problem of anxiety/panic spells and leads to the additional problem of addiction in the long term. While many clinicians treating vestibular disorders may feel uncomfortable in managing anxiety and panic disorders associated with vertigo, referral to primary care or psychiatry for management would be appropriate.

Migraine has been associated with vertigo, dizziness, and superior canal dehiscence in particular. Migraine has significant symptom overlap with TMWD and has been shown to prolong recovery after SSCD surgery [19]. Given the possibility of overlapping conditions, medical management of migraine prior to planning surgical intervention for TMWD seems prudent, since resolution of migraine may obviate the patient's desire for surgical intervention. Furthermore, given the prolonged recovery noted among migraine patients after SSCD surgery, it would also seem sensible to treat migraine preoperatively. However, it remains to be seen whether preoperative treatment of migraine will improve the longer recovery, and hopefully this will be delineated with future research. Management of migraine entails trigger avoidance, dietary modifications, and medication. For clinicians not comfortable with migraine management, referral to a neurologist is advised.

Other medications we employ are typical of supportive care given to vertiginous patients, including vestibular suppressants and anti-nausea agents. However, we impress upon our patients that these medications are for use only when symptomatic and not for routine use. Routine use of these medications will cause adverse effects on central compensation.

Microprism Lenses

More recently, we have come to recognize that a subset of TMWD patients have visual misalignment. There can be a multitude of symptoms arising from this and these patients can be identified using the binocular vision dysfunction questionnaire [20]. We refer to Neuro-Optometry/Ophthalmology for further evaluation and treatment in these cases. For an in-depth discussion of management of binocular vision dysfunction, see Chap. 14.

Sleep Apnea Evaluation

With the rising incidence of obesity in our society, we have seen a rise in sleep related breathing disorders, such as obstructive sleep apnea (OSA) [21]. OSA has been associated with a higher incidence of peripheral vestibular disorders (particularly Ménière's, BPPV, and sudden hearing loss) than those who do not have OSA [22, 23]. OSA has also been associated with Idiopathic Intracranial Pressure (IIP), transient increases in intracranial pressure, and spontaneous CSF leak [24, 25]. While IIP has been implicated as a possible etiologic role in the development of SSCD (see Chaps. 2 and 19), changes in intracranial pressure (ICP) is one of the main triggers of vertigo in TMWD. Treatment of OSA with CPAP has been demonstrated to improve symptoms and audiometric outcomes in Ménière's [26] as well as non-Ménière's spells of vertigo [27]. Whether OSA and/or IIP have any association with TMWD, however, is immaterial considering the other, far-reaching negative health effects of untreated OSA. Careful assessment of patients at risk for OSA, namely obese patients and those with narrowed airways, should include polysomnography and appropriate referral to a sleep specialist.

Other Medical Problems

The frequent finding of endolymphatic hydrops among SSCD patients suggests that medical measures aimed at its treatment may be reasonably employed. Additionally, control of any associated medical problems would also seem judicious. Many medical problems have been associated with endolymphatic hydrops, including allergy, autoimmune, metabolic, and endocrine (in particular hypothyroidism) disorders [28]. The basic tenant of treating Ménière's disease is to medically optimize treatment of these medical problems prior to considering surgical intervention. We think this same approach is reasonable for TMWD.

Postoperative Medical Therapy

Immediately postoperative, surgeons usually have routine medications dispensed for the expectations of pain, nausea, constipation, etc. In addition to these we prescribe high dose prednisone for 7–10 days with a subsequent taper. The rationale for this is to reduce any inflammatory response which could result in hearing loss and vestibular loss. As to how much, and for how long, we are simply speculating. This would be a good area for future research to help determine (1) are steroids effective for this purpose? (2) if so, are they more beneficial than the potential side effects encountered? (3) and which steroids and for how long? At this point we do not know.

SSCD surgery has been demonstrated to be quite effective for control of vestibular symptoms and autophony but much less so for other symptoms. We have witnessed this in our patients and have found the medical measures detailed above have frequently resulted in resolution or improvement in these symptoms. Again, we are not aware of any detailed analysis of this, but the symptoms that seem most amenable to these medical measures include aural fullness, otalgia, pulsatile tinnitus, hyperacusis, and residual autophony.

Physical Therapy

Vestibular rehabilitation therapy is most successful when treating a fixed peripheral vestibular lesion. By nature, TMWD are fluctuant, due to changes with sound and pressure evoked stimuli. As noted above, many of these patients will have symptom resolution with the medical measures discussed earlier in this chapter. However, in some patients, vestibular loss has occurred, and the patient will exhibit some symptoms attributable to an uncompensated vestibulopathy. If the lesion can be stabilized with medical and/or surgical intervention, vestibular rehabilitation should be employed to complete central vestibular compensation.

