The Journal of
Laryngology and Otology
(*Founded in* 1887 *by* MORELL MACKENZIE *and* NORRIS WOLFENDEN)
July 1929

VERTIGO.*

By SYDNEY SCOTT, F.R.C.S.

THE scientific study of vertigo began with the researches of Flourens a hundred years ago. The work still continues, and yet much remains for the clinician to study and learn, and for future researchers to survey and explain. The symptoms of vertigo are well known. Patients who have had both labyrinths completely removed, and deaf-mutes who have complete loss of function of the vestibular nerves, do not suffer from vertigo. Patients who have bilateral auditory nerve tumours which implicate the whole of each eighth nerve cannot be made giddy by rotation or galvanic stimulation, even if we employ a trans-cephalic current of 20 milliamperes.

On the other hand, normal individuals can become giddy without any physical disturbance of the labyrinth, as shown by the tests with a prism before one eye, which Dr Hughlings Jackson and Mr Paton described.

After walking with a prism held before one eye, while the other eye is closed, the prism is suddenly removed, the individual reels to one side. Again, when walking with one eye closed, while the open eyeball is momentarily displaced digitally, release of the pressure causes a feeling of vertigo, accompanied by stagger and nausea. Yet these are not common causes of spontaneous vertigo. They serve to remind us that every case of vertigo is not necessarily labyrinthine in origin, but that other factors which disturb muscle sense and produce want of

* Paper read in opening a discussion on Vertigo at the meeting of the Section of Otology, Royal Society of Medicine, London, 1st February 1929.

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press

harmony between afferent impulses from ocular-motor tracks may also cause vertigo. So, modifying Hughlings Jackson's definition, we will define vertigo as the state of consciousness of a false sense of orientation of ourselves in relation to our environment. The individual affected by vertigo feels "as if" he is moving, or "as if" his surroundings move at a certain rate or in a certain direction, while realising that such inferences are really erroneous. He will say he felt "as if" he were being whirled, or "as if" falling in this or that direction, or "as if" the room or surroundings were going round and round, or over and over.

Such sensations may last a few moments, or persist for hours, to recur with variations. When these symptoms are slight in intensity they are often spoken of as feelings of dizziness or giddiness, and it is convenient to use these terms as representing lesser forms of vertigo.

Sir William Gowers and Dr J. A. Ormerod always maintained that vertigo was aural in origin in over 90 per cent. of cases. Others have said it is almost invariably due to some disturbance in the ears. The term "gastric vertigo" indicates the frequency with which gastric disturbances may prove prominent features, but it would be an error to look upon the gastric disturbance as a cause of vertigo, in the light of Gower's teaching, and from what we know from the work of Holmes and other modern neurologists.

By over-stimulating the labyrinth in normal subjects we can induce not only feelings of vertigo, but symptoms of visual and ocular disturbance, gastric disturbance, cardiovascular symptoms, flushing, pallor, signs of collapse, all of which are secondary reflex results of the excessive stimulation of the vestibular centres in the medulla, and not causes of giddiness.

We have seen, with Sir Henry Head, cases of spontaneous vertigo, with dyspnœa, due to aural disturbances, also of diarrhœa after severe vomiting, following otherwise typical attacks.

The diagnosis must present difficulties to some, for I have seen a patient who submitted to gastro-jejunostomy in order to cure attacks of vomiting which were subsequently proved to be due to aural vertigo. We have seen the appendix removed for similar reasons, which, too, proved erroneous.

In the course of clinical practice we find that giddiness is associated with different disorders of the ear. As otologists

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press

we connect vertigo with labyrinthitis because we realise the great importance of making a correct diagnosis so that we can act upon it early in order to save life. Therefore we would point out that vertigo and nystagmus sometimes occur in acute otitis media before the tympanic membrane has been opened. This condition, as proved by physiological tests, is not due to invasion of the labyrinth. But we must also avoid overlooking invasion of the labyrinth, a danger often leading extremely rapidly to fatal lepto-meningitis. In such cases spontaneous nystagmus, which is present at first on deviation of eyes to the same side, soon changes over to nystagmus directed to the opposite side, and caloric tests give no reaction.

