indicate some relationship between the two, it may be mentioned that so far they appear to be both chemically¹² and physiologically unlike.

I wish to express my thanks to Sir F. Gowland Hopkins for his interest in this article.

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MÉNIÈRE'S DISEASE*

BY

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HARROGATE

For many years it was taken for granted that Ménière's disease was due to haemorrhage into the labyrinth. This misconception was due to a faulty reading of Ménière's original paper, in which there is no mention of this condition. He described the material found in the labyrinth of his well-known case as being composed of reddish plastic lymph. Alexander of Vienna considers that Ménière's patient suffered from leukaemia complicated by a leukaemic haemorrhage into the labyrinth. Escat thought that Ménière's disease occurred only in arterio-sclerotics, and ascribed the paroxysmal attacks to an intermittent spasm of the internal auditory artery, and the apoplectiform attacks to haemorrhage.

The exclusion of labyrinthine haemorrhage as an etiological factor placed this disease in a less formidable light, for haemorrhage from one intracranial blood vessel implies the possibility of haemorrhage from another, secondary to arterio-sclerosis and high blood pressure. But haemorrhages, arterio-sclerosis, and high blood pressure are absent in Ménière's disease. The vertigo due to spasm of the cerebral arteries in patients with arterio-sclerosis must be distinguished from the vertigo of Ménière's disease in which tinnitus and deafness are prominent symptoms.

INTRACRANIAL CAUSES

The rejection of haemorrhage and arterio-sclerosis as causes of this disease was followed by many theories and, naturally, by as many treatments. Thornval, a recent writer, considers that the main disturbance is in the medulla oblongata. Charcot (1874) and Dandy (1928) put forward the view that there was a lesion of the auditory nerve and not of the semicircular canals. Charcot recommended division of the nerve, and Dandy divided the nerve in nine cases with a resulting cure. Aboulker thinks that the syndrome is due to pressure on the nerve from a collection of fluid around it—that is to say, to an encysted meningitis or hypertension of cerebro-spinal fluid in the lateral cisterna. He consequently does a decompressive trephine behind the mastoid, with or without incision of the meninges. Babinski, and, later, Blake, must have had the same idea, as they treated their patients by lumbar puncture. Quix also believes that this disease is due to intracranial hypertension, which acts

on the otoliths via the ductus endolymphaticus instead of on the nerves. Kobrak also subscribes to this theory of an intracranial origin. According to his view the trouble is localized behind the labyrinth mainly in the cisterna pontis, and the increased pressure is transferred to the labyrinth via the porus acusticus or saccus endolymphaticus. He thinks that the excess of fluid is caused by an irritation of the choroid plexus or is due to a transudation resembling a circumscribed oedema of the skin. Kobrak uses atropine, pilocarpine, and adrenaline in examining his patients to find out whether the syndrome is associated with a vagotonic or sympathetico-tonic condition.

INTRA-AURAL CAUSES

The other theories to be considered all agree that the origin is intra-aural rather than intracranial. Thus Witmaack is of the opinion that toxins produced by a catarrhal condition of the middle ear diffuse into the labyrinth via the windows, and so produce an intra-labyrinthine hypertension. Quincke believes that the symptoms are due to an angioneurotic oedema of the labyrinth, which brings about an increase in tension of the endolymph. According to Portmann the hypertension originates in the labyrinthine blood vessels. He calls this hypertension of the labyrinth "glaucoma of the labyrinth," and considers that Ménière's syndrome is closely connected with vasomotor phenomena, and hence with sympathetic equilibrium. He believes that the vertigo is caused by a vaso-constriction of the blood vessels of the labyrinth, while tinnitus and deafness are produced by vaso-constriction of the vessels of the cochlea. This vaso-constriction elsewhere would produce pain, as in frostbite or Raynaud's disease. The sign of labyrinth disturbance is vertigo, just as the sign of a diseased cochlea is tinnitus. This vaso-constriction disappears after a sympathectomy, the consequence of which is a vaso-dilatation of the blood vessels of the labyrinth. He also considers that the constriction of the blood vessels may be caused by adrenaline. Portmann does not know whether the vascular stasis produced by vaso-constriction acts directly on the nerves or, as the result of an increase of pressure, on the semicircular canals. With the idea that vertigo was due to an increase of pressure in the semicircular canals he opened the saccus endolymphaticus after performing a mastoid operation without opening the antrum. On the supposition that vertigo was purely a vasomotor reaction he performed a perivascular sympathectomy on the common and internal carotid arteries, and injected drugs which inhibited the sympathetic system, attempting to cause peripheral vaso-dilatation by hot baths. Halphen, on the same grounds, applied cocaine (Bonain's solution) to the sphenopalatine ganglion, and stated that this led to the disappearance of the vertigo after an hour or two, due, presumably, to the inhibition of the sympathetic system. Lermoyez recommended liquor trinitrini in cases of suspected spasm of the internal auditory artery. Amyl nitrite and benzyl benzoate have also been recommended. Muck agrees with Portmann in that he has found his vaso-constrictor nasal reflex present in Ménière's disease. This indicates, according to Muck, the presence of an angiospastic condition of the auditory artery. He has successfully treated five patients with a meatless diet.

