# VERTIGO

*Tuesday, June 18th*

WITH the Master of the Conference, Mr. I. SIMSON HALL, in the chair, Lord BRAIN opened a discussion on Vertigo.

He used the term vertigo to include all forms of a sensation of physical disorientation in space and would not restrict it only to a sense of turning. With unimportant exceptions it was a symptom of a disorder of the vestibular part of the internal ear or of its afferent pathway up to the cortical representation in the temporo-parietal region of the brain. In the cortical area, lesions such as neoplasms may cause vertigo but usually it was not a prominent symptom in such cases. Of greater importance was its association with epilepsy. When it occurred as the aura of an attack it did not differ from any other kind of sensory aura and had been called vestibular epilepsy. Rarely epilepsy may be associated with organic disease of the internal ear which on occasions would cause vertigo followed by a typical epileptic attack, described as vestibulogenic.

He then turned to the problem of the elderly patient complaining of various symptoms which included attacks of giddiness, usually brief and lasting for a few seconds to a minute but sometimes up to several hours. The brief attacks were frequently brought on by head movements such as rotation to one or other side, extension or flexion as in taking off a dress over the head. They could follow change of posture, particularly lying down or sitting up in bed. The patient may stagger but did not fall. The next type of attack, the "drop attack", consisted of a sudden fall without warning which might occur spontaneously or be precipitated by head movement. The patient was not aware of falling but loss of consciousness was brief and he could get up again at once. The third type of attack, which may or may not occur in patients who suffered from the other two varieties, was one of syncope. The syncope attack differed a little arbitrarily perhaps, from the drop attack in that the drop attack did not appear to occur when the patient was standing up. The syncopal attack on the other hand might occur when the patient was sitting down or standing and occurred spontaneously or could be precipitated by head movement. The speaker believed that in the great majority of cases all the types of attack he had described were of vascular origin. To understand how they were produced he described the normal cerebral vascular circulation, particularly through the brain stem, the effect of atheroma and of cervical spondylosis. The vertebro-basilar circulation, derived from



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the two vertebral arteries, supplied all structures below the tentorium and the vertebral arteries were rarely equal in size. A cinematograph film was then shown of a dissection of the vertebral arteries in the sub-occipital region of a cadaver. Rotation of the cadaver head demonstrated the large loop between the foramen in the transverse process of the axis and the foramen magnum and showed how this provided the necessary slack to prevent tension on the artery on rotation. Even in normal subjects temporary occlusion of the vessel on the contra-lateral side might occur on rotation. Atheromatous narrowing could interfere with circulation either in the subclavian artery, proximal to the vertebral artery or anywhere in the course of the vertebral arteries. Most commonly atheroma produced symptoms when it occurred with cervical spondylosis which caused pressure on the vessel from osteophytes at the neuro-central joints or kinking from disc narrowing and shortening of the cervical spine. A further cause was abnormal movements of the vertebrae, often painless, when the upper vertebrae moved forwards or backwards on the lower one during flexion or extension.

There was no doubt that vertigo, drop attacks and syncopal attacks may all be due to ischæmia of the brain stem but it was not yet possible to decide whether ischæmia of the internal ear played any part. When undertaking tests to differentiate vertebro-basilar ischæmia from labrynthine conditions such as Ménière's disease or vestibular neuronitis, it was necessary to stimulate the labyrinths without disturbing the vertebro-basilar circulation. This could only be done by rotating the patient as a whole. Cervical spondylosis and atheroma could be present without causing symptoms which were due to something else, such as a tumour. A sensitive carotid sinus could also cause vertigo or syncopal attacks.

Dr. J. R. LINDSAY (U.S.A.) read a paper on Positional Vertigo which reviewed the work done in his laboratory during the past decade and made some comments on the types of positional nystagmus found in the human. Animal experiments on the central nervous system suggested that the frontal and temporal lobes were not a likely cause for positional nystagmus. Neither did lesions of the superior or inferior colliculi in rabbits and the superior colliculi in cats produce either positional nystagmus or disturbance of caloric, rotatory or optokinetic responses. Stereotaxic lesions of the caudal mid-brain in a small series of cats showed a high incidence of direction-fixed positional nystagmus. Surgical ablation of the nodulus was followed mainly by a positional nystagmus of paroxysmal type, whilst irradiation by Palladium 109 resulted usually in type 1 positional nystagmus. The presence of peripheral labyrinthine responses were necessary for the production of nystagmus after nodulectomy.

