MÉNIÈRE'S DISEASE

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It is almost the consensus of opinion that Ménière's disease is caused by an irreversible distension of the membraneous internal ear, which I have called " hydrolabyrinth " in order to stress the relationship to hydrocephalus. Although it was known that a hydrolabyrinth may be discovered in patients having suffered from spells of vertigo (Bruehl[1], Alexander, F. and Manasse[2], Mayer[3], Brunner[4], Yamakawa[5]), it was Hallpike and Cairns[6] who stated, for the first time, that the hydrolabyrinth constitutes the essential morbid anatomy of a specific disease of the internal ear, viz., of Ménière's disease. The concept of Hallpike and Cairns was confirmed by several investigators (Rollin[7], Hallpike and Wright[8], Altmann and Fowler[9], Lindsay[10], [11], Cawthorne[12]). However, there are cases on record, having suffered from dizzy spells, which did not show a hydrolabyrinth at autopsy (Hellmann[13], Berggren and Gray[14], Brunner and Grabscheid[15], Arnvig[16]). The latter findings render the problem again controversial and warrant an analysis of all of these findings.

The subject of this paper is an attempt to evaluate the hydrolabyrinth from the pathological as well as from the clinical point of view. For this reason two cases of Ménière's disease are presented which were associated with an osteitis deformans of the temporal bones. The osteitis deformans was studied in previous papers by Dr. Grabscheid and myself. In these papers the changes of the internal ear were only briefly mentioned.

1. Pathological Aspect

According to the studies of Siebenmann[17], Herzog[18], Zange[19], Wittmaack[20] and others hydrolabyrinth is, in general, not a pathological entity, but the result of a serous labyrinthitis and, for this reason, it is a comparatively frequent finding in congenital deafness (" Type Scheibe " after Siebenmann), in deafness due to cholesteatoma (Siebenmann, Nager)[21], to tuberculous otitis (Herzog) to scarlatina otitis, in chronic progressive labyrinthine deafness (Manasse[22]) and in other types of deafness. Wittmaack claims that a hydrolabyrinth may be caused not only by a serofibrinous labyrinthitis, but likewise by a hydrops of the labyrinth which consists of a distension of the membranes due to a hypersecretion of endolymph. However, this does not imply that a serous labyrinthitis or a hydrops must invariably cause a hydrolabyrinth.

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It is rather a firmly established fact that a serous labyrinthitis may eventually be a reversible disease. Some otologists like Alexander even claim that a serous labyrinthitis, in contrast to a purulent labyrinthitis, is invariably a reversible disease. This categorical point of view is useful from the clinical point of view, but it is not confirmed by pathological findings. For unknown reasons, serous labyrinthitis does cause eventually irreversible changes in the labyrinth and among these, an irreversible hydrolabyrinth. Herzog has emphasized that very virulent, particularly purulent infections never cause a hydrolabyrinth. Only these instances of serous labyrinthitis are supposed to result in a hydrolabyrinth in which the infection is of low virulence and is active for a long period of time as for instance the tuberculous otitis. It seems that the opinion of Herzog is correct.

If there is an abnormal amount of serous or serofibrinous fluid in the endolymphatic inner ear, the endolymphatic pressure increases, the membranous walls, particularly these which are not endowed with sensorial epithelium and nerves, become distended and eventually the sensorial epithelium is damaged. Since serous labyrinthitis is never a localized disease, the entire inner ear is involved. In other words, the increased endolymphatic pressure must be effective upon all walls of the internal ear. But the distension involves particularly and primarily the pars inferior, viz., cochlea and saccule, for various reasons: (*a*) the endolymph is chiefly produced in the cochlea; (*b*) in the cochlea and saccule there are thin walls, not endowed with sensorial epithelium and nerves, which may bulge far into large perilymphatic spaces, if the endolymphatic pressure rises; (*c*) there is no perilymphatic tissue in the cochlea and saccule, interfering with the distension of the membranous walls. For this reason, there is primarily a reversible or irreversible distension of Reissner's membrane and of the free wall of the saccule, if the endolymphatic pressure rises. The influence of the accumulations of fluid upon the sensorial organs was ably analysed by Herzog and Zange who emphasize that Corti's organ and its nerves are more extensively and more rapidly damaged than the maculae and cristae.

