Section of Otology

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MÉNIÈRE'S DISEASE

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INTRODUCTION

After we have learned to walk we expect to be able to move freely in an environment which is solid and stable. Though we may voluntarily give up this comfortable state, as when we travel by sea or by air, at least we know what to expect. For the patient with Ménière's disease this trust in the behaviour of his surroundings no longer exists. From time to time he has experienced the sensation of movement of his environment or of himself when reason tells him that they should be still. His feeling of security is further undermined by nausea and vomiting, and in addition his distress is increased by deafness and tinnitus. It is not surprising that these symptoms often suggest a widespread disorder involving at least the nervous and digestive systems, but it is now generally agreed that in almost every case the disorder has its source in the internal ear.

Although the syndrome which we now recognize as that of acute labyrinthine failure had aroused the interest of many workers, it was not until 1861 that Prosper Ménière of Paris published a series of papers showing that a disordered labyrinth could cause paroxysmal attacks of vertigo and vomiting associated with unilateral deafness and tinnitus.

Ménière had passed an interesting and not uneventful life during which he had been physician-accoucheur to the wife of the Pretender to the French throne, a friend of Balzac's, and superintendent of a Deaf and Dumb Institution (Wells, 1947), but it was not until the last year of his life that he announced the discovery which was to earn him fame.

After this discovery it became the custom to attach the label "Ménière's syndrome" to cases of paroxysmal vertigo, with or without deafness and tinnitus, no matter what the cause; while in more recent times bouts of vertigo without deafness have earned the sobriquet "pseudo-Ménière's syndrome". Despite this general appreciation of Ménière's discovery the labyrinth guarded its secrets well. More than three-quarters of a century passed before Hallpike and Cairns in 1938 were able to demonstrate a deformity of the endolymphatic system of the labyrinth on the affected side in certain sufferers from what was now called Ménière's disease. They concluded that this appearance was the result of recurrent distension of the endolymphatic system caused by a failure of the mechanism governing the production and disposal of endolymph. They felt that as an anatomical basis for the disorder had been demonstrated it was appropriate to use the term Ménière's disease instead of Ménière's syndrome. Since then several other workers have confirmed their findings and the descriptive subtitle of endolymphatic hydrops is now often used.

There are, of course, other labyrinthine disorders, with vertigo as their principal symptom, which may mimic Ménière's disease but we have found that endolymphatic hydrops is responsible for most cases of aural vertigo.

That the ear was often responsible for vertigo was clearly appreciated by Sir William Gowers, who in 1893, when writing of his experience at the National Hospital for Nervous Diseases, said: "Of 106 consecutive cases in which definite vertigo made the patient seek advice, in no less than 94 ear symptoms were present.... It is exceedingly rare for definite vertigo to occur apart from aural symptoms, and it is certain that in the majority of cases in which vertigo has been ascribed to other causes, these have only had an exciting influence, and the symptom has been essentially due to the effect of unobtrusive labyrinthine disease."

Our experience has been the same as that of Sir William Gowers and in Table I it will be seen that out of 1,902 cases of vertigo referred to us in the course of the past ten years, 1,169 or 61% were due to Ménière's disease, and altogether 87% were due to a peripheral labyrinthine lesion.

Although it may be that there are more cases of central vertigo than Table I suggests, it is interesting to note that the proportion of central to peripheral causes where vertigo is the predominating symptom is almost the same in this group as in that of Sir William Gowers.

The central causes include epilepsy, disseminated sclerosis, certain temporal lobe tumours, some tumours in the posterior cranial fossa usually of the cerebellopontine angle or in the mid-line where the vestibular connexions in the cerebellum are involved, and thrombosis of the posterior inferior cerebellar artery. Migraine has also been considered as a cause.

TABLE I.—CAUSES OF VERTIGO IN A SERIES OF 1,902 CASES

Central		130
Peripheral		1,657
Ménière's disease	1,169	
Trauma	137	
Vestibular neuronitis	152	
Positional	130	
Infective	69	
Not classified		115

In the peripheral groups under the heading of trauma are included patients with persistent vertigo following head injuries, and for a more detailed consideration of this group *see* Cawthorne and Cooksey (1946).

Vestibular neuronitis is a condition in which patients suffer a sudden loss of vestibular function, as a rule on one side only, but without any impairment of hearing. Dix and Hallpike (1952) who first used the term vestibular neuronitis, believe that the lesion lies somewhere in the vestibular neurones, and it may be situated either in Scarpa's ganglion or more centrally. It may be associated with a focus of sepsis or may accompany a respiratory infection, though in many cases there is no obvious causal factor. This may well be the condition which is known as "epidemic labyrinthitis".

Paroxysmal positional vertigo can be due either to a lesion in the utricle as shown by Dix and Hallpike (1952), or it may accompany lesions involving the vestibular connexions in the cerebellum. In the peripheral cases the vertigo and nystagmus appear some seconds after the head has been placed in a certain position, usually backwards, and to one side. The vertigo and nystagmus last a few seconds—rarely more than ten—can be fatigued easily by repetition and do not reappear for at least half an hour even when the offending position is resumed. It may sometimes follow a head injury. In the central cases, the vertigo and nystagmus are not fatigable and the direction of the nystagmus may vary with the position of the head. In some of the central cases the sensation of vertigo is slight but on the other hand we have seen a few cases in which the vertigo was intense and was accompanied by vomiting.

Further information about these two interesting groups of aural vertigo which are sometimes confused with Ménière's disease can be obtained from an outstanding paper by Dix and Hallpike (1952).

The infective group includes patients in whom past or present middle-ear infection has resulted in an erosion of the bony labyrinth or where an opening has been accidentally made in the course of an operation for infection.

There is an interesting little group of patients in this infective series who exhibit what appears to be a functional disturbance of balance often with a bizarre gait. The connexion between the labyrinth and the clinical picture may not be obvious and in fact is not infrequently overlooked. There may have been a former mastoid operation now well healed, but for some reason or another there is a slight but continuing fluctuation of vestibular activity. This may be due to a fistula in the bony labyrinth, and there may be depression of vestibular function of the affected side though it is never absent. We have been in the habit of using the term perilabyrinthitis for this group of cases. By destroying the offending labyrinth, we have been able to restore to an active life several patients who had been literally crippled by this complaint. In this connexion we must mention the effect of recurrent bouts of vertigo upon the psychological state of the sufferer. In many patients repeated attacks of vertigo for which no cause is readily demonstrated can easily induce a feeling of insecurity which may masquerade as a psychological disorder. In such patients it is important not to fall into the trap—not infrequently set by the patient or his relatives—of attributing the "attacks" to "nerves" when in reality the "nerves" are due to the "attacks".

