MÉNIÈRE'S DISEASE.

INTRODUCTION

P. Ménière, 1799-1862, read a paper to the Académie de Médecine on 8th January 1861 entitled "On a particular form of severe deafness resulting from a lesion of the Inner Ear". This was the first description of a condition which had never previously been recognised and which was subsequently to be named after its discoverer. In the course of time much confusion was to arise and numerous conditions associated with vertigo and deafness were referred to as "Ménière's Disease"; until finally at the present time we have "Ménière's Disease" as a single pathological entity and "Ménière's Syndrome" a loose collection of unrelated conditions associated with Vertigo and Deafness, ranging from wax in the ear to the cerebello-pontine angle tumour.

HISTORICAL

Prosper Ménière was born in Angers in 1799. In 1828 he received his doctorate from the University of Paris, and in 1832 he was installed as head of the clinic of the Faculté
under the direction of Chomel. He was made medical director of the Deaf and Dumb Institute of Paris in 1838.

He was a man of wide culture, interested in mediaeval medicine, on which he published a number of papers, and a classical scholar whose translations of Latin prose and verse are still well worth reading.

He died suddenly in Paris on the 7th February 1862, just a year after the presentation of the paper which was to make him famous.

We have no record of this original paper apart from the Author's summary which was published in the Gazette Medicale de Paris of 1861, and it is of such interest as to merit quotation in full:

"I. A previously healthy auditory apparatus can suddenly become the seat of functional disturbances consisting of variable noises, continuous or intermittent, which soon are accompanied by greater or less hearing loss.

II. The functional disturbances having their seat in the mechanism of the Inner Ear can give rise to symptoms previously regarded as cerebral. These are vertigo, giddiness, uncertain gait, staggering and falling, accompanied by nausea, vomiting and a state of collapse.

III. These symptoms, which are intermittent, are
soon followed by an ever increasing deafness and often all hearing is suddenly and completely lost.

IV. Everything leads to the belief that the actual lesion which is the cause of these functional disturbances is situated in the semi-circular canals."

This communication aroused considerable interest and numbers of cases were referred to Ménière from which he selected the following for publication.

"Case I. M.X., medical practitioner, aged 47 years, had suffered for fifteen years from variable noises in the ears, particularly the left, which gradually lost its function in spite of the most energetic treatment. The right ear was failing in the same way. Neither had ever been affected by inflammation or even catarrh of the tubes or the drums; air had always reached the middle ear freely.

There had been frequent attacks of congestion of the external ears, which became hot and red. This the patient had treated by simple remedies and he had abandoned all treatment since 1854 in the hope that the deafness would not progress.

For three years the disease made no progress, until, on 26th December last, after a day spent in the open air, M.X. while quietly reading was taken by a violent fit of sneezing. Then, when he wished to rise he found that his gait was staggering and that he could not freely steer himself in a straight line. Three hours later, the same symptoms persisted although the patient had taken a strong
mustard foot bath. He made a light meal and went to bed hoping that sleep would put an end to this troublesome incident.

He woke at 2 a.m. feeling that walking was even more difficult than on the night before. There was a feeling of weight in the left occipito-mastoid region, of pressure, and the patient turned involuntarily from right to left. There was a threat of falling, as if the left side no longer obeyed his will, in spite of the fact that in bed both sides of the body freely performed all voluntary movements.

Intelligence was not affected; the patient was able to write immediately to a colleague for assistance. The latter came at once, and as he was about to let blood from an arm the patient was overcome by nausea and vomited. He vomited repeatedly and very frequently during the following day; the stomach was unable to keep anything down.

On the 28th and 29th and the following days, under the influence of bleeding, of wet cupping and a purgative, the disorder of locomotion improved progressively and on the 2nd January the patient was able to go out while remaining a trifle unsteady in his gait. To-day his general health is perfect, but his hearing is gradually weakening.

The doctor who observed exactly the above related succession of phenomena upon himself believed at first that it was a cerebral congestion. Two of his colleagues who saw him during the illness thought as he did that the congestion was located in the cerebellum. They now believe, however, that the symptoms more probably depend upon a lesion of the inner ear, and we are fully convinced of this."
Discussion. In the light of subsequent investigations one may consider this a true case of Ménière's Disease, involving both labyrinths and with a relative preponderance of function of the Right labyrinth in the attack described. The feeling of weight in the affected occipito-mastoid region has been quite commonly noted by later observers. It is tempting to suggest that the flushings of the ears might hint at a vasomotor instability and the prolongation and severity of the attack suggest the vaso-dilator or allergic type of this disease.