The most significant feature, however, is the distension of Reissner's membrane. The distended Reissner's membrane may be mobile or may be fixed in an abnormal position. The fixation may be caused by newly formed connective tissue which originates from the organization of the serous exudate, or by serofibrinous exudate. In the microscopic specimen the distended Reissner's membrane will be seen, therefore, in various position when the endolymphatic pressure has ceased. In extreme cases the enormously distended membrane is fixed toward the walls of the scala vestibuli, causing an enormous dilatation (ectasia) of the cochlear duct. In the vestibulum the increased pressure may even cause a rupture of the membranous walls. In less extreme cases a part

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of Reissner's membrane is fixed above the stria vascularis, the rest of the membrane is mobile. This is called " the high insertion of Reissner's membrane ". In other cases, the entire membrane is mobile and wavy. Or, the distended membrane may sag toward the basilar membrane of the cochlea and may become fixed by fibrinous exudate to Corti's membrane, Corti's organ, external spiral sulcus and vascular stria, causing a considerable narrowing of the cochlear duct. This is called " collapse of Reissner's membrane ".

The distension of Reissner's membrane is frequently associated with a similar distension of the free wall of the saccule and eventually with a marked degeneration of Corti's organ and spiral nerves. These findings constitute the hydrolabyrinth, which is a partial hydrolabyrinth because it involves cochlea and saccule but not the other parts of the internal ear. In more advanced cases the utricle and eventually one or more semicircular canals become likewise dilated. These findings constitute the complete hydrolabyrinth. The ampullae of the semicircular canals almost never become dilated (Alexander[23]).

It is the merit of Hallpike and Cairns to have stated that a hydrolabyrinth, partial or complete, represents the pathology of Ménière's disease. Regarding the pathogenesis of the hydrolabyrinth, they believe in the absence of an infection of the tympanic cavity or the meninges or the blood " that the endolymphatic dilatation in these cases is due, at any rate in part, to some failure in the normal absorptive mechanism ". The cause of this failure is supposed to be a fibrosis of the loose vascular tissue surrounding the endolymphatic duct. This concept is not tenable because this type of fibrosis may be found without hydrolabyrinth as emphasized by Hallpike and Cairns and because there is experimental evidence indicating that the normal endolymph is probably not absorbed in the endolymphatic sac (Lindsay[24]). Hallpike and Cairns apparently feel likewise not sure about their own concept, because they continue by saying that " the primary change may still be either an increased production of the endolymph itself, or else some inscrutable type of alteration in its physico-chemical constitution ".

Obviously the problem what causes the hydrolabyrinth in Ménière's disease is not solved. It is likely that the following cases may answer this question, at least for these individual cases.

Case I.

K.B., white female, age 68. The patient had no serious illnesses. About two weeks ago she suddenly became ill with severe attacks of whirling dizziness, vomiting, tinnitus and an abrupt diminution of hearing on both sides. The diagnosis of " Ménière's disease " was made, but a thorough examination of the ears was not performed. However, a spinal puncture was made which revealed a positive Wassermann reaction in the spinal fluid. Two weeks later she became delirious and expired after four days.

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Hans Brunner

Autopsy revealed an osteitis deformans of the skull, arteriosclerosis of the cerebral blood vessels, sclerosis of the coronary arteries, mesaortitis luetica, aneurysm of the aorta and an arteriosclerosis of the kidneys.

Microscopic examination of the temporal bones.

The changes are on both sides essentially the same.

All spaces of the cochlea are filled with a sero-fibrinous fluid which stains with eosine and contains a few lymphocyte-like cells, adherent to the walls of the osseous inner ear (Fig. 1). The fluid enters likewise the channels of the modiolus and the meshes of the spiral ligament. In the specimen the exudate within the endolymphatic spaces has a granular appearance; in the perilymphatic spaces the fluid stains darker with eosin and contains fibrin. Corti's organ shows post-mortem changes; in the vestibular portion of the cochlea (Fig. 2) it consists only of one layer of flat cells. In the other coils the pillar cells can be clearly distinguished and, when the amount of exudate is small, the internal sensorial cell can be likewise recognized. Stria vascularis consist of a loose connective tissue, harbouring exudate, and contains large capillaries. In the vestibular portion Reissner's membrane is bulged toward the basilar membrane and adherent to the stria. Corti's membrane is in normal position. In the lower part of the basilar coil Reissner's membrane is adherent to the stria and the cochlear duct is very narrow. Corti's membrane is adherent to Corti's organ. In the upper portion of the basilar coil and in the middle coil Reissner's membrane is bulged toward the basilar membrane, but not adherent to the stria. Corti's membrane is adherent to Corti's organ. In the tip of the cochlea Reisner's membrane is slightly bulged toward the scala vestibuli; Corti's membrane is œdematous and raised from Corti's organ. The nerves and ganglia cells of the cochlea are extremely small in number. In the vestibular portion ganglia cells are entirely absent. The cochlear nerve is markedly atrophic. The vestibular ganglion is apparently normal.