Now that we have referred to the problem of aural vertigo in general we would like to enquire a little more fully into the incidence, and clinical features of Ménière's disease, and then to consider the possible underlying causes of the disorder; and finally to discuss the various forms of treatment.

CLINICAL FEATURES

General incidence.—There are many and varied views on the incidence of Ménière's disease, and our own impression has been that it is not so rare as is sometimes believed; though of course we have always appreciated that our experience would not give a true impression of the incidence.

There is, however, now to hand a most informative monograph by the General Register Office on General Practitioners' Records (Logan, 1953). This is an analysis of the clinical records of eight practices with a population of 27,365 persons made during the year ending March 1952.

From this we have chosen five disorders including Ménière's disease which are comparable in frequency (Table II).

Thus it will be seen that while it is not among the commoner group of diseases, it is encountered and diagnosed more frequently than might be expected from the attention given to it in the past. It is now grouped by the General Register Office under "Diseases of the Ear".

There is a slight preponderance of males over females in about the same proportion as was noted in a previous review of 424 cases (Cawthorne, 1947), most of which are included in the present series (Table III). This suggests that, although the preponderance of males is slight, it is consistent. However, women at the menopause tend to attribute symptoms of giddiness to "the change", and therefore they may not always seek advice. As a result of this attitude the predominance of males may be unreal.

TABLE II.—DISORDERS OF COMPARABLE FREQUENCY WITH MÉNIÈRE'S DISEASE IN 27,365 GENERAL PRACTICE PATIENTS DURING ONE YEAR

Scarlet fever	45
Ménière's disease	43
Hyperplasia of prostate	27
Thyrotoxicosis	26
Nephritis and nephrosis	19

TABLE III.—SEX INCIDENCE IN 900 CASES OF MÉNIÈRE'S DISEASE

Male	489
Female	411

Table IV shows quite definitely that the majority of patients (65%) have their first attack before the age of 50.

The disease does not favour one ear more than the other, and in 13% both ears were affected (Table V). It was interesting to find that in half of this bilateral group both ears were obviously affected from the onset of the disorder. In the other half, the second ear was not affected until some years after the first. In one patient the interval was thirty-seven years.

TABLE IV.—AGE AT ONSET OF FIRST SYMPTOM IN 900 CASES OF MÉNIÈRE'S DISEASE

Age group	Up to 25 years	26–50 years	51–75 years	over 75 years
Number of patients	63	521	307	9

TABLE V.—LATERALITY IN 900 CASES OF MÉNIÈRE'S DISEASE

Left ear	408
Right ear	371
Both ears	117
No record	4

It has been held by some that the deafness usually precedes the bouts of vertigo, while others have postulated a different disease when the vertigo precedes the deafness. We do not attach any significance to the order of appearance of the symptoms and, as Table VI reveals, we found that the commonest state of affairs was for the deafness to be noticed simultaneously with the original bout of vertigo; though in the event of one symptom appearing before the other, deafness was the more likely to be first.

TABLE VI.—ORIGINAL SYMPTOM IN 900 CASES OF MÉNIÈRE'S DISEASE

Deafness first	237
Vertigo first	173
Deafness and vertigo simultaneously	449
No record	41

TABLE VII.—OTHER SYMPTOMS IN 900 CASES OF MÉNIÈRE'S DISEASE

Unconsciousness	30 cases
Diplopia	9 cases
Allergy	41 cases
Former head injury	29 cases

Other symptoms are relatively infrequent in Ménière's disease, and in this series a careful note was made of any unconsciousness, diplopia, allergy or head injury (Table VII). Despite the severity of what might be termed the vagal stimulus, genuine loss of consciousness is very unusual in Ménière's disease. In this series it was noted only in just over 3% of cases; but in all of these, the loss of consciousness was only momentary. Anything more than a fleeting loss of consciousness should always lead to an enquiry into the possibility of epilepsy being present. Diplopia is rare, and again is only transitory, unless the real cause of the attack is disseminated sclerosis.

As an allergic basis has been held responsible for the attacks, and as in a recent monograph Williams (1952) has argued strongly in support of this, it may be worth while going into the matter in some detail. An enquiry was always made by us into the presence of any allergic symptoms such as hay fever, rhinorrhœa, asthma, eczema, or colitis. That such a small proportion—under 5%—have other allergic symptoms is not in favour of this ætiology. Though in individual patients the allergic response tends to be localized in a particular organ, an exceptional dosage of antigen is likely to call forth a response from other organs, and this surely would be expected to occur during severe bouts of Ménière's disease. Some 50% of allergic patients have a positive family history of allergy as compared with 7% of normal individuals. Although no specific note was made of the family history we can call to mind only 4 cases of Ménière's disease in which there was a history of a similar disorder in the family. Lastly, the age of onset of allergic symptoms differs from that of Ménière's disease, being less than 40 years in about 80% of cases while that in Ménière's disease,

in our series, averaged 44 years. In this connexion the state of affairs in migraine, a condition in which an allergic basis seems to have been established, may be noted. Here the age of onset is usually in the first two decades, there is a strong family history, and the attacks tend to die out before the age of 60 years.

An association between Ménière's disease and head injury has been suggested, but here again the number of cases in which a head injury might have played a part is probably not more than might be accounted for by the average incidence in the general population, though it is only fair to say that in at least 3 cases, the symptoms first appeared soon after a head injury.

When we consider the cochlear symptoms more closely (Table VIII), it will be seen that nearly all have some degree of deafness, whilst the majority have tinnitus as well. In a bilateral case, tinnitus is often an important indication as to which ear is in the active stage of the disease.

TABLE VIII.—COCHLEAR SYMPTOMS IN 900 CASES OF MÉNIÈRE'S DISEASE

Deafness	859
Tinnitus	764
Distortion of hearing	402

It is quite common to be told that warning of an impending attack is given by an increase or alteration in the tinnitus. Some patients find that just before an attack their constant tinnitus has added to it a clanging machinery-like noise which may prove very distressing.

Distortion of hearing is a valuable distinguishing feature of endolymphatic hydrops which has been noted in nearly half the patients in this series. It is probable that the incidence of such distortion of hearing is higher than the figure just given, as the significance of this feature of the deafness was not always appreciated in the earlier cases. The distortion is usually noticed for loud, shrill and musical sounds, and it is not uncommon to be told by a patient that, previous to the onset of the disease, he enjoyed listening to music, but that during its active phase he finds it impossible to listen any longer to music on the wireless because of the jarring and discordant effect on his ear. To our knowledge, the only other disorder which may cause distortion of hearing is acute acoustic trauma during the stage of recovery. This distortion may seriously impair hearing so that, after labyrinthectomy, in which the distorted remnant of hearing in the affected ear is destroyed, the patient may volunteer that his hearing is better, and that he can enjoy music and other sounds again. A serious effect of this distortion is to reduce the ability to hear speech much more than the ability to hear pure tones. This is well shown in Fig. 1, in which it will be seen that, although there seems to be a potentially useful amount of hearing as measured by the pure-tone audiometer, a speech audiogram reveals that such hearing as remains is of little use for understanding speech.

