1912

Labyrinthitis

Edwin Cobb
State University of Iowa

This work has been identified with a Creative Commons Public Domain Mark 1.0. Material in the public domain. No restrictions on use.

This thesis is available at Iowa Research Online: https://ir.uiowa.edu/etd/3571

Recommended Citation
Cobb, Edwin. "Labyrinthitis." MS (Master of Science) thesis, State University of Iowa, 1912.
https://doi.org/10.17077/etd.uwm0htwg.

Follow this and additional works at: https://ir.uiowa.edu/etd
"LABYRINTHITIS"

A THESIS

PRESENTED TO THE

GRADUATE FACULTY OF THE STATE UNIVERSITY OF IOWA

In partial fulfillment of the requirements

for the

Degree of Master of Science.

by

Edwin Cobb, B.S., M.D.,

Department of Otology, Ophthalmology and Rhino-Laryngology.

Iowa City, Iowa.

June 1912.
OUTLINE.

---

History.
Anatomy.
Experiments of Ewald.
Caloric reactions.
Etiology.
Channels of infection from middle ear to labyrinth.
Pathology.
Channels of infection from labyrinth to brain.
Division of labyrinthitis.
Symptoms.
Diagnosis.
Complications.
Prognosis.
Terminations.
Treatment.
Report of Cases.
Conclusions.
Drawings.
HISTORY.

Suppurative Labyrinthitis following otitis media suppurativa is the form to which reference is made in this paper.

The development and study of labyrinthine suppuration from the earliest experiments to the scientific studies of the present day, is a most interesting work. The studies of Floueus in 1824 to 1828 and followed later by the experiments of Ewald were the foundations upon which our present day literature is founded. Later several noted scientists, among whom are the names of Jansen, Hinsberg, and Richards added greatly to the subject. Among the more recent writers, the name of Robert Barany of Vienna perhaps stands foremost among the investigators of Labyrinthine suppurations. He has conducted many experiments and has given much valuable time to the study of the labyrinth. Much credit is due Phillip D. Kerrison of New York for his many valuable contributions to the literature on this subject.
ANATOMY.

That we may understand this subject better, perhaps a brief outline of the anatomy of the labyrinth would not be out of place.

The inner ear is composed essentially of two structures, the (1) Osseous Labyrinth and (2) the Membranous Labyrinth. (1)-The Osseous Labyrinth being made up of a series of cavities continuous with each other, namely the cochlea, vestibule, and semicircular canals. The vestibule makes up the central portion, communicating posteriorly with the semicircular canals and anteriorly with the cochlea. In the antero-inferior portion is the recessus sphaericus which lodges the saccule. The recess has numerous perforations which transmit filaments of the auditory nerve to supply the saccule. Posterior and above is the recessus ellipticus, lodging the utricle. Below this recess is a canal, aquaductus vestibuli, which passes posterior through the petrous bone to open at a point mid-way between the internal auditory meatus and the groove for the lateral sinus. This canal carries the ductus endolymphaticus and a small vein. Posterily are the five apertures of the semicircular canals. Anteriorly the vestibule opens into the scala vestibuli of the cochlea.

The three semicircular canals lie posteriorly and above the vestibule, being differentiated by position, namely, superior or anterior vertical, posterior and external or horizontal. They open by five apertures into the vestibule. The inner extremity of the anterior vertical and the upper
extremity of the posterior unite and form one canal opening into the vestibule.

The cochlea is a cone-shaped structure, having the base corresponding to the internal auditory meatus and apex pointing outwards and forwards. It is sort of a spirally arranged tube surrounding a central portion, the modiolus. The lamina spiralis ossea is a thin bony process which divides this spiral tube structure into two parts, the one above called the scala vestibuli and the one below the scala tympani. Below the crista semilunaris, near the round window, is found the aqueductus cochlea, opening internally on the posterior surface of the petrous bone, through which communication is established between the scali tympani and the subarachnoid space.

