Mr CYRIL HORSFORD (in reply) said that he saw the patient for the first time six days previously, and there was no evidence of tuberculosis. He thought there must have been some disease of the vocal cord previously, as there was a history of eighteen months of hoarseness; he did not suppose it was a growth during all that time. In the anterior part of the right cord there was an excavation, and in the posterior part corresponding, a firm and distinct swelling. The excavation was evident when the cords were opposed, and the right cord was much infiltrated. The congestion and slight irritation seemed to be due to the irritation of the lump on the opposite side. He thought it was a case of growth superimposed on chronic inflammation. Tuberculosis caused more destruction of the voice than this man showed: and malignant disease would produce a harder voice than the patient had.

He would have a Wassermann reaction done, and show the man again, carrying out no treatment in the meantime.

Postcript. — Since the Meeting the following notes have been obtained: "Wassermann reaction negative; slight evidence of phthisis both apices; tubercle bacilli in sputum."

ABSTRACTS

EAR.

The Psychology of Vertigo. W. F. MENZIES. (*Lancet*, 1924, i., 949.)

The author formulates the hypothesis that vertigo is a purely subjective sensation, and is the realisation in consciousness of the instinctive emotion of anxiety connected with the maintenance and adjustment of antigravity postures. He gives clinical examples of the emotional nature of the condition, and draws attention to the facts that the reaction time of vertigo is two to three seconds, and that hitherto no direct fibre connection has been traced between the vestibular nuclei and the thalamus. On this hypothesis he suggests that vertigo processes are: (1) a peripheral afferent stimulus, ocular, aural, or somatic; (2) a vestibular stimulus; (3) an adjustment response; (4) an emotion; (5) its realisation, consciousness. He states that vertigo "is rare in the dark," also that he "cannot pretend to any opinions concerning intra-aural states beyond the routine neurological examination." The latter statement excuses the fact that the paper does not square completely with the etiological concept of vertigo. MACLEOD YEARSLEY.

The Ear

A Series of Cases of Tinnitus Aurium associated with Abnormalities of Blood Pressure. H. M. WHARRY. (*Lancet*, 1924, i., 893.)

The author believes tinnitus associated with too high or too low blood pressure is more frequent than is supposed. Almost without exception middle age or beyond is the age for such cases, which he divides into four main groups: (1) Tinnitus associated with low blood pressure; (2) that with high blood pressure improving on administration of trinitrin; (3) that with high blood pressure where trinitrin had little or no effect; (4) that with high blood pressure in which local treatment caused disappearance of the symptoms. He considers that trinitrin is of great value in cases associated with high blood pressure, as it appears to have a specific effect in relieving the tinnitus.

MACLEOD YEARSLEY.

Contributions to the Study of Experimental Injury from Noise. MOTOGORA KIMURA, Jena. (*Zeitschrift für Hals-, Nasen-, und Ohrenheilk.* Bd. viii., Heft 1, p. 13, May 1924.)

Kimura has carried out on white mice and pigeons Yoshii's and Wittmaack's experiments on guinea-pigs, and has found the effects on Corti's organ to be the same although some other experimenters have had negative results. The changes in the ganglion cells and nerve fibres consist in a progressive breaking-down of these structures as in toxic degeneration. He does not find the chromatolytic changes insisted on by Yoshii. The paper is well illustrated.

JAMES DUNDAS-GRANT.

Results obtained from one year's use of the Audiometer in the Otological Clinic of Iowa University. By L. W. DEAN and C. C. BUNCH.

The "Pitch Range Audiometer" consists essentially of an instrument for furnishing an oscillating electric current, whose oscillations are of the *sine* curve pattern. The current, acting on a telephone disc, produces a pure tone corresponding in pitch to the frequency of the oscillations. The tone can be varied continuously over a range of from 30 to 7000 d.v. The oscillations of the current are produced by two electric generators in the form of two toothed wheels, one of which rotates in the near neighbourhood of, but not in actual contact with, the other. One of the wheels is magnetised, and the variations in the magnetic field induced in the other wheel are due to the varying distances apart of the generators—(1) when the apices of the teeth on the two wheels are opposite, (2) when the apices of one set are opposite the hollows of the other.

The pitch of the test sound is increased according to the speed of rotation of the wheels, and the intensity is regulated by a shunt resistance.

