MÉNIÈRE'S DISEASE AND ITS SURGICAL TREATMENT

REPORT OF 300 CASES*

FRANCESCO CASTELLANO, M.D.

Neurosurgical Clinic, Serafimerlasarettet, Stockholm, Sweden†

(Received for publication July 14, 1950)

Olivecrona in 1943 described all cases of Ménière's disease observed in the Neurosurgical Clinic of Serafimerlasarettet from 1930 to December 31, 1942, a total of 160 cases. This paper is a continuation of the previous work, including all cases observed up to December 31, 1949. The total has now increased to 300, averaging 20 cases a year. Of these patients, 254 were operated upon. This represents 2.5 per cent of all surgical operations performed at this Clinic.

Ménière's disease is a symptom-complex characterized by sudden attacks of vertigo usually accompanied by nausea and vomiting, unilateral reduction of hearing, and tinnitus. The cause is probably irritation of the acoustic nerve. The exact localization of an abnormal stimulus is not yet known but the most likely situation is in the terminal receptors. A similar symptom-complex may be caused by any space-occupying lesion around the eighth nerve; in such a case we prefer to call it Ménière's syndrome. Frequency and duration of attacks are very variable, a peculiarity of the disease being long periods of remission. Examination almost always reveals defect of the acoustic nerve, more marked in the cochlear than in the vestibular branch.

The space-occupying lesions that may cause a Ménière's syndrome are acoustic neurinomas, other cerebellopontine angle tumors, and aneurysms of the vertebral artery or, less frequently, of the posterior choroidal artery. These lesions, however, simulate Ménière's disease only at their onset, because during the course of the disease other symptoms develop, such as of a lesion of other cranial nerves and often of the cerebellum, while in Ménière's disease only the acoustic nerve is affected. Only in cases of vertebral aneurysm may Ménière's syndrome be simulated for a long time (Olivecrona). Processes affecting the ear (otitis media with extensive perforation of the tympanum, otosclerosis) may also exhibit symptoms of Ménière's syndrome.

Sex and Age. The disease is more common in men. Of our patients, 186 were men and 114 women. The onset is usually between the ages of 31 and 55 years; 211 of our cases (70 per cent) came within this group. The youngest patient was 13 years of age, the oldest 72. The age distribution appears in Fig. 1.

Laterality. In 147 patients the disease was on the right side, and in 142 on the left. In 11 patients it was difficult to determine the affected side.

* The surgical results of the same material were presented at the “Giornate Mediche Internazionali,” Verona, Italy, July 20–30, 1950.
† Chief: Professor H. Olivecrona.
Occasionally a bilateral acoustic examination revealed no abnormalities, so that if the patient did not have a definite unilateral tinnitus it was impossible to determine the side of the disease. Bilateral Ménière’s disease does occur, and was found in 7 of our patients (2.3 per cent). Dandy found a rather higher percentage (10 per cent).

Predisposing Factors. (a) Trauma. Previous trauma was ascertained in 37 of our patients. Dandy considered trauma of no importance. This has not been our experience. In the majority of cases trauma is important only because it calls attention to a pre-existing process, but some of our cases present interesting considerations. In 1 patient (No. 60, History: 233.38) unilateral tinnitus and deafness developed immediately after a cranial trauma. Ten years later he had typical attacks of Ménière’s syndrome. Operation revealed an arachnoiditis of the cerebellopontine angle, bridling the nerves and vessels (a branch of the anterior inferior cerebellar artery was very adherent to the eighth nerve).

(b) Otitis. Ear infections affecting only the diseased side, or both sides, occurred in 26 instances. In several of our cases an earlier otitis media, particularly if accompanied by a defect of the tympanum, without doubt played an important role. One patient (No. 17, History: 2752.35) had bilateral otitis media with extensive perforation of the tympana. Examination of the vestibular function was impossible because even gentle blowing into the external ear, especially on the right side, caused nausea and typical dizziness. The right side was most affected and section of the right vestibular branch was performed. Several years later, however, the patient again had attacks of dizziness, probably due to irritation of the left acoustic nerve. This case was diagnosed as bilateral Ménière’s disease.

Blood Pressure. A rise in blood pressure is often considered to be the cause of dizziness. The average blood pressure in 270 patients was systolic 138, and diastolic 82, corresponding almost to that found by Crowe in 68 patients, systolic 137, and diastolic 83. Only in a few instances did the pressure rise to nearly 200. We therefore do not consider that blood pressure plays any important role.