Not long ago I saw a patient with early acute otitis media who was intensely giddy and had nystagmus to the opposite side. Her doctor, who was also an aural surgeon, was convinced it was purely hysterical. After carrying out tests, I urged him to operate on the labyrinth without delay, for the affected labyrinth was defunct. Afterwards I heard that he did not take my advice until the patient developed marked clinical signs of meningitis, when an operation proved, I regret to say, too late. We must be careful to guard against the mistake in diagnosing labyrinthitis as "pure hysteria," for a patient with vertigo is likely to become hysterical. What we should do is to seek the cause of hysterical manifestations.

Erosion of the labyrinth may occur, after some weeks in acute, and also in chronic otitis media, especially when cholesteatomatous accumulations are present.

Apart from otitis media, we meet with attacks of vertigo in patients who suffer from otosclerosis, especially in the early stages, and the disease is perhaps more frequent when the signs of otosclerosis are unilateral, or more advanced on one side than on the other.

And now we come to a class which, in my experience clinically, is the largest class—recurrent vertigo associated with inefficiency of the Eustachian tube mechanism. If we examine patients liable to giddiness with sufficient care, we discover that Eustachian inefficiency is present on one side or the other far more frequently than many have suspected. Here I would recall that the late Mr Mark Hovell, at the London Hospital, noticed that the great majority of patients who were referred to him with vertigo without suppurating ears had nasal or post-nasal catarrh. Also Dr Mayer, who read a paper before

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press

this Section a year or so ago, mentioned the frequent association of Ménière's disease with nasal and post-nasal catarrh.

I presume we agree that Ménière's symptom-complex is not due to hæmorrhage into the labyrinth, and that when indeed spontaneous hæmorrhage does occur, it is extremely rare, whereas the symptoms of Ménière's disease are common in aural practice. As I have given my reasons elsewhere, I will only repeat that I am still firmly convinced that Ménière's fatal case was one of infective labyrinthitis which occurred in a patient whose tympanic membrane did not perforate, and whose death was due to so-called meningitis serosa. We have seen and described parallel cases.

Ménière's symptoms are met with in the Air Force, and in the Navy, in submarines, and caisson divers. During the Great War we found invariably that vertigo and nausea occurring in an airman immediately he landed after high flying, were associated with some degree of invagination of the tympanic membrane from the excess of air pressure in the meatus, resulting from obstruction of the Eustachian tube. Dr James L. Birley (then Lt.-Col. Birley), who was chief medical officer to the R.A.F. in France, suspected this, before it was definitely proved to be so. We found that when both Eustachian tubes were inefficient, and both tympanic membranes were compressed inwards, there was deafness without vertigo, but when only one Eustachian tube was obstructed, then the deafness was not noticed, while the symptoms of vertigo were prominent, due to the heterogeneous stimuli. This is most important in the case of an air pilot, for it must be remembered that vertigo in the air is associated with reflex forced movements, and a pilot will be seen sometimes trying to land with one wing down, quite unintentionally, when he thinks he is on a dead level keel. From my experience with the Air Force, I have no doubt whatever that one cause of many fatal spins resulted from the reflex forced movements with vertigo due to unilateral Eustachian obstruction. The prevention and treatment are self-evident. All pilots should be capable of inflating both Eustachian tubes by Valsalva's method, or else of opening the Eustachian tubes by the act of swallowing, otherwise they should not risk high flying.

What happens to airmen can also happen to submarine and caisson divers. During the period of artificially increased compression of the atmosphere, Valsalva's test should be

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press

applied if swallowing fails to relieve the feeling of increased pressure on the tympanic membranes. But air can be absorbed from the tympanic cavity without the application of pressure in cases of Eustachian obstruction.

I will give an example of a young lady patient who had attacks of giddiness. She had signs of unilateral otosclerosis. When I saw her she had no Eustachian obstruction, nor was she giddy at that time. A few days later I was called to her as she had been seized with violent giddiness when shopping. The shopman thought she was intoxicated, and sent her home in a taxicab. I found her lying on her right side, afraid to move her head. She had nystagmus on looking to the right, but not to the left. The left ear, which was the deaf one, was uppermost, and I could easily see the tympanic membrane inflating when she performed Valsalva's test. So we slowly rolled her on to her left side, and I found the right tympanic membrane displaced inwards, and immobile when she attempted Valsalva's method of self-inflation. As she remained lying on her left side, I passed a Eustachian catheter, and inflated the right ear. The nystagmus instantly disappeared, and the vertigo ceased; she sat up and telephoned to a friend to explain why she had failed to keep an appointment.