Animal experiments on the peripheral labyrinth indicated that stimulation of the otoliths caused conjugate movement of the eyes to a



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fixed position and suppression of nystagmus. Section of the utricular nerve, or the nerve to the lateral semicircular canal, or the nerves to the utricle and superior and lateral canals combined, each produced positional nystagmus, at first type I or type II but later with latency and transitory. The presence of signals from the affected labyrinth on changes of position was necessary for the release of positional nystagmus due to a peripheral labyrinthine lesion.

Clinical observation on man indicated that in contrast to some lesions in the posterior fossa, the gradual onset of a peripheral lesion was not likely to produce a positional nystagmus. The distinction between persistent and transitory types was theoretically questionable but in practice provided useful information. Persistent types in man did not usually present latency but might do so in the cat. Paroxysmal types had been observed in man and the cat in both central and peripheral lesions. Most cases of paroxysmal type in man followed a benign type. The origin, however, was not definitely indicated by the paroxysmal character.

Dr. C. S. HALLPIKE read a paper on Ménière's Disease.*

Ménière's penetrating clinical studies provided strong evidence that the cause of the disease which has since come to bear his name is limited to the inner ear and carries no direct danger to life.

The nature of the disease process was not established by Ménière, and for further information we have had to await the results of a number of histological studies carried out in the last 25 years. These have done much to clarify our understanding of the clinical problems of vertigo. In addition, they are related in an instructive way to the impressive additions which have since been made to our knowledge of the Anatomy, Physiology and Biophysics of the Inner ear. The results of histological studies are well known and can be summarized very briefly as a distension of the endolymph system, with degenerative changes in the end organs. The cochlear and vestibular neurones are unaffected; the picture, in fact, is one of an end-organ derangement. Although the cause of the cell changes has not yet been established, the knowledge that they exist has had far reaching effects.

Thus, the clinical features of Ménière's disease have been re-studied, more closely defined and interpreted more critically in terms of organic damage to the cochlear and vestibular end organs. From this point it has been possible to build up a pattern of functional disorder characteristic of end organ disease which we could identify with confidence and put to use for its differential diagnosis from other organic affections of the VIIIth nerve system.

Among these are included tumours of or affecting the VIIIth nerve,

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vestibular neuronitis—a toxic affection of the vestibular neurones—and positional nystagmus of the so-called benign paroxysmal type.

This differentiation has called for numerous clinical and histopathological studies. In the course of these, much valuable information has been obtained relating to the physiological mechanisms which underlie a number of our best known clinical tests of hearing and equilibrium, in particular the Loudness Recruitment Test, the speech intelligibility tests and the caloric tests.

Though the cause of the histological changes described by Cairns and Hallpike in the labyrinths of their subjects with Ménière's disease has not yet been elucidated, many valuable observations have been published, particularly by the late Seymour of the Ferens Institute of Otology and by Golding-Wood. Both adduce strong arguments in favour of vascular occlusion as the primary cause of the inner ear changes.

However, other and more subtle possibilities need to be considered, particularly in view of the demonstration within recent years of the remarkable electrolyte composition of the endolymph and its high potassium/low sodium contents. This suggests that certain cell elements of the endolymph system, in particular the stria vascularis and Reissner's membrane, have as their essential function the transport across their cellular partitions of water and electrolytes, playing in this respect a role comparable to kidney tubule cells.

This approach to the problem of the pathogenesis of Ménière's disease has been systematically followed by Spencer Harrison and Naftalin of Lincoln, who have studied the effect upon the clinical manifestations of Ménière's disease of changes, induced by dietary control, in the Na and K contents of the body. They report that although they were able by these means to induce vertigo in their subjects with Ménière's disease this did not depend upon a high level of body Na. Instead, the occurrence of the vertigo was constantly related to a certain phase in which an Na diuresis was occurring in association with water retention.

The authors have related their findings in an interesting way to the homeostatic mechanisms for water and Na which are known to operate elsewhere, particularly within the kidney.