Symptomatology. As stated previously, Ménière’s disease is characterized
by unilateral deafness, tinnitus, and typical attacks of vertigo accompanied by nausea and vomiting. The average duration of symptoms at the time of admission to hospital was from 6 to 7 years. The shortest period was 4 months, the longest 30 years.

The first symptom in the 300 patients in this series is shown in Table 1.

<table>
<thead>
<tr>
<th>Symptom Combination</th>
<th>No. of Pts</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness</td>
<td>175</td>
<td>58.2</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>49</td>
<td>16.3</td>
</tr>
<tr>
<td>Deafness</td>
<td>28</td>
<td>9.3</td>
</tr>
<tr>
<td>Tinnitus and deafness</td>
<td>41</td>
<td>14.0</td>
</tr>
<tr>
<td>Dizziness and tinnitus</td>
<td>4</td>
<td>1.3</td>
</tr>
<tr>
<td>Dizziness and deafness</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>Dizziness, deafness and tinnitus</td>
<td>2</td>
<td>0.6</td>
</tr>
</tbody>
</table>

Table 1 shows that dizziness was usually the earliest symptom. However, it is quite probable that the disease began with a slight deafness often revealed by a casual examination. Dandy\textsuperscript{5} states that deafness is usually present when the first attack appears, and Wright\textsuperscript{20} maintains that the first symptoms are not pertinent to the vestibule, but to the cochlea, probably due to the presence of the "vascular area" in the cochlea, which secretes endolymph. Examination further shows that hearing is affected much more often than the vestibular function (see later).

In the majority of cases the three characteristic symptoms appear a short time apart, but often the first appears much earlier than the others. Table 2 shows the number of patients in our material in whom the first symptom appeared at least 3 years before the others.

<table>
<thead>
<tr>
<th>Symptom Combination</th>
<th>No. of Pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness</td>
<td>20</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>8</td>
</tr>
<tr>
<td>Deafness</td>
<td>10</td>
</tr>
<tr>
<td>Tinnitus and deafness</td>
<td>7</td>
</tr>
</tbody>
</table>

Dizziness is the main symptom. The attacks of vertigo are usually characteristic, and are referred to as Ménière's attacks. The frequency of the attacks is highly variable. They may occur several times in one day, or years apart. In some forms of Ménière's disease there may be mild and more or less permanent giddiness, with a tendency to recrudescence. We found 2 such cases in our material. In similar cases Crowe\textsuperscript{3} considered it advisable to suspect an acoustic tumor. The duration of the attacks is also very variable. They may last for a few minutes, or for several days. We have found that the average attack lasts about 2 hours. Typical attacks of vertigo begin
suddenly. The patient must hold on to something to keep from falling. He feels that everything is spinning, often in one definite direction. This fact we were able to ascertain in 44 patients. In 23 patients the objects seemed to whirl towards the diseased side, in 21 towards the healthy side. This sign is therefore of no importance in determining the side of the lesion. Several patients felt the necessity of lying down on a certain side during the attacks to avoid the recurrence of dizziness, but this fact was also of no practical importance. Nausea, cold sweats, and diarrhea often occur, and are probably explained by a lowering of the blood pressure. In 2 instances there was loss of consciousness. In Crowe’s material loss of consciousness occurred in 12.8 per cent of the patients. Vomiting is usually associated with dizziness and occurred in 70 per cent of our patients. It may occur several times during the same attack. The onset of deafness is insidious. It usually has a slowly progressive course and may result in complete deafness. Of our patients 43 (13 per cent) were completely deaf. Tinnitus may be constantly present or may disappear between attacks. It usually becomes worse just before an attack, and grows more intense during and after the attack. It does not disappear with complete deafness, and sometimes constitutes the warning aura of attacks of vertigo. The term “pseudo-Ménière’s disease” is used to indicate characteristic attacks of vertigo without deafness and tinnitus. In such cases it is always difficult to determine the side of the disease, especially when the auditory and vestibular tests are completely normal. We had 2 such cases. In one, the patient vaguely remembered having had a slight unilateral tinnitus for a short time. Section of the vestibular branch of the acoustic nerve was performed on the affected side with good result. The other patient was not operated upon. In similar cases Dandy’s advised bilateral section of the vestibular portion of the acoustic nerve. He sometimes performed the bilateral procedures at the same time, with satisfactory results. Bilateral operation usually causes disturbances of equilibrium, especially in the dark. Romberg’s sign is positive. There is no ataxia, but inability to coordinate the movements of the eyes. Headache, usually of the frontal type, was present in about 30 per cent of our patients. Mygind and Dederding found this symptom in a very high percentage of their patients, and attribute it to an accumulation of cerebrospinal fluid in the posterior cranial fossa. This phenomenon could also be due to changes in the hydrosaline metabolism causing a considerable retention of water.

