indicates cerebellar abscess. Tubercular or syphilitic disease of the temporal bone may produce oculo-motor paralysis, causing direct pressure on the trunk of the nerve. Otogenic paralysis of trochlearis has been recorded by Schwartze, De Laparsonne (5), and others. Jacques twice observed external ophthalmoplegia follow acute otitis media in infants, accompanied by ptosis; one died with all the signs of acute meningitis.

To sum up, it appears that otogenic paralysis of the oculo-motor and other orbital nerves is clearly established, but our difficulty arises when we endeavour to establish the pathological lesion present. When paralysis exists with an ipsolateral otorrhœa, accompanied by headache and vomiting, an exploration of the auditory apparatus should be at once undertaken to determine the extent and limits of the disease, whilst at the same time it should be borne in mind that the examination of the nasal fossæ and accessory cavities may reveal the focus of disease, in so much as nasal disease infection is far more common than aural infection in the production of paralysis of the oculo-orbital nerves.

REFERENCES.

(1) ONODI.—"The Rhinogenic and Otogenic Lesions of the Third, Fourth Fifth, and Sixth Cranial Nerves," JOURN. OF LARYNGOL., RHINOL., AND OTOL., 1914.

(2) LANDOLT.—"Etude sur les mouvements des yeux, etc.," *Archiv. d'ophtal.*, 1881.

(3) MICHEL.—*Lehrbuch der Augenheilk.*, 1890.

(4) LOMBARD.—*Internat. Med. Congress*, 1908, Otologie.

(5) DE LAPARSONNE.—*Société française d'ophthalmogie*, 1907.

SOCIETIES' PROCEEDINGS.

ROYAL SOCIETY OF MEDICINE—SECTIONS OF NEUROLOGY, OPHTHALMOLOGY, AND OTOLOGY.

Combined Meeting, February 26, 1914.

Discussion on Nystagmus.

The discussion was preceded by a demonstration of cases illustrating the different varieties of the disorder.

(We select only those portions of the discussion which are of special interest.)

Mr. SYDNEY SCOTT: It is perhaps well to remark that normal or what one may speak of as everyday movements of the head suffice to stimulate the semicircular canals, setting up afferent impulses which are trans-

https://doi.org/10.1017/S1755146300014293 Published online by Cambridge University Press

mitted to the basal nuclei, and so forth, and reflex influence the co-ordination of muscular movements, *not only of the eyes but of other parts of the body.* The ocular movements known as nystagmus are in most cases with which we are concerned in otology due to *excessive* stimuli applied to physiologically normal vestibular systems. When we meet with spontaneous nystagmus we have to consider the relation between the spontaneous and induced nystagmus. We shall first devote attention to induced nystagmus in so far as the ear is concerned.

Nystagmus produced by Syringing the Ear.—Irrigation of one ear with hot water when the head is erect will produce involuntary pleurothotonus towards the contra-lateral side, with rhythmic rotatory nystagmus towards the ipsi-lateral side. The direction of these reflex movements is reversed by keeping the head inverted during the progress of irrigation, and they can also be reversed by using cold water instead of hot, without inverting the head. The legitimate explanation is that, in accordance with the laws of heat, convection currents are set up in the endolymph, and that the direction of these currents determines the direction of the reflex movements. When the head is erect or inverted the currents would be induced in the superior semicircular canal, and this would account for the rotatory nystagmus. When the individual under examination lies with face upwards when the ear is irrigated with hot water, he will tend to roll towards the contra-lateral side, and horizontal rhythmic nystagmus will be noticed towards the ipsi-lateral side. If we use cold water instead of hot, the face remaining uppermost, the direction of the forced movements will be reversed. We can also reverse the direction of the movements by placing the head with face downwards during the procedure of irrigation with hot water, and again reversal of the direction of the reflex movements may be induced by employing cold water instead of hot, while the face remains prone. I have ventured to express the reactions in the form of a law based upon the convection hypothesis: "*The deviation of the head and eyes in the same direction as the current in the endolymph, and the nystagmus is in the opposite direction.*"

Nystagmus produced by Rotation.—The phenomena produced by rotation tests are in harmony with those produced by caloric tests, and are explicable by the laws of inertia and the production of currents in the endolymph.

