REFRESHER COURSE FOR GENERAL PRACTITIONERS

VERTIGO

BY

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Vertigo is a common and often disturbing symptom in which the senses are deceived into seeing or feeling movement. The patient is likely to refer to this hallucination of movement as "giddiness" or, if slight, "dizziness." Although the term "vertigo" suggests by derivation a "turning," it can quite properly be applied to apparent movement in any direction. There is no need to postulate a turning or whirling movement, nor in consequence is it necessary to add the terms "true" or "false." Vertigo is always accompanied by a disturbance of balance which usually causes the sufferer to seek support, and if sudden and severe may throw him to the ground. It may even occur at rest, making him clutch at his chair or the bedclothes for added support. Vague disturbances of balance without any sensation of movement, particularly when associated with heights, flights of stairs, and edges of railway platforms, are rarely accompanied by signs of organic disease and should not be called vertigo.

Though the symptom of vertigo leads so often to an inquiry into the efficiency of the cardiovascular system, the digestion, the condition of the eyes, and even the state of the endocrine organs, it will be found that in most cases of severe vertigo some part of the vestibular system is at fault. If the central nervous system is normal a cause of the vertigo is likely to be found in the peripheral part of the vestibular system in the labyrinth or eighth nerve. This is not really surprising when we recall that the vestibular apparatus in the labyrinth is the receptor organ for the sense of balance. As long ago as 1893 Gowers in his *Diseases of the Nervous System* observed :

"Of 106 consecutive cases in which definite vertigo made the patient seek advice, in no less than 94, ear symptoms were present. . . . We have seen that 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 such 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."

The Vestibular System

As, therefore, vertigo so often implies some interference with the proper working of the vestibular system, either within the labyrinth or with its central connexions in the brain, it will be useful at this stage to refer briefly to the form and function of this system. It includes the vestibular end-organ housed in the posterior part of the labyrinth, the vestibular division of the eighth nerve, and the extensive central connexions in the brain stem with the cerebellum, the nuclei of the eye muscles, and the vestibulo-spinal tract of the spinal cord. The vestibular nuclei in the brain stem lie near the vagal centres, and it is not surprising to find that a prominent, disturbing, and often misleading feature of a vestibular disorder is the vagal effect which is discussed below.

The vestibular end-organ in the higher orders of the animal kingdom shares the bony labyrinth with the organ

of hearing ; consequently deafness, tinnitus, and vertigo are often seen together and usually indicate a labyrinthine disorder.

In normal circumstances man is not accustomed to using his labyrinths for balancing. He finds that with his feet firmly planted on the ground in well-lighted surroundings visual and skin-muscle-joint impressions enable him to balance without his labyrinth. This is why, when he has to use it, as when swimming or riding a bicycle, he feels insecure until he has had plenty of practice.

Here then is an elaborate and delicate sense organ which in our waking and moving hours is constantly receiving and transmitting impressions of the very existence of which we are quite unaware, until it goes wrong. Its object is to assist in maintaining balance by stabilizing the position of the eyes, head, and body in that order of importance. It reacts to the slightest movement or change in posture either by a movement of the fluid endolymph in the semicircular canals or by a shift of the otoliths. Impulses are constantly flowing up each vestibular nerve in such a manner as to balance one another, travelling to the eye nuclei, to the cerebellum, and to the body muscles without impinging upon consciousness.

Acute Vestibular Failure

Any lack of harmony between the two labyrinths will result in a disturbance of balance, and the most severe upset will follow sudden and complete loss of function on one side. The effect of this can be summarized as follows :

(1) *Vertigo*.—Objects appear to move in one direction and the subject feels he is moving or turning in the opposite direction.

(2) *Nystagmus*.—The eyes move slowly in one direction and quickly flick back to their former position. This alternate and rhythmic oscillation of the eyes may be rotary, horizontal, or vertical.

(3) *Overbalancing*.—The patient falls or deviates in the direction of the apparent movement (which is towards the affected labyrinth).

(4) *Vagal*.—Under this heading are included the variable and often misleading associated features of excessive endolymph movement, or of sudden and severe interference with the normal stream of impulses from one or other labyrinth. The pallor, nausea, and vomiting, the slow thready pulse, and the fall of blood pressure are all due to a disturbance spreading from the vestibular nuclei in the brain stem to adjacent vagal centres. Additional features of such a disturbance may include epigastric pain from vomiting on an empty stomach, activity of the biliary apparatus resulting in the vomiting of bile, and in an unfortunate few excessive intestinal movement causing diarrhoea.