(2)- The Membranous Labyrinth, lying within the bony labyrinth, contains a fluid called endolymph and the space between it and the real bony labyrinth is called the perilymphatic space which contains perilymph. The utricle and saccule are the two membranous sacs in the vestibule, the former receives the endolymph from the membranous canals and the latter communicates with membranous cochlea. All portions of the membranous labyrinth contain endolymph and communicate with each other. The ductus endolymphaticus from the saccule uniting with the ductus utriculosaccularis from the utricle are transmitted by the aqueductus vestibuli to communicate with the meningeal lymphatics. The inner layer of this membranous labyrinth throughout is composed of a supporting layer of cells, upon which rest the hair cells proper.
Experiments of Ewald.

To make clear the action of the vestibular apparatus it is essential that one understands the experimental work conducted by Ewald. They are given below along with Barany and Kerrison's revision.

The experiments of Ewald on each semicircular canal alone were conducted as follows:— the selected canal being exposed, a very small opening is drilled into the most prominent part. Then a second opening is made similarly between the first and small end and into this is placed a lead mass. A small cylinder apex at both ends is introduced into this first opening. A small piston works inside the cylinder. A rubber tube with a bulb on the end is attached to the cylinder, so that compression of this bulb drives the piston inwardly thereby compressing the endolymph in that particular canal experimented upon. Since this canal is closed in the direction of its small end, it is very evident that the movement of the endolymph must be directed towards its ampulla. With a release of pressure on the bulb the endolymph movement is induced in the opposite direction, away from the ampulla and toward the small end of the canal.

These experiments in the different canals gave the following reactions:—

Right External, or Horizontal Canal.—Compression of bulb (causing endolymph displacement toward ampulla) was followed by strong, gradual movement of the head, exactly in the plane of the canal, to the left. Coincidently with
this head movement, the eyes were moved, also in the plane of the canal, to the left. On release of pressure the head and eyes quickly regained their normal position.

Suction (i.e., endolymph movement in right horizontal canal toward small end of canal) gave rise to gradual movement of head and eyes, always in the plane of the canal, to the right. But this latter movement to the right caused by suction was much less forcible than the opposite movement caused by compression.

Right Posterior Vertical Canal. Compression (i.e., endolymph movement towards ampulla) was followed by gradual movement of the head, exactly in the plane of this canal and toward its ampulla, i.e., to the right. Suction gave rise to movements of the head and eyes in the same plane but in the opposite direction, i.e., to the left. But in this canal, suction was followed by much stronger movements than those produced by compression.

From Ewald's experiments the following deductions are made:—

(a) Excitation of a single canal produces a nystagmus only in a plane that corresponds to the plane of the canal.

(b) By reverse movements of the endolymph in any canal, the induced nystagmus is reversed.

(c) The strongest nystagmus which can be induced by irritation of a single canal is always in the direction of the ear experimented upon.

Kerrison has formulated the following rule which can be readily applied providing the relative positions of the ampullar
and small ends of each canal are fixed in mind. - "Displacement of endolymph in any canal gives rise to nystagmus in which the eyes move in a plane parallel with the plane of the canal, and of which the slow movement is always in the direction in which the endolymph moves."

A nystagmus of the vestibular origin has the following characteristics -

1- Is composed of a quick movement (quick component) in one direction and a slow movement (slow component) in the opposite direction.

2- When the eyes are turned voluntarily in the direction of the quick component, the nystagmus is increased in rapidity and in length of excursion.

3- On turning the eyes in the direction of the slow component the nystagmus becomes very weak and at times disappears. These characteristics are usually to be elicited during the course of any acute suppurative labyrinthitis.

In acute labyrinthine disease we obtain a characteristic rotary nystagmus which is very easy to recognize having seen it once. On the other hand vestibular nystagmus may be vertical, horizontal or oblique. On eliciting the nystagmus the patient is directed to follow your finger which is moved to the right, straight ahead and to the left and the effects noted. The patient is then asked to voluntarily turn his eyes in the same positions and the character of the nystagmus noted.
THE CALORIC REACTIONS.

To Dr. Barony of Vienna belongs the honor of establishing the significance and the technique of the caloric reactions and of the conclusions drawn. They are dependent on the following conditions.