In the perilymphatic cisterna the fibrinous exudate forms a wall which has the same localization as the vestibulo-cochlear septum of Alexander (Fig. 2). Utricle and saccule and ductus reuniens are filled with a granular exudate; their walls are covered from without by a layer of homogeneous exudate. Macula utriculi shows post-mortem changes; it is normal. The utricular nerve seems to be slightly atrophic. The semicircular canals are partially filled with granular exudate. Cristae are normal. The endolymphatic duct is dilated, contains granular exudate: its epithelium covers an œdematous connective tissue, poor in nuclei.

The temporal bones show an osteitis deformans. Tube, tympanic cavity and antrum are normal. The drums do not show the normal tension; malleus and incus are osteoporotic. Stapes and windows are normal and so is the pneumatization.

In this case the diagnosis of Ménière's disease was made by a physician about 14 days prior to the death of the patient. According to the description, given by the patient, it was a grand attack, consisting of labyrinthine and cochlear symptoms. At autopsy the labyrinths were filled with a fluid which was neither pus nor peri- or endolymph, which

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had rather the appearance of serofibrinous exudate, containing a few lymphocyte-like cells.

The question which has to be answered concerns the origin of the fluid in the presented case. At present, this question can be answered more in the negative than in the positive. The fluid is not produced by a hypersecretion of the stria because the fluid contains fibrin and cells. For this reason the microscopic findings cannot be classified as a hydrops labyrinthi. However, the term " serous labyrinthitis " is likewise not quite correct because there is no infection of the tympanic cavity, of the meninges or of the blood stream which could have caused an inflammation of the labyrinth. The positive Wassermann reaction in the spinal fluid is likewise not responsible because a serous labyrinthitis in a case of late tertiary syphilis which is not associated with a syphilitic infection of the temporal bone, would be extremely unusual. I have believed that in this particular case the accumulation of fluid may have been caused by the osteitis deformans. The transformation of bone may have released metabolites which exerted a toxic effect upon the labyrinth. This concept may be plausible, however, there are several objections. In Paget's disease the transformation of bone frequently extends to the endosteum of the internal ear. Nevertheless, the finding of accumulation of fluid in the internal ear is an exceptional finding. In addition, hydrolabyrinth in Ménière's disease was frequently discovered in cases, not afflicted with Paget's disease. For this reason, Paget's disease cannot be the only cause of the serous labyrinthitis. At present, it is not possible to determine the other factor which rendered the blood vessels of the labyrinth permeable in the presented case. Obviously the concept of an allergic or hormonal influence upon the blood vessels immediately turns up. However, there is no definite proof in this respect. For this reason, I have used the preliminary term " otitis interna vasomotoria " (vasomotor labyrinthitis) to designate the accumulation of serofibrinous fluid in the internal ear in instances which have no infection in the tympanic cavity or in the meninges or in the blood stream (Brunner[25]). Since the arteries of the internal ear are branches of cerebral arteries the presented concept may explain also the headache which frequently is associated with Ménière's attacks and which even may replace the dizzy spells. Encephalographic studies, however, performed immediately after the attack failed to show pathology in the posterior cranial fossa.

The last question which has to be answered is the relationship between vasomotor labyrinthitis and hydrolabyrinth. The following case is supposed to answer this question.

Case II.

P.J., white male, age 58. In 1912, following an influenza the patient became suddenly hard of hearing. Ever since he suffers from attacks of dizziness

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and headache. For a certain period of time there was vomiting. He also suffered from gallstones and general œdema. He frequently looses the balance in walking and gives the impression of being drunk.

