handicapped or assisted by our hereditary equipment. What sort of transmutation does it suffer? Does it make us more, or less, capable of adaptation? If we consider these questions in relation to racial types, again we find an immense variety. We find the imaginative range of the Celt which makes him the least hopeful victim of the obsessional neuroses. There is the intellectual brilliance of the Jew; his facility in adaptation, notably economic; yet with all this, Jewry has produced an immense proportion of hysterics. We have the stolidity of the Teuton, his incapacity to see beyond the objective; and on the other hand, the instability of the Latin, with his remarkable power of blending an objective outlook with creative vision.

We could continue endlessly, and the more we study this problem of adaptation the more we realise its significance, not only for the individual, but for every group, for every community, for every race.

VERTIGO.*

BY

C. P. SYMONDS, M.D. OXON., F.R.C.P. LOND.,

ASSISTANT PHYSICIAN FOR NERVOUS DISEASES, GUY'S HOSPITAL.

VERTIGO may be quite briefly defined as a subjective sensation of instability. It is a departure from the normal sense of equilibrium, which in health is hardly perceptible, though it plays a part in the general sense of well-being. The normal sense of equilibrium is dependent upon the proper coördination of afferent impulses from nerve-endings in the retina, the labyrinth, and muscles and joints. From these three sources impulses are constantly passing upwards. They are brought into relation with one another in the brain-stem whence they exert an unconscious control over bodily position and postural tone.

Vertigo has been defined more explicitly by Hughlings Jackson as a state of consciousness of the effect upon motor functions of the want of harmony between these afferent impulses.

PATHOLOGY.

Vertigo may be caused by disease or physiological disturbance in either the sense organs and afferent nerve-fibres coming from them, or the coördinating centres themselves.

Anyone may test for himself the effect of ocular disturbance in producing vertigo as follows. Fix the eyes upon an object in the near distance and start walking towards it. While doing this, displace one eyeball inwards by pressure with the finger upon the outer canthus, and continue walking; the result is diplopia and a sense of giddiness, sometimes associated with nausea. Vertigo may also be produced experimentally by artificial stimula-

tion of the labyrinths by means of rotation or syringing the ears with hot and cold water.

In various diseases we find examples of vertigo due to diplopia, and to irritation or destruction of the labyrinth from infective, toxic, or degenerative causes. Tabes supplies an example of vertigo due to the loss of muscle and joint sense. It is interesting to compare the effects upon equilibrium of complete deprivation of any one of these special senses.

Diagram to Illustrate the Nervous Mechanism of Equilibrium.

R. = Retina. L. = Labyrinths. M.J.S. = Muscle and joint sense.
C.C. = Coördinating centres in brain-stem and cerebellum.
E.M.N. = Efferent motor neurons.

With the eyes shut a man hardly feels unsteady. Complete destruction of both labyrinths leads at first to intense giddiness, but in time this disappears. Not long ago I had the opportunity of examining a man who had suffered from an acute infection of both labyrinths, for which, in order to save his life, a complete double labyrinthectomy had been performed. To my surprise he was able to walk firmly and steadily with no complaint of giddiness, and could continue to do this even when his eyes were blindfolded. These observations lead to the conclusion that muscle and joint sense is the most important leg of the tripod which supports equilibrium. This explains why the tabetic, who has lost sense of position and of passive movement in the lower limbs, is unsteady with his eyes open; when they are shut he falls. It is also clear from such observations that the coördinating centres have considerable powers of adaptation. The vertigo produced by diplopia, by labyrinthine destruction, and even by disease of the posterior columns in the cord varies with the suddenness with which the lesion occurs. For disturbance of ocular balance and loss of labyrinthine function the nerve-centres can apparently compensate completely if given time.

Failure of the coördinating centres themselves may be caused by anoxæmia or by destructive lesions. Common examples of the former are the vertigo experienced upon getting out of bed following an illness, that which occurs in aviators at high altitudes, or that felt as the result of confinement in a hot and stuffy room. Or a defective oxygen supply may depend upon such causes as anæmia, or the low blood pressure of Addison's disease—in which case it is of a more permanent

* A Lecture delivered before the Fellowship of Medicine on June 26th, 1925.

nature. Destructive lesions of the coördinating centres may be due to acute or chronic infections, vascular disease, or new growths.

CLINICAL VARIETIES OF VERTIGO.

Although disturbance of vision is an uncommon cause of vertigo, it should invariably be looked for. I have met with this complaint as one of the earliest signs of ocular palsy, and I can well imagine that it might be caused by a defective muscle balance or gross error of refraction.

The vertigo of tabes is characteristic. To some extent the patient learns to compensate for the loss of postural sense by means of vision. Deprived of this he becomes immediately aware of instability; hence the typical story of the man who falls while washing his face, and staggers in a dim light. By the time that vertigo becomes a permanent symptom of tabes associated symptoms and signs are, as a rule, unmistakable in the form of lightning pains, pupillary changes, and loss of tendon-jerks.