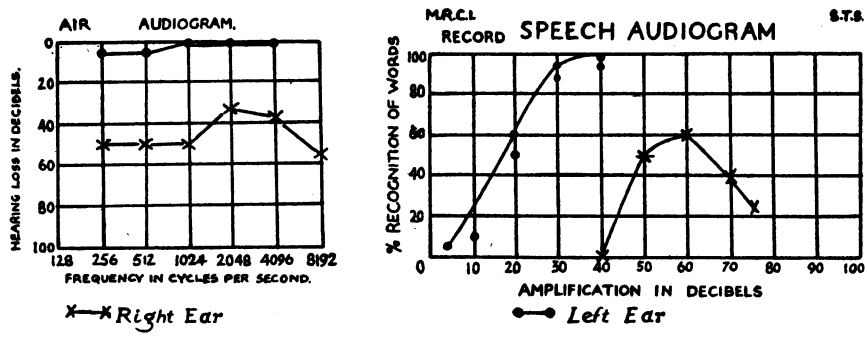

FIG. 1.—Hearing for speech and for pure tones in a case of Ménière's disease. (Reproduced by permission, Cawthorne, T., 1954. In: Modern Trends in Diseases of the Ear, Nose and Throat. Editor: Maxwell Ellis. Butterworth and Co. Ltd.; p. 31.)

The pure-tone audiogram shows the threshold hearing for certain pure tones, while the speech audiogram measures the percentage of words repeated correctly against the loudness of the speech. Normally and in many forms of deafness the louder the speech the better it is understood, but in Ménière's disease there may be but little difference in loudness between just hearing speech and finding it too loud for clarity or comfort.

We regard distortion of hearing as a valuable clue as to the nature of the deafness; and in a certain proportion of cases in which the vestibular signs are not prominent, it is possible to make a diagnosis of endolymphatic hydrops on the cochlear symptoms and signs alone.

The deafness in Ménière's disease is, as would be expected from what we know of the pathology, perceptive, and characteristically affects the lower tones. In the few cases in which there was an

overlying conductive deafness there was also an underlying internal ear condition which was responsible for the attacks.

It is noteworthy that in only one case out of 900 was there clear evidence of a blocked eustachian tube; though we would not deny that this may be occasionally associated with giddiness of mild degree.

Associated ear diseases include congenital deafness and mumps deafness. Although the number of patients in whom Ménière's disease developed in an ear previously affected by mumps is small, there may be a connexion because in 2 cases the attacks followed soon after the mumps neuro-labyrinthitis.

In addition, mention must be made of the possibility of hydrops developing after the fenestration operation. Shambaugh (1949) has noticed this in a definite proportion of cases, and we have seen it, too, in a small number of patients. In most of such cases, however, the cochlear labyrinth has been mainly affected, and apart from slight dizziness the vestibular symptoms have, fortunately, rarely been pronounced.

A test of vestibular function should always be carried out, as it may give the only physical sign of a disordered vestibular labyrinth. It occasionally happens that the vestibular abnormality is found in the better hearing ear. In such a case further careful enquiry usually brings to light former attacks and bilateral disease is assumed.

We employ the caloric test according to the technique developed at the National Hospital for Nervous Diseases, by Fitzgerald and Hallpike (1942) and the results in Ménière's disease were at that time given by Cawthorne, Fitzgerald and Hallpike (1942). Further experience shows that a reduced response to caloric stimulation on the affected side is the usual finding (Table IX).

TABLE IX.—CALORIC TEST IN 900 CASES OF MÉNIÈRE'S DISEASE

Canal paresis	640
Directional preponderance	75
Combined canal paresis and directional preponderance	108
Normal	51
No record	26

PATHOLOGY

The distension of the endolymphatic system which is the visible relic of Ménière's disease is the result of recurrent bouts of endolymphatic hydrops. Let us for a moment inquire into the natural history of the disease, and see how this can be compared with what we believe may be going on inside the labyrinth. The attacks tend to appear in groups, with intervals of complete freedom. During what may be called the active phase of the disease, when the attacks are likely to take place, there is often a feeling of pressure in the affected ear, and patients may declare that the ear is blocked, or that it feels as though it was "blown up". This blocked feeling, particularly when it is combined on testing with a false negative Rinne may account for the former belief that a blocked eustachian tube was commonly associated with Ménière's disease. There may be increased deafness and tinnitus, and a general feeling of unsteadiness. These symptoms may build up in a crescendo, ending in an attack of vertigo. When the attack subsides the premonitory symptoms may also disappear. Sometimes, however, a residue of symptoms remains over a period of days, weeks, or even months, and during this time a succession of attacks may be experienced, until quite suddenly one day the ear clears and a quiet phase is entered upon. During the quiet phase, the sensation in the ear, the cochlear symptoms, and the unsteadiness are reduced or absent. We interpret this as follows:

In the active phase, there may be a disturbance of the normal balance between the production and disposal of the endolymph which leads to a rise in endolymphatic pressure. At any time during this period there may be a critical increase in the endolymphatic pressure, which if sufficient to squeeze the blood out of the capillaries, will result in an attack, as suggested by Hallpike and Cairns (1938). This obliteration of the capillaries may, at the same time, hinder the production of more endolymph so that the pressure subsides, circulation returns, the attack passes, and function is restored to the labyrinth. So long as the abnormal state responsible for the disturbance of balance between the production and absorption of endolymph continues, the endolymphatic pressure is liable to be kept above normal. During this active phase, the labyrinth is sensitive to any increase in endolymphatic pressure, as Mygind and Dederding (1932) found when they drew attention to the apparent disturbance of water metabolism in sufferers from Ménière's disease. We have also found that during an active phase the patient is very sensitive to anything which tends to favour the retention of fluid within the body (Cawthorne and Fawcett, 1938). During the quiet phase, the normal balance between the production and disposal of endolymph is restored, so that the labyrinth is no longer at the mercy of any factor liable to affect the pressure within it, and apart from any residual deafness and tinnitus the patient feels well.

There is no rule or regularity about these phases. Sometimes the active phase only lasts for the period of a single attack, while at other times it may extend over weeks or months and include several attacks.