On July 9th, 1921, there was an œdema of the lids, of the legs, of the scrotum and ad sacrum. The patient is very deaf and a communication is only possible by writing. The drums are retracted. There is a pastpointing to the left in the right arm. There is no adiadochokinesis, but the gait is unbalanced. There is no Babinsky. The labyrinths are excitable, perhaps hyperexcitable to the caloric stimulation. The patient complains of being dizzy. On July 23rd, the dizziness has decreased. On July 29th, the patient is stone deaf on the left side, on the right side he understands words, shouted into the ear. He does not hear high tones or low tones by air conduction. The a^1-fork is heard by bone conduction on the left side for 3 to 4 seconds, on the right side for about 10 seconds. There is no spontaneous nystagmus. There is a hypo-excitability of high degree of both labyrinths which is on the left side more marked than on the right. Practically there is non-irritability on both sides. Examination on the turning chair was not performed. On October 14th, the patient expired.

At autopsy an arteriosclerosis of the coronary arteries, a fibrous myocarditis, a pneumonia, a chronic hydrocephalus and an osteitis deformans were discovered.

Microscopic examination of temporal bones.

On the left side the cochlear duct is very much dilated, particularly at the base and at the tip of the cochlea (Fig. 3). In the vestibular portion of the cochlea Reissner's membrane is separated from the bony wall of the scala vestibuli only by a narrow slit. At the tip Reissner's membrane is not only adjacent to the walls of the scala vestibuli, but bulges through the helicotrema into the scala tympani. In the superior portion of the basilar coil and in the middle coil approximately two-thirds of the scala vestibuli are occupied by the cochlear duct. In the inferior portion of the basilar coil the dilatation is less marked, but Reissner's membrane forms pouches which extend into the scala vestibuli of both the middle coil and the vestibular portion of the cochlea. The entire Reissner's membrane of the basilar portion has a wavy appearance and the pouches are simply excessive outgrowths of Reissner's membrane.

In Corti's organ the sensorial cells are absent; the supporting cells are fairly preserved. Corti's membrane is in normal position. Only in the vestibular portion Corti's membrane is reflected toward the surface of the crista spiralis and ensheathed by connective tissue. Ligamentum spirale consists of a loose connective tissue, which only on some places has a more dense texture. Stria vascularis shows post-mortem changes. The spiral nerves and the spiral ganglia show a considerable loss of nervous elements which is particularly marked at the base of the cochlea. In the walls of the channels of the modiolus there is a formation of osteoid tissue. The cochlear nerve in the internal meatus is atrophic. The saccule is dilated, the ductus reuniens is normal. The utricle is enlarged toward the windows. The lateral wall of the utricle forms a small pouch which is connected by connective tissue with the oval window. The cisterna perilymphatica is reduced to a small space between utricle and saccule. The macula sacculi consists only of a small

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amount of epithelial cells. Sensorial cells and otoliths are absent. The macula utriculi shows marked post-mortem changes; seems to be smaller than under normal conditions. Otoliths are absent. Utricular nerve is slightly atrophic. The endolymphatic duct is slightly dilated; it is surrounded by firm connective tissue. Cochlear aqueduct is closed by connective tissue and contains corpora arenacea. The cristae show post-mortem changes. Cupulae are absent. Semicircular canals are normal.

There is no infection or remnants of infection in the tympanic cavity. On the right side the tympanic cavity is likewise normal.

The cochlear nerve shows a moderate atrophy. The vestibular ganglion is perhaps less rich in ganglia cells as under normal conditions. The facial is normal. In the cochlea there is a moderate atrophy of spiral ganglia and spiral nerves which is marked only in the basilar and vestibular portion. Corti's organ shows post-mortem changes, but the supporting cells are apparently normal. Stria vascularis shows a loosening of the cells; the spiral ligament consists of a loose network of connective tissue. The meshes of the network are large, almost cyst-like. Reissner's membrane is in normal position and there is a small amount of granular exudate in the scalae of the basilar portion. The arterioles in the internal meatus, in the modiolus and in the laminae cribrosae show a thickening of the adventitia and small strips of calcium within the adventitia. The cochlear aqueduct shows the same changes like on the left side.

