

Pain on pressure located particularly over superior part of mastoid process.

Frequency of associated facial paralysis.

Weber test localized in the diseased ear, as pointed out by Neumann.

#### NEGATIVE

Absence of the fistula symptom or attacks of vertigo such as occur in circumscribed labyrinthitis.

Infrequency of intracranial complications.

It is difficult to diagnose perilabyrinthitis really early. The importance of the diagnosis relates particularly to operative interference; that is, to the kind of operation.

Perilabyrinthitis is an extension of mastoiditis into cells which communicate directly with the mastoid cells. A typical theoretic case is, as follows:

Spontaneous nystagmus first directed to the diseased side probably in the frontal plane, accompanied or not by nausea and vomiting. Reactionary falling backward, forward or side-ward—progressive rather quick loss of hearing, but not absolute. In the meantime change of plane of the nystagmus to compound horizontal and rotary and directed to both sides but strongest to one side; finally rotary and directed to the good side only, with reactionary movements characteristic of rotary nystagmus. Absence of fistula symptom, probable absence of intracranial complications, unless it is of tuberculous origin; facial paralysis coming on late in the disease. Weber test localized in bad ear. Frequent association with systemic disease. Probable recovery of hearing after simple mastoid or radical operation. Possible recovery of facial palsy in the course of a year after radical operation if the systemic disease, when present, is cured or improved.

This is a new subject and I advance this picture for what it may be worth to those who may have opportunity to observe cases frequently from the very beginning. Acute severe cases of mastoiditis are very promising for such observations. In conclusion, for the sake of brevity I will say only that the conclusion arrived at is that the study of symptoms set forth enables us to make an earlier and more exact diagnosis.

100 State Street.

### THE PRESENT STATUS OF VERTIGO CONSIDERED FROM A DIAGNOSTIC STANDPOINT \*

GEORGE E. DAVIS, M.D.  
NEW YORK

In the discussion of suppurative labyrinthitis before this section at the Chicago meeting in June, 1908, it was my privilege and good fortune to present the first paper to appear in the medical literature of this country, reporting the recent progress made in the diagnosis and treatment of labyrinthine diseases, as taught in the Vienna schools. Since then the literature on this subject has been added to very materially, notably by MacKenzie<sup>1</sup> of Philadelphia, Fridenberg<sup>2</sup> and Richards<sup>3</sup> of New York, and Mr. Scott<sup>4</sup> of London, the last contributing some valuable new research data relating to the functions of the semicircular canals.<sup>5</sup>

\* Read in the Section on Laryngology and Otology of the American Medical Association, at the Sixty-first Annual Session, held at St. Louis, June, 1910.

1. MacKenzie: *Homeop. Eye, Ear and Throat Jour.*, 1909, xv, 17.

2. Fridenberg: *Ann. Otol., Rhinol. and Laryngol.*, St. Louis, 1908, xvii, 670.

3. Richards: *Ann. Otol., Rhinol. and Laryngol.*, St. Louis, 1907, xvi, 561.

4. Scott: *Proc. Roy. Soc. Med., Otol. Sec.*, p. 41, 1909.

5. From Scott's most excellent paper on "The Problems of Vertigo, etc." (*Proc. Roy. Soc. Med.*, April, 1909.) I have quoted freely and adopted his tables, showing the detailed results of his research illustrating the three main types of labyrinthine nystagmus and comparing the hypotheses of convection currents and momentum currents.

My first contribution was rather a foreword, very briefly outlining the methods of functional tests of the semicircular canal system developed by the researches of Barany, Alexander and Neumann of Vienna. The basis of these tests was founded on the fact that stimulation of the peripheral end-organs of the eighth nerve produces vertigo, nystagmus, and disturbances of equilibrium. It is in the interest of the solution of the mechanism of these several manifestations, when dependent on pathologic conditions, that the present discussion is undertaken.

For a correct interpretation of vertigo, a phenomenon ubiquitous in extent and protean in type, it is necessary, first of all, to have a clear conception of the term. Equilibration, or the adjustment of the individual to space, and orientation, or the recognition of that adjustment, are very complex functions.

#### ORIENTATION

The faculty of orientation is exercised through the central and peripheral sense organs and any marked or sudden disturbance of this function, induced experimentally or pathologically, creates that disagreeable sensation of confusion, known as vertigo, due to a false conception of one's relative position to space or motion, and relatively its concomitant sensation of disturbance of equilibrium. It is self-evident that vertigo and disequilibration are made manifest through the same sense organs as are equilibration and orientation. For convenience of discussion these peripheral sense organs may be classified into three subdivisions: (1) the static labyrinth, the special equilibrium sense organ; (2) the eyes, or visual sense organs; (3) the deep muscles, joints, viscera, etc., the kinesthetic sense organs.

It is a well-recognized fact that the impulses conveyed by these several peripheral sense organs and their centripetal tracts are correlated in the cerebellum, by which the function of equilibration is established and maintained. It is a further well-recognized fact that a perversion of this function, when occasioned by a permanent lesion in one of these three sets of sense organs, will eventually be corrected by the other two, perhaps not absolutely but sufficiently for all practical purposes. It follows also that with a loss of the function of two sets of these sense organs there will be a relative increase in disturbances of equilibration and orientation.

This paper will be chiefly concerned with the type of vertigo (rotatory) which results from stimulation or irritation of the static labyrinth, the special sense organ of equilibration. Since pathologic phenomena are better comprehended when we have a clear conception of the physiologic functions and anatomic structure and arrangement of the particular organs or tissues involved, perhaps it were well, from a diagnostic standpoint, to make a brief reference to this phase of the subject—particularly as to the arrangement of the semicircular canals and the origin and distribution of the nerve and blood supply to the same. As regards the blood supply to the labyrinth the works of Shambaugh, Siebenmann, Schwalbe and Eichler should be consulted, and a most excellent paper by Shambaugh<sup>6</sup> treating this subject from a clinical standpoint, is worthy of consideration in this connection.

The geometrical arrangement of the semicircular canals in three principal directions of space gave rise to the original theory as to the functions of the labyrinth. At first the assumption was that the canals were asso-

6. Shambaugh: *Arch. Otol.*, New York, 1906, xxxv, 11.

ciated with audition, and they were supposed to mediate sensations of sound coming in any of the three main directions corresponding to their axes, or to analyze atypical sound directions by a proportional representation in the three dimensions.

Later investigations, from the time of Purkinje to Barany, by careful experiments on animals and man, developed the theory of a specific equilibristic function of the labyrinth. In analyzing the mechanism of this function it is important to bear in mind the anatomic structure, arrangement and relation of the labyrinth, and to remember we have to do with a bilateral, symmetrical structure; that each canal of a side corresponds in the same plane to a canal of the opposite side; the two external canals in the horizontal plane, the anterior or superior canal of one side with the posterior canal of the other side; that the ampullary bulbs of corresponding canals are at opposite ends; the ampulla of the right horizontal canal being on the right, while that of the contralateral canal is on the left; the ampulla of the right superior canal being in front, above and beyond that of its synergist, the left inferior being behind and below.

The anatomic structure, and the relations of the membranous labyrinth to its corresponding bony container, the osseous labyrinth, may be had by reference to the texts, and I will not discuss this phase of the subject more than to call attention briefly to the origin and communications of the eighth nerve. We shall appreciate the fundamental significance of this when we undertake to differentiate the manifestations or impulses due to stimulation or irritation of the static labyrinth, or the special equilibrium sense organ, from correlated manifestations or impulses originating from stimulation of the visual sense organs, the eyes, and the kinesthetic sense organs, the deep muscles, joints, etc.

