

NEUROOTOLOGY.

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Neurotology is a study of the internal ear and its associated intracranial nerve pathways. While it is strictly an ear study and essential to the proper diagnosis of any pathologic ear condition, it is of much broader application as a means of obtaining data for diagnosis and treatment in general medicine and surgery. The internal ear consists of two distinct organs, the cochlea, which is the organ of hearing, and the kinetic static labyrinth, which is the organ of equilibration. The cochlear portion offers a very limited field from a diagnostic standpoint, while the examination and study of the kinetic static labyrinth, consisting of the utricle, saccule and the three semicircular canals, together with their intracranial nerve pathways, affords, on the other hand, a very wide field for diagnosis and treatment.

We are indebted to the Vienna group of otologists for the present methods of examination of the internal ear. Robert Barany was awarded the Nobel prize in 1915 for his original work in this field. His name will be forever associated with the clinical relation of the internal ear and the central nervous system.

For centuries we have recognized only five special senses. Within the last two decades, a sixth sense, the muscle, joint and splanchnic sense, has been recognized. It is unlike the other special senses in that it does not make its impression on our consciousness. By means of this sixth sense the individual performs coordinate acts automatically and unconsciously. The researches which have been made have revealed a seventh special sense, the kinetic static sense, which is a special sense, just as truly as sight and hearing are special senses.

The seventh special sense, in common with the other special

senses, is presided over by a special sense organ, which likewise has an end organ, a brain center and nerve pathways connecting them. The end organ is in the labyrinth, and the brain center has been postulated by Mills to be located in the posterior portion of the first and second convolutions of the temporosphenoidal lobe near the cortical center for hearing. It is believed to be more highly developed on the right side.

The nerve pathways or vestibular tracts have not been entirely worked out. From anatomic and physiologic studies and from operative and postmortem findings these tracts may be said to have a definite course from the labyrinth to the cortical center. The fibers from the horizontal canals and those from the vertical canals join the fibers from the cochlea in the vestibule and form Scarpa's ganglion, which gives origin to the eighth nerve. The eighth nerve passes through the internal auditory canal to the brain stem, where it divides into the cochlear portion and the vestibular portion.

The fibers from the horizontal canal and those from the vertical canals have a different course within the brain stem. The horizontal canal fibers go to Deiter's nucleus in the upper part of the medulla and here divide into two separate tracts. The vestibulocerebellocerebral tract ascends by way of the juxta restiform body in the inferior cerebellar peduncle to the dentate nucleus in the cerebellum. The vestibuloocular tract ascends to the posterior longitudinal bundle and goes to the sixth and third eye muscle nuclei in the pons. The fibers from the vertical canals ascend into the pons and divide into two tracts. The vestibulocerebellocerebral tract goes to the dentate nucleus in the cerebellum by way of the middle cerebellar peduncle, and the vestibuloocular tract ascends to the fourth and third eye muscle nuclei in the pons.

The vestibulocerebellocerebral tracts ascend from the dentate nucleus through the superior cerebellar peduncle and the crura cerebri to the pyramidal tract and hence to the cortical center of the opposite side. A few of the fibers go to the cortical center of the same side. The cortical center for the quick component of nystagmus has not been definitely located, but the cerebroocular or supranuclear tracts descend through the internal capsule to the posterior longitudinal bundle and

hence to the eye muscle nuclei. The vestibuloocular and the cerebroocular tracts preside over nystagmus, and the vestibulo-cerebellocerebral tracts preside over vertigo.

When the endolymph in the semicircular canals is stimulated and made to move, two reactions occur in a normal individual, namely, nystagmus and vertigo. Past pointing and falling are the objective evidence of the subjective vertigo. The stimulus is brought about by turning the individual or by douching the ears with cold or hot water for the purpose of changing the temperature of the endolymph. The turning and the temperature changes produce a movement of the endolymph in a given direction. The turning is applied by placing the individual in a specially adapted chair and turning with the head in the proper position, at a certain rate of speed and for a definite number of times. In a normal individual nystagmus and vertigo of a proper type and duration will invariably result. If one or both are absent or changed in character it has pathologic significance. This means that the stimulation did not go through so as to produce a normal reaction.