"Case II. Here is another case which seems to us well suited to throw light upon this question.

M.X., medical practitioner, age 45 years, small, thin, dark, of wiry constitution and hasty southern temperament, suffered in 1858 attacks of intermittent fever severe enough to require the use of sulphate of quinine in large doses. The fever abated, but there remained in the ears noises which ended up by becoming sufficiently intense to draw the patient's attention. He was the less concerned at the beginning of the trouble as previously on several occasions in analogous circumstances the noises had promptly disappeared. This time it could not have been the same, the noises persisted and soon the hearing was felt to be failing. He suffered some irritation of the skin of the external ears, which consisted of itching and scanty discharge. Soon the patient suffered attacks of vertigo, coming on suddenly and followed by vomiting.
This type of cerebral symptom recurred very often, even under the influence of a journey, or a sudden change of temperature. The giddiness with nausea and vomiting persisted through January and February and the patient had to remain in bed because of general weakness and exhaustion. During March the vertigo ceased. On August 25th it recurred and with such severity that on several occasions Doctor X. fell suddenly in the roadway on his way to hospital. Deafness rapidly increased, although apart from a slight mucoid discharge the ears were healthy, requiring only a slight expiratory effort with nose and mouth closed to introduce air into the middle ear.

All the most active therapeutic measures were deployed against these pseudo-cerebral symptoms without success and to-day the hearing is almost lost. The general health is good, cerebral functions normal, and everything proves that the cause of the symptoms lies in the internal auditory apparatus."

It is rather more difficult to be sure of the condition in this second case. The history of tinnitus following heavy quinine dosage suggests a toxic neuritis, and the extremely short and severe course of the labyrinthine disease is rather suggestive of a serous labyrinthitis. On the other hand, the abrupt recurrence of attacks after a five months' remission, with the sudden dramatic onset and obvious lack of warning as shown by the collapse in the streets is not altogether
inconsistent with the vaso-spastic type of Ménière's Disease to be described later. A third possibility is that of an undiagnosed chronic otitis media with dry cholesteatoma and a labyrinthine fistula, in spite of the patency of the Eustachian tubes.

Ménière proceeds: "We possess a great number of similar observations from people of an intelligence to observe and recount their symptoms.

Some of these patients are quick to note the coincidence between the tinnitus, vertigo and deafness, and having established for themselves the prognosis of this singular malady no longer worry over cerebral catastrophe. Others remain deeply uneasy as they do not understand that the disease has its principal focus in the ear. The popular knowledge of the seriousness of brain lesions, with the expectation of apoplexy, paralysis and imbecility which follow in their train, causes horrible torments of worry to some patients. The doctors are warned. Let those who observe such symptoms in their patients take the trouble to explore carefully their auditory apparatus. Let them measure the amount of hearing in each ear, let them question the patients to find out what has happened in these organs since the onset of the attacks of congestion of the brain. They
will soon recognise that the internal auditory apparatus is the site of the trouble, the starting point of symptoms which have seemed at first to be so fearful, and against which has been employed a rigorous treatment of which the uselessness is not the least of its inconveniences.

We have said, and do repeat that accidents of this nature are fairly common. We have seen a great number of them. They constitute a perfectly distinct morbid entity with the specific characteristic of loss of hearing .... in a word, if one studies the auditory apparatus the diagnosis of the malady will be arrived at, and thereafter one can dispense with all the efforts of a violent therapy of which the least inconvenience is to harm the patient and cast doubts upon the sagacity of the doctor."

It is unnecessary further to quote from Ménière's case histories; all show the same skilful observation and accurate reporting, but we cannot leave him without mentioning the too well known fatal case, which is remembered and quoted when so much more valuable and important work is forgotten. Taken in its context, as will be seen, it is merely given as an example bearing out in man the experiments of Flourens on pigeons localising certain symptoms to the labyrinth. Nowhere does Méniere claim that this is an autopsy on a case of Ménière's Disease.
"I have spoken elsewhere, a long time ago, of a young girl who, having travelled at night in winter on the outside of a coach, while in her catamenia, suffered, as a result of severe exposure, from a sudden and complete deafness. She was admitted to the wards of M. Chomel presenting symptoms of continuous vertigo. The slightest effort to move herself produced vomiting, and death ensued on the fifth day. Necropsy demonstrated that the brain, cerebellum and spinal cord were all entirely normal. As the patient had become completely deaf, after hearing perfectly, I removed the temporal bones to seek carefully for the cause of this sudden and complete deafness.

