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Menière's disease

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Forward

I WOULD like to present a rather unusual case of attic cholesteatoma which, because of its specific pathological features, may throw indirect light on the current trend of ideas concerning the pathogenesis and therapy of an essentially unrelated condition, Menière's disease. While animal experimentation, statistical analysis of a large number of case histories, and anatomical studies of a certain medical entity are the usual means whereby we try to explain pathological or pathophysiological situations, we should not forget that disease itself can serve as Nature's experiment, from which basic facts can be learned. The case described herewith is not an isolated one—four similar cases were described by us only two years ago (Bumsted *et al.*, 1977)—only at the time I did not perceive what can be deduced from them concerning the fascinating entity of Menière—the so-called endolymphatic hydrops.

Material

The case: L.Y., male, 39 years old, had suffered intermittently from mild discharge from both ears for 20 years. Tinnitus was absent, but very slight positional dizziness was noticed at times. On examination: the right ear presented an atelectatic drum, grade II, of the *pars tensa* (Sadé and Berco, 1976), as well as of the *pars flaccida.* These were 'plastered' over the neck of the malleus and the long process of the incus. Some bone of the scutum was missing around the periphery of the retracted *membrane flaccida.* The left ear was similar to the right, except for an additional 4×4 mm. 'central' perforation in the anterior part of the drum. The middle-ear mucosa was seen through the perforation to be of normal texture.

Audiometry showed 10 db. bone and 30 db. air in both ears—SRT 35–40 db. left, 30–35 db. right. Discrimination was 92 per cent bilaterally. A patch test over the perforation in the left ear showed elevation of the threshold by 10 db. and a ventilating tube inserted in the right eardrum accomplished the same elevation of the threshold. Mastoid X-rays were not taken pre-operatively.

I planned to do a myringoplasty on the left side. It was carried out under general anaesthesia through a postauricular incision—an approach I find convenient in anterior perforations with a slim anterior tympanic

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margin, especially in the presence of some anterior bony overhang. Once the tympanomeatal flap was elevated, I found, surprisingly, a hidden cholesteatomatous sac, medial to the incus and to the head of the malleus. Part of the long process of the incus was eroded and replaced by fibrotic tissue. The sac was closed and intact, forming a complete epidermoid cyst. At first the head of the malleus was nipped off from its neck and the incus disarticulated from the stapes. The head of the malleus, the incus and the epidermoid cyst were then slowly rolled out of the epidermoid cyst bed. Once the sac was elevated, the facial nerve was found to be exposed through its entire horizontal course. The sac continued posteriorly and superiorly toward the antrum and mastoid, which were completely filled by it. However, the most dramatic finding was the destruction of the greater part of the semicircular canals—the lateral semicircular canal, the posterior semicircular canal and the common crus were destroyed, as was the postero-superior wall of the vestibule. Granulation tissue separated the cholesteatomatous sac matrix from what was left of part of the partly exposed superior semicircular canal. Most of the bone separating what remained of the inner ear and the internal auditory canal (anteriorly) from the sigmoid sinus, its bulb and the cerebellum (posteriorly) had disappeared, leaving a deep crater. The cholesteatomatous epithelium was removed, save for that part of the cholesteatoma which 'sealed' the deep antero-inferior part of the eroded vestibule. Surprisingly, no perilymph leak was seen. Altogether there was hardly any bone work done—most of the destruction, and therefore the exposure, had occurred previously, due to the cholesteatoma and the underlying granulation tissue. The amount of bone destruction can be appreciated from the X-rays which were taken post-operatively (Fig. 1). Once the ear was cleaned, the handle of the malleus was rotated over the stapedial head and the drum repaired with temporal fascia—a rotating tympanomeatal flap was swung over the fascia.

During the first post-operative day, the patient suffered from vertigo, but he was up and about on the third day. Once the dressing was removed and epithelization complete, hearing in the operated ear was 35 db. SRT; discrimination did not change—92 per cent. His bone conduction had dropped to the 20 db. level up to 2,000 Hz., and to the 70–80 db. level in the 3,000–8,000 Hz. range (Fig. 2). Today, one year after the operation, he has a dry, medium-sized self-cleansing mastoid cavity and an aerated tympanic cavity. When a small piece of cotton wool soaked in ice water was placed in the cavity, slight vertigo and nystagmus were elicited, implying that the utricle, saccule and/or one or more ampullae were not completely destroyed!