Dr. W. H. JOHNSON (Canada) read a paper on the Testing of Vestibular Function. He described how, after full audiometric examination including tests for recruitment had been carried out in the adjacent Audiological Department, the history and findings of tests conducted in the Vestibular Clinic at St. Michael's Hospital, Toronto, were recorded in a special three-page chart. The following tests were made:

(1) A check was made for the presence of spontaneous nystagmus with the patient in the upright position and the eyes looking straight ahead and to each side.



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(2) Drift reaction. The patient stood with the eyes closed and the arms raised parallel at shoulder level. Any tendency to movement of the arms or body was noted.

(3) Past pointing. Deviation from the vertical and the relationship to the slow phase of any existing nystagmus was noted. Perversion of response aroused suspicion of a lesion involving the central pathways. At the same time dysdiadochokinesis of the hands was tested.

(4) Walking tests with the eyes open and closed. In addition the ability to walk on a spring mattress was of value to detect minor vestibular deficiencies.

(5) Rhomberg Sign. Observation was made to determine if the direction of falling was influenced by the head position.

(6) Spontaneous Nystagmus was observed not only as in the first test but with the patient lying on a litter mounted on gimbals. The patient could be moved to any position at a subliminal rate so as to prevent stimulation of the semicircular canals. The response was compared with the effects of rapid movements to the same position.

If a continuous nystagmus, which could be repeated with further tests, was produced by tilting, the cause was central and if the nystagmus was rapidly fatigable it was thought to be due to an end-organ lesion.

McNally's tilt test was no longer employed.

(7) Caloric Testing, using the technique developed by Cawthorne, Fitzgerald and Hallpike at Queen's Square in 1942. If no responses were obtained at the normal temperatures, water at 20°C., iced water or the Dundas Grant tube was used. The last named had elicited a response when iced water had failed to produce nystagmus. This method was also used if there was a perforation in the eardrum. Simultaneous bilateral stimulation of the ear had sometimes shown a response when there had been a central nervous system lesion which did not cause abnormality when each ear had been tested separately.

(8) Rotation tests, with the head upright, as described by Bárány. Ewald's law probably did not apply to man. The speaker described his modification of the test in which the subject was seated in a rotating chair looking at a closed circuit T.V. camera or alternatively an electrically driven cine camera, fixed in front of him. If, during clockwise rotation, the head was tilted forwards the subject felt as if he were rolling sideways. If the right vestibule was not functioning he did not experience this sensation but a response would occur if the direction of rotation was reversed.

(9) The Oculokinetic Nystagmus test was recommended prior to caloric stimulation and indicated the integrity of extra-ocular muscle action. He now used a modification of the Queen's Square rotating drum consisting of a flat white background across which black bars attached to a belt could be moved in any direction.



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(10) Fistula test.

(11) Electrical tests of labyrinthine function were not of great value but were used on occasion. Direct current with a square wave was applied with the anode in the ear or on the mastoid and the indifferent electrode on the back of the hand. The patient stood on a tilt board and when the response was normal would fall towards the side of the anode. It had been found of use in these cases where the end organ was completely inactive to other tests.

(12) Tests of Otolith Function. Recent experiments in Toronto were described of a new method of controlled stimulation of the otoliths without canal stimulation. The subject was seated on a revolving chair mounted eccentrically on a turntable which was turned in the opposite direction to the chair. The relative speeds of the chair and the turntable were adjusted so that the subject faced in the same direction throughout the rotation of the turntable. As a result the otolith organ was subjected to a linear centrifugal force. Although no nausea, vertigo or nystagmus resulted, a tilting sensation from side to side and an upward and downward sensation was produced. Several deaf mutes however failed to experience this illusion.

Mr. SPENCER HARRISON (G.B.) spoke of the deliberate production by biochemical means of attacks of vertigo in patients with Ménière's disease which he and his biochemist colleague, Dr. I. NAFTALIN, had carried out on patients in their wards. In his address Dr. Naftalin suggested that the hormonal system played a controlling influence in correlating events in the labyrinth, particularly in Ménière's disease, with other body functions. A steady state of sodium chloride balance was obtained in a patient for several days by maintaining a fixed sodium intake. The balance was then upset by administering 5 grammes of salt per day. It was found that about 15 days at the higher rate of sodium intake were needed before equilibrium was reached. It was preceded by a marked positive (increased blood concentration) phase and then a negative (diminished concentration) phase before reaching equilibrium. The sodium curve was known to be controlled by aldosterone—a steroid hormone secreted by the glomerular zone of the adrenal gland. This in turn was controlled by a number of factors, one of which was a hormone emanating from the epithalamus. When aldosterone secretion was at a low ebb, sodium diuresis occurred. Water control was mediated by vasopressin, or A.D.H., secreted by the posterior pituitary gland in response to stimuli from the hypothalamus in nervous centres sensitive to changes in osmotic pressure. The speaker then demonstrated the effect of adding a large amount of salt to the diet of a patient with active Ménière's disease who had been made to reach a steady state of salt balance. Despite increasing intake, profuse sodium loss occurred in the urine and water retention took place. At the moment of