The only abnormality I found was that the semi-circular canals were filled with a red plastic material, a sort of sanguinous exudation of which hardly a trace appeared in the vestibule and none in the cochlea. The most careful search enabled me to state with precision that the semi-circular canals were the only part of the labyrinth revealing an abnormality and this consisted, as I have said, in the presence of a red plastic lymph replacing the liquid of Cotungo.

Can one, on the authority of a single fact, establish the necessary correlation between vertigo, deafness and a lesion of the semi-circular canals? We would not have been bold enough to reply in the affirmative if there were no confirmatory evidence. However, the experiments of M. Flourens on section of the canals, with resulting turning of the animals, support the view that the symptoms in man consisting of vertigo, nausea and collapse, accompanied by
As knowledge has progressed it has become possible to differentiate a number of general conditions which may give rise to focal labyrinthine symptoms, such as arteriosclerosis, disseminated sclerosis and some of the blood diseases. It has also become possible to recognise certain local pathological conditions such as tumour of the eighth nerve and labyrinthitis, which also give rise to giddiness, deafness and vertigo. When all these known conditions have been eliminated there remains a large group of patients in which, in the words of Ménière, "a previously healthy auditory apparatus becomes the site of functional disturbances associated with vertigo, deafness and tinnitus. These symptoms are intermittent and progressive and lead to a greater or less hearing loss."

The investigation of this condition which Brunner has defined as "Idiopathic Ménière's Syndrome" in contrast to "Symptomatic Ménière's Syndrome" (where there is a known cause) has been hampered by the fact that it is not in itself a fatal disease. The first report of the pathological findings in two authenticated cases of idiopathic Ménière's Syndrome appeared in 1938 by Hallpike and Cairns. Both patients had died as a result of cerebellar oedema and haemorrhage following section of the eighth nerve. In the first case both temporal bones showed a chronic non-suppurative otitis media. The left
labyrinth, which in life had been diagnosed as the site of the disease, showed a generalised gross dilatation of the endolymph system, with obliteration of the perilymph spaces. There was also degeneration of the left organ of Corti. In both there was described an obliteration of the normal peri-saccular connective tissue around the saccus endolymphaticus. The right labyrinth did not show any endolymph dilatation.

In the second case only the left temporal bone, which was the site of the lesion, was examined. Here also there was a gross dilatation of the endolymph system with obliteration of the perilymph spaces, degeneration of the organ of Corti, and a reduction of the normal peri-saccular connective tissue around the saccus endolymphaticus. There was also degeneration of the sensory epithelium of the vestibule and the stria vascularis, and degeneration and rupture of the wall of the anterior vertical canal. Confirmation of the findings of gross endolymph dilatation was supplied by a further case described by Hallpike and Wright in 1939, and by a report on six cases by Rollin in 1940.

This demonstration of the labyrinthine changes in idiopathic Menière's Syndrome is of great importance and interest, but yet it does not explain the underlying pathology.

A very large series of cases have now been submitted to section of the eighth nerve, either complete or partial, and it
has been found that complete section does not relieve all cases of their tinnitus, and in some it even becomes more severe, as we shall see later when we consider more fully the results of surgical treatment of this disease.

There remain now to be considered the clinical features and response to empirical methods of treatment of idiopathic Ménière's syndrome. The paroxysmal character of the attacks with the functional involvement of all the labyrinthine end organs has suggested to observers from the time of Cheatle and Knapp that there might be a temporary increase of endolymph pressure associated with some vascular instability. In 1900 McBride was treating Ménière's Disease with rest in bed, purgation, sweating and injections of Pilocarpine. He noticed that attacks of vertigo with syncope and sweating appeared to be due to vagal spasm. In 1908 Politzer remarked on the occasional association of Ménière's disease with angioneurotic oedema, and suggested that the vertiginous attacks might be due to a vasodilation of the labyrinthine capillaries.

Between 1919 and 1929 Portmann turned his attention to the possibility of drainage of the endolymph system, on the analogy of the trephination of glaucoma. He perfected a trans-mastoid approach to the saccus endolymphaticus and claimed to cure the vertigo and relieve the tinnitus in cases of Ménière's disease.
Musgrave Woodman in 1939 reported very favourably on his results of Portmann's operation on a series of eleven cases of Ménière's disease.