The auditory nerve really consists of two nerves, the cochlear and vestibular, and has its origin from three roots situated in the medulla. The cochlear division has its origin from the accessory nucleus and is distributed to the cochlea. The vestibular division has its origin from the chief nucleus and Deiter's nucleus and is distributed to the vestibule and semicircular canals. The peculiar arrangement of the distribution of the labyrinthine nerve end-organs, or the hair cells, in the maculae of the utricle and saccule and ampullae of the semicircular canals, bears a basic relation to the mechanism of the phenomena of vertigo, nystagmus and disturbances of equilibrium. The last two manifestations can be readily understood when we remember that the portion of the vestibular nerve which arises in Deiter's nucleus communicates fibers to the nucleus of the motor nerves of the eye muscles and to the motor nucleus of the spinal cord on either side. Nystagmus is caused by impulses conveyed to the former, and disturbances of equilibrium by impulses conveyed by the latter.

#### THE TYPE OF VERTIGO AND THE VARIETIES OF NYSTAGMUS RESULTING FROM LABYRINTHINE STIMULATION

Labyrinthine vertigo, whether produced experimentally or pathologically, is always rotatory.

Labyrinthine vertigo is always associated either with nystagmus, or disturbances of equilibrium. The reverse of these phenomena does not hold, however, as disturbances of equilibrium may occur without vertigo.

All forms of vertigo may be associated with nystagmus, but some forms of vertigo may not be associated

with nystagmus; that is, the ocular and kinesthetic forms.

Moreover, while rotatory vertigo associated with nystagmus is characteristic of labyrinthine irritation or lesion, it is not pathognomonic of it, for, as pointed out by Stewart<sup>7</sup> and Panse,<sup>8</sup> and verified by other observers, we may have rotatory vertigo and nystagmus during convalescence in cases of paresis of the eye muscles. In the latter instance the mechanism of the vertigo and nystagmus may be plausibly explained by assuming that the labyrinth is reflexly influenced by the stimulation resulting from the innervation of the eighth nerve communicated through Deiter's nucleus to the oculomotor muscles. The nystagmus occurs when the patient endeavors to turn the eyes attentively in the direction of the partly paralyzed muscle's action. I have recently observed a case with this rotatory type of nystagmus occurring during convalescence following paralysis of the left abducens muscle.

#### LABYRINTHINE NYSTAGMUS

We may conveniently divide labyrinthine nystagmus into three main types according to the direction of the excursive movements; however, there may be subdivisions as a resultant of a combination or association of the main types.

The three main types are designated, respectively, as horizontal, vertical and rotatory, according to the planes of direction of the nystagmus. A combination of the horizontal and vertical types results in a compromise between the two, or oblique nystagmus, or frequently we find the horizontal and vertical associated but not combined.

In discussing the directions of the planes of excursive ocular movements reference is had in relation not to space but to the planes of the orbit or eyeball. Thus in horizontal nystagmus the plane of oscillation is horizontal in relation to the horizontal plane of the orbit; in vertical nystagmus the plane of the oscillation is in relation to the vertical plane of the orbit; whereas in rotatory nystagmus the plane of oscillation is in the meridian of the eyeball at right angles to the antero-posterior axis.

Labyrinthine nystagmus is characterized by rhythmic movements and consists of a rapid and slow component. The direction of the nystagmus corresponds to the direction of the quick component. Thus we designate horizontal nystagmus as horizontal nystagmus to the right or left, according to the direction of the rapid movement. Similarly in regard to the vertical nystagmus according to the direction of the rapid movement up or down we designate it as vertical nystagmus upward or downward. The same rule applies to rotatory nystagmus.

The rapid movement is usually referred to as the major movement; however, the slower movement is the primary movement, or the true labyrinthine reflex, while the rapid movement is secondary or voluntary.

#### MECHANISM OF NYSTAGMUS

"Stimulation of only one ampulla produces a simple, primary reflex deviation of the eyes in one direction without nystagmus. If now the endeavor is made to turn the eyes in the opposite direction a sequence of fleeting images sweeps across the retina in the opposite direction and the eyes are jerked in rapid pursuit of the

7. Stewart: *Deutsch. med. Ztg.*, 1895, p. 511.

8. Panse: *Arch. Otol.*, 1902, xxxi, 467.

seemingly moving object which is momentarily fixed; but instantly the primary labyrinthine movement again gains control, a repetition of the fleeting images across the retina occurs, followed by the secondary rapid or major movement of pursuit, and this continues until the stimulation is annulled and the nystagmus ceases."<sup>5</sup> We have an illustration of this in the phenomenon *dévi-  
conjugée*, which represents a true reflex phase of nystagmus produced by unilateral irritation in the semicircular canals, vestibular nerve, or Deiter's nucleus. This may be demonstrated by placing the patient with a rhythmic nystagmus under general anesthesia, or may be observed sometimes in the comatose stages of cerebral abscess complicating labyrinthine suppuration. In both instances we have deviation of the eyes in the direction of the slow movement, proving that it is the primary or unconscious reflex movement, occurring during the unconscious state, while the quick movement, though the rapid and more intense, is the secondary or voluntary movement occurring during the conscious state. Therefore, the slower movement represents the vestibular reflex, while, on the other hand, the rapid movement represents the voluntary or cortical one. The maximum intensity is experienced when the eyes are turned in the direction of the quick component, and *vice versa*.

With ocular vertigo and nystagmus, closure of the eyes affords marked relief, but in the rotatory or vestibular form it has no influence; whereas in the kinesthetic form both the vertigo and the disturbance of equilibrium are exaggerated. This is a most important differential diagnostic test in the several forms of nystagmus.

Before discussing the pathologic forms of labyrinthine nystagmus let us get a physiologic view by analyzing the mechanism of some of the functional tests in developing experimental nystagmus: (1) thermal tests; (2) momentum tests.

The physical effects of the thermal stimulus depend on the variations in density of the watery solutions at different temperatures. According to well-known physical laws, when water cools the density of the cooler particles increases and they fall, and conversely, when water is heated the density of the heated particles is decreased and they rise. If we cool the surface of a bowl of water we get a descending convection current, and if we heat it we get an ascending convection current. The walls of the labyrinth and the fluid therein—the perilymph and the endolymph—are no exception to this rule. By cooling or heating the middle ear and outer wall of the labyrinth we get descending or ascending convection currents passing through the ampullæ of the superior and external semicircular canals, respectively, depending on the position of the head; through the former when the head is erect and through the latter when the face is directed upward or downward. On account of the depth of the ampullæ of the posterior semicircular canal the thermal tests are not practicable. The intensity of the convection currents, and reactions (vertigo, nystagmus and disturbances of equilibrium) resulting therefrom, depends on the suddenness, degree and duration of the alteration of the temperature of the outer wall of the labyrinth and the fluid adjacent thereto.

The momentum tests have a wider field of application than the thermal tests. The character of the physical effects of momentum depends on the direction of the motion, either in curved lines and circles or in more or less straight lines, vertically, antero-posteriorly, or laterally; also on the position of the head.

In case of the circular motion or rotation the semicircular canals, with their ampullæ, are affected, and the intensity and extent of this effect depends on the angular velocity, tangential speed and duration of rotation. As observed by Scott:<sup>6</sup>

Viewing the matter physically in order to estimate the momentum set up by rotation we must pay regard to the angular velocity, tangential speed and duration of rotation. The angular velocity is determined by computing the time of one complete revolution of 360°, and stated as so many degrees per second. If we adopt a uniform angular velocity of, say, 360° in five seconds, the approximate tangential speed varies directly with the distance between the semicircular canals and the axis of rotation; this distance can be obtained by measuring the radius of the curve of revolution described by the external auditory meatus, which will give the approximate relative position of each set of the semicircular canals. It will be obvious that when the patient reclines on the turntable with the head half a meter from the axis of rotation, and the table is turned at the uniform angular velocity of 360° per five seconds, the tangential speed of the head, and therefore the labyrinth and of the endolymph, will be greater than when the head is only a quarter of a meter from the axis of rotation. Similarly, if the patient be seated over the axis of rotation with the labyrinth equidistant from the axis of rotation, the tangential speed will be equal in the two labyrinths.