The ductus reuniens is slightly enlarged and so is the saccule at its junction with the ductus reuniens. At this side the wall of the saccule is wavy (Fig. 4). The macula sacculi shows post-mortem changes; the nerve is slightly atrophic. In the macula utriculi there are only a few sensorial cells; there are no otoliths. The utricular nerve is moderately atrophic. The inferior recess of the utricle is slightly dilated and shows a small pouch at its lateral wall. In addition, there is a crista quarta in the lateral wall and an atypical epithelial spot in the mesial wall of the inferior recess of the utricle. The epithelium of the crista quarta contains small cysts. There is a slight dilatation of the endolymphatic duct at its junction with the saccule. Otherwise the endolymphatic duct is normal and is, like the endolymphatic sac, surrounded by firm connective tissue.

There is a serofibrinous exudate in the vestibulum (Fig. 4). The exudate fills the scalae of the vestibular portion of the cochlea, the perilymphatic cisterna and the saccule. In the specimen it has a granular appearance; in the lumen of the saccule it forms a fine network (Fig. 4). There is no exudate in the utricle, in the ductus reuniens and endolymphatic duct.

The cristae show post-mortem changes; their nerves are normal except in the inferior crista the nerve of which is slightly atrophic. There is a small amount of serous exudate in the frontal and horizontal ampullae.

The footplate of the stapes is slightly bent toward the vestibulum, but the anterior portion of the stapes is slightly displaced toward the tympanic cavity. The endosteum of the footplate is œdematous. The membrane of the round window is wavy.

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In this case there was again a vasomotor labyrinthitis on the right side, viz., there was again an accumulation of serofibrinous fluid in the internal ear. The difference against case I concerned only the quantity and the localization of the fluid. The amount of fluid was much smaller than in Case I and it occupied the vestibulum almost exclusively. The greater part of the cochlea and semicircular canals did not contain a great amount of exudate. In other words, this was a subsiding vasomotor labyrinthitis.

In contrast to the right side there was no exudate, but a marked hydrolabyrinth on the left side. The hydrolabyrinth concerned the cochlea and the saccule and, to a lesser degree, the utricle. It is obvious that there is the same disease on both sides which is on the left side much further advanced than on the right side. In favour of this statement are (*a*) the clinical findings which indicate the same disease on both sides although in a different stage of progress, (*b*) the finding of a slight dilatation of the ductus reuniens, endolymphatic duct and part of the utricle on the right side ; (*c*) the finding of a degeneration of the sensorial organs on both sides, although in a different stage of progress. The latter finding concerns particularly Corti's organ, the spiral nerve, spiral ganglion and cochlear nerve. Corti's organ shows on both sides an almost normal supporting structure, a loosening of epithelium of the stria vascularis which is probably artificial, and an atrophy of the spiral ligament which is not artificial. The hair cells cannot be recognized on either side. Spiral nerve, spiral ganglion and cochlear nerve are atrophic on both sides, on the left side more than on the right. The sensorial organs of the vestibulum and the semicircular canals are probably more or less atrophic, but it is not possible to make a definite diagnosis because of the artificial changes. The atrophy of the sensorial organs is caused primarily by the vasomotor labyrinthitis and the hydrolabyrinth respectively ; but, in addition, the changes of the blood vessels of the internal ear should not be underestimated. The walls of the arterioles are thickened, partially calcified. These changes certainly must interfere with the normal cellular metabolism.

So far as the presented cases are concerned the conclusion can be drawn that the hydrolabyrinth in Ménière's disease and perhaps in other types of deafness, is caused by an accumulation of serofibrinous fluid in the internal ear. This fluid is not peri- or endolymph because it contains cells and fibrin. For this reason it cannot be produced by the stria vascularis ; it must rather escape from the blood vessels which run in the stria as well as in the walls of the perilymphatic spaces. It is likely that the fluid enters simultaneously, both the peri- and endolymphatic spaces. The preliminary term " otitis interna vasomotoria " (vasomotor labyrinthitis) seems to be suitable for this finding. It is not known what causes in some cases the vasomotor labyrinthitis to subside without

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leaving traces behind it, and in other cases to change into a hydrolabyrinth. So far as Ménière's disease is concerned, it is likely that this depends on the intensity, frequency and duration of the acute attacks.