With this apparatus it is a simple matter to plot curves of the relative acuity of hearing in a series of cases. Each of the various types of deafness give curves having a general similarity of form. So far no striking new clinical facts have emerged. The results may be summarised as follows: lowering of the upper limit of tone perception is the marked characteristic of the curves of the neuro-labyrinthitis group, as exemplified in cerebro-spinal syphilis, meningitis, cerebro-spinal or otherwise, in mumps, influenza, pellagra, generalised tuberculosis, leukæmia, and hysteria. In otosclerosis, the deafness is chiefly at the lower end of the scale, the upper end being only involved in advanced cases. Otosclerosis is also specially characterised by the presence of islands of deafness. In middle-ear deafness the depression of the curve involves both the upper and lower end, the middle reach being less affected.

The results are perhaps more valuable from the purely scientific than from the clinical side. The curve of normal hearing shows very clearly the preponderance of acuity for tones between 300 and 3000 d.v. This covers the range of tones of normal speech. Another remarkable fact, insisted upon by all who have used the new methods of audiometry, is the frequency of islands of comparative or absolute deafness in apparently normal-hearing individuals. Dr Dean determined the presence of these islands of deafness in 43 per cent. of all the cases examined by him, normal or pathological, and in 66 per cent. of otosclerotics. F. W. Kranz of the Geneva Laboratories notes the same fact. These facts have an important bearing on the theory of hearing, as they demonstrate conclusively the well-marked discontinuity of the pitch receptors in the cochlea, and of the nerve tracts associated with them; in other words, they are consonant only with the resonance theory of hearing. They confirm the conclusions of Yoshii and Witmaack drawn from their experiments on the production of degeneration of localised areas in the cochlea and spiral ganglion, as the result of continued exposure of animals to sounds of one pitch.

It is noteworthy that the curves obtained by Dean and Bunch show no trace of the acute peaks present in those given by Gordon Wilson's audion oscillator in the neighbourhood of 2000 and 4000 d.v. Probably the latter were adventitious resonance effects.

G. WILKINSON.

The Pharynx

PHARYNX.

The Incidence and Pathogenesis of Tonsillar Concretions. CARL VERNON WELLER, M.S., M.D. (*Annals of Otology, Rhinology, and Laryngology*, Vol. xxxiii., March 1924, No. 1, p. 79.)

After referring to some of the more important contributions to the literature on this subject the writer draws the following conclusions: tonsilloliths of considerable size are comparatively rare. They occur much more frequently in adults approaching middle life. A history of repeated attacks of tonsillitis in earlier years is usually obtained. Advice is sought because of constant pain in the throat aggravated by deglutition, and it is frequently noted that the dysphagia is worse with solid than with liquid food. The breath is often very fœtid. The tonsillar swelling may be mistaken for an abscess or neoplasm and is frequently in the supratonsillar region. In nearly half of the reported cases the calculus has been expelled spontaneously. Recorded cases emphasise the tendency to recurrence if tonsillectomy be not performed. The calculi are opaque to the X-rays, and chemically consist of the phosphate and carbonate of calcium with an organic nucleus.

The writer, from a microscopical study of 16,000 pairs of faucial tonsils and about one-half as many pharyngeal tonsils, draws the following conclusions:—

(1) While tonsilloliths large enough to attract attention are rare, smaller calculi are very common, showing an incidence of 8 per cent. in all tonsils submitted to tonsillectomy for all reasons.

(2) Microscopical study of the genesis of tonsillar concretions shows that such deposits may occur in any portion of the tonsil. With very rare exceptions, however, it is only in the crypts that concretions reach such a size that they become clinical tonsilloliths.

(3) In the faucial tonsil the most common modes of origin within the crypts are in the dead interior of colonies of mouth organisms and in masses of keratohyalin. The former method is slightly more common than the latter. These are the chief sources of clinical tonsilloliths.

(4) Vegetable material, food debris, inflammatory exudate, desquamated epithelium, and old blood from hæmorrhage may also serve as the organic nucleus for calculus formation in the crypts. With any of the substances mentioned cholesterin may be present.

(5) In the pharyngeal tonsils, calculus formation occurs especially in the mucopurulent exudate and in keratohyalin.

(6) In the lymphoid tissue of the tonsil calcification of old blood, pus, or caseous necrosis may occur, as in lymphoid tissue elsewhere.

Small intracellular concretions in the giant cells of true tubercles may cause them to be mistaken for foreign body pseudo-tubercles.

(7) Tonsil calculi conform to the general laws of calculus formation in that they develop only upon an organic nucleus in a state of necrosis or necrobiosis, and that they may show, although but faintly, concentric lamination and radial striation.

(8) The very common association of cholesterin crystals with the beginnings of calculus formation in the tonsil indicates that the presence of lipoids and the formation of soaps may be an important part in the chemical mode of their development.