The theory most accepted now as to the effects of thermal stimulus and rotation on the semicircular canal system is that they cause currents of endolymph through the canals, either from the canal through the ampulla to the utricle or from the utricle through the ampulla to the canal. These currents, pressing or dragging on the hair cells in the ampullæ, stimulate the cells and provoke nystagmus, vertigo and disturbances of equilibrium. Owing to the observation that it requires greater effort to provoke nystagmus by rotation in one direction than by rotation in the opposite direction, particularly if the labyrinth on one side is destroyed or not functioning, the theory has been advanced that the several ampullary nerves have a principal and subordinate function. The more plausible assumption is, considering the marked inequality of the sectional areas of the semicircular canal and its ampulla, the ratio being about 1 to 25, that it requires more effort and time by rotating the head in one direction to establish a current from the ampulla to the canal—that is, from a broad to a narrow channel—than when rotating the head in an opposite direction to establish a current from the canal to the ampulla—from a narrow to a broad channel.

In case of motion in more or less straight lines the semicircular canals and ampullæ are not affected, but the utricle and saccule and their contents, i. e., the endolymph and the sensory hair cells of the maculæ of the utricle and saccule. In this instance the intensity and extent of the effect depends on the suddenness, speed and duration of the motion. We may cite as an illustration the nystagmus and vertigo of *mal de mer* or sea-sickness. Ordinarily the arc described by the rolling and pitching of a ship is too small and the angle of velocity and tangential speed too slow to occasion momentum currents in circles as in the semicircular canals, but such currents as are established are in more or less straight lines in the sacs of the utricle and saccule, corresponding to the direction of the lines of the motion of the ship but opposite in direction, and the vertigo, nystagmus, etc., is due to the irritation occasioned by the increase or diminution of weight on or dragging of the hair cells of the two maculæ.

The following tables from Scott illustrate the methods of provoking the three main types of labyrinthine nystagmus and indicate the direction of the deflection of the three sets of fibrillae which would be produced in accordance with the laws of convection and momentum considered in relation with known anatomic structure:

TABLE 1.—ROTATORY (COUNTER-CLOCKWISE) NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES TO THE RIGHT \*

BY THERMAL METHODS

Cold water irrigation.....	Left ear.....	Head erect
Hot water irrigation.....	Right ear.....	Head erect
Hot water irrigation.....	Left ear.....	Head inverted
Cold water irrigation.....	Right ear.....	Head inverted

BY ROTATION AROUND A VERTICAL AXIS

Rotation counter-clockwise .....	Face directed downward
Rotation clockwise .....	Face directed upward

\* For an account of technic adopted by Barany see Thomas Guthrie's digest (Brain, 1906, p. 383); also Mackenzie's abridged translation (Jour. Laryngol., Rhinol. and Otol., xxiv, No. 2). The present series of observations was carried out by methods evolved independently of Barany's, though essentially similar. The conclusions were also formulated before the results of Breuer's investigations were studied.

TABLE 2.—ROTATORY (CLOCKWISE) NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES TO THE LEFT \*

BY THERMAL METHODS

Cold water irrigation.....	Right ear.....	Head erect
Hot water irrigation.....	Left ear.....	Head erect
Hot water irrigation.....	Right ear.....	Head inverted
Cold water irrigation.....	Left ear.....	Head inverted

BY ROTATION AROUND A VERTICAL AXIS

Rotation clockwise .....	Face directed downward
Rotation counter-clockwise .....	Face directed upward

\*The effect of rotation on the eye-movements depends on the angular velocity and tangential speed. A certain minimum velocity is necessary, and this varies somewhat in different persons. Given the appropriate angular velocity and appropriate tangential speed, one finds that rotation will induce the same type of nystagmus with the same directional characters whether both labyrinths are functional or whether one labyrinth had become defunctive.

TABLE 3.—HORIZONTAL NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES TO THE RIGHT \*

BY THERMAL METHODS

Cold water irrigation.....	Right ear.....	Face downward
Hot water irrigation.....	Left ear.....	Face downward
Hot water irrigation.....	Right ear.....	Face upward
Cold water irrigation.....	Left ear.....	Face upward

BY ROTATION AROUND A VERTICAL AXIS

Rotation counter-clockwise .....	Head erect
Rotation clockwise .....	Head inverted

\* Inversion of the head was conveniently obtained by complete backward extension of the head over the end of a special rotating table, so that the head was about 10 in. from the axis of rotation.

TABLE 4.—HORIZONTAL NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES TO THE LEFT

BY THERMAL METHODS

Cold water irrigation.....	Left ear.....	Face downward
Hot water irrigation.....	Right ear.....	Face downward
Hot water irrigation.....	Left ear.....	Face upward
Cold water irrigation.....	Right ear.....	Face upward

BY ROTATION AROUND A VERTICAL AXIS

Rotation clockwise.....	Head erect
Rotation counter-clockwise.....	Head inverted

TABLE 5.—VERTICAL NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES UPWARDS (IN RELATION TO THE ORBIT)

BY ROTATION METHODS

Rotation counter-clockwise.....	Right side of head downward
Rotation clockwise.....	Left side of head downward

TABLE 6.—VERTICAL NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES DOWNWARD

Rotation clockwise.....	Right side of head downward
Rotation counter-clockwise.....	Left side of head downward

TABLE 7.—DEFLECTION OF THE FIBRILLÆ OF THE SUPERIOR SEMICIRCULAR CANAL ON ONE SIDE \*

BY THERMAL METHODS

Direction of Deflection of Superior Fibrillæ.	Produced By	Position of Head.	Resulting Type of Nystagmus.	Direction of Maximum Intensity.
From canal to utricle.....	Cold.....	Erect.....	Rotatory.....	Non-stimulated side
From canal to utricle.....	Hot.....	Inverted.....	Rotatory.....	Non-stimulated side
From utricle to canal.....	Hot.....	Erect.....	Rotatory.....	Stimulated side
From utricle to canal.....	Cold.....	Inverted.....	Rotatory.....	Stimulated side

\* No reactions are obtained where the method is applied to a defunct labyrinth.

TABLE 8.—DEFLECTION OF THE FIBRILLÆ OF ONLY ONE SUPERIOR SEMICIRCULAR CANAL BY ROTATION WHEN ONLY THE RIGHT LABYRINTH IS FUNCTIONAL, THE LEFT BEING COMPLETELY DEFUNCT

Direction of deflection of superior fibrillæ	Direction of rotation.	Position of head.	Type of nystagmus.	Direction of maximum intensity.
In right side.				
From canal to utricle.....	Clockwise.....	Face downward.....	Rotatory.....	Left
From canal to utricle.....	Counter-clockwise.....	Face upward.....	Rotatory.....	Left
From utricle to canal.....	Counter-clockwise.....	Face downward.....	Rotatory.....	Right
From utricle to canal.....	Clockwise.....	Face upward.....	Rotatory.....	Right

TABLE 9.—DEFLECTION OF THE FIBRILLÆ OF ONLY ONE SUPERIOR SEMICIRCULAR CANAL BY ROTATION WHEN ONLY THE LEFT LABYRINTH IS FUNCTIONAL, THE RIGHT BEING COMPLETELY DEFUNCT \*

Direction of deflection of superior fibrillæ	Direction of rotation.	Position of head.	Type of nystagmus.	Direction of maximum intensity.
In left side.				
From utricle to canal.....	Clockwise.....	Face downward.....	Rotatory.....	Left
From utricle to canal.....	Counter-clockwise.....	Face upward.....	Rotatory.....	Left
From canal to utricle.....	Clockwise.....	Face downward.....	Rotatory.....	Right
From canal to utricle.....	Counter-clockwise.....	Face upward.....	Rotatory.....	Right

\* Deflection of the fibrillæ of both superior semicircular canals simultaneously. The nystagmus evoked by rotation clockwise and counter-clockwise with the face upward and face downward is precisely the same in type and directional characters when both labyrinths are intact as when only one is functional.