A question which has been asked is: Why is distension of the membranous semicircular canal not found when gross dilatation of the scala media and saccule, and to a lesser extent of the utricle, is usual? The answer given by Hallpike and Cairns was that the membrane of the canals was much thicker than that of most endolymphatic structures. In some, rupture of the scala media may occur with relief of pressure but without recovery of function. Such an effect could account for those cases in which a single apparently typical Ménière's attack leads to permanent loss of hearing and vestibular responses.

In the foregoing account of the mechanics of Ménière's disease, we have been relying on Guild's (1927) theory of the formation, circulation and removal of endolymph. So long as we continue to accept this theory, then it would seem that some hindrance to the disposal of the endolymph through the wall of the saccus endolymphaticus must be the most likely reason for the hydrops. However, the work of Lindsay (1947, 1952) suggests that in animals the saccus endolymphaticus can be obliterated without the subsequent development of hydrops, so there must be other routes for the absorption of endolymph.

That ischæmia of regions of the labyrinth is caused by spasm of the internal auditory artery or of its branches, is the contention of those who attribute the disease to allergy. They suggest that as a result of ischæmia damage is done to vessel walls and this leads to transudation of an endolymph of altered character and higher osmotic pressure. In consequence, dialysis of fluid through Reissner's membrane occurs and produces distension of the endolymphatic system.

This hypothesis would fit the histological findings but it would certainly be strange if the vasospasm so persistently favoured one group of vessels, unless there were some anatomical predisposition. Even in migraine in which vasospasm has been proved the attacks show little lateralization, either side being affected at different times.

Much has grown out of the original observation of Sheldon and Horton (1940) that histamine has a favourable effect on the progress of the disease. This was attributed by some to its effect in allergy, and by others to the fact that it caused peripheral vasodilatation, and so increased the circulation within the internal ear. Other vasodilating drugs, such as nicotinic acid and procaine have been used, and Miles Atkinson (1945, 1946, 1949) and Hilger (1950) have advanced attractive and ingenious hypotheses as to how these drugs may act. Before, however, accepting these views, should we not enquire a little more closely into this matter of vasodilatation of the labyrinthine blood vessels? First of all, we are asked to assume that vasodilatation of the labyrinthine arteries will reduce, or in some way disperse, the fluid hydrops in the labyrinth; and then we are asked to assume that certain drugs do indeed produce vasodilatation of the labyrinthine vessels. If, however, endolymph is secreted by the stria vascularis of the cochlea, then surely the physiological response to any increase of blood flow through the secreting organ should be an increase in the amount of endolymph produced.

There is evidence which suggests that the nervous control of intracranial blood vessels differs from that of the rest of the body (Schmidt, 1952), and it has been shown that peripheral vasodilators have a much less noticeable effect on the intracranial blood flow than on the peripheral circulation. In fact, it seems as though CO_2 is the most, if not the only, certain and potent vasodilator of intracranial blood vessels, and we have observed at operations a dilatation of the blood vessels within the bony labyrinth following the inhalation of CO_2. Mygind and Falbe-Hansen (1951) found that in the guinea-pig adrenaline caused a vasodilatation, and histamine a vasoconstriction, of the labyrinthine blood vessels. If this observation is applicable to man, then the influence that histamine sometimes has on Ménière's disease might be explained by vasoconstriction, and not by vasodilatation.

Following up an observation of Hallpike and Cairns (1938), Lempert and his associates (1952) were impressed by the frequent presence of vesicles in the wall of the membranous semicircular canals in material from cases of Ménière's disease. They suggested that these blebs might be due to "a chronic herpetic neuritis of the vestibular labyrinth of toxic or trophic origin". That similar appearances are present in the canals of many labyrinths from those over the age of 40, who have had no symptoms of Ménière's disease, together with the absence of pathological changes in the neurones, is not in favour of this ingenious hypothesis. Recent work at the Lempert Institute (Rambo, *et al.*, 1953), shows that neither the severance of the vasodilator nor yet of the vasoconstrictor nerves to the ear in monkeys has any lasting effect on the blood vessels of the labyrinth. All this suggests that much more work needs to be done before we can accept without question, hypotheses concerning the effect of altering the blood flow through the labyrinth in Ménière's disease. Thanks to Hallpike and Cairns we know the effect on the delicate endolymphatic structures of attacks of Ménière's disease, but of the events which lead up to such attacks we are still ignorant.

Treatment

Treatment may be considered under the headings of sedation, anti-retentional regime, measures to encourage vasodilation of the labyrinthine arteries, removal of foci of sepsis, drainage of the labyrinth and finally destructive procedures.

(*a*) *Sedation.*—All will be agreed that sedation, either by the barbiturates or by certain of the antihistaminics, of which Dramamine and Avomine seem to be the most effective, is a necessary part of the treatment which, though it may not prevent attacks, will often reduce their severity.

(*b*) *Anti-retentional regime.*—During the active phase of the disease, we have often found it to be of great help to reduce the intake of salt and fluid. Strict adherence to a low sodium diet often discourages attacks though some patients find the regime tiresome.

(*c*) *Vasodilatation.*—This is sought either by drugs, such as histamine, procaine and nicotinic acid, or by paralysing the cervical sympathetic nerve (Passe and Seymour, 1948). We have tried repeated injections of the stellate ganglion on the affected side as reported on by Hoogland (1951) using 2% xylocaine. This is a simple procedure which rarely fails to produce a full Horner's syndrome with suffusion of the eyeball and dilatation of the vessels of the tympanic membrane and of other branches of the external carotid. We have not, however, observed in such cases any change in the retinal blood vessels nor of the vessels within the labyrinth at operation. Apart from a transient alteration of the tinnitus in some of the patients we have not noted any definite and lasting benefit as the result of injecting the stellate ganglion. With regard to surgical interruption of the sympathetic fibres going to the internal ear, Mr. Roland Lewis has kindly operated on some of our cases and he tells us that in half of these the attacks of vertigo have either ceased or have been reduced. Though the hearing for pure tones has not improved, the distortion of hearing has been reduced in about one-third of the cases. Tinnitus was relieved immediately in most, and this reduction in the tinnitus was maintained in about one-third. Thus it seems that in most of the cases something happens within the labyrinth as the result of sympathectomy, though the proportion of cases in which there is sustained relief of symptoms is not sufficiently high to make it certain that such relief is not part of a natural remission.

We have had 3 patients referred from elsewhere because of severe vertigo unaffected by sympathectomy for whom labyrinthectomy was needed. Our feeling at present is that until we know more about the effects of sympathectomy upon the labyrinth and upon the patient generally, we must continue to regard it as an experimental procedure, and not by any means a minor one.