(9) As tonsillar calculi become larger, their presence favours the origin and continuation of an active inflammatory process in the wall of the crypt in which they are situated. F. HOLT DIGGLE.

The Bacteriology of Extirpated Tonsils and its Relation to Epidemic Tonsillitis. T. NAKAMURA. (*Collected Papers of the Mayo Clinic*, 1923, Vol. xv., p. 921.)

The bacteriology of 2048 tonsils was studied by the writer. The tonsils were removed on account of recurring attacks of tonsillitis, or more often, because they were believed to be foci of infection in patients suffering from ulcer of the stomach, chronic infectious arthritis, myositis, nephritis, etc. The size and consistency of the tonsils varied greatly. In many instances pus was expressed from the crypts and on section one or more small abscesses were found near the capsule, even though the patient had not had symptoms referable to the tonsils, and when the examination *in situ* was largely negative.

Streptococci predominated. They were classified in three groups, (i.) the hæmolytic, (ii.) the viridans, (iii.) the indifferent. The percentage of the different streptococci varied according to the change in the seasons. Thus, during October, the green-producing colonies predominated. The curve became depressed until the latter part of May, when it rose. Whereas the curve representing the hæmolytic streptococci was low during October, but rose to a slightly predominating level during November and the early part of December, and then passed to a still higher level, where it remained until the early part of April, when it dropped to a low level.

In view of the fact that most of the patients came from widely separated regions, there is reason to believe that the increase in hæmolytic streptococci during the winter months occurs generally, and that the increased incidence of hæmolytic streptococcus tonsillitis is due more to climatic conditions than to contact infection.

A. LOGAN TURNER.

The Pharynx

Direct Blood-Stream Infection through the Tonsils. S. J. Crowe, M.D., Baltimore. (*Archives of Internal Medicine*, April 1924, Vol. xxxiii., pp. 473-482.)

The author had previously insisted (1916) that tonsils or adenoids could not be responsible for a general systemic disturbance, such as infectious arthritis, unless there was a palpable enlargement of the lymph glands in the neck. For many years, therefore, he advised patients against operation of removal of tonsils in these circumstances. As a number of his patients subsequently informed him that, acting upon other advice, the tonsils were removed and the general symptoms improved and ultimately disappeared, Crowe proceeded to re-examine his histological material previously obtained from several thousand tonsil operations, in order to see whether he could demonstrate microscopically a direct infection of the vascular system.

Re-examination of the microscopic sections of the tonsils revealed certain points which undoubtedly appeared to favour the view that infection might pass directly into the blood stream. The epithelium lining the tonsillar crypts differs from that upon the surface of the tonsil in being provided with papillæ and a rich network of capillaries. In chronic tonsillitis there is commonly destruction of the crypt epithelium, and around the margins of the ulcerated areas there is a network of thrombosed capillaries and lymph channels, and the conditions seem favourable for the passage of organisms into the lymphatic system and for the liberation of infected emboli into the blood stream. The author has found these ulcerated areas most frequently in the tonsils of individuals suffering from infectious arthritis. A few of these arthritic patients whose tonsils show histological ulcers in the crypts have no palpable enlargement of the cervical lymph glands, and the findings in these cases suggest a direct blood-stream infection. Further, the removal of the tonsils with this type of lesion in the crypts is followed most frequently by general improvement. A. Logan Turner.

Indications for Tonsillectomy, with a Criticism of the Operation. Gilbert H. Lansdown, M.R.C.S. Eng. (*Canadian Medical Association Journal*, May 1924, page 379.)

The author summarises the indications for tonsillectomy under three heads:—

(1) Interference with respiration, deglutition, or voice production.
(2) Interference with middle-ear ventilation.
(3) Focal infection.

He places considerable importance on the bacteriological examination of the tonsils. He advises the preliminary use of calcium lactate

for four days before operation. He describes his technique which is dissection, the finger being used to free the tonsil. Hæmorrhage, if severe, is controlled by ligation of the bleeding point.

E. HAMILTON WHITE.

Causes of Failure in the Operation for Removal of Tonsils and Adenoids, and their Avoidance. H. MORTIMER WHARRY, F.R.C.S. (*Brit. Med. Journ.*, 5th April 1924.)

There are three places where remains of adenoids are commonly found.

(1) Anteriorly in the upper part of the choanæ.
(2) Laterally in the fossæ of Rosenmüller just above and behind the Eustachian cushions.
(3) Posteriorly just behind the soft palate on the anterior tubercle of the atlas vertebra.