The sudden failure of one labyrinth may be likened to the effect produced when one engine of a twin-engined aeroplane fails. The plane is driven round by the normally running engine and great confusion may ensue before, by suitable adjustments to the controls, the pilot is able to bring the plane back to its former course. He will, in all probability, be able to hold it more or less on a straight course, though at first this may be difficult and even tiring. In some respects an even more disturbing event would be if the defective engine, rather than stopping altogether, spluttered and functioned intermittently or gave out when the nose of the plane was pushed up or down.

The same sort of thing happens in man with sudden vestibular failure on one side. This is followed by intense vertigo, collapse, nystagmus, and vomiting. These distressing features slowly disappear as central compensation is

established, and after some days the patient is able to creep cautiously about and gradually to regain his balance. If, however, the labyrinth fails only momentarily or with the head in a certain position, the effect should not be so devastating and may last only a few minutes or hours. Nevertheless, these attacks, if recurrent, may engender such a feeling of insecurity that the sufferer may feel compelled to limit his activities.

These effects take place when the vestibular end-organs in the labyrinth have been overstimulated by excessive movement, as in motion sickness, or when normal activity is impaired as the result of injury or disease affecting one or other labyrinth. In the extreme case of sudden and complete unilateral vestibular failure the effect is startling, and it may be difficult to persuade the patient, his friends, and even his medical attendant that a disordered labyrinth is responsible for all the trouble.

Actually the overwhelming giddiness, the awful sickness, and the turbulent eye movement—all enhanced by the slightest movement of the head—that accompany acute vestibular failure form a picture of helpless misery that has few parallels in the whole field of injury or disease.

This lurid clinical scene, typical though it is of acute vestibular failure, as a rule only follows sudden and complete loss of function in a previously active end-organ. When the lesion is incomplete, and particularly when it is gradual in onset, the symptoms are less intense.

Diagnosis

Vertigo is such a disturbing complaint that both patient and doctor will naturally wonder whether there is any serious underlying cause for the trouble. Though custom has led to the earliest inquiries being made into the state of the cardiovascular and digestive systems, it will be found that when vertigo is a prominent symptom these systems are rarely affected, though in cardiovascular disease minor forms of dizziness are often encountered. The visual hallucination of movement may lead to the suspicion of an ocular cause, though here again the eyes are usually innocent. The menopause may sometimes be held responsible because of the transient dizziness that is often experienced at this time.

This leaves the central nervous system, including the vestibular nerve and the vestibular end-organ in the labyrinth, as possible sites for disease causing vertigo. As it is necessary to find out whether there is any serious underlying cause for the vertigo it will be convenient to consider first of all the causes for vertigo that may be found within the central nervous system.

Central Vertigo

Any involvement of the central vestibular connexions, particularly if it is sudden, may lead to vertigo. The most likely causes are a vascular lesion or a patch of sclerosis. Tumours, being more gradual in their onset, are not so likely to produce severe vertigo. These disorders are rarely limited to the vestibular tracts in the central nervous system, so that associated symptoms will suggest the true nature of the condition. In disseminated sclerosis the picture may sometimes be one of acute vestibular failure, but there is usually some accompanying sensory disturbance and diplopia. An epileptic fit may be preceded by a vertiginous aura, but loss of consciousness, which is rarely seen in labyrinthine vertigo, offers a clue to the cause. Tumours in the posterior cranial fossa, particularly if they involve the roof nuclei in the cerebellum, may cause postural vertigo and nystagmus; whilst tumours pressing on the vestibular nuclei in the brain stem will usually cause nystagmus and a varying degree of dizziness. The commonest of these tumours is a neurofibroma of the acoustic nerve, and though the vestibular nerve is always involved, with loss of peripheral vestibular function, vertigo is rarely a prominent symptom, probably because the gradual loss of vestibular function has enabled compensation to take place. Arachnoiditis in the posterior fossa may be the reason for vertigo and it may simulate an acoustic neurofibroma.

Thus when vertigo is associated with loss of consciousness, diplopia, sensory disturbances, failing vision, inco-ordination of movements, or persistent spontaneous nystagmus, it is necessary to search for a possible cause within the central nervous system. Spontaneous nystagmus due to a labyrinthine lesion never lasts for more than three weeks, and a vertical nystagmus always means a lesion within the central nervous system. Apart from an acoustic neurofibroma, it is unusual, though not impossible, for deafness to be associated with vertigo due to central causes.