Of the peripheral causes of vertigo, labyrinthine disease is without doubt the commonest. Gowers stated that in his experience 90 per cent. of all patients complaining of vertigo were suffering from this cause. Fortunately for purposes of diagnosis (though not for the patient) the labyrinth and cochlea are so closely connected that in almost all cases of disease both structures are simultaneously affected. Hence the association with labyrinthine vertigo of tinnitus and deafness, the triad of symptoms constituting Ménière's syndrome. This term I use designedly rather than "Ménière's disease." The story is an old one, but since so many people speak of Ménière's disease, it seems worth repeating: Ménière did not describe a disease, but recorded his observations, in ten cases, of the association of these three symptoms—tinnitus, deafness, and vertigo. In one case the symptoms appeared acutely, and the patient died. At autopsy Ménière found what he supposed was a hæmorrhage into the labyrinth. As the result of his paper others jumped to the quite unjustifiable conclusion that in the remaining nine cases the cause was the same, and began to speak of Ménière's disease as being due to labyrinthine hæmorrhage. Actually the symptoms are due to the situation of the disease, not its nature. Any irritative or destructive disease of the internal ear will give rise to Ménière's syndrome. In Ménière's fatal case the cause was probably not a hæmorrhage, but an acute hæmorrhagic infection. Similar cases have since been recorded. Chronic infection from otitis media may produce intermittent symptoms of the same kind. Certain poisons may also produce the syndrome, notably quinine and salicylates; or it may be caused mechanically by a blocked Eustachian tube, or impacted wax in the meatus.

But by far the commonest cause of Ménière's syndrome is that which at present remains obscure. In the absence of any other signs of local disease the symptoms appear; deafness is progressive, tinnitus intermittent, vertigo, as a rule, paroxysmal. The

patient is frequently seized without warning, and usually gives an account either of objects moving around him or a sensation of himself spinning round. The attack may last for a few minutes or for several hours. It is often accompanied by nausea and sweating, sometimes by vomiting. After the attack the patient may feel unsteady for several days.

Several suggestions have been made as to the cause of this the commonest variety of Ménière's syndrome. It has been suggested on the one hand that it is due to some unknown toxæmia, possibly of a gouty nature; on the other hand, that there is in the internal ear a condition analogous to glaucoma, in which the pressure of the endolymph is increased leading to pressure atrophy of the nerve-endings with bouts of irritation. Yet another theory supposes that in some cases degeneration of nerve-endings is secondary to arterial thickening with consequent obstruction of the blood-supply.

Whatever the pathology, it is a most intractable complaint. There are two possible lines of treatment—palliative and radical. The former aims at damping the excitability of the vestibular nerve-endings by means of sedatives. Amongst these luminal is undoubtedly the most valuable, and can be taken easily, as a rule, without any unpleasant symptoms in a tablet of half to one grain night and morning.

CASE 1.—A man of 38, perfectly healthy in other respects, began in 1916 to suffer from tinnitus in the left ear. This symptom recurred at intervals for the next seven years, and then became persistent. About the same time he discovered himself unable to hear the telephone with the receiver to the affected ear. In 1924 he began to have attacks of giddiness with nausea and vomiting. These would occur quite suddenly and at varying times of day, lasting sometimes for a few minutes, sometimes for several hours. During a severe attack he was obliged to lie down, and the least movement of his head would cause vomiting. In February, 1925, attacks were of daily incidence. On examination the only physical signs were a slight degree of nerve deafness on the left, and a little nystagmus on looking to the right. In walking he inclined to deviate to the left. He was advised to take 1 gr. of luminal night and morning, and from the time he commenced this he was free from attacks for six weeks. During the year that has since elapsed he has had occasional attacks. Some of them have been severe, but they have never been so frequent as they were before he began taking the luminal. He continues to take this regularly without any unpleasant effect, save an increased tendency to fall asleep if he rests after the midday meal.

Radical treatment aims at abolishing the attacks of vertigo by destruction of the irritable nerve-endings. This involves a severe operation and complete deafness. The latter is of little account since it is only an anticipation of that which is inevitably brought about by the disease. The severity of the operation is the chief objection, and to this may be added some uncertainty as to the result. In some cases, however, it appears to be a justifiable experiment.

CASE 2.—A single woman, who had had otorrhœa as a child, and had a strong family history of migraine, began at 34 to have attacks of giddiness. She described it as a feeling as if she were moving up and down. This would occur at any time of day and sometimes in bed at night. Later the attacks became more severe, and were sometimes

associated with vomiting. Occasionally objects appeared to be whirling round her from right to left. Once or twice she fell at the onset of an attack. She also had occasional clicking, booming, or hissing noises in her ears. When seen at the age of 38 she presented no physical signs except nerve deafness on the left. The attacks were threatening her livelihood. She therefore decided to submit to operation. The left labyrinth was extirpated in July, 1924. She returned to her work in the following October, and has since been free from attacks.