There should be three definite steps in the operation—preliminary digital examination, removal with the curette, and after-examination with the finger which can generally crush and scrape away anything left. Complete removal of tonsils is advocated, and the blunt-guillotine method is clearly described. Bleeding points should be secured by forceps—"this is the only operation in which it is still common for a patient to be carried off the table still bleeding." Methods of dealing with secondary hæmorrhage are discussed. The author is of the opinion that gas and ethyl chloride are suitable anæsthetics only when adenoids alone are to be removed. For the tonsil operation they give too little time, and their use is responsible for most of the failures—incomplete removal, injury to uvula or faucial pillars, pharyngeal tags, and unnecessary loss of blood.

T. RITCHIE RODGER.

Lingual Thyroid in an Infant of three and a half months. G. B. NEW. (*Collected Papers of the Mayo Clinic*, 1923, Vol. xv., p. 917.)

The writer reports upon an infant of three and a half months who had had difficulty in breathing and swallowing since the second week of life. The symptoms had progressed. On examination there was slight inspiratory stridor and grunting on expiration. A soft-rounded tumour was seen at the base of the tongue on laryngeal examination. It was covered by normal epithelium on the surface of which were a few large blood vessels. The larynx was normal. The symptoms were not such as to require interference, and it was thought that as the child grew older and the pharynx enlarged, the air-space would increase and no treatment would be necessary.

A. LOGAN TURNER.

The Larynx

THE LARYNX.

Complex Laryngeal Syndromes in cases of Wounds of the great Vessels of the Neck. Prof. GIOVANNI TRITTO. (*Archiv. Italiani di Laringologia*, Anno xliii., Fasc. 3^{e} 4, p. 81.)

Professor Tritto reports the case of a man who was wounded in the left side of the neck with a knife and who had a series of severe hæmorrhages over a period of two months. Eventually the common carotid artery was tied, and it was found that this vessel had become necrotic following sepsis. The original wound had apparently injured the lingual and facial branches of the external carotid, and the internal jugular vein. The patient left the hospital two and a half months after the infliction of the wound. Shortly after his discharge the patient's voice deepened, became bitonal, disappeared altogether for a short time, and then reappeared as a hoarse, bitonal voice. Examination at this stage showed: (1) paralysis of the left vocal cord which lay in the cadaveric position; (2) anæsthesia of the left half of the vestibule of the larynx; and (3) paralysis of the left half of the soft palate.

As the paralysis only came on gradually after the ligature of the carotid artery, the lesion could not have been due to the original wound or to the ligature, but to implication in scar tissue formed during the healing of the wound. Cutting or ligation of the nerve would give rise to immediate and complete paralysis of the cord, but in the more gradual types due to pressure or to infiltration, the fibres leading to the posterior crico-arytenoid muscle are affected first and a mid-line position is taken up. Later the constrictors—lateral crico-arytenoids, internal thyro-arytenoids, and the inter-arytenoids—are paralysed and the cord takes up the cadaveric position. The tension exerted by the crico-thyroid tends to draw the arytenoid cartilage forwards and downwards, and the antagonistic pull of the posterior crico-arytenoid is lost so that the affected vocal cord takes up a position inferior to that of the healthy one.

The anæsthesia of the vestibule included the whole of the left side and a small portion of the right side, showing that there is a certain amount of overlapping in the sensory nerve supply of the larynx. There is a very complicated interchange of fibres between the last four, and particularly between the X and XI nerves, and a remarkable correlation between the centres for respiration and for phonation. In anæsthesia of the vestibule it can be said that section of the recurrent nerve does not give rise to anæsthesia, and that the recurrent is not a mixed nerve. The anæsthesia is in the region of distribution of the superior laryngeal nerve, and is a secondary manifestation. It has been shown in the dog that section of the recurrent gives rise to an ascending degeneration of the recurrent nerve and a descending

degeneration of the superior laryngeal. It is probable that a similar phenomenon occurs in man and, including the sensory fibres, gives rise to the anæsthesia.

The author suggests that when the motor paralysis is followed by sensory paralysis, the degenerative processes have spread to the sensory centre in the dorsal nucleus of the vagus from the motor centre in the nucleus ambiguous, and that there is a complete degeneration of the arc. The motor paralysis of the soft palate is probably due to a similar spread of degeneration to the centre and fibres supplying it.

It is just possible that in the case recorded the spread of degeneration was in the opposite direction, and that injury of the superior laryngeal was the primary nerve lesion; but the author thinks that the recurrent nerve was implicated in the scar tissue and that the superior nerve was also implicated in scar tissue in the upper part of the wound. There was in this case a troublesome cough reflex, which, in the absence of the function of the vagus fibres, was probably due to a conduction of impulses by the anastomosing branches of the sympathetic.