The eye movements are of secondary importance compared with the primary movements of the head, trunk and limbs, which result from rotation stimuli. By attending only to the nystagmus we are studying only part of the reflex movements produced by labyrinthine stimulation.

Nystagmus produced by Galvanic Stimulation.—When the electrodes of a galvanic system are applied to one or both sides of the head, in the former case the one electrode being in a neutral position, a sustained current of 2 to 5 ma. is sufficient to provoke definite reflex movements of the head and trunk, but insufficient to provoke nystagmus. If the strength of the current is increased to 5 or 10 ma., or sometimes 15 ma., rotatory nystagmus will be set up in addition to the forced movements of the head and trunk. This mode of stimulus is occasionally of considerable value. It will be observed that the direction of the nystagmus varies with the position of the electrodes. When the anode is in contact with the head in the region of the ear, and the kathode is in the hand, on the back or on the other side of the head, the nystagmus is directed *away from the anode.* When the position of the electrodes is reversed, the nystagmus is *towards the kathode.* The anodal current gives a stronger stimulus than the kathodal current.

https://doi.org/10.1017/S1755146300014293 Published online by Cambridge University Press

The responses to galvanism are observable in cases in which the labyrinth has been disorganised or removed, but the reactions are absent in cases of destruction of the vestibular nerve by a neoplasm.

Nystagmus produced by Meatal Compression.—Nystagmus can also be momentarily provoked occasionally in patients who have middle-ear disease associated with a fistulous communication between the middle and internal ears, by sudden compression of the meatal contents, provided the neuro-epithelium of the vestibular nerve is intact and that the local condition remains favourable for the transmission of sudden changes of pressure to the interior of the labyrinth.

Dr. Hughlings Jackson's Case, "Fistel Symptom."—It is now about thirty-five years ago since Hughlings Jackson described examples of nystagmus in association with ear disease. Schwabach in Germany, Blake, and later Kipp in America, also described cases about the same time. Hughlings Jackson's case was that of a woman, aged forty-nine, with suppurative disease of the ear. Digital compression of the meatal contents caused immediate giddiness, which was associated with deviation movements of the eyeballs towards the contra-lateral side. Rhythmic nystagmus followed, with the rapid component towards the ipsi-lateral side; some rotatory movement was also noticeable. Dr. Jackson did not have the opportunity of verifying the existence of a labyrinth fistula, but there can be little doubt that one did exist. The term "fistel symptom" is now employed to designate the phenomena resulting from meatal compression which Jackson described. During a period of about ten years I have met with only twenty-three examples of the "fistel symptom," but in nearly every case an opportunity occurred of verifying the actual existence of a fistula which generally led into the external semicircular canal.

Many cases of labyrinth fistulæ occur, of course, without being associated with the "fistel symptom," simply because the internal ear has already become disorganised and ceased to react, or because the fistula may be protected by epithelial accumulations which hinder the transmission of sudden changes of pressure from the middle to the internal ear, although the latter may retain its normal sensibility. Rarely the "fistel symptom" may appear about a month after the mastoid operation has been performed for chronic suppuration of the middle ear. At the time of the operation the semicircular canals and the vestibular walls may be quite intact, and it would seem that the sign develops with the appearance of vascular granulations upon the outer wall of the labyrinth. The gentlest possible application of a probe, in such cases, to the granular area over the external semicircular canal immediately provokes the nystagmus and also a sudden jerking movement of the head. It is several weeks before the sign disappears, and I have little doubt it arises from the formation of porous bone during the growth of granulations from the thin outer wall of the external semicircular canal. The "fistel reflex symptom" can be provoked not only with the probe, but by means of a Politzer bag, connected with the external auditory meatus with a rubber tube which accurately fits the meatus. Sudden slight compression of the rubber bulb, when the patient is seated erect, causes the head to be suddenly and involuntarily jerked about a vertical axis, the face turning away from the stimulated side; simultaneously the eyeballs rotate in the orbits in the same direction as the head. The ocular movements are more sensitive to the pressure than the head movements, so by carefully regulating the compression one may evoke ocular movements without head movements. It will then be seen that