TABLE 10.—DEFLECTION OF THE FIBRILLÆ OF THE EXTERNAL SEMICIRCULAR CANAL, EITHER SIDE SINGLY

BY THERMAL METHOD

Direction of Deflection of External Fibrillæ.	Produced By	Position of Head.	Resulting Type of Nystagmus.	Direction of Maximum Intensity.
From canal to utricle.....	Cold water.....	Face downward.....	Horizontal.....	Stimulated side.
From canal to utricle.....	Hot water.....	Face upward.....	Horizontal.....	Stimulated side.
From utricle to canal.....	Hot water.....	Face downward.....	Horizontal.....	Non-stimulated side.
From utricle to canal.....	Cold water.....	Face upward.....	Horizontal.....	Non-stimulated side.

TABLE 11.—DEFLECTION OF THE FIBRILLÆ OF ONLY ONE EXTERNAL SEMICIRCULAR CANAL BY ROTATION WHEN ONLY THE RIGHT LABYRINTH IS FUNCTIONAL, THE LEFT BEING COMPLETELY DEFUNCT \*

Deflection of the External Fibrillæ.	Direction of Rotation.	Position of Head.	Type of Nystagmus.	Direction of Maximum Intensity.
From canal to utricle.....	Counter-clockwise.....	Erect.....	Horizontal.....	Right
From utricle to canal.....	Clockwise.....	Erect.....	Horizontal.....	Left

\* The corresponding reactions are reversed when the rotation is made with the head inverted.

TABLE 12.—DEFLECTION OF THE FIBRILLÆ OF ONLY ONE EXTERNAL CANAL BY ROTATION WHEN ONLY THE LEFT LABYRINTH IS FUNCTIONAL, THE RIGHT BEING COMPLETELY DEFUNCT \*

Deflection of the External Fibrillæ.	Direction of Rotation.	Position of Head.	Type of Nystagmus.	Direction of Maximum Intensity.
From utricle to canal.	Counter-clockwise.	Erect.	Horizontal.	Right
From canal to utricle.	Clockwise.	Erect.	Horizontal.	Left

\* The corresponding reactions are reversed when the head is inverted. Deflection of the fibrillæ of both external semicircular canals simultaneously when both labyrinths are functional. The nystagmus produced by rotation clockwise and counter-clockwise with the head erect or with the head inverted is precisely the same in type and directional characters when both labyrinths are intact as it is when only one labyrinth is functional.

TABLE 13.—DEFLECTION OF THE FIBRILLÆ OF THE POSTERIOR SEMICIRCULAR CANAL, EITHER SIDE SINGLY, THE ONE BEING DEFUNCT, OR BOTH SIDES SIMULTANEOUSLY, BOTH LABYRINTHS BEING FUNCTIONAL

Direction of Deflection of the Inferior Fibrillæ, i. e., of Posterior Canal.	Direction of Rotation.	Position of Head.	Type of Nystagmus.	Direction of Maximum Intensity.
From utricle to canal.	Clockwise.	Right side downward.	Vertical.	Downward
From utricle to canal.	Counter-clockwise.	Left side downward.	Vertical.	Downward
From canal to utricle.	Clockwise.	Left side downward.	Vertical.	Upward
From canal to utricle.	Counter-clockwise.	Right side downward.	Vertical.	Upward

#### DEDUCTIONS FROM THE TABLES

Study of the accompanying tables reveals that stimulation of the ampullæ of the horizontal and posterior semicircular canals or deflection of the fibrillæ from utricle to canal, or *vice versa*, imparts similar impulses, provoking deviation of the eyes in attentive fixation, and, when the stimulation is sufficiently intense, nystagmus in the direction of the deflection of the fibrillæ. With stimulation of the ampullæ of the superior semicircular canals the conditions are reversed; the nystagmus provoked takes place in the direction opposite to the deflection of the fibrillæ. Thus in the former instances of the horizontal and posterior canals, deflection of the ampullary fibrillæ from the utricle to the canal produces nystagmus toward the opposite side or in the direction of the deflection of the fibrillæ; but in the latter instance of the superior canal, deflection of the fibrillæ from the utricle to the canal produces nystagmus to the same side, or in the opposite direction to the deflection of the fibrillæ.

The diagnostic importance of this observation is evident from a clinical viewpoint. Thus spontaneous horizontal nystagmus to the left would indicate some probable lesion in the right vestibule, while rotatory nystagmus to the left would indicate some lesion in the left vestibule. A combination of horizontal nystagmus to the left and rotatory nystagmus to the right would be stronger evidence still of some lesion of the right vestibule. Again, rotatory nystagmus to the right, associated with oblique nystagmus downward and to the left (the latter being a combination of horizontal and vertical nystagmus), would indicate some irritation or lesion in the right vestibule. Similarly other deductions may be drawn from a study of the directional character of nystagmus. We must be careful in our analyses, however. For instance, in a case of spontaneous nystagmus associated with labyrinthine fistula outside the vestibule (say the fistula is situated in the right osseous horizontal canal), provided the membranous canal and ampulla are intact, thermal and momentum tests will give the

normal reaction (see tables); but in this case should spontaneous nystagmus be present it would be horizontal in character and directed to the right or to the same side as the lesion, whereas usually with spontaneous horizontal nystagmus to one side we would infer vestibular lesion on the opposite side. The confusion occasioned in such cases, particularly if bilateral middle-ear suppuration or involvement exists, is strikingly illustrated in a similar case cited by Scott. In such a condition, however, if the osseous fistula happens not to be protected by cholesteatomatous masses, etc., meatal air compression will elicit horizontal vertigo to the same side and clear the diagnosis. The mechanism of spontaneous nystagmus in this case, according to the above tables, Ewald's experiments and other clinical observers, is due to the impulse proceeding from the canal to the ampulla. Scott explains the mechanism of the nystagmus in these cases by assuming that the impulse is produced by the vascular granulation tissues situated at the seat of the fistula. Should the fistula be situated in the superior canal under like conditions (vascular granulation, and ampullæ functioning), if spontaneous nystagmus occurred it would be rotatory, but to the opposite side. With the fistula situated in the vestibule, under like conditions, the presence of spontaneous nystagmus would be rotatory to the same side and horizontal to the opposite side.

Furthermore, functional tests, in cases of normal labyrinths, have shown that if the stimulation be the same on either side no nystagmus results, however intense the stimulation. This may be demonstrated by the thermal test, by using hot or cold water from the same container, to which is attached two tubes, one tube being inserted in either ear, and the injections made simultaneously. In other words, to produce vertigo, nystagmus and disturbances of equilibrium, there must be a preponderance of stimulation on one side sufficiently intense to imbalance the function of the mediating sensory organs and cause a perversion or misinterpretation of the impulses produced by said stimulation.

This brings us to the consideration of a new hypothesis promulgated by an eminent London authority in explanation of unilateral ablation nystagmus.

#### ABLATION NYSTAGMUS

Preliminary to an analysis of this hypothesis attention may be called to some of the characteristics of ablation nystagmus. It is well known that spontaneous nystagmus does not occur after removal of the labyrinth in children, rarely after ablation in adults of 35 or 40 years of age who have normal vascular systems, but that it is more or less frequent for a limited period following destruction of the labyrinth in persons over the latter age—provided the remaining labyrinth is functioning. The character of the nystagmus may be rotatory, horizontal or oblique, or a combination of these, and vary in intensity and duration. If there is but one of the types present it is usually the rotatory, and directed toward the sound side; if two types are present they are the rotatory and horizontal, the rotatory directed to the sound side and the horizontal to the side of the ablation; if all three types are present the horizontal and vertical are usually combined into the oblique.