Conclusion

As mentioned earlier, there are no controlled trials of non-surgical treatment of TMWD. However, the otologic literature is filled with medical measures employed in the treatment of other vestibular disorders with varying success—and many of the patients in these studies almost certainly had TMWD. Our collective experience with TMWD patients over the past 24 years strongly suggests that medical therapy has a place in the management of TMWD. Especially for patients with mild symptoms and for patients who have persistent symptoms postoperatively, medical management may fill the void that has been present in TMWD.

References

1. Minor LB, Solomon D, Zinreich JS, Zee DS. Sound- and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal. Arch Otolaryngol Head Neck Surg. 1998;124(3):249–58. https://doi.org/10.1001/archotol.124.3.249. PMID: 9525507.
2. Smullen JL, Andrist EC, Gianoli GJ. Superior semicircular canal dehiscence: a new cause of vertigo. J La State Med Soc. 1999;151(8):397–400. PMID: 10554474.
3. Ossen ME, Stokroos R, Kingma H, van Tongeren J, Van Rompaey V, Temel Y, van de Berg R. Heterogeneity in reported outcome measures after surgery in superior canal dehiscence syndrome-a systematic literature review. Front Neurol. 2017;8:347. https://doi.org/10.3389/fneur.2017.00347. PMID: 28790965; PMCID: PMC5523725.
4. Ray A, Hautefort C, Guichard JP, Horion J, Herman P, Kania R, Houdart E, Verillaud B, Vitaux H, Attyé A, Eliezer M. MRI contribution for the detection of endolymphatic hydrops in patients with superior canal dehiscence syndrome. Eur Arch Otorhinolaryngol. 2021;278(7):2229–38. https://doi.org/10.1007/s00405-020-06282-3. Epub 2020 Aug 14. PMID: 32797276.
5. Gordon RT, Vining WD. Active noise control: a review of the field. Am Ind Hyg Assoc J. 1992;53(11):721–5. https://doi.org/10.1080/15298669291360427. PMID: 1442563.
6. Sone M, Yoshida T, Morimoto K, Teranishi M, Nakashima T, Naganawa S. Endolymphatic hydrops in superior canal dehiscence and large vestibular aqueduct syndromes. Laryngoscope. 2016;126(6):1446–50. https://doi.org/10.1002/lary.25747. Epub 2015 Nov 3. PMID: 26525170.
7. Johanis M, De Jong R, Miao T, Hwang L, Lum M, Kaur T, Willis S, Arsenault JJ, Duong C, Yang I, Gopen Q. Concurrent superior semicircular canal dehiscence and endolymphatic hydrops: a novel case series. Int J Surg Case Rep. 2021;78:382–6. https://doi.org/10.1016/j.ijscr.2020.12.074. Epub 2020 Dec 26. PMID: 33421957; PMCID: PMC7804363.
8. Feinberg D, Gianoli G, Rosner M. Inverse tullio phenomena: a novel approach to identifying vestibular disorders in patients using a noise cancelling device (NCD) in a binocular vision specialty clinic. Submitted for publication.
9. American National Standards Institute. Maximum permissible ambient noise levels for audiometric test rooms, ANSI S3.1 1991. New York: ANSI; 1991.
10. Kitajima N, Sugita-Kitajima A, Kitajima S. Superior canal dehiscence syndrome associated with scuba diving. Diving Hyperb Med. 2017;47(2):123–6. https://doi.org/10.28920/dhm47.2.123-126. PMID: 28641325; PMCID: PMC6147222.
11. Derebery MJ. Allergic management of Meniere's disease: an outcome study. Otolaryngol Head Neck Surg. 2000;122(2):174–82. https://doi.org/10.1016/S0194-5998(00)70235-X. PMID: 10652386.
12. Jafari Z, Kolb BE, Mohajerani MH. Hearing loss, tinnitus, and dizziness in COVID-19: a systematic review and meta-analysis. Can J Neurol Sci. 2022;49(2):184–95. https://doi.org/10.1017/cjn.2021.63. Epub 2021 Apr 12. PMID: 33843530; PMCID: PMC8267343.
13. Cooper CW. Vestibular neuronitis: a review of a common cause of vertigo in general practice. Br J Gen Pract. 1993;43(369):164–7. PMID: 8323804; PMCID: PMC1372362.
14. Hussain K, Murdin L, Schilder AG. Restriction of salt, caffeine and alcohol intake for the treatment of Ménière's disease or syndrome. Cochrane Database Syst Rev. 2018;12(12):CD012173. https://doi.org/10.1002/14651858.CD012173.pub2.
15. Hindiyeh NA, Zhang N, Farrar M, Banerjee P, Lombard L, Aurora SK. The role of diet and nutrition in migraine triggers and treatment: a systematic literature review. Headache. 2020;60(7):1300–16. https://doi.org/10.1111/head.13836. Epub 2020 May 25. PMID: 32449944; PMCID: PMC7496357.
16. Crowson MG, Patki A, Tucci DL. A systematic review of diuretics in the medical management of Ménière's disease. Otolaryngol Head Neck Surg. 2016;154(5):824–34. https://doi.org/10.1177/0194599816630733. Epub 2016 Mar 1. PMID: 26932948.