F. C. ORMEROD.

On Necrosis of the Cartilage of the Larynx in Diphtheria. KARL BECK. (*Acta Oto-Laryngologica*, Vol. III., fasc. 1, 2, February 1924).

Necrosis of the cartilaginous framework of the larynx is not unusual in syphilis, tuberculosis, and suppurative diseases, but the possibility of it occurring in diphtheria has only been mentioned incidentally by a few authors. Even in the case of necrosis occurring in diphtheria a mixed infection might be present, and hence it is not unlikely to be of streptococcal origin.

Beck describes a case of septic diphtheria of the upper air passage where, in addition to external abscess formation suggestive of cartilage necrosis, there occurred after four weeks a detachment and spontaneous coughing up of an arytenoid cartilage, but the symptoms and discomfort attending this very serious process were astonishingly slight.

H. V. FORSTER.

Concerning the After-Treatment of External Operations on the Larynx. M. HAJEK. (*Acta Oto-Laryngologica*, Vol. iii., fasc. 1, 2, February 1924.)

The author compares the different amount of interest shown between the actual operation and the after-treatment of these cases. He deals with cases of tracheotomy and partial and total extirpation of the larynx.

He speaks of the performance of cricotomy instead of tracheotomy, with the damage likely to follow cricotomy. Errors in introducing the cannula in a wrong position should be corrected early. He urges special cannulas for individual cases; advises a window resection of the tracheal wall to prevent granulation formation, and refers to the purifying of air by the use of Haslinger's cannula.

The after-treatment of thyrotomy and partial extirpation depends upon whether the operation has been performed in one or two stages, and must deal with the treatment of the wound and the management as regards decannulation. With regard to the question of a two-stage operation, the merits of the individual case must be considered. The larynx should be closed after the operation only when the less extensive procedures are carried out. When more has been done, packing should be used to control secretions and for adaptation of soft parts to the cartilaginous framework. Decannulation is carried out with due regard to possible obstruction to breathing following granulation formation and scarring inside the tracheal wall.

He completes the extirpation operation in two stages, first of all anchoring the trachea by his special method. After extirpation he always leaves a pharyngeal fistula from which to draw secretion by suction. The results were gratifying.

He does not deal with the more permanent after-results in this paper. H. V. FORSTER.

VERTIGO.

DISCUSSION AT THE CONJOINT MEETING OF THE SECTIONS OF MEDICINE, NEUROLOGY, OPHTHALMOLOGY, AND OTOLOGY, ROYAL SOCIETY OF MEDICINE, 26TH FEBRUARY 1924.

(Abstract by J. S. Fraser, F.R.C.S. Ed.)

IN opening the discussion Sir Humphry Rolleston said that neuro-otology, or analysis by the vestibular tests, enabled a diagnosis of organic lesions of the vestibular apparatus to be made, and thus narrowed down the points to be discussed by a physician to two main categories: (1) What evidence was there that the action of toxins, transient vascular changes, or functional disturbances of that part of the cerebral cortex where the sensations concerned in equilibration normally meet in harmony, might cause vertigo? That toxic, vascular, or functional disturbance might occur appeared probable from consideration of epileptic auræ and the borderland cases of epilepsy, namely, the occurrence in an epileptic of vertigo without any fit, and from the occasional alternation of migraine and vertigo. Just as disturbances of vision might be due to toxic influence

or to vascular spasm affecting the cerebral cortex or the retinal vessels, so might an analogous cerebral localisation explain vertigo. Poisons due to defective metabolism in gout, nephritis, arteriosclerosis, or intestinal stasis, were capable of causing vertigo.

(2) What were the ways in which general diseases and visceral disorders might affect the vestibular apparatus? *Anaphylactic conditions* might affect the semicircular canals; Sergent had recently grouped vertigo with asthma, epilepsy, and paroxysmal tachycardia, as manifestations of humoral dyscrasias. Jones quotes a case which manifested vertigo from gastro-intestinal disturbance due to fish and eggs, *i.e.*, an angioneurotic manifestation in the labyrinth.