https://doi.org/10.1017/S1755146300014293 Published online by Cambridge University Press

each compression causes the eyeballs to oscillate horizontally. A very slight stimulus may cause undulatory horizontal nystagmus, a stronger compression will set up rhythmic horizontal nystagmus, and a still slightly stronger compression will introduce a rotatory element, so that we obtain combined horizontal and rotatory nystagmus. In each case the nystagmus is quite transient, lasting only a few seconds. In applying the test there is a possibility of forcing pus into the labyrinth and setting up diffuse labyrinthitis and risks of meningitis. One should be extremely cautious in the application of the test for the " fistel symptom."

Effect of Anæsthesia.—In considering the relation of the two components in labyrinthine nystagmus it is important to notice that the rapid or secondary component is abolished by general anæsthesia. One sometimes has the opportunity of observing the effect of direct pressure upon a semicircular canal fistula when operating for disease of the ear. If the labyrinth is active the eyeball moves in response to the pressure, not in an oscillatory manner, but by simply deviating to one side. Sometimes one may evoke alternate deviation movements by applying pressure alternately to a semicircular canal fistula and to the fenestra ovalis. In one case I was able to provoke at will alternate movements of the eyeball away from the stimulus when the probe was applied to the canal, and a movement towards the stimulus when the probe was applied to the stapedial fossa. One regards such alternate movements as being two separate primary reflex movements, the secondary reflexes being in abeyance so long as the patient is anæsthetised.

Spontaneous Nystagmus.—In discussing labyrinthine nystagmus, and comparing it with nystagmus associated with disease of the central nervous system, or of the eye itself, we concentrate our observations on nystagmus which occurs within the binocular field.

The less intense forms are sometimes seen in cases of acute or chronic otitis media. If the infection does not spread to and destroy the labyrinth, the nystagmus is restricted to the side of the infection. The nystagmus in these cases is usually transient, and may be overlooked. The complaint of giddiness leads one to look diligently for sustained nystagmus, although giddiness is experienced more frequently than nystagmus can be recognised. If one applies the tests for inducing nystagmus to such a case, the labyrinth in question appears to be hypersensitive. The existing nystagmus can be temporarily abolished or intensified, according to the test, more easily when the stimuli are applied to the affected ear than when applied to the other side.

Spontaneous rhythmic rotatory nystagmus to one side is also met with when the opposite labyrinth has become functionless. This form of nystagmus develops the moment the one labyrinth is destroyed, but disappears when both labyrinths become defunct. It does not usually occur in young children, and Sir Victor Horsley has pointed out its absence or very early disappearance in monkeys. In the adult human being this contra-lateral nystagmus is an invariable sign of unilateral ablation, and can be observed, if sought for, several weeks, months or sometimes years after the onset. It certainly tends to become less intense as time goes on, but is liable to be renewed in intensity temporarily whenever the patient's general health fails. In a few instances contra-lateral nystagmus, due to ablation of one labyrinth, is accompanied by a fine horizontal nystagmus toward the defunct side. Dr. James Taylor has pointed out the importance of observing well-marked nystagmus toward the diseased side with fine nystagmus to the contra-lateral side in cases of cerebellar tumour, and we should remember that the same sign is observed in cases of cerebellar or

https://doi.org/10.1017/S1755146300014293 Published online by Cambridge University Press

extra-cerebellar abscess, which generally arise from an infection of the middle or internal ear.