1- No objective or subjective phenomena result when a normal ear is irrigated with water at ordinary body temperature.

2- Irrigating an ear with water at 70 - 78°F the following phenomena occur -
   a- A rotary nystagmus to the opposite side (towards the ear not irrigated).
   b- Patient becomes dizzy and at times nausea and vomiting may set in.

3- Irrigating an ear with water whose temperature is 110° - we note the following phenomena-
   (a)- a rotary nystagmus to the same side (toward ear irrigated).
   (b)- ataxia and vertigo and if severe enough and nausea and vomiting. I have seen patients become very nauseated and yet never reaching the vomiting stage.

Barony explains the cause of the above caloric reactions on this theory - he regards the labyrinthine cavity as a vessel filled with fluid whose temperature is the same as that of the blood. Now the specific gravity of this contained fluid will be altered, that is, increased or decreased as the hot or cold water is brought in contact with any wall of this vessel. The
portions of the semicircular canal most exposed to such temperatures are -

(a)- Anterior portion of external or horizontal canal.
(b)- An external small portion of anterior vertical canal.

The caloric reactions are direct in accord with Ewald's experiments. Kerrison states "that the causation of caloric nystagmus is further supported by the fact that if the head is quickly inverted so that the top of the head is directly downward toward the floor, the direction of the nystagmus is reversed, i.e., is now toward the ear irrigated. That the use of hot water, which by reducing specific gravity would cause an endolymph movement in the opposite direction, invariably gives rise to rotary nystagmus toward the ear irrigated, leads further support to this view as to the causation of these phenomena".

The real value of all these caloric experiments is that we can determine to what extent if any, the vestibular loss has been. This of course, depends on the fact that if the vestibular apparatus has been impaired or destroyed, we find no caloric reactions on applying water whose temperature is 78° or 110° (cold or hot).

The rotation experiment depends on the fact that when a normal person is placed in an easy revolving chair and turned either to the right or to the left a nystagmus, horizontal in nature, is set up. If turned to the right, usually about twenty turns, a horizontal nystagmus results with the quick movement in the direction to which the patient is turned (here to the
right). Now when the patient is suddenly stopped the nystagmus is reversed, the quick movement being directed to the left. The nystagmus usually appears in 20 - 45 seconds in the experiments conducted here, while Dr. Barony reports that the average duration is longer, being about 40-45 seconds. Kerri-son explains this rotational nystagmus in accordance with Ewald's experiments - "The mechanical influence of sudden turning of the head in the horizontal plane upon the fluid in any particular canal depends upon the rotation which the plane of this canal bears to the horizontal plane. Thus, with the head erect the two horizontal canals are approximately in the horizontal plane. When the head is suddenly turned in either direction - let us say to the right - the fluids in these canals by reason of its inertia at first lays behind, i.e., suffers an initial displacement in the opposite direction. This initial endolymph movement in the right horizontal canal is toward its ampulla, while in the left horizontal canal it is toward the small end of the canal. Now according to Ewald's experiments, these are precisely the endolymph movements which in these canals, give rise to nystagmus to the right, and this phenomenon is always present during ten rotations to the right. When however, the rotations are suddenly stopped there occurs, again by reason of its inertia, an endolymph displacement in the opposite direction with the result that the direction of the nystagmus is reversed, i.e., rotation to right is followed by nystagmus to left".

By placing the head in the different positions one is able to thus test each semicircular canal and note its normal
Dan McKenzie in studying some 42 cases examined according to Dr. Barony's caloric test as to the time of the appearance of the nystagmus, states that in health nystagmus appeared in from 20 to 40 seconds and in any condition of hypersensitivity of the vestibular organs in from 5 to 15 seconds. In cases of depressed excitability the induction period was from 50 to 120 seconds. He states that in chronic uncomplicated suppuration of the middle ear, the vestibular reactions are normal.