From the foregoing very incomplete survey we can conclude that there exists a certain agreement on the pathology of idiopathic Ménière's Syndrome. It is generally admitted that the lesion in the labyrinth is associated with a gross dilatation of the endolymph system. There is some evidence to show that this dilatation may be due to a vasomotor imbalance in the capillaries of the stria vascularis.

There is also evidence that in some cases there are permanent central changes, the nature of which is at present unknown.

**TREATMENT OF MÉNIÈRE'S DISEASE.**

As there is at the present moment no common agreement on the etiology of Ménière's Disease, the methods of treatment depend on the choice of the individual clinician.

Mygind stated flatly that the characteristics of the attacks of Ménière's Disease made it certain that the underlying pathology must be concerned with an intermittent increase of endolymph pressure. His associate, Dida Dederding, launched upon one of the most careful and painstaking investigations
ever conducted on Ménière's Disease to find evidence to prove or disprove this assertion, and to elaborate some rational method of treatment.

Dederding turned her attention to an exhaustive study of the hearing in a very large series of cases, and noted the occurrence of great variations in this from day to day. She particularly noted variations in the duration of bone conduction, which she was able to demonstrate, after Gellé, to be concerned with pressure on the footplate of the stapes. She made a pressure chamber and tested the effect of atmospheric pressure on the bone conduction of the normal ear, the otosclerotic ear, and the ear in Ménière's Disease. She was able to show that during the active phase in Ménière's Disease the footplate of the stapes was dislocated outwards and fixed by the increased endolabyrinthine pressure. This caused an elevation of the lower tone limit, a shortened bone conduction and impaired air conduction.

Mygind and Dederding restricted the fluid intake of their patients and exhibited Salyrgan in an effort to expel the extracellular water which they felt to be the cause of the condition. Out of 157 cases so treated, 151 were reported to have lost their vertigo.

Dederding notes that of the 157 cases in her series,
nearly all had symptoms of some general nature. Most had headaches, a third were rheumatic, a third had symptoms and signs of vasomotor disturbance, and a large number had symptoms of gastro-intestinal disturbance. She concluded by expressing her opinion that the basic lesion is probably a vasomotor condition of the labyrinth.

Furstenberg and his co-workers in 1934 criticised the methods of investigation used by Dederding in her estimation of the fluid intake and output. They pointed out that the fluid intake as such accounted for less than half the water available to the organism; the remainder is accounted for by water contained in solid food, and by tissue oxidation. The urinary output, also, accounts for less than half the fluid loss from the body; the remainder is due to evaporation from the skin and lungs and the water in the faeces.

Furstenberg reported an investigation on fourteen cases of typical, active, idiopathic Ménière's Syndrome. In each case he was able to prevent the development of attacks of vertigo on a salt free diet, regardless of the fluid intake. He was able to precipitate attacks by the exhibition of Sodium Chloride or Bicarbonate, and to control the attacks by the use of Ammonium Chloride. He claimed as the result of this work that the condition must be due to a retention of sodium ions in the
tissue fluids. He outlined a regime of high protein, low salt diet, with the addition of 3 gm. of Ammonium Chloride to each meal for three days, with a break of two days without Ammonium Chloride, between each course.

Talbott and Brown in 1940 studied in 48 cases of idiopathic Ménière's Syndrome the acid-base constituents of the blood, both during attacks and in the resting phase. They were totally unable to show any variations from the normal, even in patients who had been on a low sodium plus Ammonium Chloride regime, and had been improved by it. They claimed that this investigation showed that retention by the body of water and salt is not a factor in the attacks.

Talbott and Brown failed to induce attacks of vertigo by exhibition of sodium by mouth or intravenously, although the concentration of serum sodium was raised 5 milliequivalents per litre. This was accompanied by a simultaneous decrease in protein concentration and increase of water content in the serum. They decided that the benefit derived from Furstenberg's regime must be due to an increased concentration of Potassium ions. They suggested the daily exhibition of 6 to 10 gm. of Potassium Chloride. This caused an increased diuresis and a lowered concentration of Sodium ions. Forty patients out of the forty eight investigated were improved if
not entirely relieved of their symptoms.

Walsh and Adson in 1940 reported on the cases which had registered at the Mayo Clinic with a complaint of paroxysmal vertigo, deafness and tinnitus between 1929 and 1938. Excluding cases of arteriosclerosis, intracranial lesions and labyrinthitis, 186 cases were considered.