Discussion

A. *Nature's experiments—and their message*

The remarkable conclusion from such a case is that, contrary to what is

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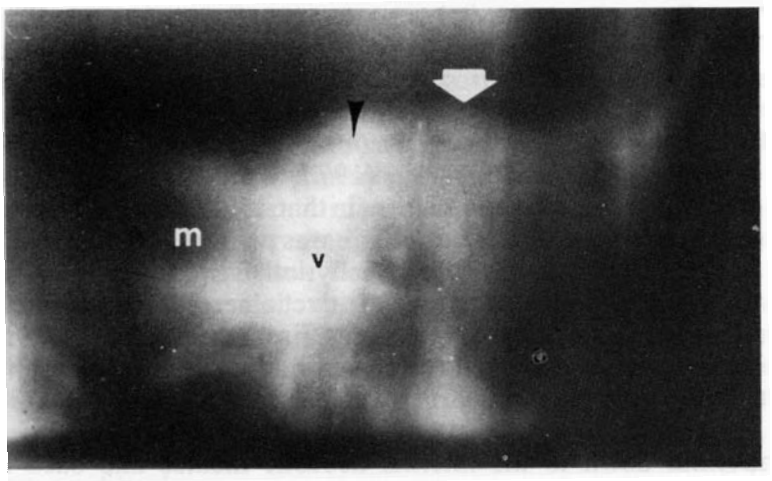


FIG. 1

Tomography showing: m—internal acoustic meatus; black arrow-head—superior part of superior semicircular canal; V—vestibule; white arrow-head—Tegmen.
Note: In between the white arrow-head and the vestibule all the labyrinth (except for part of the superior semicircular canal) has disappeared.

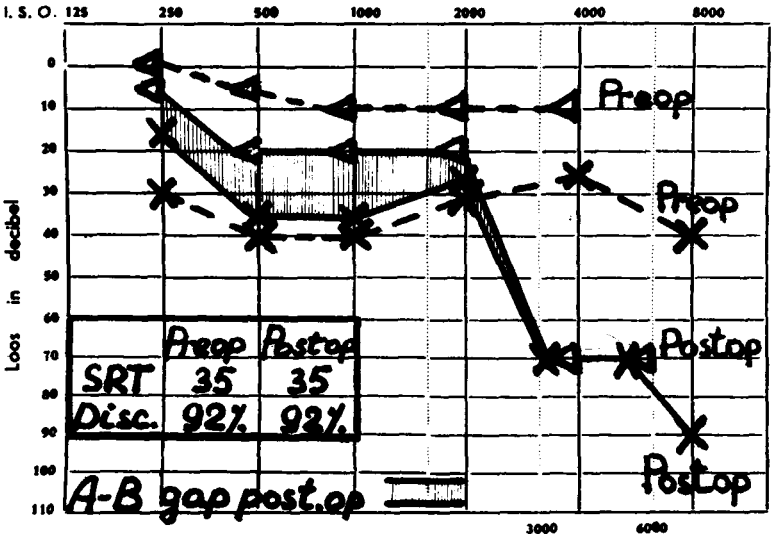


FIG. 2

Pre-operative and post-operative audiograms.

(or was) accepted, destruction of any part of the membranous labyrinth does not necessarily lead to total functional abolition of the remaining part, i.e. of the cochlea. This was discussed in detail in our former paper (Bumsted *et al.,* 1977), where four similar cases were described. These were in addition to 14 cases previously described in the otological literature (Sadé-Sadowsky, 1955; Pedersen, 1969; Phelps, 1969). Other

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otologists (Paparella, 1979; Palva *et al.*, 1976; Sterkers, 1979; Thomsen *et al.*, 1980) have also told me that they have encountered similar cases, thus one may suppose that such events are less rare than might be assumed.

The cases described by Pedersen (1969) and Phelps (1969), the four cases described by us from Iowa City (1977) and the case described above contain an important common element in that, in addition to destruction of part of the labyrinth and vestibule, there was present destruction of much of the bony tissue which separates the labyrinth from the posterior fossa. It is in this region that the endolymphatic duct emerges from the vestibule, just below and medial to the common crus. From there it courses its way to the cerebellar dura—and it is therefore almost impossible to conceive that this structure was spared in these seven ears. In fact, in two out of these cases upon which I operated myself, I did not find any bone left between the labyrinth down to the internal acoustic meatus and back to the cerebellum. This bony region was empty, destroyed by the cholesteatoma and its underlying granulation tissue—the amount of missing bone being similar to that we remove when we prepare a translabyrinthine exposure for acoustic tumour removal. In view of the current concepts concerning the role played by the endolymphatic duct and sac in the pathogenesis of Menière's disease, one would expect these patients to suffer from episodes of vertigo, tinnitus and fluctuating hearing loss pre-operatively—and certainly post-operatively. Yet this was not the case.