In this connexion we must mention section of the chorda tympani for the relief of Ménière's disease. Rosen (1949) has repeatedly advocated this procedure though it is still not clear to us how and why it should help Ménière's disease, and we feel that even more than sympathectomy this should be regarded for the present as an experimental operation. As regards vasodilating drugs, we have not found, except for histamine, which does sometimes seem to have a favourable effect upon the disease, any other of these measures of definite or lasting help. We have never been able to establish any connexion between Ménière's disease and foci of sepsis, though we would add that in certain allied conditions such as vestibular neuronitis and positional vertigo focal sepsis does seem to play a part as Wright (1937) pointed out.

(*d*) *Destructive procedure.*—(i) *Streptomycin*: The vestibulo-toxic properties of streptomycin have been used in Ménière's disease to abolish vestibular function (Glorig and Fowler, Hamberger, Ruedi, Cawthorne). Unfortunately vestibular function on both sides is affected by the drug and if function is lost in both it may well, in older patients, result in a greater incapacity than the disease for which it is given. We therefore feel that if it is to be used at all it should be limited to young people with bilateral disease; an unusual combination.

(ii) *Drainage of the saccus endolymphaticus*: The logical surgical treatment would seem to be the drainage of the saccus endolymphaticus, as first described by Portman (1927). If it can be identified, incision of the saccus may, it is true, allow an excess of endolymph to escape. But in the process of healing there is likely to be some fibrosis in the wall of the saccus which may even further hinder the disposal of surplus endolymph.

(iii) *Destruction of the labyrinth*: The labyrinth can be destroyed either by introducing into it powerful agents of tissue destruction such as alcohol or diathermy, or by simply removing a portion of the membranous labyrinth, usually, for convenience sake, the membranous external semicircular canal.

There is no doubt that alcohol, as first suggested by Mollison (1931) and diathermy, by Day (1943) are effective agents of destruction, but at times they are too effective—as when their destructive properties are allowed to extend beyond the confines of the labyrinth, causing facial palsy. We prefer to abolish labyrinthine function in Ménière's disease by the simple removal of a piece of endolymphatic labyrinth in the manner first described before this Section in 1943 (Cawthorne, 1943). This operation is simple and safe and quite within the competence of any aural surgeon equipped with adequate magnification and a pair of fine "hair-spring" forceps. In Ménière's disease this operation always results in complete and irreversible loss of both cochlear and vestibular function, though this does not apply necessarily to other disorders (Cawthorne, 1949).

(iv) *Nerve section*: Section of the vestibular portion of the acoustic nerve has the advantage that the hearing may be spared, but it is an intracranial operation which is not without risk. We think it should only be advised when the hearing on the affected side is remarkably good.

In many cases the hearing is badly distorted, so that for speech it is useless, and there seems to be no object served in embarking on a serious operation merely to preserve a distorted remnant of hearing.

Table X gives the results of the membranous labyrinthectomy which we practise.

TABLE X.—MEMBRANOUS LABYRINTHECTOMY FOR MÉNIÈRE'S DISEASE

Total operations	288
Healed by first intention	284
Post-operative infection	4
Transient facial paresis	1

This has been carried out now in 288 cases, and of these 284 healed by first intention, and there was only a slight post-operative infection in 4, which soon yielded to appropriate antibiotic treatment. In one recent case there was a transient facial weakness but otherwise the post-operative course has been quite uneventful, and in none of these cases did the patient ever get another attack of vertigo from the operated labyrinth. In 6% of the patients, however, the other ear subsequently became affected.

CONCLUSION

Ménière's disease, despite its widespread symptoms, is really an otological disorder. The internal ear is at fault, and proof of the true nature of the disease is only forthcoming after a detailed otological examination. The effect of conservative treatment is difficult to assess because of the natural tendency of the disease towards spontaneous remissions. In stubborn unilateral cases, destruction of the affected labyrinth by the simplest and least harmful method at our disposal is the treatment of choice; and in our experience this has been necessary in just over a quarter of the cases we have seen.

Though we still do not know the cause of Ménière's disease, we are now well aware of its effects upon the labyrinth. Despite the cloud of pessimism which surrounds the treatment, we have found that in the majority of the patients who have come under our care it has been possible by one means or another to prevent or subdue the attacks.

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Mr. Miles Foxen: *The Use of Streptomycin in Ménière's Disease*

Very many of the victims of Ménière's disease are vastly improved, if not entirely relieved of their symptoms by non-surgical methods of treatment (Discussion, 1950).

Some cases remain which do not respond to simple methods such as vasodilatation, or histamine desensitization, and, of these, a small number are so obviously bilateral as to preclude labyrinthotomy. It is in this group that certain cases suitable for streptomycin treatment are found, and it will be evident that these cases are few and far between. Indeed it would be most imprudent to apply this method of treatment to cases of Ménière's disease which might respond to less dangerous methods.

As far as can be ascertained it was first suggested by Hawkins of the Merk Institute that the toxic action of streptomycin might be utilized in the treatment of aural vertigo, and in 1948 Fowler reported 4 cases which he had treated during the previous year. He used a dosage rate of 4 grammes *per diem* until the total dose of 25 to 33 grammes had been given, and he was well satisfied with the results.

In 1949 Hamberger, Hydén and Koch reported a further series of 4 cases using a dosage rate of 3 grammes *per diem* until a total dose of 90 grammes had been given. 2 of the cases were reported as being successful. In 1951 the subject was reviewed by Ruedi who added a further series of 3 cases, and again in 1951 Hanson published 5 cases. In the latter series streptomycin was given at the rate of only 2 grammes *per diem* until total doses varying from 40 grammes to 76 grammes had been administered and improvement was recorded in every case. At a meeting of this Section in May 1952 (Discussion, 1952) the toxic effects of streptomycin and dihydrostreptomycin on the acoustic and vestibular systems were discussed. Mr. Terence Cawthorne spoke of the use of streptomycin in Ménière's disease, drawing attention to the fact that the toxicity of different batches of streptomycin might vary. In our own literature I have been unable to discover any case reports though I am aware that a number of British otologists have used this method.

The case to be described was treated three and a half years ago and has remained symptom free. At the age of 17 she underwent left radical mastoidectomy and twenty years later, in 1945, commenced to suffer from vertiginous attacks.

These increased in frequency and severity, and concurrently the hearing deteriorated in both ears.

In 1948, she first attended hospital, but simple sedation and treatment along general medical lines was of no avail. In the following year therapy of a more specific nature was instituted, and included the use of nicotinic acid combined with salt deprivation and fluid limitation, a method of treatment which has been of tremendous value in most of our cases.

The patient was improved for a few months, but the attacks recurred with such severity that she was totally incapacitated, whereupon she was readmitted to hospital, and treatment with streptomycin (calcium chloride complex) was instituted. For the first fifteen days the drug was given at the rate of 2 grammes *per diem* in divided doses 6-hourly and for the remainder of the period at the rate of 2·5 grammes *per diem* until a total of 53·25 grammes had been administered. By that time caloric reactions had been abolished on the right side and very considerably reduced on the left, the side, in fact, of a radical mastoid cavity.