The disease was noted to be one of middle life, without sex preponderance.

In 175 cases of Ménière's Syndrome treated by Medical measures their results are as follows:

<table>
<thead>
<tr>
<th>Type of Treatment</th>
<th>Disappearance of Vertigo</th>
<th>Improvement of Vertigo</th>
<th>Unchanged</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Per Cent</td>
<td>Number</td>
</tr>
<tr>
<td>Low salt diet and Ammon. Chloride (128)</td>
<td>45</td>
<td>36</td>
<td>33</td>
</tr>
<tr>
<td>Low salt diet and Potass. Nitrate (13)</td>
<td>4</td>
<td>31</td>
<td>6</td>
</tr>
<tr>
<td>Low salt diet alone (11)</td>
<td>3</td>
<td>27</td>
<td>4</td>
</tr>
<tr>
<td>Other general Measures (23)</td>
<td>14</td>
<td>61</td>
<td>4</td>
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</tbody>
</table>
The effect on hearing of the Medical treatment roughly paralleled that on the vertigo: 28% stated that their deafness and tinnitus improved, 25% that the deafness became worse and 47% that their hearing was unchanged.

From this survey Walsh and Adson conclude that remissions in Ménière's Disease are common, and spontaneous disappearance of the vertigo is not infrequent. Two thirds of the patients treated by low salt diet alone or combined with Ammonium Chloride or Potassium Nitrate have their vertigo either relieved or very much improved.

There are many reports in the literature of Ménière's syndrome occurring in patients with allergic manifestations. In one of the cases described by Ménière and previously cited the vertiginous attack was preceded by a severe bout of sneezing. Dederding noted that a third of her patients showed signs and symptoms of vasomotor instability, and numerous cases have been described where a known allergan precipitated the attacks. Dohlman in 1939 recorded four cases, two of which were due to specific allergans (in one case milk and in the other wheat), and two to a "general" allergy. In all four the vertigo was cured by treatment directed to the allergic condition.

Miles Atkinson in 1940 found two patients sensitive to
food proteins, withdrawal of which prevented the vertiginous attacks and exhibition was followed by a recurrence of the vertigo. Both patients had been relieved for many months by avoidance of these foods without any other treatment.

Atkinson suggested that idiopathic Ménière's Syndrome is a symptom complex produced by at least two causes, in one group allergic and in the other vaso-spastic. He pointed out that allergic manifestations in man are the result of capillary dilatation and increased permeability, and that they are paroxysmal. He suggested the use of an intradermal histamine skin test as a general indicator of sensitivity, and claimed that seven histamine-sensitive patients with Ménière's disease had been entirely relieved of their symptoms over many months by histamine desensitization.

Atkinson proceeded to investigate the histamine insensitive patients in his series. He found that he was able to produce nystagmus by hypodermic injection of Adrenaline, and to abort the vertiginous attacks by hypodermic Acetyl Choline. He treated nineteen cases with the vaso-dilator drugs Nicotinic Acid and Thiamine Hydrochloride and claimed that seventeen had been completely relieved or much improved, and two showed early improvement with subsequent relapse. He pointed out that there is a group of patients in the vaso-constrictor series who respond
poorly to treatment. In them the age of incidence is markedly lower and the course of the disease much more acute. These he compared to Reynaud's Disease in the severity of their symptoms and the slightness of their response to the vaso-dilator drugs.

Shelden and Horton reported in 1940 on the dramatic success of intravenous histamine in the treatment of acute attacks of Ménière's Disease in eleven cases. Atkinson criticised these results in a subsequent paper, claiming that the cases must have been vaso-spastic in origin and that once the temporary vaso-dilatation caused by the histamine had passed off the end result was likely to be an increased severity of the attacks owing to a raised blood histamine esterase as a response to the treatment. One of the eleven cases had been seen by him subsequently and he had great difficulty in restoring her to a state of equilibrium by massive dosage with Nicotinic Acid.

Wright believes that idiopathic Ménière's Syndrome is a result of a focal labyrinthitis secondary to sepsis, and that eradication of sepsis will arrest the condition. In his first series Wright claimed 88% of cures following removal of septic foci. However, in a recent paper Wright has reported 60 cases of alcohol injection of the labyrinth, with relief of symptoms, where previous elimination of sepsis had failed to cure.
SURGICAL TREATMENT.