Pathological Anatomy. This constitutes a rather unknown chapter. Hallpike and Cairns (1938) examined the labyrinths of 2 patients with Ménière’s disease who had died following operation. In both cases, in addition to signs of chronic otitis, they found gross dilatation of the membranous labyrinth and degeneration of the organ of Corti and the vestibular receptors. According to them the dilatation of the labyrinth was due to increased endolymphatic tension which caused compression of the receptors and their subsequent degeneration. In another case of Ménière’s disease, in which death was due to pneumonia, Berggren and Gray were unable to find any
pathologic changes in either the labyrinth or the central nervous system. Berggren refutes the hypothesis of Winkler who, on the basis of animal experiments, maintains that lesions of the nuclei of Goll and Burdach may cause a disease similar to Ménière’s syndrome. To this effect he cites a case of Stenwers in which complete degeneration of these nuclei was found, whilst the patient, when alive, had never had attacks of dizziness. The hypothesis that Ménière’s disease is due to central lesions is difficult to accept because it is hard to understand how a metencephalic process can affect only the vestibular and cochlear nuclei, which do not even lie in close proximity, without causing any change in other adjacent structures. Sometimes dizziness persisted even after section of the vestibular nerve. This may suggest a central lesion, but is probably due to incomplete division of the vestibular nerve or to a bilateral affliction.

Histological examination of the acoustic nerve gave contradictory results. Dandy⁴ consistently found signs of fibrosis, and maintained that the pathological stimulus is situated in the nerve trunk. Berggren² (2 cases), Crowe³ (2 cases) and Furstenberg⁴ (11 cases), on the other hand, never found any pathologic changes in the nerve removed at operation. Furthermore, mechanical stimulation with a dissector of both branches of the acoustic nerve in patients who had been locally anesthetized caused only slight nausea and, occasionally, vomiting, but never dizziness. In 1 patient the cochlear nerve was stimulated with faradic current without any effect. Wright¹⁰ is of the opinion that the lesion is caused by dilatation of the labyrinth and chemical modification of the endolymph system.

Attention should also be given to the alterations in course of the anterior inferior cerebellar artery. This artery may pass either above the eighth nerve, between its two branches, or below the eighth and seventh nerves. In 1 patient McKenzie¹³,¹⁴ found a small venous plexus above the acoustic nerve. He considered the anomalous course of the anterior inferior cerebellar artery to be one of the causes of Ménière’s syndrome. Watt and McKillop¹⁹ described the great variability in the course of this artery in relation to the acoustic nerve, and considered deafness in some patients, especially the older ones, to be due to sclerosis of the artery which, owing to its abnormal hardening and thickening, compresses the nerve. Dandy, in 20 per cent of 550 patients operated upon, found an encircling artery constricting the nerve, or a large artery lying on it. In several of our cases an artery was found lying close to the acoustic nerve, which appeared sometimes to be the internal auditory artery, and sometimes the anterior inferior cerebellar artery.

Etiology. The original hypothesis that Ménière’s disease was due to an intralabyrinthine hemorrhage has always been the least acceptable. Not long ago Öhnell¹⁶ again studied this hypothesis, and considered the possibility of a relationship between Ménière’s disease and scurvy. He examined 87 patients, and found evidence of scurvy in 45 of them. A further 8 patients probably had scurvy; in the remaining 34 patients it was doubtful. From a
therapeutic point of view he appears to have obtained favorable results with the administration of vitamin C. Investigations in Macacus rhesus in which he produced artificial scurvy, did not permit any conclusions being drawn.