The other method of applying stimulation to the endolymph is by douching with cold water at 68 degrees or hot water at 112 degrees. This procedure results in the same phenomena that we obtain from turning, namely, nystagmus and vertigo of a given type and duration. Here again if the reaction departs from the normal or is absent it has pathologic significance. Turning stimulates the endolymph in both labyrinths, while douching stimulates only the labyrinth douched. The reaction from the cold douch is the opposite from that obtained from the hot douche.

It is important to remember that a neurorhino examination not only investigates the kinetic static labyrinth, but also its widely distributed nerve pathways. We can from such an examination gain information regarding the pathology of the labyrinth, eighth nerve, medulla, pons, the six peduncles, the cerebellum, the crura cerebri, the pyramidal tracts, the internal capsule and some parts of the cerebrum. It is this wide distribution, all so wonderfully coordinated in health, that has opened up new possibilities to the clinician. There is, in fact, no part of the body musculature unaffected by the ear stimulation. When the reaction is normal it means that the nerve

pathways are intact, but when there is a changed reaction or an absence of reaction it means an impairment of either the terminals or the lines of communication.

To the clinician the study of vertigo is an ever present problem. We are dependent for a perfect balance mechanism on the sight sense, the muscle sense and the kinetic static sense. This sense trilogy makes possible estimations of all sorts, orientation, locomotion, the rate of motion, estimation of weight and the realization of posture and position. The kinetic static sense is most important and it has as its sole function the maintenance of balance. After impairment or loss of one of the three, compensation may occur to a certain degree. The tabetic may learn to avail himself of his sense of sight and his kinetic static sense. The blind man is able to walk until deprived of his muscle sense or his kinetic static sense. Deafmutes in whom the kinetic static sense is destroyed maintain their balance by sight and muscle sense, and develop incoordination only in the dark or in the water. Full compensation, however, cannot develop unless two of the trilogy remain normal, and perversion of any one of them may be more provocative of trouble than its loss.

Previous to the realization of this important new function of the ear we regarded the ear as purely an organ of hearing. The physician was concerned with the disturbance or loss of the hearing function or with the effects of the extension of infection from the ear to adjacent structures. The general practitioner is concerned with the new development in diagnosis because of the value of the ear tests in determining the cause of vertigo, no matter what its origin or type, and in recognizing pathologic conditions of the internal ear itself. Vertigo is a subjective sensation of disturbed relationship of one's own body to the surrounding objects in space. Although it has been recognized for many years that vertigo may result from ear disturbances, the conception that all vertigo, from whatever cause, is peculiarly an ear study, is the result of the study of the new otology. These dizzy cases suffer acutely with apprehension and, with no explanation at hand, their apprehension increases. The vague terms in which vertigo is often spoken of is an index of the uncertainty in our minds regarding this distressing condition. Often we speak of inter-

tinal or stomach vertigo or dizziness from refractive errors or from Bright's disease or neurasthenia without thinking of the real mechanism of its production.

It has been impossible to tell, in any given case, whether the vertigo was due to a functional or organic disease and still less to recognize whether it was of trivial significance or the forerunner of some serious disease. Vertigo should be considered as a distinct clinical entity, deserving as careful investigation and analysis as any other distressing pathologic condition.

All conscious sensations are cerebral. Vertigo is a disturbance perceived within the brain, just as taste or smell. If a disturbance of any organ is accompanied by vertigo it is because of a direct attack on the apparatus capable of producing vertigo. If the same condition fails to involve the ear and its pathways, there will be no vertigo. If a patient with vertigo gives an exaggerated reaction of normal character, it means that the patient is suffering from an irritation of the vestibular organ, either distally or centrally. This is usually toxic in character. The source of the toxicity may be a focal infection or a gastrointestinal toxicity or some specific disease, such as syphilis or any of the acute infections. If the patient gives an abnormal reaction it signifies an organic lesion. It is assumed that any toxicity may in time produce an organic change in the tissue involved. It has been proven beyond doubt that vertigo is not a "cerebellar pull," but the cerebrum's conscious effort to compensate.