Labyrinthine Nystagmus in cases of Absolute Blindness.—When the labyrinth is stimulated in cases of total blindness, due to a local cause, such as primary optic atrophy, and when the blindness is not accompanied by intracranial disease, rhythmic nystagmus can be provoked by rotation or irrigation in the usual way, but the primary component comes into greater prominence as the eyeball aimlessly deviates or rolls to one side in response to the labyrinthine stimulus. In the absence of retinal stimuli the patient is directed to look towards his own hand, which is passively held to the right or left as desired, and we shall then notice that the oculo-motor effort to deviate the ocular axes at once induces true rhythmic nystagmus, having characters similar to those met with in patients with normal vision subjected to the same tests.

Methods of Measuring the Strength of the Stimulus and the Intensity of the Nystagmus.—It is sometimes found that nystagmus can be more easily produced by stimulating one ear than by stimulating the other. Just as the knee-jerks are described as being equally or unequally brisk, sluggish or abolished, so may we describe the labyrinthine reflexes as being equally brisk, sluggish, or abolished. It is difficult to choose a criterion for briskness and sluggishness, and so to a great extent we have been satisfied by concluding that the labyrinthine reflexes are either present or absent. There is a possibility of estimating and recording degrees of activity by elaborating the following methods:

Rotation.—(1) Employing a constant stimulus; that is, constant rate and number of rotations and noting the duration of the nystagmus which follows the sudden stop. (2) Ascertaining the minimal number of rotations which induce nystagmus.

Caloric Method.—(1) By employing a constant temperature of the thermal agent and observing the period of induction before the onset of nystagmus. (2) By measuring the quantity of the fluid at a constant temperature which flows before nystagmus can be recognised. (3) By noting the extremes of temperature employed to provoke nystagmus.

Galvanic Method.—By observing the current in milliamperes which is required to induce nystagmus.

Dr. DAN MCKENZIE: *Vestibular Nystagmus.*—There are one or two points connected with the *physiology* I should like to touch upon. First of all, with regard to the mechanism by which rotation induces nystagmus, I agree with those who hold that the movement of the endolymph in the semicircular canal is never a steady round-and-round circulation. That there must be some movement of the fluid is undoubted, but the movement is that of a wave rather than that of a current—a local rise or fall in fluid pressure.

Arguing from the anatomical conformation of the canal, I suggest that under the ordinary conditions of normal life the rise or fall in endolymphatic pressure in the ampulla, consequent upon the ordinary changes in position of the head, is quickly relieved by the local excess of fluid passing into the finer position of the canal, and so round to its undilated end, and the pressure being thus rapidly equalised an excessive stimulation of the end-organ is avoided. (The process is similar but reversed, of course, in direction in the case of fall in intra-ampullary pressure.) But when, as in continued rotation, this change in pressure is sustained for a time, or when there is a repercussion, so to speak, the finely calibred canal will be incapable of providing a sufficiently rapid relief and excessive stimulation of the nerve-ending will result, with

https://doi.org/10.1017/S1755146300014293 Published online by Cambridge University Press

nystagmus, vertigo, etc. This theory necessarily refers the period of equilibrium of eye-movements *during* rotation to the exhaustion of the nerve centre. The ampulla may be regarded as a device to favour the reception of small waves, while the rest of the canal provides a method of rapidly equalising the pressure, and so of calming the oscillation.

I turn now to the *pathological* aspect of the question, and first of all to the *spontaneous nystagmus* of the "labyrinth storm." In this particular I have only one remark to make, and that is that I agree that the direction of the spontaneous nystagmus should *not* be taken as the faithful guide to the labyrinth that is affected, or as the means of distinguishing acute labyrinthitis from cerebellar abscess. In acute labyrinthitis I have more than once observed spontaneous nystagmus to the affected side, although, of course, in most cases it is directed to the opposite side. The reliable distinction lies in this, that in acute labyrinthitis the nystagmus declines in severity as time goes on, whereas, in cerebellar abscess, the symptom persists unaltered, or it may even become more marked.