Nowadays the generally accepted hypotheses regarding the cause of Ménière’s disease are: the allergic, the bacterial, and the disturbance of water metabolism. Mygind and Dederding maintained that Ménière’s disease is caused by a biochemical alteration of the endolymph. According to them an increase of sodium ions in the endolymph produces a considerable fluid retention and, thereby, dilatation of the membranous labyrinth. This concept is corroborated by the anatomo-pathological investigations of Hallpike and Cairns. According to Furstenberg, Richardson and Lathrop, sodium ions cause dizziness. In several of our cases we have tested Furstenberg’s therapy, a diet excluding sodium chloride but with large doses of ammonium chloride, with fairly good results, but the material is too small to draw any conclusions.

The allergic hypothesis, held chiefly by Horton, is based on a casual discovery. A patient with migraine and Ménière’s disease who was being treated with histamine for the former condition, recovered not only from the migraine, but also from Ménière’s disease.

The bacterial hypothesis advanced by Wright suggests a focal infection in the internal ear as the true and only cause of Ménière’s disease. According to Wright all other mechanism may be considered as determining the attack, but not the essential cause of the disease.

The hypothesis of an angiospastic origin of dizziness also deserves mention. During the examination of the fundus oculi in a patient, Berggren noticed a sudden spasm of the arteries and pallor of the papilla. A few seconds later the patient had a typical attack of dizziness. Apparently the same phenomena have been observed in cases of migraine.

Objective Findings. Audiograms and vestibular tests were carried out. Neurologic studies and general examinations seldom reveal anything of note. Results from bilateral audiograms and vestibular tests in the majority of the 300 patients of this series are shown in Table 3.

Conclusions from these data are: (1) There is an obvious difference between the diseased and healthy side, although in 12 cases the findings were normal on both sides (7 of these patients were not operated upon, and in the other 5 patients the proper side for operation was determined only by the tinnitus. (2) Tinnitus does not disappear with complete deafness; 43 patients who were totally deaf stated they had tinnitus. (3) Complete absence of vestibular excitability does not preclude dizziness. (4) The hearing is affected more often than the vestibular function. This may support the hypothesis that auditory disturbances occur much earlier than vestibular dysfunction.

Nystagmus was observed in about 10 per cent of the patients, but was of no aid in determining the side of the disease. In McKenzie’s group of 12 patients, 5 had nystagmus; in 3 of them the gaze towards the side of the
lesion increased the nystagmus. In another patient nystagmus was visible only during gaze to the affected side. The last patient had nystagmus only during attacks of vertigo and it was then much more evident towards the side later ascertained as the side of the lesion.

**Diagnosis.** In the majority of cases no difficulties were encountered in diagnosing the nature of the disease. X-ray examination of the internal auditory meatus should always be carried out. Encephalography is indicated in all cases in which a pontocerebellar tumor is suspected. These examinations were carried out in 30 patients, and in each case the findings were normal.

In only 1 case was an incipient papilloedema observed.

The diagnosis of Ménière's disease is based on the rather characteristic

**TABLE 3**

<table>
<thead>
<tr>
<th></th>
<th>Diseased side</th>
<th>Healthy side</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Pts.</td>
<td>Percentage</td>
</tr>
<tr>
<td>Hearing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complete deafness</td>
<td>43</td>
<td>13</td>
</tr>
<tr>
<td>Considerably decreased</td>
<td>178</td>
<td>61</td>
</tr>
<tr>
<td>Slightly decreased</td>
<td>54</td>
<td>20</td>
</tr>
<tr>
<td>Normal</td>
<td>70</td>
<td>7</td>
</tr>
<tr>
<td>Vestibular excitability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>20</td>
<td>7</td>
</tr>
<tr>
<td>Considerably reduced</td>
<td>73</td>
<td>23.5</td>
</tr>
<tr>
<td>Slightly reduced</td>
<td>70</td>
<td>24.5</td>
</tr>
<tr>
<td>Normal</td>
<td>128</td>
<td>43</td>
</tr>
</tbody>
</table>

history, the auditory and vestibular tests, the negative result of the neurologic examination (careful study of the trigeminal sensibility and corneal reflex, and facial motility), the X-ray findings and the encephalography.

**SURGICAL TREATMENT**

Ménière's disease can be treated either medically or surgically. It is not our intention in this work to deal with the medical therapy. Surgical treatment is indicated in (1) all patients who have not improved on medical treatment and (2) patients whose working capacity is impaired due to the severity of the symptoms. The operation is contraindicated in patients who show progressive improvement with increasingly low periods of remission.