The following classification of vertigo is, as far as we know, complete:

1. Involvement of the ear mechanism by a lesion in the ear itself. "Meniere's symptom complex" is due to a sudden destruction of the labyrinth by hemorrhage or by effusion into it, such as may occur in nephritis or diabetes or other conditions affecting the cardiovascular system. The usual symptoms of deafness, tinnitus, vertigo, staggering and, if severe, nausea and vomiting, are all explained by either a toxic irritation of the labyrinth or by cardiovascular disease producing anemia or hyperemia. Any inflammatory condition affecting the labyrinth gives the same picture in a varying degree. Slow

degenerative changes within the labyrinth cause attacks of vertigo from time to time.

2. Involvement by a lesion affecting the intracranial pathways from the ear. Lesions within the brain causing vertigo may be tumor, hemorrhage, thrombosis, infarct, abscess, gumma, tubercle, leukemic infiltration, specific neuritis, multiple sclerosis, syringomyelia, polioencephalitis and meningitis.

3. Involvement by ocular disturbances, either through the eye muscle nuclei or through association fibers from the cuneus to the cortical terminus of the fibers from the ear in the posterior part of the first and second temporal convolutions. This group includes all the cardiovascular conditions which produce either congestion or ischemia within the cranium.

4. Involvement by toxemias from any organ or part of the body. This group includes ptomaine poisoning, alcoholism, chemical poisoning, such as lead, quinin, salicylates, the toxemia of nephritis, rheumatism (focal infections), syphilis, infectious fever, such as mumps, scarlatina and typhoid fever. These toxemias may be grouped in two classes: (a) Evanescent toxemias, which have caused no degeneration of the cellular elements within the ear or the nerve pathways. This constitutes a large group of the cases of vertigo seen by the practitioner. (b) Toxemias which have caused a definite damage to the internal ear or its nerve extensions, such as in mumps or syphilis, or repeated attacks of a milder toxin, such as from gastrointestinal conditions or focal infections.

In aviation we have a practical example of the function of the ear in maintaining equilibrium. It is not necessary for an individual to have a perfect muscle sense, perfect sight and a perfect kinetic static sense in order to have an equilibrium sufficiently good to take care of the ordinary pursuits of life. In aviation, however, it is imperative. When flying above the clouds or in the dark the sight sense is of no avail, and the muscle sense in an unstable and moving machine affords little or no sense of balance. It is essential, therefore, that the aviator have a perfect vestibular apparatus, as it is upon this alone that his balance when flying depends. Some of the unexplained accidents in aviation are very likely due to a sudden concussion of the internal ear from the deafening

roar of the motor or by a decrease in the air pressure at great heights. In a rapid ascent from a denser to a rarer air there occurs an oxygen insufficiency which has a direct effect on the ear mechanism through the blood stream. Unquestionably many an aviator has gone to his death because he did not know that he did not possess a normal internal ear. The United States was the first nation to appreciate the need of the neurootologic examination of aviators. During the late war this branch of the service was very highly and efficiently developed. The world owes a great deal to Isaac Jones of Philadelphia, not only to his pioneer work in this field during the war, but as well to his original researches and contribution to neurootology for the past few years.

Seasickness or mal-de-mer is a phenomenon resulting from the movement of the endolymph within the internal ear. As long as man has traveled by water, seasickness has been a source of distress to thousands of travelers. It would be impossible to enumerate the theories advanced for the cause of the malady, and the remedies have been as numerous as the theories. With the discovery of the physiology of the kinetic static labyrinth and a study of the reactions attendant upon stimulation of this organ the true etiology of seasickness was evident. The first symptom of seasickness is dizziness, which is followed by staggering on attempting to move. The face becomes flushed and soon is changed to a pallor, followed by nausea, and if the condition continues, by vomiting. This is entirely due to the endolymph movement brought about by the motion of the ship. This is the same picture obtained by douching the ears. Women are more susceptible to seasickness than men, little children are very little affected and infants not at all. This corresponds with our experience following douching. Infants never vomit, no matter how long the ears are douched. The vestibular organ of infants is not fully developed, which accounts for the absence of this reaction. From repeated experiences most people develop what may be termed an immunity to seasickness, which is also true of repeated stimulation by artificial means. An individual who cannot be nauseated by the examination of the vestibular apparatus will not become seasick.