FAULTY WATER METABOLISM

Finally, Dida Dederding and Dr. Mygind of Denmark have originated a theory which solves all these problems. They definitely state that the symptoms of Ménière's disease are due to increase of pressure in the semicircular canals, which produces vertigo and deafness by displacement of the stapes. They attribute this increased

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pressure to a faulty water metabolism, which allows water to collect intracellularly, resulting in subcutaneous infiltrations, overloading of the fluid in the labyrinth, and increase of pressure in the cerebro-spinal fluid. To the well-known signs of Ménière's disease—namely, tinnitus, vertigo, and deafness—they add the attribute of variability of these signs, the variability being due to the amount of water retained at the time. Thus, after exercise sufficient to produce perspiration, or following a decreased intake of water and salt, air conduction and bone conduction improve; while after an increased intake of fluid, air conduction and bone conduction decrease, and the latter may even be absent. Low notes are usually not audible, while high notes are well heard. This shows that the cochlea is intact and that hearing can be saved. In late cases the high notes are lost. These writers find the tympanic membranes retracted as a result of stenosis of the Eustachian tube, and so they look upon catheterization as an essential part of the treatment. The stenosis is caused by waterlogging of the lining mucous membrane. The examination of the labyrinth reveals abnormality of the semicircular canals; nystagmus occurs spontaneously, or it can be induced by the inhalation of amyl nitrite, and the turning chair shows a difference in the direction of right and left after-turning nystagmus. The proof that these patients suffer from a faulty water metabolism is that if 1,000 c.cm. of water is administered, only 400 to 500 c.cm. will be passed in four hours, whereas a normal person will pass the 1,000 c.cm. or more during this period. Furthermore, a patient with Ménière's disease will increase in weight corresponding to the amount of water retained; that is to say, if a patient retains 500 c.cm. he will increase in weight by 400 grams, allowing 100 c.cm. for loss through the skin and lungs.

A haemocritic test also shows that in a normal person the blood becomes diluted half an hour after intake of water and regains its normal concentration an hour later, while in a waterlogged patient with Ménière's disease there is no alteration in the dilution of the blood. A waterlogged patient also shows a loss of haemoglobin, the haemoglobin being about 73 per cent. The third proof offered is that an intravenous injection of salyrgan (1 c.cm.) causes diuresis, which commences immediately and lasts two days in a patient with Ménière's disease, while in a normal person the diuresis lasts one day, and there is a decreased output of urine on the second day. In certain patients with only tinnitus and catarrhal otitis disordered water metabolism has been found. These cases can be recognized by noticing an alteration from hour to hour in the perception of low tones or in the air conduction.

TREATMENT

The treatment of Ménière's disease, based on this theory, consists in diminishing the intake of fluid so that the patient loses weight and is actually thirsty. For dietetic purposes fruit and vegetables are considered to be fluid. One also must promote the loss of water by carbonic acid, fir needle, and mud baths, and by the Scotch douche, by exposure to the carbon arc up to one hour three times a week, and by massage. Diet must be salt-free. Local treatment consists in catheterization of the Eustachian tube. The drug treatment in this Danish school is unimportant. Like others they treat the acute attack with atropine, but have found ergotinine also useful. An injection of salyrgan is followed up by a long course of calcium-diuretin; while calcium, ovarian, and thyroid extracts are also used.

I myself have had great success in employing a salt-free diet with a decreased intake of fluid. I have also used a drug (in a few cases) which has been investigated by

Berggren—namely, bulbo-capnin (Merck). This drug is as valuable in Ménière's disease as ephedrine is in asthma. My patients take one tablet (0.1 gram oral tablet) a day, and this is sufficient to prevent vertigo, while the acute attack is treated by a hypodermic injection (0.1 gram hypodermic tablet), which will almost at once relieve the patient.

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MINERS' NYSTAGMUS

BY

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The following investigation favours the theory that defective illumination is the major factor in the causation of miners' nystagmus.

I have investigated the same seam in two pits which are about a mile apart; they are steam-coal pits. In Pit A 0.303 per cent. of the workers are on compensation for nystagmus; in Pit B 1.27 per cent. are on compensation. I have examined the men who are working on the day shift in both pits: suffering from nystagmus—Pit A, 26.25 per cent.; Pit B, 41.42 per cent. In both pits the greatest incidence is among colliers, but there are many more acute cases in Pit B than in Pit A.

Comparison of Conditions

	Pit A	Pit B
Number of men employed ...	1,300	1,280
Number of oil lamps ...	550	590
Number of l.e.p. electric lamps ...	804	770
Depth of seam ...	1,500 ft.	1,800 ft.
Average thickness of seam ...	4 ft. 2 in.	3 ft. 1 in.
Mechanically handled ...	100 %	100 %
Barometric reading ...	28.35	28.43
<i>Analysis of Air</i>		
Carbon dioxide ...	0.24 %	0.14 %
Methane ...	0.78 %	1.63 %
Oxygen ...	21.62 %	20.36 %
Nitrogen ...	78.36 %	77.87 %
Hygrometer ...	68° F.	73.75° F.
<i>Analysis of Coal</i>		
Moisture ...	0.6 %	0.5 %
Ash ...	3.6 %	3.0 %
Volatile matter ...	14.9 %	16.5 %
Fixed carbon ...	81.5 %	80.8 %
Sulphur ...	0.83 %	0.84 %
Phosphorus ...	0.042 %	0.007 %
Quantity of air ...	1,000 c. ft. per miner	4,920 c. ft. per miner
Velocity of air ...	17 ft. per min.	122 ft. per min.