I would here emphasise that it was the *good* ear that was at fault, and I am convinced that recurrent vertigo in unilateral otosclerosis often occurs through faults on the unsuspected side. Patients often say the noises in the deaf ear are greater when they are giddy, and this complaint is apt to distract attention from the good ear.

But we must not put down every case of recurrent vertigo to actual obstruction in the Eustachian tube. We have seen vertigo in patients who, at the time, had no obstruction, but there was visible fibrillary tremor of the facial muscles on the same side. Is it not possible that the stapedius was also affected?—for certain movements of the stapes can produce giddiness. In some cases it seems highly probable that a reflex spasm—it may be tremor—occurs in the tensor tympani or stapedius.

Vertigo associated with disease of the maxillary sinuses may obviously fall into the class of those cases due to Eustachian inefficiency, but it is also possible that, in some cases, where there is no Eustachian block, the *fifth nerve* plays a reflex part with the seventh nerve, setting up disturbance in the intrinsic

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press

muscles of the ear, and, through the movements of the stapes, causing commotion in the labyrinthine fluid.

The frequent association of "dead teeth" with vertigo is very suggestive of some such trigemino-acoustic reflex. This deserves further investigation.

Lastly, we must avoid missing cases in which vertigo and nystagmus are due to syphilis, to disseminated sclerosis, syringobulbia, and other diseases of the central nervous system. But the greatest mistake, considering the tests we can now rely upon, is to attribute the cause, in a case of acute labyrinthitis, to hysteria!

I shall not attempt to cover the contentious ground of treatment. We shall here be guided, first, by whatever in our opinion is the cause; secondly, by the effect of attempts to remove that supposed cause. Personally, I regard destruction of the *intact* labyrinth only as the very last resource, and must admit feeling humiliated by previous failure whenever I have performed that operation.

Illustrative Cases.

Case I.—C. T., male, clerk, born 1870.

When aged 43 began to be troubled with deafness and tinnitus in left ear.

At age of 49 attacks of giddiness began, associated with occasional deafness in right ear. Became depressed and worried owing to the frequent repeated attacks of giddiness. Was treated medically, without benefit, for two years, then attended the National Hospital at Queen Square, and St Bartholomew's Hospital.

Aural Examination.—Drum membranes intact, but middle-ear deafness left side, and, at the time of examination, normal hearing right side. Much nasal catarrh and nasal obstruction due to deflected septum and nasal spur.

Accessory sinuses investigated; no evidence of infection found.

Patient was seen repeatedly during attacks of giddiness, and during intervals when free from vertigo; it was definitely established that the attacks were invariably associated with Eustachian inefficiency. Treatment for the catarrh was begun, nasal obstruction removed, and the Eustachian tubes inflated twice a week at the hospital. Owing to the necessity for repeated inflations, it was decided to make a small fenestration of the left tympanic membrane. This did not make him deafer but it did not prevent the recurrence of vertigo. He decided to risk its making the hearing defective in the right ear, and the right tympanic membrane was fenestrated. The attacks ceased.

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press

The nasal catarrh did not disappear until after being treated for five years. The fenestration of the tympanic membranes closed after three years. He learnt to self-inflate both ears, and now has had no return of vertigo for over four years. The hearing in the right ear is normal. The defective hearing in the left ear is the same as it was nine years ago.

CASE II.—*Hypersensitive Labyrinth.*—Mrs B. Patient had had Küster's mastoid operation performed on the right ear. The meatus is well open. She is now always giddy in a cold wind, and even when in a room if air is fanned by the side of the right ear it causes temporary giddiness, and nystagmus in less than a minute. No fistula sign present. Patient has to keep wool in the right meatus to prevent giddiness. The nystagmus induced by the wind has the usual characteristics—that is, when facing forwards the nystagmus is clockwise, and increased by deviation of the eyes to the left. There is no nystagmus "to the right."