The ordinary endolymph movements, such as accompany the usual pursuits of life, are responsible for our body balance because the brain through countless repetitions has learned to interpret the significance of these movements. When the movements become unusual the brain cannot interpret them and confusion and vertigo result. If the unusual movements recur frequently the brain learns to interpret them and vertigo no longer occurs, hence immunity to seasickness. The mechanism of vomiting is chiefly through the vagus and phrenic nerves. Nausea is a different reaction and is the cerebral interpretation of the unusual stimulus. Cumulative nausea may produce vomiting by a motor impulse from the cerebral cortex to the nuclei of the vagus and phrenic nerves. Besides the cerebral impulse we have association fibers directly from the ear to the vagus and phrenic. The vestibular nerve enters the posterior longitudinal bundle, which in turn is directly connected with the vagus nucleus. Other vestibular fibers enter the medulla and descend to the motor cells of the anterior horns of the spinal cord. The phrenic nerve has its origin in the cells of the anterior horns of the second, third and fourth cervical vertebræ. It is thus that the impulse travels direct to the vagus. Certain olfactory and visual impulses are contributing factors in overcoming the cerebrum's attempt to keep from vomiting. We may, of course, have nausea and vomiting without endolymph movement. If one could place himself in such a position that the motion of the ship would cause endolymph movement only in the horizontal plane there would be no seasickness. Unfortunately this is impossible, as the ship movements are in more than one plane. However, the seasickness is often mitigated by lying in the plane of the greatest movement. Any treatment or drug that will lessen nerve irritability will tend to lessen the seasickness. By a vestibular examination we can determine if an individual will become seasick.

It is well known that the toxin of syphilis has a predilection for the eighth nerve, and oftentimes this will be a very early manifestation of the disease. In any stage of the disease it will often be the first part of the nervous system to become involved, and this may occur in the entire absence of any other clinical evidence of the disease. In the past we have

had to depend upon the functional tests for hearing to detect the presence or absence of eighth nerve involvement. At the best this is uncertain, and its unreliability has placed this method of diagnosis in ill repute. In the examination of the labyrinth by the Barany method we have an objective measurement of the degree of function of the eighth nerve. The following data may be presented as an aid in the diagnosis and treatment of syphilis. In the absence of the microscopic diagnosis of the initial lesion, or a Wassermann reaction, we can by these tests detect a slight diminution of function of the eighth nerve and by repeated examinations detect a progressive condition. In this way we may gain information that is suggestive in the presence of an uncertain Wassermann. We may detect by this means a lesion of the eighth nerve or the central nervous system which might not be evident for some time. In some cases this has been demonstrated years before any clinical evidence, and in the presence of a negative spinal fluid Wassermann. The early diagnosis of syphilis of the central nerve system is so important that the use of this means of diagnosis cannot be too strongly urged. We may demonstrate this before gross changes have taken place and while there is still hope of therapeutic help.

Recurrence of syphilis of the central nervous system is very insidious. The blood and spinal fluid Wassermann may be negative. The inconvenience of repeated spinal fluid examinations for control of recurrences is such that it is often not done, but this method affords an opportunity for repeated tests without discomfort or inconvenience to the patient. We may also by repeated examinations during the course of treatment of syphilis of the nervous system determine the efficiency of the treatment. We know how difficult this measure is by the usual means. If the patient is not cured the vestibular examination of the central nervous system will accurately reveal it.

The value of the eye examination to the neurologist has long been recognized. Based on opinions of neurologists and ophthalmologists who have had experience with the Barany examination, it is safe to say that the ear examination affords as a rule more definite and exact information than the eye. In Vienna no neurologic examination is considered complete

without a vestibular examination. The ear tests are very valuable in making differential diagnoses between labyrinth and intracranial lesions and in furnishing additional data for intracranial localization. Nystagmus and vertigo, often associated with nausea and vomiting, may be due to either a labyrinth lesion or an intracranial lesion. In many instances the symptoms of internal ear disturbance and cerebellar lesion are identical. It is in such cases that the ear tests are valuable.