It was Michael Paparella (1979) who also drew my attention to the fact that in Mundini malformation, the whole posterior part of the inner ear is missing, but these patients do not suffer from a Menièriform syndrome; neither do these ears present the histological features of a dilated membranous labyrinth, i.e. of a labyrinthine hydrops. This observation, which in essence is similar to that derived from our cholesteatomatous cases, also suggests that the critical physiological and pathophysiological role which is attributed to the endolymphatic duct and sac should be re-examined.

B. *The concept of hydrops*

The concept that Menière's disease is secondary to hyperaccumulation of fluid in the membranous labyrinth, which consequently creats excessive pressure there, is not new. The basic idea promulgated is that endolymph is secreted by the stria vascularis and in turn is constantly reabsorbed by the endolymphatic duct and sac system. Some evidence does indeed exist to support this chain of events (Lundquist, 1963). It is further postulated that when reabsorption through the endolymphatic duct and sac is defective, excessive fluid accumulation becomes inevitable. Intra-labyrinthine pressure will now be augmented—resulting in Menière's disease. Already in 1927 an operation designed to relieve such an over-pressure was devised by Georges Portmann (1927). When Hallpike and

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Cairns (1938) described a histological specimen of Menière showing dilatation of part of the membranous labyrinth, little doubt was left that Menière's syndrome is in effect a hydrops (or 'glaucoma') of the inner ear. Indeed, with time, the two (hydrops and Menière's disease) became almost synonymous. Two additional factors to support the hydrops concept were advanced: experimental induction of histological hydrops was accomplished by obliterating the endolymphatic duct and sac system (Kimura, 1976); and further multiple reports have accumulated claiming relief of Menière's symptoms through measures (shunts) which were supposed to reduce the endolymphatic pressure (House, 1964; Arenberg, 1979).

While the foregoing cause-and-effect relationship is logical and attractive, it is nevertheless worthwhile from time to time to re-examine the details of such an accepted medical theory.

C. *Hydrops histology*

Histologically, hydrops is described as a situation where the saccule is ballooned while Reissner's membrane is displaced towards the scala vestibuli. Collapse and rupture of the saccule is also found at times. Dilatation of the utricle is much less marked—if present at all—*and the semicircular canals have never been seen to be affected.* It is interesting to note that only in exceptional instances are cochlear hair-cells found to be missing—and then, notwithstanding profound hearing loss, only over a rather limited area. The macular and cupular hair-cells have so far not been found to be affected at all. Such histological features have been reported from most Menière autopsies (Schucknecht, 1974).

These findings could be significant if dilatation of the saccule and displacement of the Reissner membrane were found only in cases of Menière's disease, but this is not so. Similar histological features have also been found in syphilitic ears (Schucknecht, 1976), in a large proportion of ears with chronic otitis media (Paparella *et al.,* 1979), in some otosclerotic ears and in other unspecific entities (Lindsay, 1946; Lindsay, 1958; Kimura, 1976; Sando *et al.,* 1976). These reports were from patients who did not suffer from episodes of vertigo, feelings of pressure in the ear or tinnitus, or from other features typical of Menière's disease—indeed, some had no sensorineural hearing loss at all. The fact that the histological features of hydrops are present in a multitude of conditions indicates that these features do not represent a specific pathological picture for any particular disease—Menière's disease being no exception. It is possible, and even plausible, that this is the way the inner ear react non-specifically (as an end result?) to many pathological conditions of various natures—in analogy to the non-specifity of inflammatory reaction. On the other hand, it is also possible that hydrops manifests itself also as a postmortem change, i.e. that we are dealing with a histological artefact (Kelemen, 1979). This thought could be also

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entertained because, when the stapes is extracted in hopeless Menière's disease cases, when destruction of the labyrinth is the best way out, the saccule is not seen to bulge any more than in an ordinary stapedectomy ear.