II. Clinical Aspect

In ear-pathology it is an extremely difficult task to establish the relationship between pathological and clinical findings. This difficulty is redoubled in Ménière's disease because of the fluctuation of the symptoms.

In the writer's opinion, Ménière's disease is a chronic disease of the inner ear which advances chiefly by means of acute attacks, the attacks being different in character and intensity. For this reason, the pathology of Ménière's attack is the central problem. If I understood Hallpike and Cairns correctly, these authors believe that the attacks are brought about by extremely rapid rises of fluid pressure within a hydrolabyrinth. In other words, the attacks can initiate only if there is primarily a hydrolabyrinth. A great number of otologists seem to share this opinion (Lindsay, Cawthorne, Altmann and Fowler, Williams[26]). Nevertheless, this concept is apparently not tenable. The cases of Berggren, Arnvig, Alexander and Manasse, Hellmann and the presented Case I prove that Ménière's attacks may occur although there is no hydrolabyrinth. In agreement with Vernet[27] and Ferreri[28] the hydrolabyrinth can not be considered as a necessary prerequisite, but rather as an eventual result of Ménière's attacks. Case II is supposed to prove this concept. Nor is the hydrolabyrinth invariably caused by Ménière's attacks. Lindsay[29] noticed a hydrolabyrinth in a case of labyrinthine deafness which, according to the history of the case, had not suffered from dizzy spells. Lindsay believes that the dizzy spells may have occurred a long time prior to the death and had been simply forgotten by the patient. This conclusion would be correct of the case of Lindsay would be a sporadic finding. However, this is by no means a sporadic case because, as mentioned before, a hydrolabyrinth has been noticed in several types of deafness which are not associated with vertigo of considerable degree.

Another argument against considering the hydrolabyrinth as the pathological basis of Ménière's attacks, is offered by the cases of Ménière's disease showing only a partial hydrolabyrinth (Bruehl, Hallpike and Cairns, Rollin, Altmann and Fowler, Lindsay). In these instances the hydrolabyrinth concerns only cochlea and saccule; utricle and semicircular canals have a normal configuration. In contradistinction, the clinical symptoms, viz., the spells of vertigo concern the utricle and the semicircular canals, but not cochlea and saccule. This suggests the conclusion that the hydrolabyrinth does not play the important role in the pathology of Ménière's attacks as many otologists claim. It rather seems that the relationship between Ménière's disease and hydrolabyrinth

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is similar to the relationship between otosclerosis and stapes ankylosis. There is otosclerosis without stapes ankylosis and there is Ménière's disease without hydrolabyrinth. But, on the other hand, stapes ankylosis is most frequently caused by otosclerosis and likewise hydrolabyrinth is frequently caused by Ménière's disease.

More important for the pathogenesis of Ménière's attacks than hydrolabyrinth is the vasomotor labyrinthitis. This finding was obtained only in the presented cases. Consequently the following discussion is based on the findings in the presented cases, although Hallpike and Cairns have likewise noticed albuminous coagula in the inner ear of their cases. However, they refer this finding to the operation on the vestibular nerve which was performed a short time prior to the death. The presented cases prove that the albuminous coagula may be found although no operation has been performed. The finding of newly-formed connective tissue in the perilymphatic spaces of the cases of Hellmann, Hallpike and Cairns and others is an additional indication that vasomotor labyrinthitis does occur in cases of Ménière's disease.

From this discussion the following concept would emerge: Ménière's attack is caused by a vasomotor labyrinthitis which may resolve without leaving any traces or which eventually may cause a hydrolabyrinth associated with a degeneration of sensorial organs. This implies that vasomotor labyrinthitis causes essentially the same symptoms as does, for instance, an acute serous labyrinthitis due to an acute otitis media, viz., an acute attack of dizziness, nystagmus and deafness. This concept simplifies considerably the problem of Ménière's attack, but unfortunately in this case simplicity is not a test of accuracy. I [30] have noticed a vasomotor labyrinthitis in a case of osteitis deformans of the temporal bone who suffered from a profound deafness, but never complained of dizzy spells; and I [31] have noticed likewise a vasomotor labyrinthitis after experimental head injuries in animals which did not show labyrinthine symptoms. From these facts the following conclusion must be drawn: Granted that my findings in Ménière's disease should be confirmed by others, it is likely that there is invariably a vasomotor labyrinthitis or the result of it, viz., a hydrolabyrinth, if there is a Ménière's attack. But this statement should not be reversed. The finding of a vasomotor labyrinthitis or hydrolabyrinth respectively does not necessarily indicate that there has been a Ménière's attack. A vasomotor labyrinthitis may cause deafness and tinnitus, but may fail to cause dizziness and nystagmus. In this respect vasomotor labyrinthitis is not unique, it is rather analogous to the acute serous labyrinthitis of bacterial origin, because in a serous labyrinthitis due to a tuberculous or scarlatinous otitis media or due to a meningitis, the labyrinthine symptoms are likewise frequently absent or inconspicuous, the principal symptom being deafness, complete or incomplete, transitory