The normal physiological function of the vestibular apparatus is one that informs us of the relative position of the head to a line of gravity. With every forward movement and with each inclination of the head the relatively heavy otoliths remain in the medium in which they are suspended, thereby causing a bending of the delicate hairs which support them; this bending produces specific sensations, which become apparent by means of numerous nerves. (Breuer)
ETIOLOGY.

As is well known, the etiology of suppurative labyrinthitis is very rarely primary. It is usually secondary to diseased conditions of the middle ear, especially otitis media suppurativa which so frequently following the acute exanthematous fevers as measles, mumps and scarlet fever, particularly the latter. Suppurative labyrinthitis may follow pneumonia, meningitis, and even cases of thrombus in the petrosal sinus of the temporal bone.

In the acute labyrinthine suppuration the etiological factor is that of a previous suppurative otitic disease. The cases observed at the University hospital all could be traced to a middle ear suppuration. Undoubtedly a suppurative process might be set up as a result of meningitis, the infective agent gaining access through the lymph channels, the aqueduct of the vestibule and the cochlea. Only occasionally are suppurative processes set up in the labyrinth as a result of acute otitic infections, more commonly they occur as a sequence of long standing middle ear suppuration. They occur very frequently as an acute exacerbation of a chronic process of tympanic suppuration.
CHANNELS OF INFECTION FROM MIDDLE EAR TO LABYRINTH.

The labyrinth may become infected systematically through the blood vessels and the lymphatics, and indeed, many of those unexplainable inflammations are in all probability due to such channels. Shambaugh has worked out the circulation of a guinea pig and has demonstrated that there is quite a definite anastomosis of the blood vessels of the labyrinth and those of the middle ear. It must also be remembered that toxins of a systemic disease may become localized in the labyrinth, being carried by the vessels, and thus a localized labyrinthitis becomes apparent and which soon by lowering the vitality of the structure, makes it a ready playground for the many pyogenic bacteria which are constantly in the circulation.

There are no direct communications of the middle ear and the labyrinth. The two openings present in the osseous bony structure, foramina rotundum and ovale, are covered by a dense tough membrane which stops the progress of all germs. Many different accidents may occur to cause a perforation of the outer labyrinthine wall and thus afford an easy channel of entrance to the vestibule of any existing middle ear trouble. Cases have been reported of knitting needles being the cause. In a few of the cases the needle might have gone directly through the membrane covering the foramina ovale. Anatomically speaking though, a probe passing directly into the middle ear through the external auditory canal points directly at the promontory. In cases of fracture of the base of the skull through the petrous bone, microorganisms can invade
the labyrinth from the naso-pharynx by way of the eustachian tube and also externally through the auditory canal. During the operative attempt of extracting the stapes when necrotic especially if in unskilled hands, the labyrinth becomes infected. Even in the removal of foreign bodies from the ear, the stapes has become dislodged and the labyrinth infected. Also during the radical mastoid operation, great care must be exercised in removing the malleous and the incus, lest the stapes should be pulled away and the membrane covering the foramina ovale become perforated. Grunert in reporting Schwartze's clinic for 1895-1896 relates of an interesting case in that following the accidental removal of the stapes, there was a period of marked improvement as to condition. On the eighth day, the pulse became irregular; vertigo appears on the ninth day; on the tenth, intense headache; on the fourteenth day, symptoms of diffuse meningitis appear suddenly and patient dies on the twenty-third day following the operation. Autopsy revealed the oval window open and the labyrinth filled with turbid serum which on examination was found to be cocci. He states that the infection had reached the meninges by way of the acoustic nerve.

Victor Hinsberg of Breslau after carefully searching the literature for labyrinthine suppurations complication suppurations of the middle ear found only 198 cases which had been carefully recorded. The entrance of the infection from the middle ear to the labyrinth could be determined in only sixty-one cases of the one hundred and ninety-eight. Below are given the locations as he reports them.
Fistula in horizontal semicircular canal ..........27 cases
Fenestra ovalis...........................................17 "
Fistula on promontory................................. 7 "
Fistula in posterior or superior semicircular canal 5 "
Fenestra rotunda and ovalis.........................3 "
Fenestra rotunda....................................... 2 "

From the above statistics, it will be readily noticed that the infection entered the labyrinth most frequently through erosion of the horizontal semicircular canal, then through the oval window, the infection gaining entrance in only two cases through the round window.