Peripheral Vertigo

Most of the patients in whom severe vertigo is the presenting symptom will be found to have a lesion in the peripheral part of the vestibular system, usually within the labyrinth. It has been customary to refer to this group as aural vertigo, or Ménière's syndrome, in honour of the French physician who first drew attention to the labyrinth as the seat of the disorder responsible for paroxysmal attacks of vertigo usually accompanied by unilateral deafness and tinnitus. Attacks of vertigo without deafness earned the sobriquet pseudo-Ménière's syndrome. Under these headings there has been grouped, irrespective of their pathology, a variety of conditions, most of them otological, and it will be useful to consider them under headings based on their pathology.

Hydrops of the Labyrinth.—Hallpike and Cairns in 1938 found that the pathological process responsible for paroxysmal attacks of vertigo accompanied by unilateral deafness and tinnitus, where the ears appeared normal and there were no signs of disease within the central nervous system, was an obstructive distension of the endolymphatic system. They called this Ménière's disease, and the additional descriptive subtitle of endolymphatic hydrops has since been added. The sexes are equally affected, and in more than half the cases the symptoms first appear before the age of 50. The attacks of vertigo may be preceded by deafness and tinnitus or may occur without warning and at any time, even when asleep. The vertigo can be violent and may last for some hours, often being accompanied by severe vomiting. After a rest and possibly a good sleep, recovery is the rule. Attacks often occur in groups at intervals of days or weeks with periods of complete freedom for months or years. The hearing in the affected ear is usually and significantly distorted so that loud, musical, and high-pitched sounds are distressing. When examination reveals a normal-looking auditory apparatus but perceptive (nerve) deafness and an impaired response to a caloric test of vestibular function, without any sign of disease within the central nervous system, the diagnosis of hydrops is established. A careful examination of both cochlear and vestibular function is necessary to locate the lesion, which is usually in one labyrinth, though in about 15% of cases both are obviously affected.

Injury.—The labyrinth is very susceptible to damage in a head injury, either by a skull fracture involving the labyrinth or following severe concussion when the delicate membranous labyrinth is shaken up and suffers damage which may be permanent. That is why giddiness is a prominent feature of the post-concussion syndrome and of some basal skull fractures. In such cases examination will usually reveal a vestibular defect. After the obvious effects of the injury have passed off persistent dizziness and a disturbance of balance should lead to a suspicion of vestibular damage, even if there is no deafness. In some cases vertigo may be induced or aggravated by movements of the head or by holding the head in a certain position, usually backwards and to one side.

Infection.—In labyrinthitis secondary to otitis media the infection will spread inwards, either through natural preformed spaces in acute infections or more commonly by erosion of the bony labyrinth in chronic suppuration. In the latter event the final invasion may be preceded for some time by slight dizziness which is accentuated by pressure on the ear, the so-called fistula sign. This warning sign in chronic suppurative otitis media calls for early operative treatment if the labyrinth and sometimes life are to be saved. Once the labyrinth is invaded by infection all the features of acute labyrinthine failure will be present, and will be likely to continue for some days. There will also be some accompanying fever and constitutional disturbance, with the prospect of the infection spreading to the meninges. In meningococcal meningitis, infection may spread outwards from the meninges to the labyrinth, though this complication is often overlooked at the time because of the severity of the meningitis, and it may be revealed only by residual deafness and some disturbance of

balance. Unfortunately neurolabyrinthitis is often bilateral, in which case total deafness results. *Mumps* may also be complicated by neurolabyrinthitis, though here fortunately only one labyrinth is likely to be affected. Here, again, the symptoms of acute labyrinthine failure are usually attributed to the causal infection. In another small group there are signs of acute vestibular failure accompanied by facial palsy and possibly a vesicular eruption around the affected ear, the so-called *geniculate herpes* or Ramsay Hunt syndrome.

Toxic.—The vestibular system is undoubtedly vulnerable to certain toxins, in particular to streptomycin. This may cause permanent impairment of vestibular function, both labyrinths usually being affected. In an effort to overcome this selective toxic action of streptomycin, the dihydro compound was introduced. Unfortunately this has proved to have a similar effect on the cochlear apparatus, so it seems that until a non-toxic compound has been found it is wiser to use the original brand cautiously, as it is obviously better to be giddy than deaf. In another and not uncommon group there is a sudden and inexplicable loss of vestibular function on one side, with all the clinical features of acute vestibular failure. Hearing is unaffected, and it seems that the site of the lesion is somewhere in the vestibular neurons. Dix and Hallpike refer to this condition as *vestibular neuronitis*, and they have found that in a proportion of cases there is an active focus of sepsis which may be responsible. The condition is easily recognized as one of acute vestibular failure by the sudden onset of severe vertigo accompanied usually by vomiting, which demands absolute rest in bed, as the slightest movement of the head or body causes further vertigo. The symptoms slowly subside over the days, and after two or three weeks all is well apart from a little residual dizziness with sudden movements of the head. It is probably the cause of so-called "epidemic labyrinthitis," though in many cases there is no evidence of an infection.