Vernet has suggested that *endocrine disorders* may induce vertigo and has advocated the oral administration of adrenalin. Ovarian disturbance may induce otospongiosis (otosclerosis), and by affecting the semicircular canals may cause vertigo. Escat suggests that pituitary extract, by controlling the ovary, may be beneficial. The *gastro-intestinal origin* of vertigo has been much discussed. The gastric manifestations and the vertigo are now widely regarded as concomitant effects of vestibular disturbance instead of standing in the relation of cause and effect. But because many cases of vestibular vertigo have been regarded as stomachic in origin, it does not follow that gastro-intestinal disturbance cannot be a primary cause of vertigo, either reflexly through the brain-stem or by generating poisons which act directly on the vestibular system. Hepatic insufficiency and the resulting failure to stop or neutralise enterogenous poisons, such as indol, may play a part in inducing a toxic vertigo. Lermoyez has argued in favour of vertigo due to *local spasm* of the vestibular artery in gouty, neuro-arthritic subjects, and suggests that the intermittent character of a vertigo points to a vasomotor nature.

The association of *arterial disease* with vertigo may be explained in several ways; syphilis may cause endarteritis obliterans. High blood-pressure is associated with increased pressure of the cerebro-spinal fluid and of the intralabyrinthine fluid. Great variations may occur in the systolic blood-pressure, and so presumably in that of the intralabyrinthine fluids. In cerebral arteriosclerosis, attacks of vertigo may be due to transient cortical anæmia depending on vascular spasm. Bonnier's syndrome of Deiters' nucleus consists of vertigo, oculo-motor disorders, trigeminal pain, and symptoms due to implication of adjacent nuclei, *e.g.*, thirst, polyuria, and vasomotor disturbance. In arteriosclerosis combined with renal disease vertigo may be explained by increased exudation of fluid in the semicircular canals, corresponding to œdema elsewhere, or to changes in the vestibular nerve comparable with those in the optic nerve, or to the action of uræmic poison on the brain stem, cerebellum, and cortex.

In *anæmia* it might at first sight appear probable that the medullary centres and the cortex are affected, but anæmia may lead to degenerative changes in the retina and also, possibly, in the end organs of the vestibular nerves.

Leukæmic infiltration of the semicircular canals, with hæmorrhage, is a well-recognised cause of Ménière's syndrome.

In conclusion, Sir Humphry Rolleston referred to an outbreak of *paralysing vertigo* which occurred around Geneva in the eighties of last century. The disease appeared in the summer and was confined to men who were agricultural labourers. It was thought to be due to infection in stables. Miura described the occurrence of this disease in the Northern Provinces of Japan. In addition to vertigo there were severe pain in the neck, transient loss of muscular power and ocular manifestations (ptosis, dimness of vision and diplopia). The disease was never fatal either in Switzerland or Japan.

Dr Gordon Holmes, who dealt with the neurological aspect of vertigo, said that giddiness was a common symptom in local lesions of both the brain-stem and the fore-brain. It might arise from local lesions involving the intracranial portion of the vestibular nerve, either the nerve itself on the side of the pons, its intermedullary portion, or its terminal nuclei. In the second place, vertigo might result from disease of those portions of the central nervous system which were intimately, though indirectly, connected with the labyrinths, as parts of the medulla oblongata, the cerebellum, and the mid-brain. Vertiginous attacks were among the most prominent symptoms of eighth nerve neuroma, meningitis, gummatous infiltration, or pressure of an arteriosclerotic vessel. In one of the latter cases there had been other evidences of an intracranial aneurysm. In these cases there was usually a sensation of rotation of self from the healthy side towards the side of the lesion, and of external objects towards the opposite side. Acute vascular lesions of the pons and medulla were often heralded by intense attacks of giddiness. A patient with thrombosis of the posterior-inferior cerebellar artery almost invariably gave a history that, without warning, he became suddenly and intensely giddy, with vomiting, cardiac and vasomotor disturbances, and intense prostration.

Lesions of those portions of the pons, mid-brain, and cerebellum which are connected with the vestibular nuclei gave rise to vertigo of the same type. Small cortical softenings of the cerebellum seldom caused giddiness, but when they extended to the central nuclei and the peduncles, vertigo was very common. Many patients with tumours of the cerebellum never complained of giddiness.

Vertigo occurred with disease of the forebrain only when the cortex or subcortical white matter was involved. It was sometimes assumed that there existed in the cerebral cortex a centre for

equilibrium, to the disturbance of which vertigo might be attributed, but there was no real evidence of this; equilibrium was a much more complex function than could be assigned to any one centre. On the other hand, lesions involving any portion of the cortex from which spasmodic phenomena, especially movements of the head and eyes, could be excited, might give rise to giddiness. Holmes had found that vertigo was frequently complained of by men with gunshot wounds of the occipital lobes, who were subject to epileptiform seizures. In these affections vertigo might, however, be present apart from convulsive manifestations.