D. *Expungement of the ductus endolymphaticus—experimental and other*

Many experiments have been performed to simulate Menière's disease or histological hydrops of the inner ear. The most elegant results were obtained by Kimura (1976), who was successful in producing hydrops in most of his guinea pigs after obstructing the endolymphatic duct or sac. However, most of Kimura's experimental animals showed no vestibular symptoms. It is interesting to note that the same histological hydrops has apparently been obtained in numerous other experiments in which the endolymphatic duct or sac system was not touched. In fact, various experiments, having nothing in common with one another, on the external, middle and inner ear, or the internal auditory meatus, all ended in the same fashion, i.e. a histological hydrops resulted. Thus injection of chloroform into the external auditory canal (Mygind, 1948), the injection of distilled water, tuberculin and horse serum (Naito, 1959) or sodium chloride (Arslan, 1969) into the middle-ear, and injection of pilocarpin or acetylcholin into the internal auditory canal (Naito, 1959), all produced distension of the saccule and displacement of Reissner's membrane. Even intravenous injection of egg albumin (Sadamitsu, 1963) or polystyrene (Alford *et al.,* 1965) and a multitude of other insults, reviewed by Kimura in his excellent paper (Kimura, 1976), all produced histologically an inner ear 'hydrops'. This brought Kimura (1976) to conclude that hydrops can develop from almost any kind of insult or injury to the ear and that its occurrence is unpredictable. Later, when Kimura (1979) produced hydrops in animals by obstructing the duct, and then relieved the obstruction, the histological hydrops created by him did not disappear.

E. *Treatment of Menière's disease*

The history of the treatment of Menière's disease is of importance to the patient, to doctors and indeed to those who are interested in the philosophy and history of medical treatment. Actually, there have been dozens of treatments proposed to cure, control or alleviate Menière's disease, some having their days for a short time, others for longer periods, but none has withstood the test of time.

If we exclude the relatively small number of Menière's cases in which the patient has troublesome and repeated vertiginous attacks and loss of practically all hearing or discrimination, so that a destructive operation is indicated, we are left with the majority of cases which suffer from some hearing loss and noise intolerance, occasional vertigo, fullness (or pressure) and tinnitus. Indeed, the greatest misery and burden of most

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Menière patients does not emanate from their vertigo, but from their hearing loss, intolerance of noise and tinnitus. Destructive labyrinthectomy, if properly done, will stop all further vertigous attacks, but usually none of the other symptoms. Selective vestibular neurectomy can also accomplish the same and may save, *at times,* some of the remaining hearing.

Thus, the majority of Menière patients do not suffer to the extent that a destructive operation is warranted, and therefore other therapeutic measures have been resorted to. Both medical and surgical attacks on the vascular system have had their days. Various medications supposed to being about 'inner ear vasodilatation' have been employed, and later patients were submitted to cervical sympathectomy. Anti-histamines had their proponents too, yet others thought (and some do so today) that histamine itself would be more helpful. The chorda tympani has been suspected of being the culprit, and has been sectioned—with excellent results; seventy per cent of patients so treated were relieved of their symptoms (Rosen, 1951). This percentage of successes (or thereabouts) is found in many other treatments for Menière's disease (Guilford, 1964).

But the main trend has been towards a more logical approach, based on the pathophysiology of the inner ear increased pressure—namely, relief of endolymphatic pressure. Impressive success has been reported by several methods. At first, low salt diets were in vogue, but this was superseded by more than one method which attempted surgical drainage or shunting of the endolymphatic system into the subarachnoid space. It has been stated that the 'endolymphatic subarachnoid shunt has shown that equalizing endolymphatic pressure against the spinal fluid pressure by shunting these two fluids together will control Menière symptoms' (House, 1965). Support for the endolymphatic duct 'insufficiency' concept has also been obtained from X-ray studies, which claimed the canal to be narrowed in Menière cases, but this finding was challenged by others, who could not confirm it (Øigaard *et al.,* 1975). Others, like Schindler (1979), have also raised questions. They reported a poor correlation between the surgeon's ability to identify the sac and its actual presence in histological material. Schindler's study throws doubts on the accurate placement of drainage tubes in the lumen of the endolymphatic sac. Plantenga and Browning (1979) noted that, in 80 per cent of their samples, the sac 'was not a sac or bag-like structure' and their Menière cases 'showed no obvious pathological features in the endolymphatic sac, nor in the vestibular aqueduct, in temporal bones with idiopathic endolymphatic hydrops when compared with normals'.

Evidence of intralabyrinthine hyper-pressure has also been sought by feeding glycerol to Menière patients—the glycerol supposedly drawing fluid out of the inner ear and improving thereby the hearing rather promptly. Reports of hearing improvements in Menière after glycerol injection encouraged many. However, when Thomsen and Vesterhange (1979) applied this test to two groups of Menière's patients they found,

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that while the hearing improved in those who were told it would improve, it actually decreased in those patients who were told that this is what would occur.