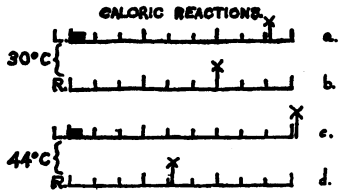

FIG. 1.—Prior to treatment with streptomycin. In tests *a* and *c* the stimulus was applied for 10 seconds only, but was followed by vomiting.

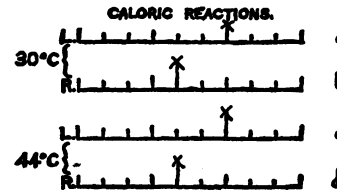

FIG. 2.—After 25 grammes of streptomycin. Tests *a* and *c* were accompanied by nausea.

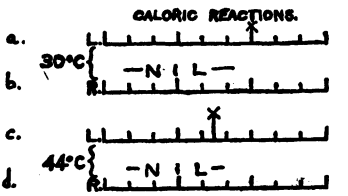

FIG. 3.—After 53·25 grammes of streptomycin. The stimulus was applied for 40 seconds in each case, but no nausea was experienced.

Audiometry was carried out at intervals and showed no obvious cochlear deterioration as a result of treatment.

Cooksey exercises were commenced early in treatment but had to be abandoned for a time, as the patient had a number of minor attacks of vertigo and was generally unwell after the second week. Physiotherapy was, however, recommenced and was continued in all for nine months. Owing to her inability, at first, to go out in the dark she did not commence work for six months after treatment, but since that time, three years ago, she has pursued her normal occupation—clerical work; has had no untoward symptoms and is extremely grateful.

SUMMARY

The two essential requirements in any particular case are: firstly that the possibility of relief with all other more conservative methods of treatment should have been thoroughly explored, and

secondly that the affliction should be bilateral. Prior to the commencement of treatment the renal function should be carefully assessed as toxic effects seem to occur more rapidly when this is impaired. The age of the patient must be taken into consideration, for the process of adjustment to an "avestibular life" is very slow in the elderly.

Throughout the period of treatment cochlear and vestibular function should be determined with regularity, and it is now my practice to have pure-tone audiometric records made at forty-eight-hour intervals; any depression of cochlear function necessitating immediate withdrawal of the drug. Lastly, the part played by the physiotherapist is of supreme importance. Remedial exercises must be applied with persistence and unflagging vigour, and the patient's morale must be continually boosted, for those who suffer from severe vertigo are often, and with reason, the most miserable of men and women.

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Mr. R. L. Flett: *The Saccus Operation in Ménière's Disease*

This operation was proposed by Portmann as far back as 1926. Its good results, however, did not seem to be maintained, and it fell into disuse until Woodman and Stirk Adams reported 10 cases in 1939. In 1938 Hallpike and Cairns showed their slides of 2 cases of Ménière's disease in which the endolymph system was tremendously dilated.

I had a severe case of vertigo who was totally deaf in the other ear. All investigations and medical treatment gave no help. He was a coal-face worker, and the coal face was 2 miles from the bottom of the pitshaft. He demanded that something should be done as he had been carried by his mates on quite a number of occasions when he had started a bout of vertigo. From the language used on these occasions, he could not risk another attack.

I thought, therefore, that the saccus operation ought to be tried. On the day after the operation his hearing had markedly improved and he could hear very satisfactorily, even through the dressings and bandages. He has maintained this condition ever since, and has no tinnitus or giddiness.

I therefore considered that instead of subjecting the severe cases of Ménière's disease to destruction of the labyrinth by injection, as I had done in the past, I should try this operation first, and if it did not succeed, I would reopen the wound and inject the semicircular canal with absolute alcohol. In fact when doing the saccus operation, I always exposed the bony external semicircular canal in readiness for injection in one week's time if it became necessary.

Since this first case I have performed this operation 73 times. This is only done in very severe cases, and I was prepared to go on to destruction, in the event of failure, in all except one other case. We carried out all the routine investigations and various types of treatment, and these cases are those which showed no response and were so severe that something had to be done.

The second case in which I did not wish to destroy the labyrinth was a woman of 40, whose right ear had become totally deaf due to a supposed labyrinthitis during diphtheria in childhood. I performed a left Portmann operation in 1941 with immediate restoration to normal. The left ear remained in this condition for three years, and then the symptoms returned. The patient stated that her hearing had been so good in the interval, that she would like the operation repeated. I admitted her and again exposed the area of the saccus. She improved just the same, but recurrence took place one year later. Operation for the third time produced no improvement. This series of procedures is not to be recommended, and seems to rank with the sudden but temporary improvement in hearing in otosclerosis from the earlier trials of the fenestration operation before Sourdille.

The saccus operation has varied from time to time. At first I used to make a large exposure behind the lateral sinus. Owing to very dilated emissary veins, the hæmorrhage was often excessive and could not always be controlled by bone wax. Sometimes packing had to be resorted to, and the rest of the operation postponed for two days. There seems to be three types of arrangement of the saccus:

(1) A small cyst with thick walls about 5 mm. in diameter. This is usually found under the small spur and is visible at operation.

(2) Instead of the small spur there is a vertical slit running down to the region of the jugular bulb, and the saccus is situated at the upper end of this slit. This is the type of case in which the dura may be opened accidentally and a flow of cerebrospinal fluid ends the operation.

(3) No bony landmarks are visible at all. The posterior surface of the temporal bone is flat, and one elevates dura from the edge of the lateral sinus for 1 to 1·5 cm. This should not be exceeded, as the internal auditory meatus on an average skull is 2 cm. from the medial edge of the lateral sinus at the level of 2 cm. down from the superior petrosal sinus. In this type no cyst may be seen at all and no opening seen in the bone.

Later on, instead of making a large opening behind the sinus, I opened the mastoid, exposing the sinus plate of bone and then exposing the sinus and working medially from that. Later still, especially in the last type of saccus, I started working in the bone posterior to the posterior semicircular canal, and either using a drill or a small gouge, and for this magnification is a great help.

Owing to the possibility of failure, I always expose the bony semicircular canal at the end of the operation. The wound is sutured with no drainage. Any case that shows no immediate improvement is reopened in one week's time and the labyrinth is destroyed.

TABLE I.—RESULTS: 74 PATIENTS

Post-operative deaths, 2	(1) Meningitis (2) Bronchopneumonia
Immediate failures, 10	Destruction of labyrinth one week later
One wrong diagnosis	Epileptic aura (this diagnosis made after six years)
19 cases with perfect result	
43 cases with some improvement	

The results can be seen in Table I. There were 74 cases in fourteen years up to December 1953.