As time lapses the sensory end organs of the sound labyrinth become injured to one-sided stimulation and gradually acquire a disregard of the disproportionate unilateral impulses, and then the nystagmus begins to

lapse also. If the three types are present the vertical element disappears first, the horizontal next and the rotatory last.

An analysis of the characteristics, directional and otherwise, of ablation nystagmus (see tables) shows that it corresponds to the type of nystagmus resulting from vestibular stimuli causing a simultaneous deflection of the ampullary fibrillæ of a normal labyrinth (from the utricle toward the superior, horizontal and posterior semicircular canals respectively); the deflection being upward in the superior ampulla, and backward and downward in the horizontal and posterior ampullæ. Hence the question arises, Whence the origin of the stimuli or impulses provoking ablation nystagmus? Scott attributes the source of these stimuli, in many instances, to the influence of carotid pulsation. On account of the relation of the internal carotid artery to the labyrinth, in its passage through the petrous portion of the temporal bone, particularly where it bends sharply forward and upward in front of the cochlea, the assumption is that the force of the heart-beat transmits impact waves to the walls of the vestibule, and that the direction of such waves is chiefly upward and backward, being reflected from the convex arch of the carotid artery. Should such impaction waves occur they would be directed upward and backward and therefore cause deflection of the fibrillæ of the superior ampulla upwards, and of the fibrillæ of the horizontal and posterior ampullæ backwards, associated with the type of nystagmus such deflection would produce (see tables) rotatory nystagmus to the stimulated side, horizontal and vertical nystagmus (or combination of the last two—oblique nystagmus) to the non-stimulated side. And we would naturally infer that the intensity of the nystagmus would be proportionately exaggerated the stronger the impulse wave thus transmitted; and *vice versa*, the weaker the impulse waves, the less pronounced the nystagmus. Clinical observations and experiments have abundantly confirmed this deduction and furnished the foundation for Scott's hypothesis of impaction waves causing spontaneous nystagmus. In corroboration of the approximate correctness of this hypothesis he cites cases in which the spontaneous nystagmus following unilateral labyrinth destruction was enhanced when the force of the circulation was increased by bodily exercise and violent muscular exertion, and a diminution or cessation of nystagmus when the body was put at rest. But especially convincing was the report of the influence of the carotid pulsation on the spontaneous ablation nystagmus in eight consecutive cases. On compression of the common carotid on the side of the destroyed labyrinth no effect was noted, whereas non-compression of the common carotid on the side of the normal labyrinth showed a complete arrest of the nystagmus in each case, continuing as long as compression was maintained and returning when the compression was stopped. The direction of the nystagmus was toward the side of the destroyed labyrinth.

Furthermore, corroborative evidence of a negative character is furnished in cases where a unilateral defunct labyrinth is of long standing. In such cases the cessation of the nystagmus results from the ampullary end-organ becoming accustomed to or acquiring a disregard of the impulses conveyed by the impaction waves, which in the beginning deflected the fibrillæ from the ampullæ toward the canal. Latterly, after the cessation of the nystagmus, if we compress the carotid on the side

of the normal labyrinth, by obviating the impaction wave, we create an impulse in the opposite direction or a relative deflection of the fibrillæ from the canals toward the vestibule. In this instance we should have rotatory nystagmus toward the negatively stimulated side or the side of the destroyed labyrinth, or in the direction opposite to the deflection of the fibrillæ. We find this so in practice.

Other evidence in favor of this theory is the occurrence of spontaneous nystagmus comparatively only after the age of beginning hardening or loss of elasticity of the arteries, whereby the force of the impaction waves is increased and correspondingly the intensity of the nystagmus. Mr. Scott's hypothesis of the impaction wave causing unilateral ablation nystagmus, considered from a physiologic, anatomic and experimental viewpoint and supported by evidence of a collateral nature, is plausible, and the greatest step in advance made in the solution of the problem of vertigo and the explanation of the mechanism of nystagmus since the contributions to the subject by Barany and Alexander.

#### CONCLUSIONS

The internal ears are the special sense organs of equilibrium. With the internal ears we recognize (orientation) and maintain our relations to space (equilibration).

The visual sense organs (the eyes), and the kinesthetic sense organs, (the muscles, etc.), are accessory sense organs of equilibrium and are coordinated with the special sense organs of equilibrium (the internal ears), through the mediation of the cerebellum.

The two special sense organs of equilibrium (the internal ear of either side), are normally symmetrical in structure and function, and any factor whatever, whether it be physiologic, experimental or pathologic, which innervates, stimulates or irritates one of these twin organs in excess of the other (or on the other hand accomplishes the same thing through enervation, depression or destruction of one in excess of the other), in that measure tends to or creates proportionately a disturbance of their joint functions—equilibration. If the disturbance of equilibration is sufficiently marked or intense we also get nystagmus and that unpleasant and complex phenomenon termed vertigo.

Finally, it must be acknowledged that not yet unravelled is the mystic arcanum of the mechanism of the complex labyrinthine function of equilibration and orientation, or the mechanism of the perversion or disturbance of that function as manifested by vertigo, nystagmus and disequilibration; but the great interest in research work now being directed along these paths augurs a satisfactory solution soon.

50 West Thirty-Seventh Street.

#### ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. HOLINGER, FLETCHER AND DAVIS

DR. A. B. DUEL, New York: I have watched with keen interest the rapid advances which have been made during the past few years in this branch of our specialty. From an unknown and unexplored field, surgery of the labyrinth is rapidly developing into a well-known and well-managed one. This is due to the fact that this small and difficult field harbors the end-organs of two most delicate special senses, viz., those of hearing and orientation. By perfection of methods of examining these functions and careful observation of the alterations in them produced by various diseases affecting the



labyrinth we have learned to fix accurately the position and extension of these lesions. We are able to say with much certainty, this patient has a perilyabyrinthitis; this, an acute circumscribed suppurative labyrinthitis; this, an acute diffuse suppurative labyrinthitis; this, a manifest lesion; this, a latent one; this patient has a fistula; this probably has not a fistula.

Operative indications have been laid down in a most dogmatic way, depending on the presence or absence of certain definite indications. It needs no wise man to predict that these indications, exact as they may seem at present, will, when matured by long experience, be somewhat modified. We are too prone to be satisfied with a few rules which may be easily observed without knowing the reasons why these rules were made. Too many cases of patients operated on after observation of certain symptoms are being reported by men who have not given sufficient attention to the more difficult question of why these symptoms appeared. The phenomena of vertigo, nystagmus, ataxia, for instance, bear the same relation to the lesion or irritation in the vestibular apparatus as the absent patellar reflex and spastic gait of locomotor ataxia do to pathologic lesions in the cord causing it. No one would contend that he understood tabes because he recognized these characteristic symptoms, and likewise he should not feel that he understands suppurative labyrinthitis because he observes certain prominent features of the disease.

The function of orientation is a nice sense presided over by the vestibular apparatus; it is intimately associated with equilibration and brings this about by impressions through Deiters' nucleus, to the oculomotor centers on one hand, and to the motor neurones of the cord on the other. To accomplish this with perfect accuracy a double system is essential, just as binocular vision is necessary for judging distance, and binocular hearing for judging direction. In other words, angulation is necessary to nicety in sight, hearing, or orientation.