A differential diagnosis between peripheral and central lesions by means of the ear tests depends on certain general principles. A peripheral lesion of the labyrinth or eighth nerve is suggested by the following:

1. An impairment of the function of both the cochlear and the kinetic static labyrinth. If the examination of the hearing shows cochlear deafness, and the tests of the semicircular canals show that their function is also impaired, it immediately becomes probable that we are dealing with an end organ lesion.

2. The history or presence of tinnitus is evidence of labyrinth involvement, but its absence does not mean an unimpaired labyrinth.

3. Proportionate impairment of the responses from the horizontal canals and of the vertical canals indicates labyrinth involvement.

4. Proportionate impairment of both nystagmus and vertigo from the horizontal canals and normal responses from the vertical canals indicates a lesion involving the fibers from the horizontal canal in the eighth nerve or in the horizontal canal itself. It is the proportionate impairment of responses that speak of an end organ lesion.

A central lesion is suggested by the following:

1. Normal cochlear, but impaired or nonresponsive semicircular canals.

2. Normal responses from the horizontal canals, but absent responses from the vertical canals.

3. Normal responses from the vertical canals, but absent responses from the horizontal canals.

4. Normal vertigo, but impaired nystagmus from the horizontal canals.

5. Normal nystagmus, but impaired vertigo from the horizontal canals.

6. Normal vertigo, but impaired nystagmus from the vertical canals.

7. Normal nystagmus, but impaired vertigo from the vertical canals.

8. Normal vertigo and normal nystagmus from any semicircular canal, but impaired past pointing in any direction of any extremity.

9. Normal vertigo and normal nystagmus from any semicircular canal, but an impairment or absence of normal falling.

10. Spontaneous vertical nystagmus is pathognomonic of a central lesion and is indicative of involvement of the brain stem caused either by infiltration or pressure. A lesion of the labyrinth may produce many forms of spontaneous nystagmus, horizontal, rotatory, oblique or mixed nystagmus of these types, but an ear lesion can never produce a spontaneous vertical nystagmus, either downward or upward.

11. If there exists a spontaneous nystagmus to the right and nonresponsive semicircular canals of the right ear, an intracranial lesion is suggested. The nonresponsive labyrinth, if the labyrinth alone were responsible, would produce a nystagmus to the left.

12. A spontaneous nystagmus of increasing intensity or of long duration is indicative of a central lesion. A spontaneous nystagmus due to a lesion of the labyrinth shows its greatest intensity at the onset of the disease, becoming less and less marked and disappearing after a few days.

13. If the stimulation of any semicircular canal produces a perverted or inverse nystagmus, it is pathognomonic of a central lesion and is indicative of a brain stem involvement. Such phenomena as the following are frequently seen: Douching the right ear with cold water, with the head back, thus stimulating the right horizontal canal, should give a pure horizontal nystagmus to the left. If on such stimulation there occurs a vertical nystagmus upward or downward, a rotatory, oblique or mixed nystagmus, it may be spoken of as a perverted nystagmus. If instead of a horizontal nystagmus to

the left there is produced a pure horizontal nystagmus to the right it may be termed an inverse nystagmus. Neither a perverted nor an inverse nystagmus can possibly be produced by a lesion of the labyrinth or eighth nerve. A peripheral lesion produces a poor nystagmus or no nystagmus, but an absolutely false response of necessity demonstrates a central lesion.

14. If the ear stimulation produces a conjugate deviation of the eyes instead of nystagmus, it is pathognomonic of a central lesion.