Various operative procedures are carried out for the relief of the symptoms. The operation may be performed on the labyrinth or on the nerve trunk, but the former is now seldom done. The method employed in this Clinic consists of section of either the entire acoustic nerve or only its vestibular branch. Dandy in 1928, was the first to perform section of the acoustic nerve, and in 1931 McKenzie the first to section only the vestibular branch.

In the present series 254 patients were operated upon. In 27 of them the
eighth nerve was completely divided, and in 226 only its vestibular branch. In 1 patient the posterior cranial fossa was explored and the acoustic nerve freed from adhesions, but not sectioned. In the first 11 patients operated upon during the years 1931 to 1936 the entire acoustic nerve was cut. Since 1936, however, only the vestibular branch of the eighth nerve has been cut, complete section being done only when the patients were totally deaf on the diseased side. In a few instances the nerve had to be completely divided to arrest accidental hemorrhage.

 Operative Technic. The patient is placed in face-down position and the head fixed with strips of adhesive tape (Fig. 2). The table is adapted with lateral supports so that it may be tilted around its long axle. During operation the table can be tilted this way, whereby a good exposure of the cerebellopontine angle is assured.

Local anesthesia was almost invariably employed, but in a few instances avertin was administered rectally.

The skin incision is best described by Fig. 3.

The bone defect is made as high and as far lateral as possible. The defect must be made as small as possible (Fig. 4), with a maximum diameter of 2–3 cm. Small bone defects lessen danger of postoperative extradural hematoma. Operation is easier if the bone defect is so far lateral as to expose the
medial margin of the sigmoid sinus. Sometimes the mastoid cells are unavoidably opened, in which case they are injected with penicillin and plugged with surgical wax.

A straight lateral margin to the bone defect is advisable, so that the dural flap may be turned back on it, providing maximum exposure (Fig. 5). Complete bone hemostasis must be obtained before the dura is opened. The dura is opened by a T-shaped incision, seen in Fig. 5. At this stage the lateral cistern is opened, to further facilitate the operation. The fluid in the cistern having been drained, it is now possible to explore the cerebellopontine angle. The cerebellum is retracted medially and the acoustic and facial nerves are identified. The facial nerve is situated ventrally, and is usually hidden by the acoustic nerve. As is well known, about 1 cm. from the internal auditory meatus, the cochlear branch is located caudally and the vestibular branch cranially. Sometimes, owing to rotation of the nerve, the cochlear branch is directed dorsally and the vestibular ventrally. The farther one comes from the internal auditory meatus, the greater is the distance between the acoustic and facial nerves. As a rule the cochlear and vestibular branches of the acoustic nerve are distinctly separate. Dandy\textsuperscript{7} reports a case in which the two branches were completely detached. Sometimes, however, the acoustic nerve appears as a single structure, and it is then difficult to see the line of division. If the patient is totally deaf on the side of operation the nerve is any case completely divided. If the deafness is only partial, only the cranial half of the nerve is cut. Very frequently, as already pointed out under the heading of pathologic anatomy, small arteries are found lying close to the acoustic nerve. Occasionally an artery is situated between the acoustic and facial nerves, completely hidden by the former. The artery can therefore be damaged during section of the nerve or the vestibular branch. The resulting hemorrhage can be very troublesome, and to control the bleeding it may be necessary to apply silver clips to the cochlear and facial nerves.

The vestibular branch of the nerve, after being freed, should be lifted with a small blunt hook and divided with a small knife (Fig. 6). Dividing the vestibular nerve with a hook is not to be recommended because it is easy to damage the small arteries, and sometimes the facial nerve itself. It is very important that the function of the facial nerve be continuously observed throughout the operation. If during section of the vestibular branch
a sudden and complete facial paralysis is observed, accidental division of the facial nerve has most probably occurred. This can be verified by electrical stimulation of the nerve. In the event of the nerve being divided an attempt at anastomosis must immediately be made. Facial paralysis which appears a few hours or a few days after the operation is due to secondary causes, and regeneration of the nerve takes place within 6 months.

In retracting the cerebellum great care must be taken to avoid damaging the veins that run from the cerebellum to the dural sinus.

