Ménière’s disease with all of its clinical manifestations can be divided into two categories – those symptoms that derive from the cochlea (the anterior labyrinth) and/or those that arise from the vestibular labyrinth or the posterior part of the inner ear. The symptoms that arise from the cochlea include hearing loss, which can be fluctuant and progressive and often is, or tinnitus which can be intermittent or constant and can vary in intensity and quality. Other symptoms include aural pressure which is predominantly a symptom of the pars inferior, so far as we can tell, from histopathological studies of endolymphatic hydrops of the pars inferior, namely the saccule and the cochlear duct or the scala media, and loudness intolerance (recruitment). Symptoms that derive from the posterior labyrinth include, of course, episodic vertigo or other forms of vestibular upset. Also as we have described in the past, motion or positional vertigo is a very common aspect of Ménière’s disease invariably present during a vestibular episode; but very often in between episodes as well. Disequilibrium and imbalance can occur with or between vertiginous episodes. Collapse attacks, or so called Tumarkin attacks, occur most likely from disruption of an otolithic organ.

There is no question that Ménière’s disease when it occurs in its typical or classical form including all of the above symptoms is easy to diagnose. It is also definitely true that one can have Ménière’s disease involving either the anterior or the posterior labyrinth one to the exclusion of the other.

Moreover we have seen many patients with vestibular Ménière’s disease who years later displayed hearing loss and had typical or classical Ménière’s disease as well. Patients can have cochlear hydrops or cochlear Ménière’s disease, or symptoms referable to the anterior labyrinth to the exclusion of vertigo and symptoms that derive from the posterior labyrinth. On many occasions while studying the natural cause of Ménière’s disease, those patients often will, years later, develop vestibular symptoms also.

We have earlier described that the cause of Ménière’s disease is multifactorial inheritance, that its pathogenesis is endolymph malabsorption and that the symptoms are a combination of chemical and physical changes involving sensory cells and the membranes of both the anterior and posterior parts of the inner ear. We continue to believe this to be the case having seen many thousands of patients who have had Ménière’s disease in its various forms.

It has been described by us and others that there are anomalies in the region of the mastoid in these patients. For example, there is typically hypopneumatization. The sigmoid sinus instead of being lateral is in a medial anterior location, Trautmann’s triangle is often either reduced, or sometimes even absent. Others have described hypopneumatization around the vestibular aqueduct, and hypoplastic changes that occur in the endolymphatic sac itself. We also notice in these patients that the facial recess and aditus are more obstructive and underdeveloped than they would be in a normal mastoid.

From our conservative sac enhancement procedures, in more than 5,000 patients to date, we have noted that tinnitus is improved or absent following the procedure in more than 50% of cases. It is also true that hearing loss is improved in 40% and pressure is improved in more than 50% while vestibular symptoms are absent in 75% or more and are significantly diminished in 95% or more of patients.

However, as described above, symptoms in classical or early (insipient) Ménière’s disease can completely involve the entire labyrinth. Or symptoms can be referable as mentioned, primarily to the cochlea. We have seen many patients who have complained of pressure and tinnitus; they may describe loudness intolerance (recruitment), but typically will not have dizzy spells or vertigo. It is natural for the otologist in these patients to consider Eustachian tubal dysfunction as a primary etiological problem. A tympanogram is taken in these patients and if there is any question about Eustachian tubal dysfunction the patient should be treated with decongestants and anti-hystemetics systemically, a nasal spray and Valsalva maneuvers. Usually this suffices if the Eustachian tube is at fault.

We have seen many patients, including my wife, who have normal tympanograms but complain of pressure, and periodic or constant tinnitus as the only symptoms of early endolymphatic hydrops. It should be mentioned that pressure sometimes is the chief complaint in a patient with classical Ménière’s disease. In these patients, after ruling out Eustachian tubal dysfunction, we advise the patient to avoid CATS – caffeine, alcohol, tension and salt. We also advise other means of assisting them with psychological support and sometimes this symptom of tinnitus accompanied by pressure is very disabling for the patient. In addition, in such patients we provide a diuretic, typically Triamterene Hydrochlorothiazide (Dyazide), and have found that in the vast majority of these patients that the symptoms are improved or ameliorated. Thus, this provides another group of patients with tinnitus, who can benefit from medical treatment as would be provided to a typical patient with the full constellation of symptoms resulting from Ménière’s disease.

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