If the position of the semicircular canals be carefully observed, it will be noticed that the superior verticals lie at right angles to each other, the direction of their planes being from behind, forward and outward in either direction. The posterior verticals, at a lower level, are at right angles to the superiors on the same side and to each other, the direction of their planes being from before, backward and outward. The horizontals are so arranged that movements sidewise produce a current by the same movement, in one toward the ampulla, in the other away from it. As a result the canals work in pairs, and it becomes impossible to think of experiments by movements of the head, or whirling of the individual, which do not influence more than one canal at the same time. In fact, in the normal individual all forward and backward movements of the head must produce impressions from all four vertical canals at the same time and all coordinate movements of the eyes and muscles are the resultant of all these impressions. Now all impressions of motion in space resulting normally from orientation (the vestibular sense), come from currents set up in the endolymph by these motions. This impression results reflexly in movements of the eyes and a certain tension in the muscles to maintain equilibrium. Any upset in this delicate sense will result in subjective sensation of motion and reflex efforts to compensate for them, thus bringing about the phenomena of acute vestibular irritation—vertigo, nausea, vomiting, nystagmus—the direction and character depending on the nature and extent of the irritation.

Equilibration can soon be accomplished by practice, without orientation, by compensation, by means of vision and muscle sense. Hence the phenomena of acute vestibular irritation soon disappear and altered reflex phenomena have to be sought after and brought about by the special tests, as you have heard.

The man with only one labyrinth learns to do without the missing labyrinth just as he would without one eye or one ear, and he does without both just as he would without both eyes or ears. He learns to equilibrate without orientation. Is he just as well off? Certainly not; he feels the need of the faculty in any new environment or situation. Try a man

who has just lost one labyrinth and acute symptoms have disappeared—vertigo, nystagmus, ataxia, in ordinary situations—ask him to walk up or down an incline. He will be unable to do it at first, but by repeated trials he will soon learn. He has, in other words, learned to equilibrate with faulty orientation. After he has learned to do these things in the light, try him in the dark. He will fail again, but will learn with practice. His education in compensation for lack of sense of orientation will be analogous to that of a blind man learning to walk with confidence and just as difficult. We have only touched the outside edge of the subject of labyrinthine phenomena, although we have already learned to interpret them sufficiently to recognize danger signals and have grown so bold as to invade the domain of the labyrinth with impunity.

PROF. HOLGER MYGIND, Copenhagen, Denmark: So far as etiology is concerned, there is one point which I do not think has been brought out, and that is the traumatic cause—suppuration produced through operation, and especially due to radical operation. I have seen a considerable number of these cases, and I think that the labyrinthine suppuration caused by radical operation is much more frequent than most of us think. These cases that occur after radical operations have a very different course. I may divide them into three groups:

1. The very light cases in which no symptoms at all are observed by the patient or by the medical man. Even if such patients are asked if they feel at all ill after operation, they often reply, "No, I did not notice anything at all." As Dr. Fletcher pointed out, there is no proof that labyrinthine symptoms have not been present, only they have been overlooked, and the physician observes that there has been labyrinthine involvement; perhaps a week or more after operation he discovers that the patient is totally deaf in that ear and if the function of the labyrinth be examined it is found that it is lost. These cases, I think, teach a great deal; especially that we should be careful with our indications for radical operation. My experience is that our indications for simple mastoid operation ought to be very wide, for I believe the risk we run in acute cases is very small considering the risk the patient runs when he is not operated on. On the other hand, my indications for radical operation are very limited, so limited that in a discussion with Prof. Körner of Rostock I had even a more conservative view than he has, and he is very conservative on the whole. I think that when a patient with chronic suppuration of the middle ear has fairly good hearing we should be careful about performing the radical operation. If both ears are affected, we should be exceedingly careful and only do the operation when occasion demands it very strongly.

2. In postoperative labyrinthine suppurations in which there are distinct labyrinthine symptoms, the patient, shortly after operation, or sometime after, gets distinct labyrinthine symptoms—nausea, vomiting, noises in the ear, etc.—and in these cases one not infrequently sees paralysis of the facial nerve. What I say now it is not necessary to say to surgeons of great experience, but to the younger members here I say: After an operation, either radical or simple, if in the course of a few days a facial paralysis develops, look well to the labyrinth. It is often said that paralysis is due to blood-clot, pressure of the dressing, or something like that, but in my experience it is frequently caused by labyrinthine suppuration; and, of course, there is a close anatomic connection between the facial nerve and the labyrinth.

3. There is a group of cases of postoperative labyrinthitis, which is not so large, but which is very important; this is a class in which the labyrinthine suppuration develops explosively and leads to diffuse meningitis. The prognosis in these cases is very grave indeed; I have seen only one such patient recover. That was a boy of sixteen who had labyrinthine suppuration and diffuse leptomenigitis and recovered after craniotomy, excision of the dura, etc.

I consider an operation on the labyrinth a very difficult operation. One of the greatest difficulties is to avoid breaking down the bony wall of the Fallopian canal.

DR. GEORGE F. COTT, Buffalo: An interesting point raised by Professor Mygind is the question of traumatic cases, following the radical operation. Some operators have been luckier than others. In 10 years I have done 102 radical operations and have had only one patient die. How often do these cases occur?

There are not many reported in this country. In the last 3 or 4 years there have been some isolated cases, but I suspect that there are many more than have been reported. I have seen 8 or 9; 3 of these patients were operated on, and the labyrinth drained; and in 3 the radical operation was done; the others are still under observation with no operation. If that number occurs in a city of 450,000 why have not large numbers occurred in cities like Chicago and St. Louis? They no doubt do occur, but are overlooked. I do not know whether the otologists will be more conservative or not; I am afraid not. I think these patients will have to go to operation, although it is extremely difficult, or they will die. To wait until the patient is in *extremis* is bad policy. The physician must take the chance and so must the patient. Of course, everything must be thoroughly considered.

In this country we have not yet decided the question of time to operate. Jansen in Berlin two years ago read a paper in which he told of over 100 operations on the labyrinth in which he had done with a death-rate of 29 per cent. In the next 100 cases he will have no such death-rate, because now we have more definite symptoms and the patient has a much better show.

As to the question of diagnosis, no man would think of operating on the labyrinth unless deafness were present. When deafness is complete and the other definite symptoms are present it is well to operate. The operation necessarily belongs to the otologist and not to the general surgeon, and I believe the general surgeon will never perform it as he does other operations about the ear, nose and throat.

Suppose a patient has caries of the middle ear, with a very long history; suppose he has had perilyabyrinthitis, and is in the latent condition in which the least irritation will set up further trouble—what are you going to do? If you operate radically the chances are ten to one that he will die unless the labyrinth is drained. Such cases have occurred and I do not doubt that they are the cases to which Professor Mygind refers in which the patient died after radical operation from involvement of the labyrinth.

Another point, as to the hearing after exfoliation of the cochlea in suppuration of the labyrinth: I find in Bezold's writings that he cites a number of cases in which a certain amount of hearing existed after the cochlea had been cast off. Politzer mentions that it may be possible for some hearing to exist without the labyrinth. Before Bezold's death I think he made the claim that it is absolutely impossible to hear anything after the labyrinth has been destroyed. In this country, Dench and some others have operated in a certain number of cases, drained the labyrinth, and after the patient has recovered found that there was a certain amount of hearing left. I take the stand that it is impossible to hear after destruction of the labyrinth. Bezold says that in the cases reported, bone conduction on the other side has been neglected.

DR. CULLEN F. WELTY, San Francisco: I wish to show a specimen of the complete labyrinth operation, and to call attention to the large amount of dura that is uncovered, giving free access to any operation of the cerebellum, also to call to attention the facial nerve in the canal; the cochlea and canals have been entirely destroyed. This operation is not so difficult as Prof. Mygind would have us believe. I have seen ten or twelve similar operations and have done three myself, and have not seen a case of facial paralysis. My patients are all well. Dr. Duell hit the nail on the head when he said: "On the careful examination depends the whole condition." In the various ear hospitals it has been repeatedly demonstrated that there is a mortality of from 10 to 12 per cent. Prof. Mygind says that 65 per cent. of the cases of meningitis are infected by way of the labyrinth. When to operate and when not to operate is the all-absorbing question.