The operation is not without danger as there were two post-operative deaths, one from meningitis —streptococcal in type, in a case that had to be opened twice owing to hæmorrhage the first time. The second case died of bronchopneumonia five days after the operation. There were 10 immediate failures, and these required destruction of the labyrinth one week later. There was one which I may term a wrong diagnosis; she was not improved at all, and it took us and the neurological physicians six years to decide that this was due to an epileptic aura. There were 19 cases with a perfect result, that is the hearing showing only 10 decibels loss over the speech range with slightly increased higher-tone loss. The remaining 43 cases showed some improvement. I shall now analyse these 43 cases in Table II.

TABLE II.—43 CASES WITH SOME IMPROVEMENT

Giddiness			
Disappeared	Improved	No improvement	
23	13	5	
Tinnitus			
None	Slight or used to it	Colds cause buzzing	Bad
14	8	2	10
Deafness			
Normal hearing	No improvement or worse		
7	33	50–70 decibels loss, and of these 9 have hearing aids	

With regard to giddiness: It had disappeared in 23, improved in 13, and showed no improvement in 5, and by rights these 5 cases should really have their labyrinths destroyed. The tinnitus had disappeared in 14, 8 cases complained of slight tinnitus or had become used to it, colds cause buzzing in 2, and 10 complained of severe tinnitus, as, for example, like a bomb whistling down; heavy work makes palpitation go to the right ear, especially moving the right arm. I also saw 2 cases where the tinnitus was almost unbearable. I have had some of these show delay in improvement in the noises, or noises may come on again in three months' time and can later, for no apparent reason, disappear.

With regard to hearing. 7 of these cases show hearing loss up to 10 decibels over the speech range, and I have put these down as normal. The remaining 33 show no improvement or have become worse, that is 50–70 decibels loss, and of these 9 have hearing aids.

I shall further discuss the giddiness in those cases who have improved:

(1) Complained of slight giddiness in 1950, ten years after the operation, but he used to have severe giddiness with vomiting and diarrhœa and has not had to stay off work.

(2) Had two attacks in ten years, was off work once for five weeks and another time for two weeks.

(3) Complained of slight giddiness with colds, but could carry on his work.

(4) Becomes slightly lightheaded with noises at times.

(5) Eight years after the operation had one attack of giddiness. He was operated on at the age of 15.

(6) Is aged 71 and complains of slight giddiness when doing too much work, especially with the arm upward.

(7) Had one short attack eight years later but none since.

(8) Has been off work one month, eight years after the operation, but the attack was much less severe.

(9) Complained of slight giddiness on turning over in bed or stooping suddenly.

(10) Is a miner who complains of slight giddiness on walking in the dark, especially in the coal pit.

(11) Three years after the operation complained of slight giddiness.

(12 and 13) Complained of a feeling of general swimminess with no bouts of giddiness.

In these cases there was sometimes a delay in improvement up to one year, and indeed 2 of them during this time were put on the waiting list for destruction of the labyrinth, and later reported saying that they did not wish the operation to be performed as they were much better.

I reported a series of these in Toronto at the Triological Congress in 1952 (Flett, 1952). This was a ten-year review from 1939–1949, and the results were not highly encouraging. Also Altmann at the Presbyterian Hospital in 1945 had reported 11 cases with only temporary result. To those cases I have now added 13 more. The only extenuating circumstances I can plead are these:

(1) It offers a 25% possibility of retaining the hearing and stopping the tinnitus and giddiness.

(2) It enables 61% of them to continue at work with no giddiness, or only swimminess, but with gradual loss of hearing.

(3) It fails in 14%.

From these results it can be seen that a small proportion can retain their immediate post-operative condition. I consider it is worth while to continue this saccus operation as a preliminary to doing the destructive operation, and that sometimes need for destructive operation does not arise.

Two years ago I stated that I had not destroyed a labyrinth since 1947. In the last two years I have done this twice for failure of the saccus operation. In one I removed the membranous canal. He had 30 decibels loss before the operation. Afterwards it was still 30 decibels loss and two months later went down to 60 decibels. Unfortunately, he had since had another giddy attack, but not as violent as his previous ones.

In the other case I removed the canal, and in order to promote further destruction, injected the labyrinth with absolute alcohol. In the anæsthetic room, she told me she had just started a giddy attack half an hour before, she could not open her eyes, the nystagmus was severe, and she said, "I shall be glad to have the anæsthetic to stop this attack". I had never operated on a case in the acute condition and I thought this would be a good opportunity to see the membranous canal. It was an absolute cast of the bony canal, a wide sausage-shaped tube. I divided it at both ends with a sharp needle and removed it by forceps. On section, however, the only thing remaining was the ampullary end, and everything else was distorted. I compared it with a normal semicircular canal, as I wondered whether there might be any difference in cell spacing or nuclear spacing. If, however, anyone is used to preparing these small specimens, it would be interesting to keep these patients on a waiting list until their acute attack, and admit them for immediate removal of the membranous labyrinth, and actually see and photograph in the living that pathological appearance that Hallpike described. As regards the specimen, the unsupported membranous canal, I should like advice as to the best means of securing a respectable microscopical appearance.

To return to the saccus endolymphaticus, owing to difficulty in finding it, I am going to use the frozen section technique in those cases when it cannot easily be found. Lindsay (1951) in Chicago has done a tremendous amount of research in cats conditioned to auditory stimuli, and has found that destruction of the saccus or aqueduct, has produced no change in the hearing, behaviour or histological appearance of the labyrinth. They were normal cats and I should like to suggest that a trial be made to induce Ménière's disease by either a highly fluid diet, or a high salt intake and then, if successful, to try the saccus operation.

Lempert (1952) has shown specimens of the membranous canal preserved and sectioned in celloidin, and has noted vesiculation of the epithelium. He has put forward a theory on this, that the attack is due to rupture of the vesicles, and the severity of the attack depends on the number ruptured. It could also be argued that the vesicles may obstruct the aqueduct. However, my slide does not show these vesicles, which, of course, may have ruptured in the attack.

In future cases I intend to expose the saccus and also to expose the external semicircular membranous canal or posterior semicircular membranous canal. If they are distended, I shall note whether there is any change in the condition after opening the saccus.

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Dr. Oliver Gray showed a few lantern slides illustrating the anatomy of the structures which had come under discussion, two of which are included on this page. Without claiming any originality, he emphasized the value of speaking of the semicircular *ducts* lying within the semicircular *canals*. This improvement on the old terminology was in every way desirable, and, further, took into account the fact that the semicircular *ducts* of fishes, become, in amphibians, reptiles, birds and mammals, surrounded by the semicircular *canals*.