Paroxysmal Positional Vertigo.—In this group of cases vertigo is induced only with the head in a certain position, usually when it is placed backwards and to one side. It will be found that in such cases the paroxysm of vertigo lasts only a few seconds, and does not reappear for at least half an hour, even when the offending position is resumed. Dix and Hallpike have found a lesion in the utricular labyrinth in such a case. This condition is benign and may disappear after a few months. In some cases there is a history of a head injury while in others a focus of sepsis has been demonstrated. It has to be distinguished from the central form in which fatigue does not occur.

Some idea of the relative frequency of these different causes of vertigo can be gained from the following figures, which show the various cases in a series of 1,523 patients referred for an otological examination because of vertigo:

Central	..	..	..	..	..	107
Peripheral	..	..	..	..	..	1,349
Hydrops	..	..	..	..	..	972
Trauma	..	..	..	..	..	123
Vestibular neuronitis	..	..	..	..	..	109
Positional	..	..	..	..	..	84
Infective	..	..	..	..	..	61
Not Classified	..	..	..	..	..	67

It will be appreciated that the proportion of central cases in this series is smaller than a general neurological experience would suggest, though it corresponds with Gowers's findings.

Management

The first step in the management of a case of vertigo is to exclude, if possible, any central cause. If this can be done, then it is important to ensure that the patient appreciates that the underlying cause of the condition is benign. If the explanation offered is acceptable—and it is usually thankfully received—it dispels many fears and doubts and is an important step in rehabilitating the patient.

Any infective ear condition must be dealt with, while for the remainder it is important to get and to keep them moving once the acute stage is past. Special balancing exercises are invaluable for traumatic cases, and these are most successful when carried out in a class.

During an attack of acute vestibular failure absolute rest and strong sedatives are needed. Vomiting may call for an injection, the most effective being inj. phenobarbiton. sod. 3 gr. (0.2 g.). In Ménière's disease a course of one of the barbiturates, though it does not have any specific effect, has a generally calming influence, and to a certain extent modifies the attacks. Certain seasick remedies containing hyoscine or an antihistaminic may be very useful in modify-

ing the severity of the attacks, and are often helpful in traumatic cases in which the dizziness may be more or less constant.

In Ménière's disease a serious attempt should be made to limit the intake of sodium, and a low sodium diet should be ordered. Discouraging the retention of fluid within the body often seems to ward off attacks. In the hope of influencing the blood supply of the labyrinth histamine, nicotinic acid, and operations on or injections into the cervical sympathetic are sometimes tried. Such measures may help some cases, but it is difficult to assess the value of any conservative treatment in a condition such as hydrops of the labyrinth in which long periods of spontaneous remission are apt to occur. When the attacks are not controlled by conservative measures, and when the disease is unilateral and the hearing is badly affected, some form of operation on the labyrinth may have to be considered. Opening the bony external semicircular canal and removing a portion of the membranous labyrinth is enough, in Ménière's disease, to destroy all function and prevent any further attacks from that labyrinth. It is simple in terms of general surgical principles, there need not be any complications, and recovery is quick.

In bilateral cases, and when the hearing in the affected ear is still useful, intracranial section of the vestibular nerve may be considered, though of course this is a more extensive and potentially a more serious operation than labyrinthectomy. Deliberate intoxication of the vestibular labyrinth with streptomycin has been advocated, but experience suggests that in some instances the cure may be worse than the disease, and such a form of treatment should be considered only in relatively young patients in whom the condition is bilateral.

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NURSE-TRAINING SCHEME FOR EX-TUBERCULOSIS PATIENTS

BY

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There are probably very few sanatoria which do not employ some ex-patients on their staff, and no doubt many have already developed their own schemes for employing ex-patients as nurses. The experience of the scheme at Creton Sanatorium, Northamptonshire, may be of interest to those concerned with tuberculosis and the administration of sanatoria.

There are 152 beds at Creton Sanatorium, 91 for males and 61 for females. The establishment allows for 40 on the nursing staff, but the total number of nurses employed is at present 34. When investigating the possibility of meeting the nursing shortage by the employment of ex-tuberculosis patients we found that though a valuable contribution to staffing difficulties was made by the casual or incidental employment, in various capacities, of ex-tuberculosis patients, much better results, both for the sanatorium and for the ex-patient, could be anticipated if employment were put upon a more regular and permanent basis. Further, although it was probable that without any action at all by the sanatorium authorities a few ex-patients would seek employment