CASE III.—*Labyrinthine Fistula.*—E. R., aged 24, kitchen-maid, admitted to hospital 8th November 1928, suffering from giddiness and vomiting.

Previous History.—On 7th October, after about two years' painless otorrhœa in the right ear, she began to suffer pain in the ear, and became giddy when she leant her head back to hang clothes on the line. For the next month she had intermittent pain and giddiness, with nausea and occasional vomiting, and a feeling of rapid turning round (direction not noticed). Admitted for investigation and treatment on 8th November.

Condition on Admission.—Well-nourished, looking tired and worried, and complaining of nausea and giddiness. Felt giddy sitting up in bed, and tended to fall backwards and to the right; lying flat greatly diminished the discomfort. Otorrhœa scanty, large perforation in drum, with granulations in posterior meatal wall. Next day vomited persistently all day, and felt wretched, but after this she steadily improved. Syringing the ear with hot or cold water caused increased giddiness and rapid nystagmus.

Mr Scott explored the mastoid antrum and middle ear. A cholesteatoma in the antrum was evacuated and the diseased incus only removed.

When the dressing was pressed upon, the eyes steadily deviated to the left, and remained there till the pressure was released, patient being still under the anæsthetic.

The next day, pressure upon the dressing caused the same deviation of the eyes to the left, followed by violent nystagmus and vertigo, which terrified her, causing her to clutch the sides of the bed to save herself, as she thought, from falling out of the bed to the left.

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press

This sensitiveness diminished steadily day by day, and she regained confidence.

Through the opening into the mastoid antrum could be seen a minute pink orifice on the inner wall of the aditus; touching this with a probe caused violent giddiness as before. Otherwise, and when at rest, she had no giddiness.

On 27th December 1928, she reported no giddiness except on movement, but this to a greatly diminished extent.

31st January 1929.—No fistula sign obtainable; she seems well, and can perform her duties again.

Case IV.—*Labyrinth destroyed. Cochlea normal.*—Mrs D. G., aged 32; housewife. Patient in the Ear Department, St Bartholomew's Hospital.

In *February* 1919, she had suffered from bilateral otorrhœa for ten years.

Aural Examination: *Right* drum showed mucous membrane slightly herniating; and the *left* drum a depression in front of the malleus and a perforation behind it.

In *August* 1919, a radical mastoid operation was performed on the *left* ear. By February 1920, both ears were dry.

May 1920.—Reported recurrence of the otorrhœa, with giddiness for three weeks. Spontaneous *nystagmus*. Slight horizontal to the *right*, gross rotary to the left; Weber to right.

June 1920.—No giddiness, but nystagmus persists. Some otorrhœa from right ear.

January 1921.—Headache, but giddiness less than formerly. Otorrhœa right ear. *Right drum* hyperæmic and shows small perforation.

May 1921.—Pain in both temporal regions and vertex for two weeks. Tinnitus in right ear; no otorrhœa.

Interval of Seven Years.—Treated medically for giddiness, etc.

9th September 1928.—Returned to Ear Department. Pains in head generalised. Hearing worse than usual.

5th November 1928.—Right drum membrane looked œdematous in upper margin. Cholesteatoma diagnosed. Operation advised.

1st January 1929.—Owing to pain in right ear she was admitted to undergo mastoid operation by Mr Sydney Scott.

On Admission.—*Ears: Left* ear dry. *Right* drum obscured by much pus. External auditory meatus œdematous.

Operation (Mr Sydney Scott).—Epitympano-mastoid, labyrinthotomy (right).

External, superior and posterior semicircular canals gone. Incus gone. The facial canal not opened. The vestibule not opened.

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press

Stapes hidden by tympanic membrane. No reaction to cooling vestibule with ethyl chloride spray.

Cochlea left intact. Malleus and part of tympanic membrane left intact.

Granulations and cells down to tip of mastoid.

Present Condition.—Useful hearing in right ear. Quite deaf in the *left* ear. *No vestibular reaction* either side.

Remarks.—It would appear evident from the history that the erosion by cholesteatoma and slowly progressive destruction of the right semicircular canal began in 1920, but the extent of the disease was not recognised, as the patient had such a small perforation and so little discharge.

https://doi.org/10.1017/S0022215100033806 Published online by Cambridge University Press