The above outline indicates how additional data may be furnished to the neurologist by the labyrinth examination in determining whether he is dealing with a lesion of the internal ear, the eighth nerve, the brain stem or the cerebellum. In the broad field of brain localization the examination of the internal ear is also of distinct value. The particular feature of the ear examination is that the aurist sends in a stimulus to the brain centers and then notices the responses of the different parts of the body to this stimulation. If the ear and its nerve pathways are all intact the responses will be normal. If there is a failure of any or all of the responses, it is positive evidence of an interruption along that particular path or paths, that fails to bring about these responses.

In order to utilize the knowledge obtained from these tests it is essential to bear in mind the various pathways constituting the vestibular apparatus. The horizontal canal fibers after entering the brain stem have a different course from the fibers from the vertical canals. Each tract divides into two tracts, one the vestibuloocular tract, responsible for nystagmus, and the other the vestibulocerebellocerebral tract for vertigo. By determining the specific tract or tracts which fail to respond we can locate the site of the lesion. In order for the data to be reliable it is essential that the technic of the examination be accurately and painstakingly carried out. The ear examination is not for the purpose of making a neurologic diagnosis, but merely to give additional information, by a series of refined experiments, to be added to the knowledge obtained by the neurologist by other methods at his command. In the cases which the neurologist finds no difficulty

in making a diagnosis it is useful in providing an additional evidence in corroboration. In the obscure case the neurologist may find the ear tests to provide the most accurate information obtainable.

The diagnosis of the precise location of a lesion within the cranium is probably the most difficult task with which the surgeon is confronted. Realizing this, he avails himself of every modern aid and diagnosis, such as the laboratory and spinal and corpus callosum punctures and the refinements developing therefrom, such as oxygen injections, pressure measurement and the analysis of the fluid withdrawn and the X-ray. For many years past no surgeon would consider doing an intracranial operation without an eye examination. We have previously shown the value of the labyrinth examination in differentiating between labyrinth and intracranial lesions. A point worthy of emphasis is that with the aid of these tests he is frequently enabled to determine whether a lesion is operable or not. Many lesions of the medulla, pons or cerebellar peduncles, which are inoperable by the very nature of their location, will show pronounced cerebellar symptoms, and not infrequently the cerebellum is exposed in such cases in the hope of finding a lesion near the cortex and removing it. For such a differentiation, in many cases, no method so far developed equals the Barany tests. If after turning and douching there appears a normal past pointing of both extremities in both directions, it may safely be assumed that the cerebellum is intact. This cannot be regarded as absolutely final, but is much more definite than any other known method for determining the integrity of the cerebellum.

Tumors of the cerebellopontine angle either originate from one of the cranial nerves in the angle, usually the eighth nerve, or have invaded the angle secondarily from the cerebellum or brain stem. The eighth nerve is usually involved in these cases, and our study of the eighth nerve gives a direct insight as to the condition in the cerebellopontine angle. In addition, the ear tests usually demonstrate the two following phenomena in cases of cerebellopontine angle tumor:

1. An absence of all responses from the vertical canals of the ear opposite the lesion. Given a tumor of the right cerebellopontine angle, the usual findings are as follows: The

right ear gives no responses, the cochlear showing an absence of all function and the stimulation of the horizontal canal and the vertical canals failing to produce any nystagmus, or vertigo, or past pointing or falling. The left ear shows unimpaired hearing and the horizontal canal normal nystagmus, vertigo, past pointing and falling. The vertical canals, however, fail to produce any response because the vertical canal fibers are most probably impaired owing to pressure upon the pons by the tumor of the angle.

2. Crossed past pointing. This phenomenon consists of persistent past pointing of both upper extremities either outward or inward, regardless of the ear stimulation employed.