Having obtained complete hemostasis, the incision in the dura is closed with interrupted silk sutures. Any small defects in the dura are covered with fibrin or gelatine foam. The wound is then closed in anatomic layers. The muscles must be firmly and accurately sutured to prevent leakage of cerebrospinal fluid and formation of cysts.

As a rule, during the first 4 to 5 days after operation, the patient suffers from severe dizziness, vomiting, diplopia and headache, but these symptoms are transient and quickly disappear.

Complications. There were no operative deaths. The only complications were facial paralysis and extradural hematomas. Temporary facial paralysis was observed in 14 of our patients and permanent paralysis in 2 cases.* In a 3rd case the facial nerve was accidentally severed, but was satisfactorily repaired by a graft from the saphenous nerve, held in place with fibrinogen and thrombin. This procedure gave a satisfactory result, and 2 years after operation only a slight facial weakness was noticeable.

Extradural hematomas occurred in 5 instances. The symptomatology is well known. A few hours or 1–2 days after operation the patient begins to be dazed and does not reply to questions. Usually the blood pressure is raised and the pulse rate is decreased. In 1 patient there was simultaneously an extradural and an intracerebellar hematoma, which recurred three times. A hemorrhagic diathesis was finally discovered, the patient was given large doses of vitamins K and C, and recovered satisfactorily.

Results. The postoperative observation period in this series varies from

* In 1 case the internal auditory artery was hidden along its entire length between the acoustic and facial nerves. During section of the vestibular nerve, hemorrhage necessitated the use of silver clips, which resulted in injury to the cochlear branch and facial nerve. Facial paralysis followed immediately and was permanent. In another case the facial nerve was injured by a hook, probably because the division of the vestibular branch by the hook was made too near the porus acusticus, where the facial and acoustic nerves lie in close proximity. Anastomosis according to Dott was attempted, but failed on account of necrosis of graft. Facial-spinal anastomosis was then performed, but the facial paralysis did not improve.
a few months to 19 years. The patients keep us informed yearly as regards their state of health, and sometimes attend the Clinic for control examinations. During this period only 3 patients have died, the cause of death in each case being due to irrelevant diseases.

After section of the vestibular branch it is no longer possible to stimulate the vestibule, and nystagmus towards the non-affected side is always observed. In 7 patients the vestibule could still be stimulated by injecting a great quantity of cold water, but in no case was dizziness aroused. Complete freedom from dizziness was obtained in all cases except 8, in 7 of these probably due to a bilateral affliction and in 1 presumably due to incomplete section of the vestibular fibers. In this last-mentioned patient the attacks became less frequent, but did not disappear. In no case was bilateral operation performed. The majority of the 27 patients in whom the acoustic nerve was completely sectioned were totally deaf. Of the 226 patients in whom only the vestibular branch was divided, 46 became totally deaf. In 26 patients the hearing deteriorated, but in the majority of cases it was the same as before operation. In only 5 cases was there improvement in hearing after operation. Our percentage (2 per cent), therefore, is much lower than that of Crowe (20 per cent). The majority of our patients have been examined on discharge, 1–2 weeks after operation. The tinnitus was influenced by the extent of the operation. In 4 (15 per cent) of the 27 patients in whom complete section of the acoustic nerve was performed, the tinnitus improved, in 3 of them disappearing completely. In only 5 (2.3 per cent) of the 226 patients in whom only the vestibular branch was sectioned, did the tinnitus improve. In 2 of these 5 the tinnitus disappeared completely. Dandy found improvement of the tinnitus in 50 per cent of his patients, a fact which surprises us. We maintain that the tinnitus seldom disappears. Probably, however, section of the cochlear branch would cause disappearance of tinnitus more often.

SUMMARY

A series of 300 cases of Ménière's disease is presented with an account of etiology, pathological anatomy, symptomatology, objective findings, detailed operative procedure, and follow-up. There were 254 patients submitted to operation. In 27 complete section of the acoustic nerve was performed. In 226 only the vestibular branch was divided. In 1 the nerve was freed from adhesions, but not sectioned.

Complete freedom from dizziness was obtained in all patients except 8; 7 of these 8 patients had bilateral Ménière's disease. Tinnitus rarely disappeared. There was no operative mortality. Facial paralysis occurred in 16 cases, transient in 14, and permanent in 2. Postoperative extradural hematomas occurred in 5 patients, in 1 of them also an intracerebellar hematoma. Follow-up periods varied from a few months to 19 years.

REFERENCES