17. Beh SC. The neuropsychology of dizziness and related disorders. Otolaryngol Clin N Am. 2021;54(5):989–97. https://doi.org/10.1016/j.otc.2021.05.016. Epub 2021 Jul 20. PMID: 34294432.
18. Wackym PA, Mackay-Promitas HT, Demirel S, Gianoli GJ, Gizzi MS, Carter DM, Siker DA. Comorbidities confounding the outcomes of surgery for third window syndrome: outlier analysis. Laryngosc Investig Otolaryngol. 2017;2(5):225–53. https://doi.org/10.1002/lio2.89. PMID: 29094067; PMCID: PMC5654938.
19. Niesten ME, McKenna MJ, Grolman W, Lee DJ. Clinical factors associated with prolonged recovery after superior canal dehiscence surgery. Otol Neurotol. 2012;33(5):824–31. https://doi.org/10.1097/MAO.0b013e3182544c9e. PMID: 22664897.
20. Feinberg DL, Rosner MS, Rosner AJ. Validation of the binocular vision dysfunction questionnaire (BVDQ). Otol Neurotol. 2021;42(1):e66–74. https://doi.org/10.1097/MAO.0000000000002874. PMID: 33105328.
21. Bibbins-Domingo K, Grossman DC, Curry SJ, Davidson KW, Epling JW Jr, García FA, Herzstein J, Kemper AR, Krist AH, Kurth AE, Landefeld CS, Mangione CM, Phillips WR, Phipps MG, Pignone MP, Silverstein M, Tseng CW, US Preventive Services Task Force. Screening for obstructive sleep apnea in adults: US Preventive Services Task Force recommendation statement. JAMA. 2017;317(4):407–14. https://doi.org/10.1001/jama.2016.20325. PMID: 28118461.
22. Byun H, Chung JH, Jeong JH, Ryu J, Lee SH. Incidence of peripheral vestibular disorders in individuals with obstructive sleep apnea. J Vestib Res. 2022;32(2):155–62. https://doi.org/10.3233/VES-210012. PMID: 34250919.
23. Low WK, Lim EJ. Concomitant obstructive sleep apnoea in patients with Meniere's disease: a case report and literature review. Case Rep Otolaryngol. 2021;2021:5592611. https://doi.org/10.1155/2021/5592611. PMID: 33859856; PMCID: PMC8009700.
24. Rabbani CC, Saltagi MZ, Manchanda SK, Yates CW, Nelson RF. Prevalence of obstructive sleep apnea (OSA) in spontaneous cerebrospinal fluid (CSF) leaks: a prospective cohort study. Otol Neurotol. 2018;39(6):e475–80. https://doi.org/10.1097/MAO.0000000000001805. PMID: 29889790.
25. Sugita Y, Iijima S, Teshima Y, Shimizu T, Nishimura N, Tsutsumi T, Hayashi H, Kaneda H, Hishikawa Y. Marked episodic elevation of cerebrospinal fluid pressure during nocturnal sleep in patients with sleep apnea hypersomnia syndrome. Electroencephalogr Clin Neurophysiol. 1985;60(3):214–9. https://doi.org/10.1016/0013-4694(85)90033-1. PMID: 2578929.
26. Nakayama M, Masuda A, Ando KB, Arima S, Kabaya K, Inagaki A, Nakamura Y, Suzuki M, Brodie H, Diaz RC, Murakami S. A pilot study on the efficacy of continuous positive airway pressure on the manifestations of Ménière's disease in patients with concomitant obstructive sleep apnea syndrome. J Clin Sleep Med. 2015;11(10):1101–7. https://doi.org/10.5664/jcsm.5080. PMID: 26094927. PMCID: PMC4582051.
27. Foster CA, Machala M. The clinical spectrum of dizziness in sleep apnea. Otol Neurotol. 2020;41(10):1419–22. https://doi.org/10.1097/MAO.0000000000002824. PMID: 32740553.
28. Rizk HG, Mehta NK, Qureshi U, Yuen E, Zhang K, Nkrumah Y, Lambert PR, Liu YF, McRackan TR, Nguyen SA, Meyer TA. Pathogenesis and etiology of Ménière disease: a scoping review of a century of evidence. JAMA Otolaryngol Head Neck Surg. 2022;148(4):360–8. https://doi.org/10.1001/jamaoto.2021.4282. PMID: 35142800.