OPERATIVE CHART \*

	Cochlea.	Excitability of Vestibular Apparatus	Fistulae.	Spontaneous Nystagmus.	Operation.
I	+	+	+	+	No No
II	—	+	+	+	Eventual No
III	+	—	+	+	Operation Eventual
IV	+	—	—	+	Eventual Eventual
V	—	—	+	+	Operation Operation
VI	—	—	—	+	Operation Operation
VII	—	+	—	+	No No

\* Neumann, Heinrich: Indikationen für die operative Eröffnung des Labyrinths.

I here submit an operative chart. Grouped with the other a mistaken diagnosis is almost impossible. Use this last chart until you thoroughly master the situation from a pathologic standpoint and by that time it will be very clear indeed. There is a test that has not been spoken of; indeed, it is very important for those of us who will dare to operate without a proper examination, as I did in one case only recently. However, I operated under stress of circumstances that does not often happen. As I was completing my operation I found a good-sized fistula of the horizontal canal; besides, the facial nerve was uncovered, posteriorly. I had never seen the patient prior to operation and was leaving the following morning to attend the American Medical Association Session. By removing the anesthetic for a short time so that the patient might sufficiently recover for the reaction, a stream of cold water was directed into the ear. In a short time there was a steady pull of the whole eye to the opposite side; this is the reaction of functioning canals. This cleared the diagnosis wonderfully. Dr. Shambaugh says that a patient should not be operated on so long as there is hearing left. I am sure he has not thought of this in a serious way.

At the Otologic Congress at Budapest, Dr. Neumann reported a large number of cerebral complications showing conclusively that 75 per cent. of such cases were the result of acute exacerbation of chronic ear suppuration. Now should we follow Dr. Shambaugh's suggestion we should do the radical operation on patients with a latent labyrinth suppuration (patients who hear and do not react to the caloric test). As I have shown by Neumann's publication that the acute exacerbations are particularly dangerous, so it will be seen by this radical ear operation an acute exacerbation of the latent labyrinth suppuration is produced with possible fatal termination. According to the Vienna school of otology the labyrinth should be removed at the time the radical operation is done or left alone entirely. The disturbances of equilibrium and the tinnitus that one of the speakers refers to following the labyrinth operation are due to incomplete operation; in other words all other operations except Neumann's will be followed by more or less disturbance of equilibrium and by tinnitus—such as happened to me years ago! With more definite pathologic findings we are accorded the privilege of changing our conclusions in regard to various operations.

DR. G. E. SHAMBAUGH, Chicago: Several points have been brought up in this symposium which I should like to emphasize. In the first place, we must keep in mind that in suppurative otitis media a number of different types of internal ear complications may occur, all of which give rise to distinct labyrinth symptoms such as vertigo, nystagmus, tinnitus and deafness. It is important that we make a correct diagnosis of the type of complication in a particular case because the treatment is different for the several complications. In some of these complications giving rise to labyrinth symptoms, a simple mastoid operation is indicated; in others, the labyrinth itself should be opened up, and in still others nothing beyond the treatment of the otitis media by the ordinary measures is called for. Most important is it that we differentiate between cases of diffuse suppuration of the labyrinth and cases of labyrinth fistula, or the so-called circumscribed labyrinthitis, be-



cause in the latter we do not open the labyrinth, whereas the opening of the labyrinth may be called for in the diffuse suppuration. The diagnosis between diffuse suppuration of the labyrinth and diffuse serous labyrinthitis is also important because it is not advisable to open the labyrinth in cases of serous involvement. We should never open the labyrinth when there is any function retained in the labyrinth such as a remnant of hearing or a vestibular apparatus that can be stimulated. It is a much graver mistake to open a labyrinth when no adequate indication exists than to neglect to open it when the diagnosis would seem to indicate an operation. Many of the patients with diffuse suppuration of the labyrinth will recover spontaneously without operation, whereas to open into a labyrinth not the seat of diffuse suppuration experience has shown to be of danger to the patient. It is not possible to interpret intelligently the various symptoms arising from the several complications of the labyrinth without a clear understanding of the physiology of the inner ear and without a clear idea of the pathology of these complications.

I cannot agree with Dr. Welty that the disturbances that occur after operation on the labyrinth occur because it has not been entirely destroyed. The function should be destroyed before we operate. If symptoms of labyrinth irritation occur after operation it means that we have operated when we should not have done so, because there was still function there.

In my experience I have not yet observed the condition which Professor Mygind points out, in which there is apparently after a radical operation a destruction of the labyrinthine function, without our being able to recognize it from the symptoms. He said we might recognize it perhaps weeks or months afterward. That it is possible for a patient to have a rapid destruction of the labyrinth without symptoms I have not observed. It would seem to me that such a case might be explained by the fact that before operation we did not go through the complete tests. There might have been destruction of the labyrinth without the patient being aware of it dating back perhaps many years.

DR. ROBERT BARCLAY, St. Louis: Whatever the condition of the ear—whether or not there be suppuration of the middle ear, with or without extension to the labyrinth—vertigo may arise from, and should be attributed to, another source. My attention was directed to this when the cause of vertigo was being discussed; and that it might be borne in mind in every case I thought it well to speak of the influence of the inferior cervical ganglion. From that ganglion arise not only the inferior cardiac nerve but also vasomotor fibers that control the contraction and dilatation of the vertebral artery, of which the labyrinthine artery is a branch, and from which alone the arterial supply of the labyrinth is drawn. The effect of tobacco, acute tobacco intoxication—especially in enfeebled persons or in young persons beginning to smoke—largely affects this very nerve center, the inferior cervical ganglion and its arterial and nervous distribution. Such an attack resembles the pure labyrinthine inflammatory invasion very closely; and I wish to call attention to it and emphasize the necessity for considering this possibility in all cases of vertigo.

DR. C. M. BROWN, Buffalo: I agree with Dr. Shambaugh that it is not wise to operate on the labyrinth so long as we have any function left there. The greatest problem is when to operate and when not. We are all doing mastoid work and I believe that it is not right to do it without first making an examination to find out if the semicircular canals and cochlea are functioning. We can all examine the internal ear. The caloric test is always at hand and should be used in every acute as well as in every chronic case of mastoiditis in which we are going to operate. It is a well-established fact that in syringing an ear with cold water one gets nystagmus to the opposite side if the labyrinth is functioning. Every patient can be so examined in order to ascertain if the labyrinth is functioning or not.

Recently I had a case of double otitis media in which there was a question concerning which side to operate on. Both sides were examined thoroughly; on one side the vestibular apparatus was not functioning at all—

no result on turning and no result with cold water. There was chronic suppuration on both sides following scarlet fever at the age of 2. I did not operate on the side that was not functioning. If I had I should have expected meningitis, if I had not at the same time removed the internal ear.

Do not operate in a mastoid case without testing the condition of the vestibule, and if you cannot do an operation on the internal ear leave that mastoid alone which has a non-functionating labyrinth. If the cochlea and vestibule are destroyed it may be well to remove both. We should not quibble too much on when to operate on the internal ear, but on when to leave it alone.