The most revealing information came from Shambaugh (1966) who claimed to obtain essentially as good results as in shunt operations by merely 'decompressing' the endolymphatic sac. Shambaugh chose this direction after examining patients operated by House (Shambaugh, 1966), some of whom improved in spite of the non-introduction of a shunt when the sac was not found at surgery. Later, a controlled study by Cawthorne reported by Shambaugh (1966) yielded similar results. Shambaugh interpreted the well-being of those patients to *decompression or revascularization* of the sac and advocated such an operation (with or without insertion of a 'vascularizing' muscle flap) in favor of shunting the sac. However, benefit from such an operation can also be interpreted as being due to a surgical placebo effect.

Indeed, quite a few experienced otologists have suspected that the benefit advocated from most (if not all) non-destructive therapeutic measures are in the nature of a placebo. This point of view was based on the fact (among others) that, no matter what the therapy was, the successful results remained in the 50–80 per cent range, usually around 70 per cent, which is also the rate of spontaneous remission in Menière (Stahle, 1976; Turner, Saunders and Per-Lee, 1973). This point was discussed in details in Guilford's (1964) excellent papers.

Another difficulty in evaluating the therapeutic benefit in a disease like Menière, is the well-known observation that Menière patients have frequent spontaneous remissions, some lasting for years. Thus only studies having a five-year follow-up have real value, but such controlled studies are hard to find.

Last, but not least, any physician who has treated Menière patients for many years knows that many of these patients respond well when listened to, when paid attention; I often find them very grateful and appreciative if I take time to listen to their complaints, anxieties and symptoms of stress, which they often realize are significantly related to their physical symptoms. Indeed, the real sceptic believes this to be the best and safest treatment today, excluding the few patients whose claim merits a destructive procedure.

The terms 'stationary' and 'progressive' hydrops have been suggested (Schucknecht, 1976) to try and bridge over these contradictions, but in effect they lead to no more than a semantic exercise. Also Shambaugh's (1976) division of Menière patients into 'secondary' and 'primary' is probably more confusing than helpful, as there is little in common between a patient who is vertigous secondarily to an obvious trauma to his labyrinth and an idiopathic Menière (primary) patient who presents the classical quartet of vertigous attacks, tinnitus, sensorineural hearing loss and a feeling of pressure in his ear. On the other hand, I think that it is

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probably time for us to admit that we do not know, or understand, the etiology, pathogenesis or treatment of what we term Menière's disease. All this in spite of recognizing the entity much better than before, as well as the many interesting data (experimental and otherwise) which have been added to our bank of information concerning this subject in the last generation.

Summary and Conclusions

This paper describes patients who, at surgery, were found to have their endolymphatic duct region destroyed by an extensive cholesteatomatous process—yet, these patients did not suffer from symptoms belonging to Menière's disease or the like.

The pathogenesis of Menière's disease is considered today by many to be due to a disorder or malabsorption of endolymphatic fluid from the endolymphatic sac, which supposedly results in the accumulation of an excessive amount of endolymph. The condition termed 'endolymphatic hydrops', i.e. histologic distension of the saccule and Reissner's membrane, is found in autopsies of Menière's patients and is attributed to malabsorption of endolymph. Support for this theory comes from the successful creation of a similar histological picture by tampering with the endolymphatic duct, X-ray evidence of a narrowing of the endolymphatic duct, amelioration of hearing after the injection of glycerol and, indeed, remission after shunt operations.

However, histological hydrops has also been created by many other kinds of experiments, unrelated to the endolymphatic duct, on the external, middle and inner ear. Also, the histological picture considered typical for Menière, i.e. the hydrops, has been found in a multitude of diseases other than Menière. All this information suggests the possibility that the histological features of hydrops are not really specific for Menière's disease, but may be the non-specific end result of diverse insults to the ear. Both the glycerol test and X-ray evidence showing narrowing of the duct have also lately been challenged. As to the shunt operation, its success rate is probably as high as any of the many treatments of Menière's disease advocated through the years—or indeed as that of spontaneous remission.

The conclusions which may be drawn are:

A. The critical physiological and pathophysiological role supposedly played by the endolymphatic duct and sac should be reviewed.
B. The evidence at hand that Menière's disease is either secondary to hyper-pressure or secondary to malabsorption of the endolymphatic fluid by the endolymphatic duct and sac, is probably not conclusive.
C. We should bear in mind that, notwithstanding the many interesting data (experimental and otherwise) gathered in the last generation concerning the inner ear and Menière's disease, the essential pathogenetic nature of the disease still eludes us.

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D. The efficacy of treating Menière's disease by aiming to relieve endolymphatic pressure—or, in effect, any other medical or surgical treatment aimed at changing one parameter or another in the inner ear (vascular bed, etc.)—should be re-assessed.

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