In the examination of head injury cases we are able to be of considerable assistance. If the injury involves the labyrinth, as is so often the case, we can determine accurately the extent of the damage and give an accurate prognosis. In another group of head injury cases in which the intracranial hemorrhage is the main factor in producing a serious intracranial condition, we have found a very consistent picture in our neurootologic examination. In this group the blood, regardless of the seat of the injury, gravitates to the posterior fossa above the tentorium and, filling the fossa, overflows through the incisura tentorii, into the fourth ventricle. The examination of a very considerable group of such cases has given consistent vestibular findings. Stimulation of the vertical canals gives no responses whatever. We have an absence of vertigo and nystagmus showings that the stimulation does not go through. Stimulation of the horizontal canals gives either a normal reaction or, if the hemorrhage is large, a diminished vertigo, but a normal nystagmus. In other words we have a central blocking of the passage of the stimulus. The interpretation of the above findings locates the obstruction in the fourth ventricle. The vertigo which we have in these cases is explained by the vestibular examination. The prognosis is good in most cases, as compensation occurs and the patient does not have vertigo under ordinary circumstances. The vestibular examination may be made use of in helping to determine the advisability of surgical intervention in these cases. If the vestibular findings are normal it means that there is not sufficient hemorrhage to cause the condition

above described. In the absence of blood in the spinal fluid and no increase in the pressure, a normal vestibular examination adds weight to the surgeon's decision of nonsurgical treatment.

If the ear stimulation produces normal nystagmus, vertigo, past pointing and falling, the Barany tests are of unquestioned value in eliminating a lesion in the posterior fossa and brain stem. If they render no other service than this it would be sufficient to make them a distinct contribution in the diagnosis of intracranial lesions. In addition they may prove helpful in preventing unnecessary operation. In fact, no operation on the brain should be undertaken without giving the patient the benefit of the ear examination.

The intimate relation between the ear and the eye can best be appreciated when we realize that the ocular mechanism constantly depends upon stimuli from the ear for precision of movement. Steadiness of fixation is dependent to a large extent on normally functioning ears. Tonic impulses from the right ear, for example, continually tend to draw the eye to the left. This is proven by a sudden loss of function of the right internal ear, which invariably results in a deviation of the eyes to the right because the tonic impulses tending to draw the eyes to the left are impaired and there is a resulting nystagmus to the left. The same is true of the left side, only in an opposite direction. This may be further demonstrated experimentally by electricity. By applying the anode of the galvanic current to the right ear, which depresses the function, there results a drawing of both eyes to the right, with resulting nystagmus to the left. By applying the kathode, thus stimulating the right ear, the opposite phenomenon results. It is thus shown that ocular equilibrium, just as bodily equilibrium, is normally dependent on properly functioning ears. Artificial stimulation of the ears will produce to order any type of nystagmus. The laws governing these phenomena are invariable and simple. For example, if we wish to produce a vertical nystagmus upwards, we place the patient's head over towards the right shoulder, turn him to the left and we have a vertical nystagmus upward. The fistula test also serves to illustrate. If there is a caries of the bone

in the outer wall of the labyrinth or a movable stapes, applying pressure or suction in the ear canal causes nystagmus. The tests have a practical value to the ophthalmologist in studying ocular palsies and spontaneous nystagmus.

In studying ocular palsies it is important to note that the ear stimulation is much stronger than the voluntary cerebral stimulus. If a patient is unable to look to the right with the right eye, the question arises as to the origin of the involvement of the sixth nerve. If ear stimulation causes the eye to move outward it is evident that the sixth nerve is **not** completely destroyed. This makes for a refinement of the determination of the degree of paresis and aids in the prognosis. The horizontal canal ocular fibers go to the sixth nerve, which supplies the external rectus and that part of the third nerve which supplies the internal rectus. If appropriate stimulation is applied it will show, in many cases, that the patient's inability to look to the right or left is due to only a partial impairment of the sixth nerve. The same will apply to tests for paralysis to the right or left or upward or downward. Loss of conjugate deviation in various directions should always be studied by means of the ear tests. If there is a loss of voluntary control and the patient when asked is unable to look in a given direction with either eye, and the ear stimulation causes the eyes to move in this direction, it shows without question that there are normal pathways from the ear to the eye muscle nuclei, and through the nerves of the eye muscles themselves, and that the lesion is supranuclear.