DR. LOUIS K. GUGGENHEIM, St. Louis: The statement was made by Dr. Fletcher that with a diffuse suppurative labyrinthitis, say of the right side, he got a spontaneous rotatory nystagmus to the left. On syringing the left ear with cold water this nystagmus is decreased, and if syringing is continued long enough we get a rotatory nystagmus to the right (diseased) side. A vestibular nystagmus to the diseased side in a case of diffuse suppurative labyrinthitis without an intracranial complication I believe to be impossible, for the following reason: Normally the two vestibular apparatuses exert a certain equal influence over the eye-muscle nerves through the vestibular nerves, Deiter's nucleus, the fasciculus longitudinalis posterior, and the nuclei of the abducens and oculomotor, and any disturbance in the equilibrium of this influence results in nystagmus. Now in a diffuse suppurative labyrinthitis of the right side the influence normally exerted by that side is lost, the left vestibular apparatus acts alone, the result being a vestibular nystagmus to the left side. If we now syringe the left side with cold water the influence exerted by the left vestibular apparatus is temporarily lessened by the paralyzing effect of the cold water. The result is a decreased nystagmus to this side. If the syringing is continued long enough, the stimuli which normally pass from this side to the eye muscles may be entirely lost. The result will be complete cessation of nystagmus. Now, no matter how long the syringing with cold water is continued, a vestibular nystagmus to the right (diseased) side is, I believe, impossible, for the simple reason that the right vestibular apparatus is no longer functioning. In other words, with the right vestibular apparatus destroyed by the diffuse suppuration and the left vestibular apparatus temporarily paralyzed by cold water, we again have vestibular equilibrium restored. Therefore, there can be no nystagmus of the vestibular variety, as this depends on a disturbance of vestibular equilibrium for its existence.

PROF. HOLGER MYGIND, Copenhagen, Denmark: I think the difference of opinion between Dr. Welty and myself may be explained by the difference in our operations; I do the operation more radically. Dr. Welty has passed round a temporal bone on which he has made resection of the labyrinth. Now, I want to state that Dr. Welty has not opened the whole of the superior semicircular canal. That is just where the difficulty comes in. It is important to have a very small chisel and a very sharp one. To Dr. Shambaugh I would say, the reason the labyrinthine symptoms are overlooked is likely because these postoperative suppurations often come in the course of the first twenty-four hours after operation when the patient is still suffering from the effects of the anesthetic.

DR. OTTO GLOGAU, New York: A few years ago, when I was assistant to Professor Politzer in Vienna, Dr. Neumann said: "When you go to America tell them about this labyrinthine work; you will find that they do not know very much about it." A few months ago when Dr. Neumann was here he was astonished to find how much was known about it in this country. Three years ago I suggested that all cases of chronic suppuration of the middle ear be examined before radical mastoid or labyrinthine operation, as is done in Politzer's clinic, that is, by the so-called labyrinthine schema. I am in accord with Professor Mygind that we must be conservative in the radical operation, but we can only do this by following up the cases, every day making a new test and thus detecting labyrinthine symptoms at the very onset of the inner ear involvement. There is another disease which simulates lab-

labyrinthitis and is caused by pressure symptoms only. A retention of pus or cholesteatoma behind a fragment, adherent to the promontory, or behind a new membrane formed in place of the destroyed tympanic membrane may cause all the symptoms of labyrinthine suppuration; these will readily disappear, however, when the primary cause is treated properly. I consider Dr. Neumann's method of operating on the labyrinth the best.

Dr. J. R. FLETCHER, Chicago: The fibrous connective tissue formations in the area of the oval window aid in the production of fistulas. In relation to the matter of operation on the labyrinth, the statement has been made that it is not difficult; my experience has been mainly on cadavers, and I opened the facial canal in many of them. I have made no report of operations on the living. Every test that is mentioned in this paper I have repeatedly confirmed, and I believe that I have not deceived myself. I think they are correct.

Dr. G. E. DAVIS, New York: I do not know whether I understood Dr. Ducloux or not in regard to the loss of orientation with the destruction of one ear. I tried to make it clear in my paper that the ear serves the purpose of audition, orientation and equilibration. I also mentioned two other sets of accessory sense organs, the eyes and the muscles. It goes without saying that with the loss of one ear a man may have orientation and equilibration just as when one eye is lost he may have vision. With the loss of one set of these sense organs the other two sets in a short period will enable the individual to re-establish these functions. Orientation is the recognition of our position in space and equilibration is simply the faculty of assuming and maintaining position in space. With the loss of one ear, if a man can stand, he appreciates that he is standing, and if he reclines he appreciates that he is reclining, and that is the function of orientation. Maintaining that is equilibration. Now with the loss not only of one ear, but the visual organs also, a man soon learns to appreciate his position in space and to maintain it.

Dr. Shambaugh has, I think, fully answered Dr. Welty as to the destruction of the labyrinth. I wish to answer another point of Dr. Welty's in regard to his statement that the caloric test was negative after either the cochlea or semicircular canal system is out of commission. Now, if the cochlea is out of commission and the semicircular canals are in commission and the caloric test be made, a reaction is manifested by nystagmus, with nausea and vomiting, showing that the semicircular canals are still functioning.

## FOIBLES IN SPECIALISM\*

C. F. WAHRER, M.D.  
FORT MADISON, IA.

One of the easiest tasks is to find fault with existing things, whether they be right or wrong. There is but one other thing as easy, and that is to go wrong, as nearly all the nations of the earth were tempted to depart from the paths of right, and thus prepare the way for their enemies to encompass their destruction. Even after a short season of right-doing the Israelites, as soon as Moses was out of their sight up the mountain, made them a golden calf and worshiped it. They were not the only ones who did this. Some of the most powerful nations of the earth, drunken with the wine of material success, became unmindful of their morals, and departing from the virtues of the fathers, indulged in riotous living, became vain and arrogant, and thus soon went to their destruction. As with nations, so with individuals. Their foibles are their ruin.

Thus it might fare with the foibles of specialism. Not that specialism is not a good thing; on the contrary, it is indispensable. The realms of knowledge are so large that it is no longer possible to follow the teachings of a Chesterfield: "to be a gentleman it is necessary to know a little of everything, and know all about some things." Science asks too much of us now to follow the above teaching. This is the era of specialism, and we must be content with a choice of careers. Medicine is but a branch of the natural sciences, namely, biology, and we are not able to learn all even of this one branch. We have found it needful to study more particularly some of the parts of medicine in order to do justice to our patients' needs. This leads us to medical specialism.

May I suggest that, if we could eliminate all pseudo-science, or else if we could establish a censorship of that part of our literature which masquerades as scientific, many of our burdens would be lightened and much of our precious time would be saved. Our library shelves groan under the weight of verbosity, prolixity, tautology, and literary dust, straw and chaff. This pseudo-science has begotten pseudo-specialism and specialism gone mad.

This paper is not to be construed as the usual diatribe of a generalist against specialism, as I am an admirer of true specialism, for I recognize it as an absolute necessity; but it is a warning against the foibles of the pretender and some of the better ones even, whose zeal has exceeded their discretion. The earnest and true scientific specialist also, at times, succumbs to the foibles of his class, which is an easy thing to do when off guard. For instance, it is very easy to fancy that all affections of man must fall under this or that particular branch of practice, so that, one might think, the other specialities do not amount to much. For example, I recently heard a man, who fancies he is a major surgeon and who does some creditable surgery, stigmatize some other men as finger-and-toe surgeons. You know, of course, what he meant to express, though there is some surgery about the fingers and toes that may tax the skill of the best of surgeons.

There are those in our own Section on Diseases of Children who act as if all there was to pediatrics was the artificial feeding of infants—as if all babies were fed out of bottles! Those inclined to mathematics want to reduce infant-feeding to algebraic or logarithmic formulas, the results of which, of course, agree with some children, the same as almost anything else would agree with them. Others, in writing of the management of the infant, would make us infer that all the babies they treat are the children of millionaires. They describe the duties of the first nurse, those of the second nurse, then that of the nursery maid. They then tell us when the child should spend its time in the nursery, when in the solarium, and when in the tent on the housetop, when it should have a ride in the grounds, when in Central Park, and when it should take a sea voyage. This makes us heave a sigh and exclaim "lucky dog."

I really think that the specialist spoke from his usual habit and daily practice; only he forgot that there were some mothers who do not have two nurses, no solarium except the back porch of the flat, no sea, except the mud puddle in the alley, no housetop where the little sufferer with pneumonia may be put in a tent, and no tent. The moral is obvious. It may not be amiss to say that in the rural districts most children are fed on the moth-

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