In Non-Suppurative Disease of the Internal Ear I have for several years past been accumulating experience with regard to the vestibular reactions. By adopting the method of measuring the duration of rotation nystagmus, as Bárány advises, or the method of measuring the duration of the induction period of the caloric test, in the manner I have suggested, or by both of these methods combined with an observation of the phenomenon in general, we are able to detect the presence of slight or moderate interference with the vestibular reflexes, and can often thereby obtain information of considerable moment bearing upon the diagnosis, and even upon the prognosis of our cases.

The method of measuring the induction period of caloric nystagmus consists in running into the (cleaned) external auditory meatus a gentle stream of water of a temperature of from 22° to 24° C., the time being noted when the water begins to flow and when the nystagmus is first apparent, the slow deviation of the eyes being the first to appear. The patient should be instructed to turn his eyes to the opposite side. Although, naturally, I believe the merits of this method to be considerable, I have no desire to lay special stress upon it, because while it is true that as a rule the length of the induction period varies directly with the activity of the reflex, in a few cases the induction period will be found to be of normal duration (twenty-five to thirty-five seconds), although the excursion of the ocular movements is short and the vertigo trifling or absent. Such cases are, of course, defective, although the induction period is of the normal length.

With regard to the findings. Most of the cases seem plain and straightforward, but anomalous results occur every now and again of such a nature as to defy explanation.

An effort was made to correlate the results of the cochlear tests with those of the vestibular tests. The idea before my mind has been to ascertain, if possible, whether the vestibular tests in cases of perceptive deafness, not due to labyrinth suppuration, are of any reliable value in arriving at a diagnosis of the disease or in forming a prognosis in so far as the hearing is concerned. It often happens that the vestibular tests provide us with information not only of interest but also of practical use.

Out of thirty-six cases, nineteen showed a correspondence between the loss of hearing and the impairment of the vestibular reflex. Sometimes, minor discrepancies occur—the deafer ear may show a livelier vestibular reaction than the better ear does, or on one side, although the

https://doi.org/10.1017/S1755146300014293 Published online by Cambridge University Press

deafness is considerable, the vestibular response may be normal. But, on the whole, the claim that a harmony between the two systems exists, more or less complete, is substantiated. In two cases of simple noise-deafness, where, one would suppose, the lesion must necessarily be limited to the cochlea, the vestibular responses were deficient. It is noteworthy that the same result was recorded in a former publication dealing with an earlier series of cases.[1]

Seventeen cases showed cochlear and vestibular impairment not in harmony.

Of five cases of nerve-deafness with normal vestibular reactions no fewer than three were proved to be functional and one was suspected of being functional. One case in this group was that of a young woman with very slight nerve-deafness of short duration and normal vestibular reactions, in which it may be supposed that if it should prove to be organic the cochlear lesion was too recent to have spread to involve the vestibule. Of eight cases of nerve deafness with exaggerated vestibular reactions, four were almost certainly functional, two were doubtfully functional, one was almost certainly organic, and the diagnosis in one was unknown. That is to say, out of thirteen cases of nerve-deafness with normal or exaggerated vestibular responses, seven were certainly and three were probably functional in character; while, in two, the diagnosis was that of organic ear disease (one slight, the other moderate, and both of short duration); one case was not diagnosed.

These figures, as far as they go, tend to support, with some qualification, the observation I have previously made that the presence of normal or exaggerated vestibular responses is a point in favour of regarding such cases as functional. The qualification to be made consists in this, that organic nerve-deafness, if of recent origin, may be combined with normal or excessive vestibular responses.