Vertigo might occur in diffuse cerebral lesions, such as cerebral arteriosclerosis. The immediate relief that often followed lumbar puncture suggested that the most important effect of increased intracranial pressure was an interference with the cerebral circulation. The vertigo was probably due to failure of the proper integration of the afferent impressions, on the harmony of which our sense of balance depended, because there were evidences of defective integration in other spheres of cerebral activity, such as loss of memory and confusion. In this second sub-group Holmes included the effects of cerebral concussion, and the action of certain poisons such as alcohol and neurasthenia.

Vertigo might occur as a symptom in other nervous diseases, *e.g.*, epilepsy and migraine. In epilepsy it might occur as the warning or aura of an attack, or might be an equivalent replacing the attack. Holmes stated that, in his opinion, it was never safe to regard true vertigo as an hysterical symptom.

Mr Sydney Scott, who dealt with vertigo from the otological aspect, showed slides obtained from sections of the labyrinth and middle ear in infective otitis media and interna. Such destructive processes always caused the symptom of Ménière's disease. He regarded Ménière's one fatal case as being due to infection of the labyrinth, death being caused by meningitis serosa.

The onset of vertigo in the course of otitis media was of the utmost practical importance, as it might call for immediate surgical intervention. A normal labyrinth might, however, be disturbed by processes taking place outside the labyrinth. Vertigo and nystagmus might occur in the earlier stages of acute otitis media, generally before the drum membrane had perforated spontaneously, or had been incised. This distinction between labyrinthine disturbances due to destruction, and those due to extra-labyrinthine causes, could be ascertained by means of the various tests. The surgeon could rarely have any justification for destroying the labyrinth, so long as positive caloric tests were obtainable. In unilateral eighth nerve tumours, vertigo and staggering were consistently present. When both ears were deaf to low tones, and the patient had signs of

bilateral middle ear deafness, he was not liable to be giddy as were patients who had no deafness, or only unilateral middle ear deafness. Patients with only one deaf ear were not ordinarily noticed to be deaf but many of them suffered from giddiness. By the time that there was bilateral deafness and marked raising of the lower tone limit, which generally indicated that there was bilateral ankylosis of the ossicular joints, attacks of giddiness had usually ceased.

If the superior semicircular canal of the human temporal bone from the post-mortem room was opened and a capillary glass tube, containing a coloured fluid, was sealed into the canal, as a manometer, it showed the effect of movements of the stapes in producing relatively to such a sensitive organ enormous changes in pressure in the labyrinth. Similar changes of pressure could be evoked by distending the tympanum and by compressing the drum membrane inwards. Unilateral differences of pressure in the middle ear could be transferred to the labyrinth through the movements of the stapes. His experiences at Queen Square had led him to regard temporary disturbance of the peripheral termination of the vestibular nerve as a common cause of vertigo. The caloric, rotation, and galvanic tests could be relied upon to exclude any intra-labyrinthine lesion. The commonest cause of aural vertigo was not labyrinth disease, but Eustachian inefficiency. Mr Sydney Scott urged that in all cases the efficiency of the Eustachian tubes and the state of the posterior nares and accessory sinuses of the nose should receive particular attention, as well as the reaction of the labyrinth to caloric or rotation stimuli.

Mr J. Herbert Fisher dealt with vertigo from the ophthalmological aspect, and stated that visual judgments and information obtained from different impulses generated in the extra-ocular muscles were elements which contributed to our equilibrium. An image of the field of vision was formed on the retina, and the image of each object in the field was projected outwards along the secondary axis, passing through the nodal point to its proper position in the field. This relation of objects to one another in space was called objective localisation or objective orientation. Disorder of objective orientation might lead to metamorphopsia, polyopia, and errors of judgment, but it was doubtful if it ever caused true vertigo. Making use of stereoscopic vision, as supplied by our two eyes, we were enabled to judge of the solidity of objects, and perhaps less accurately of their distance from us and of their size. Impulses from the semicircular canals contributed to balance-maintenance without affecting consciousness. The statement had been made that unaccustomed visual sensations might *per se* be a cause of vertigo, *e.g.*, that giddiness was produced by gazing at a waterfall; that it was possible to view such an object with complete immobility of the eyes seemed improbable. When a hyper-