In cases which do not respond to medical treatment and have symptoms of a severity demanding relief, recourse may be had to operative treatment. The choice of operation must depend to a great extent on the individual case.

If there remains a labyrinth which has useful function on one side only, and this is becoming progressively worse, the operation of choice would appear to be Portmann's trans-mastoid drainage of the saccus endolymphaticus. In good hands this operation produces brilliant functional results, but it is technically rather difficult and many surgeons doubt whether the improvement is likely to be maintained indefinitely. Hallpike has shown that rupture of the distended endolymph membranes does occur and apparently is followed by healing.

The further choice of operative procedure lies between an attempt to destroy the peripheral labyrinth or partial or complete division of the eighth nerve.

Mollison reported in 1939 his results in a series of 50 cases treated by alcohol injection into the labyrinth. He exposed the lateral semi-circular canal by a simple antrotomy and opened it by a stroke of the chisel. A few minims of absolute alcohol were then injected into the ampullary end of the canal.
There was one post-operative death in the series which was due to nephritis.

Of the remainder 74% claimed that their vertigo was cured and that they were fit for work: 10% were not improved, and 16% were improved but not cured, or else untraced.

Wright in 1944 gives an account of 60 cases in which elimination of septic foci had failed to cure the symptoms, or had not been indicated. He devised a technique for injection of alcohol through the tympanic membrane and the footplate of the stapes. Two minims of absolute alcohol were injected. This method involves the minimum of trauma to the tissues, can be quickly executed and requires only a short, light anaesthesia. It is thus suitable for aged and debilitated patients.

There were no fatalities in the series. In 55 cases the vertigo was improved. In 5 cases there was no improvement although the injection had been followed by a brisk labyrinthine response. The tinnitus was improved in 33, not improved in 16 and unrecorded in 9.

Frazier in 1908 divided the eighth nerve for intractable vertigo. The patient survived, but a year later was said to be not much improved as to his symptoms.

Since that time division of the eighth nerve has been regularly practised by the neuro-surgeons in the treatment of
labyrinthine vertigo. In cases where it is desired to preserve the hearing the vestibular portion of the nerve is isolated at the internal auditory meatus and divided. There is always a chance that some vestibular fibres may have been left attached to the cochlear nerve, so when the hearing is not worth preserving the entire nerve is divided.

Crowe published in 1938 the results of an examination of 72 patients who had had a sub-total section of the eighth nerve by Dandy. The average time since operation was 2.2 years. He found the hearing was worse in 30.5%, unchanged in 50% and improved in 19.5%.

Walsh and Adson reported in 1940 on a series of 20 cases which had come to operation as a result of the failure of medical treatment. Thirteen cases had complete unilateral section of the eighth nerve. Of these, nine were completely relieved of vertigo, three were much improved and one was not relieved. The tinnitus disappeared in two cases, was greatly relieved in two, unaffected in seven and worse in two. Seven patients had a subtotal section of the eighth nerve, four were completely cured of vertigo, three were greatly improved and the hearing was maintained in the latter three cases.

As far as can be judged there is no significant difference in the end results of alcohol injection as compared to complete
section of the auditory nerve. In both cases there is a significant percentage of patients who continue to have vertigo, and in the majority the tinnitus is not completely cured. It is possible in these cases that the remaining labyrinth may be the source of the symptoms.

Partial section of the eighth nerve, however, gives a good chance that the hearing may not be lost, and so in certain cases it is the procedure of choice.

**SUMMARY.**

A fresh translation has been made from Ménière's original papers and a selection taken from the literature showing the progress that has been made in the investigation and treatment of the condition he described.

Ménière's definition of a disease of the internal auditory apparatus associated with paroxysmal vertigo and tinnitus progressing to a variable deafness still remains valid.

Histological examination of the labyrinth has shown conclusively that there is a general dilatation of the endolymph system with a degeneration of the sensory epithelium. The presence of central changes is shown by the failure of a percentage of cases to respond to section of the eighth nerve.
Observation of the course of the disease has shown that it is subject to remissions and in some cases spontaneous cure.

The majority of cases can be controlled by simple medical treatment such as the elimination of sepsis and regulation of the fluid metabolism.

In some cases there has been proved an allergic or vaso-motor basis to the condition.

Cases resistant to medical treatment may be relieved by destruction of the labyrinth by alcohol injection, or by section of the eighth nerve, or its vestibular branch.

There remain a very few patients who are not benefited by the most drastic surgical treatment because of some unknown central degenerative changes.
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