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heavy sodium loss, and irrespective of the salt intake at that time, an attack of vertigo was most likely to occur. These observations agreed with earlier ones that a low salt diet prevented attacks of vertigo as a significant sodium diuresis could not take place on a low salt regime.

Dr. W. F. HOUSE (U.S.A.) considered that there was an accumulation of fluid in the endolymphatic system in Ménière's disease and discussed the control by surgical means of this excess endolymph. Attempts to effect drainage into the perilymph had been shown by many workers to result in deterioration of the hearing and it seemed that drainage of the saccus endolymphaticus into the cerebro-spinal fluid, as suggested by Portman, resulted in improvement in about one in four cases. The speaker had had encouraging results from this operation and used it in those cases where medical treatment had failed and the hearing had not fallen below 50 db.

Mr. ANGELL JAMES (G.B.) described his experiences with the use of ultra-sonic vibrations of 3 megacycles frequency to destroy the vestibular system. At first it was thought that no ill effects resulted to the cochlea but now it was known that quite serious but usually temporary effects could occur. Considerable amounts of heat were generated in the temporal bone particularly in the facial canal. There was at first a stimulation of cell system enzymes which were later destroyed and finally a destruction of the permeability of cell membranes. Ultra-sonic vibrations produced a streaming of the cell contents which probably caused the effect on the cell enzymes. In 180 cases which he had treated, vertigo had ceased in 80 per cent. and 12 per cent. had improved. The hearing improved in 22 per cent. of cases but in 28 per cent. the hearing became worse. Tinnitus ceased in 27 per cent. and improved in 34 per cent. Facial paralysis was not uncommon in the earlier cases but with the latest technique he had not had any in the last 40 cases. He was now prepared to do these cases under general anæsthesia and was commencing work on the biological rather than the destructive effects of ultra-sonic vibrations.

Mr. T. E. CAWTHORNE (G.B.) in a short review of the history of Ménière's disease said that until recently the only treatment was either destruction of the labyrinth or division of the eighth nerve. The development by Dr. House of Portman's operation of drainage of the endolymphatic system was the more natural way. Whilst praising Mr. Angell James for avoiding the production of a facial paralysis in his last 40 cases, the speaker urged caution in the use of this (and other) methods such as the injection of alcohol. He felt that the greatest hope for the future lay in the direction taken by Dr. House.



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Dr. J. W. S. LINDAHL (G.B.) said he had caused facial paralysis on several occasions with ultra-sonic vibrations but they had all recovered quite quickly. He did not consider the facial nerve was placed at such a great risk when compared with the great value of the procedure.

Dr. D. PLESTER (Germany) had operated on a few cases by draining the saccus endolymphaticus with moderate results. Recently he had heard from a colleague in South Africa of the latter's experiences with 56 cases in which 86 per cent. were better and 30 per cent. had improvement in the tinnitus. He considered that a further trial of the operation was justified.

Mr. K. G. MALCOLMSON (G.B.) considered that the immediate cessation of an attack of Ménière's disease which could be produced by the injection of a local anæsthetic into the sympathetic chain was the most cogent evidence that the condition was fundamentally a vascular phenomenon and not a slow going metabolic disorder secondary to the vascular phenomenon in the first instance.

Mr. S. W. Allison (G.B.) also spoke.

Lord BRAIN, in reply to questions, said he had not found Dramamine to be of any value in the treatment of vertigo due to vascular causes or cervical spondylosis but in some cases of drop attacks sodium phenytoin was of value. When spondylosis was present the wearing of a neck collar for periods up to 6 months had improved some of his patients.



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