metrope first wore his convex glasses he made erroneous judgments; the ground appeared too close to him, with some resulting disorder in walking. Such disturbances of judgment were not enough to produce any sense of vertigo. With regard to astigmatic errors of refraction, claims were made that the correction of 0.5 D., or even of 0.25 D. of astigmatism would eliminate the giddiness. Fisher's own experience lent little support to this view. He did not wish to suggest, however, that we should ignore refractive errors in dealing with cases of vertigo. By the muscular sense of our ocular muscles we learned, subconsciously, the position of our eyes in our heads, and their alignment in regard to objects around us. This was what was meant by subjective orientation, disorder of which function resulted in false judgment of our own position and a sense of giddiness. A patient with paralysis of the external rectus in his better eye might yet prefer to use this eye, and to exclude his non-paralysed eye of less visual acuity, to avoid diplopia. If this had been his habit, testing would reveal that the patient was the subject of false projection when using the non-paralysed eye alone, and that he walked unsteadily if attempting to guide himself with this eye: he had established a new sense of values for the function of his paralysed eye. Such cases suggested that the organ of vision played a rather subsidiary part in the maintenance of equilibrium. In birds possessed only of panoramic vision, and in whom the movements of the eyes were extremely limited, the sense of equilibrium was highly developed. Labyrinthine nystagmus was supposed to be produced by influences from the semicircular canals reaching Deiters' nucleus and, being there reflected to the ocular muscles, jerking of the eyes resulted and stationary objects appeared to be in oscillation. It was reasonable to think that abnormal impulses from disordered ocular muscles might reach the same centre, and in the cerebellum might provide reflex irritation of the labyrinthine nuclei, and thus cause a sensation of vertigo. This might explain the vertigo of insular sclerosis when nystagmus was present. Train-sickness and sea-sickness were in all probability partly due to the disturbance of ocular muscles which resulted from viewing the moving waves or the apparently moving objects from the railway carriage. This disturbance might persist at least for some hours after the journey had been completed. (The tendency to sea-sickness is greatly diminished if the voyager keeps his eyes fixed on a book, and is at once increased by looking at or over the side of the ship.—Abstractor.) The school-boy trick of bowing the head on top of a cricket stump and whirling round it six or eight times produced a sense of intense vertigo. The giddiness was more severe if the whirling was performed with the eyes open than if they were closed, because disorder of ocular muscles was then induced and its effects were superadded to the labyrinthine vertigo. In tabes the value of accurate visual judgments in the maintenance of balance was

well shown; when deficient information was conveyed from tactile and muscle sense in the lower limbs, the swaying on Rhomberg's test was inconspicuous unless the eyes were closed. Ocular judgments could, to a large extent, compensate for the disordered subconscious impressions. Vertigo appeared to be a conscious process; normal equilibrium was maintained subconsciously; a sensation of giddiness could be experienced only as a higher mental perception. The eye movements necessary to watch either the falling water of a cascade or objects seen through the window of a moving railway carriage, or to perform the duties of a compositor, were objective and volitional; the nystagmus which they induced caused stationary objects to which the eyes were next turned to appear in jerky movement. On the other hand, the movements of congenital nystagmus were reflexly produced and unconscious and, therefore, no sensation of jerking in the outside world was ever experienced, either when the nystagmus was active or when it was in temporary abeyance. Fisher held that the sense of vision contributed to equilibrium only when it affected consciousness through the higher perception centres. Equilibrium was a sub-conscious process maintained by the tactile and muscular and vestibular senses; to the total sum of muscular sense the muscles of the eyeball contributed their quota; disorder of the muscular and tactile elements of balance produced inco-ordination which might be sufficient to cause loss of equilibrium and to produce a fall without originating any perception of vertigo. Disorders of the vestibular apparatus produced a conscious vertigo. The sense of vision could, if working normally, compensate for a lack of general muscular co-ordination while, if acting abnormally, it aggravated a vertigo. Accurate visual impressions might act as an arbitrator when the stimuli derived from other sources necessary to equilibrium were inharmonious, and thereby abolished any sense of vertigo; apart from disorder of the ocular muscles, the sense of sight would not itself produce any appreciation of true giddiness.

In the discussion Sir James Dundas-Grant said that small doses of quinine had so remarkable an effect in checking attacks of aural vertigo, that if quinine did not, in a unilateral case, cause diminution of the vertigo, it was probably not a semicircular canal condition. He thought the quinine equalised the influence of the two labyrinths by its sedative action on the sound labyrinth. The late Sir Victor Horsley accepted Mills' idea of the localisation of a centre for the sense of equilibrium in the temporal lobe. Erichsen had found that in "railway spine" due to concussion great relief was afforded by small doses of perchloride of mercury. Sir James recommended its use in cases of vertigo from concussion.