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FIG. 1

Section through the cochlea on the left side. Note the atrophy of the spiral ganglion, the exudate in the endo- and perilymphatic spaces and the slight dilatation of the cochlear canal in the tip coil.

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FIG. 2

Section through the utricle, inferior ampulla, perilymphatic cisterna and vestibular portion of cochlea on the right side. Note the sero-fibrinous exudate in all cavities of the internal ear. In x the exudate forms a vestibulo-cochlear septum.

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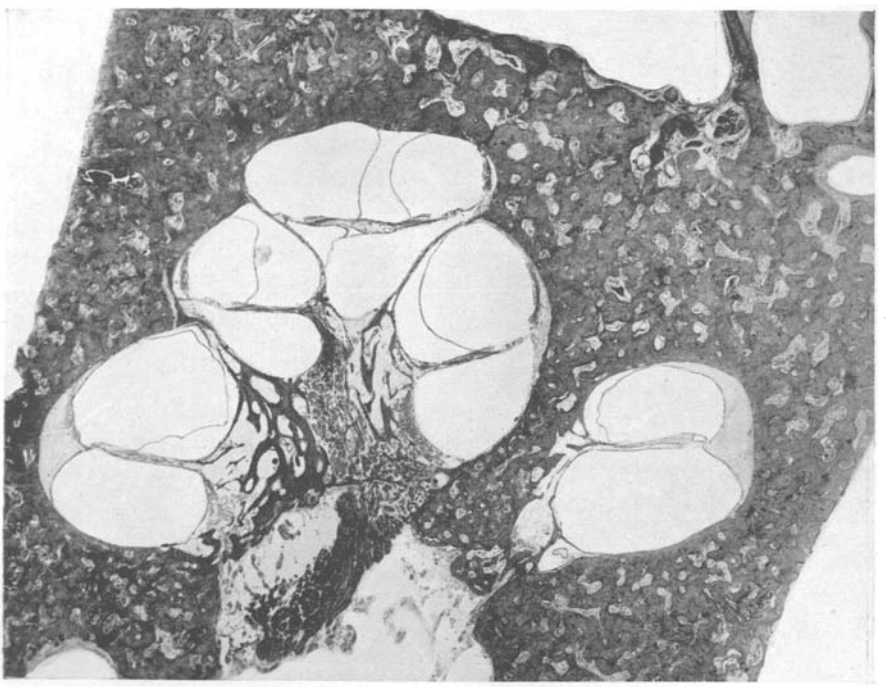


FIG. 3
Hydrolabyrinth on the left side.