Finally, in four cases the vestibule was affected more than the cochlea. In two the hearing was quite normal in one ear; in two the loss of hearing was only detected by the tuning-fork test; in one there was severe deafness in one ear and normal hearing in the other. The later history of this case and of another with severe unilateral deafness bears out a surmise that in a case with good hearing where the vestibular responses are seriously impaired, the likelihood is that the hearing also will in time become seriously affected. On the other hand, we may regard as more favourable, cases of nerve-deafness, especially if long continued, where the vestibular reactions are normal or exaggerated, bearing in mind, however, the fact that hysterical or functional deafness, like any other similar manifestation of this disorder, is often persistent.

In conclusion, I think we ought to regard the absence or deficiency of vestibular reactions as of more positive value in any given case than their presence, whether normal or exaggerated.

Mr. JOHN F. O'MALLEY described a classification of nystagmus in a series of tables, contrasting the characters of the various types, and indicating the conditions under which they arise physiologically and pathologically.

Mr. T. B. LAYTON found the tests difficult in their application. He did not think that we have in these tests a short cut to the diagnosis of nervous disease, but he claimed we may get from them evidence in a certain number of cases which, when correlated with that obtainable from other sources, will clinch a diagnosis that otherwise must have remained

[1] JOURN. OF LARYNGOL., RHINOL., AND OTOL., December, 1909, xxiv, p. 646.

https://doi.org/10.1017/S1755146300014293 Published online by Cambridge University Press

doubtful, or will exclude a gross lesion that otherwise must have been left under discussion. The eighth nerve he looked upon in the same light as is the second. No one would dream of making a diagnosis of or of excluding an intracranial tumour without examining the second nerve. Why should they do so without a complete examination of the eighth nerve. The neurologists either must learn to do these tests themselves or they should send their cases to the otologist for examination and report, in the same way as they now either examine the fundus oculi themselves or ask the ophthalmologist to do so for them.

Mr. G. J. JENKINS desired to express disagreement with the generally accepted theory regarding the association of certain forms of nystagmus with certain canals. It has been held by Bárány and generally accepted, as described in the introductory paper by Mr. Scott, that rotatory nystagmus is associated with the superior semicircular canal and vertical nystagmus with posterior semicircular canal. It appeared to him that the canal which received the maximum stimulation by rotation in lateral flexion of the neck and face turned forward and upward must be the superior canal, if the rotation be made from that side forward. This rotation would produce a vertibulo-ampullary current in the superior canal on the flexed side and an ampullo-vestibular current in the posterior canal of the opposite side. Other canals would be affected, but the resulting vertical nystagmus would probably be due to the preponderating influence of this superior canal and not the posterior canal. Similarly, he held that the posterior semicircular canal and not the superior was the important peripheral stimulus for rotatory nystagmus. He could not agree that sufficient evidence had been brought forward by the caloric test to prove that the various forms of nystagmus could be due to other canals than those described. At least, he held that anatomically the above reasoning was correct.

It is interesting to note that a nystagmoid movement artificially produced may cause vertigo with associated phenomena in certain cases. By rotating a cylinder with a series of spots on it and getting the patient to fix these spots as they come into vision vertigo can be produced. If these be done where there is an irritable labyrinth the nature of the vertigo can sometimes be changed at will of the operator. He has been in the habit for some years of testing for the irritable labyrinth by means of a mirror rapidly moved to produce a nystagmoid movement.

PROCEEDINGS OF THE SCOTTISH OTOLOGICAL AND LARYNGOLOGICAL SOCIETY.

Held in the Western Infirmary, Glasgow, June 6, 1914.

DR. WALKER DOWNIE, *in the Chair.*

Report by DR. W. S. SYME.

(*Continued from p.* 442.)

Dr. Maurice's Kinesiphone.—Its Mechanism and Mode of Use.—The Author's Claims and Results.—Effects in three cases of Catarrhal Deafness treated with its Aid.—J. Mackenzie Booth.—The

https://doi.org/10.1017/S1755146300014293 Published online by Cambridge University Press