The diagnosis of Ménière's syndrome is easy, provided that careful inquiry is made for a history of tinnitus and deafness in all cases of paroxysmal vertigo. The detailed description of whirling usually given by the patient is often a valuable clue. The association with nausea sometimes leads to the erroneous opinion that the vertigo is due to some gastro-intestinal disturbance. Patients themselves often interpret their symptoms in this manner. Gowers from his personal experience was inclined to doubt the existence of a true gastric vertigo, and there is no doubt that many of the cases so diagnosed are really instances of Ménière's syndrome.

When Ménière's syndrome has been recognised the cause has still to be sought. The common variety of obscure origin already referred to can only be diagnosed by the exclusion of other causes. The ears and nasopharynx should be examined by an aurist to exclude any mechanical condition or chronic infection which may be amenable to treatment. The functions of the other cranial nerves should also be examined. Ménière's syndrome may be caused by disease of the eighth nerve in its intracranial course, either by a tumour or a syphilitic meningitis, but in this case other nerves in the cerebello-pontine angle are usually involved—particularly the fifth and seventh.

When no cause for vertigo can be found in the peripheral sense organs or the afferent pathways from them we have to look for disturbances of the coördinating centres themselves. These centres, whose functions have recently been investigated so ably by Magnus and his fellow-workers, are clearly of an extremely complex and delicate nature. Their functional stability varies greatly in different individuals and they appear to be very susceptible to anoxæmia. Examples of the latter susceptibility have already been mentioned. The giddiness experienced on rising from the stooping posture is another familiar example. Cerebral anoxæmia from disease may produce the same symptom—as in increased intracranial pressure, cerebral arteriosclerosis, and severe anæmia.

Individual variations in the stability of the centres are a matter of great interest, especially upon a sea voyage. Sea-sickness is probably due not to any abnormal excitability of the labyrinths, but to failure of the coördinating centres to compensate for the labyrinthine over-stimulation. In some persons travelling by train is enough to produce the disturbance. I have been impressed by the frequency with which a personal or family history of migraine may be obtained in these cases. There is certainly some close relationship between migraine and vertigo, and I believe that in a

migrainous subject paroxysmal vertigo may occur in association with, or as an equivalent of, a true migraine attack.

It is in these cases that luminal is particularly effective as a preventive.

CASE 3.—A child of 13 was seen on account of giddy attacks. Her mother and maternal grandmother were subject to typical attacks of migraine. The patient, an only child, began as an infant to suffer from "bilious attacks" at irregular intervals of months. The attack would begin with a slight headache; vomiting would ensue and last almost continuously for 24 hours. As she grew up it was found that she could not travel in a tube, tram, train, or bus without vomiting. After the age of 9 the bilious attacks ceased. She then began to have attacks which were described as follows:—

"She wakes early in the morning with a headache. This continues until she suddenly feels giddy, as if her head were turning round, with a tendency to fall backwards. This lasts ten minutes or so, and is associated with nausea, sometimes with vomiting. After this the headache is relieved. There is never any disturbance of consciousness during the attacks. They occur at irregular intervals—once a week or once a month."

She presented no physical signs. Half a grain of luminal was prescribed night and morning and a favourable prognosis given. I heard six months later that since commencing this treatment she had not had another attack.

Organic damage to the coördinating centres as from vascular lesions, abscess, or tumour needs no comment, being always associated with the signs of organic nervous disease, such as headache, papilloedema, incoördination of the limbs, or alteration of the reflexes.

Finally, no discussion of vertigo is complete without a word or two concerning the differential diagnosis of true vertigo from epilepsy and from certain emotional states.

In minor epilepsy an aura of vertigo is quite common. It may even be associated with tinnitus. The distinguishing feature in epilepsy is the disturbance of consciousness. But the patient may be unaware of this, and may only remember the aura of giddiness. Therefore in a doubtful case the evidence of an eye-witness should always be taken. An account of a momentarily dazed mental state or slight lapse of behaviour is often sufficient to decide the diagnosis.

In states of morbid anxiety and depression giddiness is a frequent complaint, and must be included under Hughlings Jackson's definition of vertigo. Here, however, the trouble is at the upper end of the string—it is the state of consciousness which is disturbed. On analysis the symptom is less concrete and is described in terms of psychological rather than physiological reference. The complaint is of a constant sense of insecurity and fear of falling. The patient is unable to walk by himself, but is steady enough with a friend beside him, cannot go out into the street, but is well able to negotiate stairs and passages in the house. The sensation is constant rather than paroxysmal. Proper investigation will reveal the nature of the trouble, both by excluding disturbances at a lower level and by disclosing the associated emotional disorder, which should then be subjected to psychotherapy.