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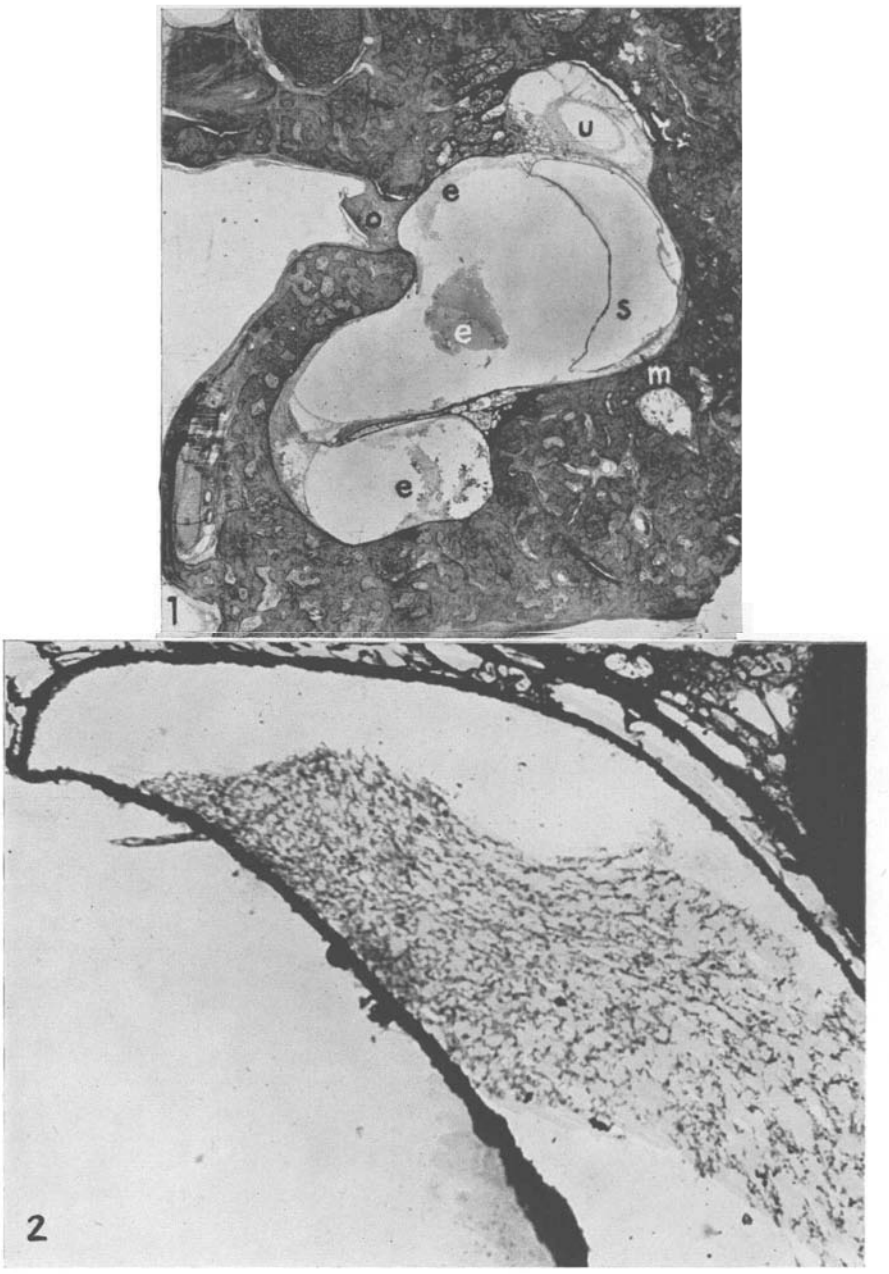


FIG. 4

Picture 1 shows a section through the utricle (u), the slightly dilated saccule (s), the macula sacculi (m) and the oval window (o) on the right side. Note the sero-fibrinous exudate (e) in all cavities of the inner ear. Picture 2 shows a part of the saccule in higher magnification. Note the network-forming exudate in the saccule.

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or permanent. This, of course, does not explain the variability of clinical symptoms in vasomotor labyrinthitis ; it rather furnishes evidence that accumulation of albuminous fluid in the internal ear does not invariably cause cochlear plus labyrinthine symptoms, regardless as to whether the abnormal permeability of the blood vessels is inflammatory in origin or not.

Conclusion

1. Ménière's disease is a type of labyrinthine deafness which is frequently progressive. It differs from other types of progressive deafness by the fact that it is associated with attacks of labyrinthine dizziness and that there is no linear increase of deafness ; there is rather a progress chiefly by means of attacks which cause a reversible or irreversible increase of deafness.

2. The hydrolabyrinth in Ménière's disease is (as in other types of chronic progressive deafness) the eventual result of a serous labyrinthitis.

3. In the absence of an infection of the tympanic mucosa or the meninges or the blood stream, the serous labyrinthitis may be—preliminary—termed " vasomotor labyrinthitis ".

4. It is likely that there is invariably a vasomotor labyrinthitis or the result of it, viz., a hydrolabyrinth, if there is a Ménière's attack.

5. Accumulation of albuminous fluid in the internal ear does not invariably cause cochlear plus labyrinthine symptoms, regardless as to whether the abnormal permeability of the blood vessels is inflammatory in origin or not.

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