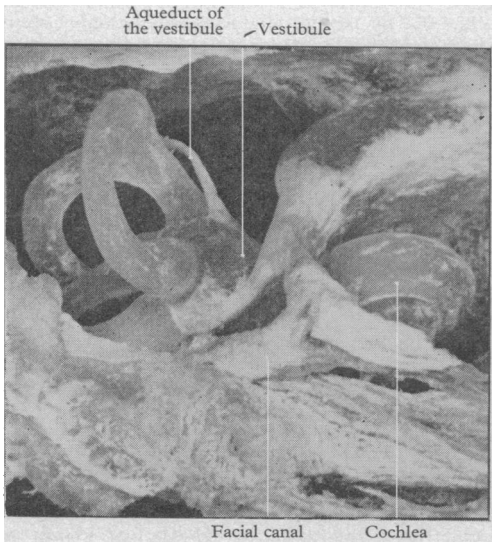

FIG. 1.—Perspex preparation of the right human labyrinth viewed from above. Note the aqueduct of the vestibule and other related structures.

FIG. 2.—Perspex preparation of the right human labyrinth looking outwards.

Mr. E. D. D. Davis said from his own experience true Ménière's syndrome, i.e. true aural vertigo, was apparently difficult to diagnose. He had collected 100 cases of vertigo and only 19 out of the 100 were true Ménière's disease or syndrome. The characters of a true aural vertigo were a paroxysmal rotatory giddiness similar to that produced by syringing the ear with too hot or too cold lotion. The tinnitus was marked before or during an attack. The deafness was unilateral and a condition of the ear known to produce vertigo was present. Labyrinthine nystagmus occurred only during the attack. Diplacusis, or a distorted hearing, might be present. Loud noises were almost painful and might bring on an attack. The vertigo of thyrotoxicosis, *petit mal* or postural giddiness were not Ménière's syndrome. Alcohol aggravated or might produce an attack of vertigo in a susceptible patient. All cases of vertigo were not aural or Ménière's disease. He would like to ask Mr. Cawthorne how he decided which ear caused the vertigo in a case where both ears were affected.

Mr. C. A. Hutchinson said there was one point he would like to ask Mr. Cawthorne: Whereas Dr. Hewlett had referred to Shambaugh's statement that there was an association between otosclerosis and labyrinthine hydrops, he had noticed that Shambaugh went further than this and suggested (*Acta Otolaryng., Stockh.*, **40**, 215) that "The fenestration operation definitely predisposes an ear to the subsequent development of a hydrops (10%)". Was Mr. Cawthorne of the opinion that, in this country at all events, there was any evidence to support this statement?

Mr. I. B. Thorburn said in his discussion of the pathology of Ménière's disease Mr. Cawthorne had, perhaps, concentrated unduly on hydrops of the labyrinth. He suggested that in the over-50 group of patients vertigo and perceptive deafness was often caused by a vascular lesion of the labyrinth associated with generalized arteriosclerosis.

He thought further that the discussion on methods of destroying the labyrinth was unnecessarily complicated. Having made an opening into the lateral semicircular canal, the simplest and most effective way to destroy the labyrinth was to apply the sucker to the opening.

Mr. S. W. G. Hargrove asked if a geographical survey had been made of the incidence of Ménière's disease in this country. He worked in a rural area where people were well fed. He looked at his own statistics before coming to the meeting and found that in the last year he had seen only 10 cases of true Ménière's disease in his outpatients and in nine years he had carried out one labyrinthectomy. Had Mr. Cawthorne any experience in sectioning of the chorda tympanic nerve such as was described by Rosen, S., 1954, *Lancet*, i, 133?

Mr. P. E. Roland spoke of the effect of stellate block. Mr. Cawthorne had explained that it did not cause vasodilatation of the vessels but he had found that it had been effective in many cases. In 2 out of 3 cases of an acute attack of Ménière's disease he was able to give the patient great relief within a few minutes and in a number of cases he had found that one injection had given prolonged relief.

Mr. Terence Cawthorne, in reply to Mr. E. D. D. Davis, said that it was not as a rule difficult to diagnose Ménière's disease. In order to arrive at a precise diagnosis, however, it was important to carry out a full otological examination including the tests not only of auditory but also of vestibular function. Mr. Davis had also mentioned the question of which ear was in the active phase in bilateral cases. Such patients could indeed be a problem but he would like to emphasize again that this was about the only time in otology when tinnitus was really useful, because it would often indicate which was the affected ear. Time and time again he had noticed in bilateral cases that tinnitus or a feeling of fullness in the ear indicated which ear was in the active phase.

Mr. Hutchinson had asked about the possibility of Ménière's disease following fenestration. Shambaugh in his monograph went so far as to say that almost 10% of his cases developed hydrops following fenestration. He had no accurate figures to show what proportion of cases followed fenestration but he would think it was something like 2%.

Mr. Thorburn had mentioned the possibility of a vascular lesion particularly in elderly cases. The blood pressure had been noted in a series of cases of Ménière's disease and was found to be rarely more than one would expect from the population at risk. Mr. Thorburn mentioned removing the labyrinth with a suction tip. This was only too easy as some had found when performing other forms of operation on the labyrinth. He felt that it was best to remove the membranous canal with fine watchmakers' forceps. In this way one had proof that the operation had been properly done. In no case of Ménière's disease in which the membranous canal had been removed or even cut across had there remained any trace of cochlear or vestibular function. He had known of cases in which the labyrinth had to be destroyed for some disorder other than hydrops, and he believed that in such cases it was wise to inject a few millimetres of alcohol as well.

Mr. Hargrove had mentioned a geographical study. He (the speaker) could say that more than half his patients came from outside the London area.

There did not seem to be any evidence at all that the chorda tympani had any other function than that of taste. He had had the opportunity of cutting the chorda tympani accidentally in cases of otosclerosis when carrying out a fenestration operation and the giddiness after the operation was just the same. He had also found that touching the chorda tympani at a fenestration or at a radical mastoid dressing might give rise to a sensation of metallic taste but not of vertigo or tinnitus. He felt a little more proof was needed that the chorda tympani had functions other than that of taste before patients were submitted to what could only be described as an extremely hypothetical procedure.

The value of stellate ganglion injections in cases of Ménière's disease, particularly during the acute phase of the attack, had been mentioned, but unfortunately he had not found it of any great value. He had been able to inject the ganglion in 2 cases of Ménière's disease during an attack but it did not seem to have any influence. However, in all fairness he should say about stellate injections that every now and again patients did say that the tinnitus had gone, perhaps only for a few minutes or a few hours, so that something must be happening within the labyrinth but what it was he did not know.