The Barany tests have taught us much that we did not know about spontaneous nystagmus. In a given case of spontaneous nystagmus, the first problem is to determine its type. Is it ocular or vestibular? If the oscillations of the eyeball consist simply of a to and fro roll, similar to the swinging of a pendulum—that is, the movement or roll in one direction is just as long and just as rapid as in the other, then it is of ocular type and suggests some local eye disturbance. It may be due to any condition that prevents the rays of light from pursuing an uninterrupted course through the various ocular media, such as corneal scars or anterior polar cataract. It is the effort to look around the obstruction. Ocular nystagmus may also result from interference with the proper action

of the rays of light at the macula, provided the lesion occurred before central fixation was established.

In sharp contrast to the foregoing, if the movement of the eyes exhibit a definite rhythm, slow in one direction followed by a quick movement or recoil in the opposite direction, the nystagmus is of the vestibular type and due to an abnormal mechanism and not a lesion in the eyes themselves. It can be produced by a lesion affecting any of the pathways between the ear and the eye muscle nuclei, either within the ear or within the cranium. Such a nystagmus is not due to an attempt at fixation but to a disturbance within the nuclei themselves or through impulses from the ear to the eye which cause the eye to deviate to one side. When the eyes are thus drawn to one side, impulses from the cerebrum to the eye muscle nuclei quickly pull the eyes back in the opposite direction. The slow component is due to irritation, impairment or destruction of the pathways from the ear to the eye muscle nuclei, whereas the quick component results from the attempt of the cerebrum to correct the altered position of the eyes.

The cause of ocular nystagmus is readily determined by an eye examination, but it is those cases in which the lesion lies not so near the surface that the ear tests are absolutely essential. No case of vestibular nystagmus can be considered to have had a proper examination without the ear tests.

The importance of the examination and study of the cochlear portion of the labyrinth is accepted by all otologists. That the kinetic static portion of the labyrinth is equally, if not more, important is not yet recognized by all otologists, or at least is not practiced by even a small percentage of them. An examination of the internal ear is certainly not complete if only a portion of it is examined. The examination and study of the kinetic static labyrinth and its pathways is just as much a part of otology as the examination of the cochlear portion. It is a misconception to regard the examination of the kinetic static labyrinth as difficult to make and hard to understand and interpret.

The entire physiology of these ear tests may be summed up as follows:

1. The eyes are always drawn in the direction of the endolymph movement.

2. The vertigo is always in a direction opposite to the endolymph movement, and past pointing and falling are always in a direction opposite to the vertigo.

By knowing these laws the otologist is master of the physiology of the subject. By making this method of examination a regular part of his practice the otologist may enlarge his field of usefulness and add great interest to his work. In the routine examination of the ear the otologist is constantly confronted with cases of deafness in which the significance of the tuning fork tests is obscure and inconclusive. By determining an involvement of the kinetic static portion of the internal ear he has at once presumptive evidence of a similar impairment of the cochlear portion. The accepted method of examination for the function of the cochlea depends on the honesty, the intelligence and cooperation of the patient, while the Barany tests of the internal ear presents objective evidence of the function of the kinetic static portion of the eighth nerve.

In the routine examination it is not necessary to make all of the tests. Merely turning the patient to the right or to the left will give sufficient information to determine the necessity for further elaboration. This can be done quickly and without any disagreeable result to the patient.

When the otologist is confronted with surgical problems of the internal ear itself, as in inflammatory conditions of the labyrinth resulting from the extension of a suppurative process in the middle ear, it is generally recognized that the turning and caloric tests, and occasionally the fistula test, are indispensable in determining the nature and degree of the involvement. The tests are the only means by which the aural surgeon can determine whether an operation on the labyrinth is necessary or whether the vestibular symptoms produced by the nonsurgical affection result from a lesion of the labyrinth itself or along the intracranial nerve pathways from the internal ear. Such a differentiation can only be made by the Barany examination and such is the otologist's problem. Vertigo is essentially an otologic study, and every otologist should be prepared to analyze such cases. In the same way he should always be prepared to give the necessary information to the internist, the surgeon and the neurologist as the possible loca-

tion and the differentiation from an internal ear and an intracranial lesion.

In the foregoing presentation of the subject of neurootology text books and the current literature have been freely drawn upon. The section on head injury cases is, as far as the writer knows, entirely new. A separate treatment of this phase of neurootology will be presented in the near future.

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