There was but one definite demonstrable fistula found among our cases. It lead into the external semicircular canal. The walls of the canals were of a very thin bony lining.

The reason for the frequency with which the horizontal canal is involved can be ascertained on studying this from the anatomical point of view. We find

(1)- The horizontal or external semicircular canal protrudes somewhat upon the floor of the aditus, like a small rounded eminence.

(2)- Being situated in this way at a point of greatest constriction between the antrum and the tympanic cavity, it is subject to the action of all the suppurative process of the middle ear.

(3)- The aditus is made up of a hard dense bony ring.

The one exception is the horizontal canal and this represents
the weakest spot in this bony ring.

(4) Richards finds that the most vulnerable point in the horizontal semicircular canal is at a point several millimeters below the summit of its ivory-like cap and not at the extreme arch as is commonly supposed, due to the fact that this vulnerable point is made up of a more cancellous bone.

In a suppurative process destroying bone, this canal is easily reached and once the infection entering the canal, it soon gains entrance directly to the vestibule and then all the symptoms become most manifest, some of which may have appeared while the infection was en route to the vestibule. This fact is readily demonstrated working it out on the cadaver.

It must be remembered also that there are cases on record where the posterior and the vertical canals have become involved. Habermann reports a case following acute otitis media, where the infection extended from the tympanic cavity to the dura and then traveled along the vessels in the fossa subarcuata where an extradural abscess was developed, which later broke through into the superior semicircular canal, and then into the vestibule. Jansen reports seven cases where the labyrinth was infected through the superior semicircular canal. Koernen mentions possibly another source of invasion, namely, involvement of the labyrinthine capsule. This, he mentions, may occur especially in those virulent forms following an attack of scarlet fever. Politzer is of the opinion that in acute middle ear suppuration, the pus gains entrance most commonly through the fenestra vestibuli. Then the fenestra cochlea and lastly through
a fistula of the horizontal semicircular canal. I regret very much that in those cases which resulted in death, no pathological report was made of the petrose portion of the temporal bone, so the knowledge gained from our cases as to the method of invasion was obtained at the time of the operation. An infection once set up in the vestibule or in the cochlea, the germs seem to take on a more virulent form, destruction is rapid and the infection is able to travel easily to the brain by way of the facial and auditory nerve sheaths and also by the vestibular and cochlear aqueducts.
The pathology of suppurative labyrinthitis following an acute otitic trouble consists in finding the inflammatory products in the endolymph and these may be localized or diffused throughout the vestibule and all the canals. The fibrin becomes organized, a vascularized connective tissue is formed around the entrance of the infection. If of a very virulent infection the process becomes quite extensive, granulations may or may not be formed in the acute form. In the chronic form or where a labyrinthine infection has been set up following a long history of otitic trouble, a great many small granulations are found. Some of the canals on being opened, especially the external semicircular canal, are seen to be practically occluded by granulation growth. This vascularized connective tissue may in time be transformed into a hard bony substance by the deposit of calcium salts. Much of the labyrinthine wall may become absorbed and broken down. Microscopically, round cell infiltration is found among the granulations, the serous membrane, lining the vestibule and the canals becomes congested and swollen; round cell infiltration is found in most of its walls. The foot-plate of the stapes may be necrosed and only a small portion left or it may be embedded in the surrounding exudate. In one case an opening the first and second turns of the cochlea, as is always performed in a complete labyrinthectomy, a purulent exudate was readily seen exuding from the opening. Politzer\textsuperscript{3} states that in some cases the absorption has gone
on to such an extent in the cochlea that only a small fragment of one turn was remaining and the modiolus was almost wholly gone.

Gorke\(^{14}\) reports a few cases where in the destruction of the labyrinth he has found evidences of a rarefying osteitis taking place. This attacked generally the outer layer of the capsule.

Healso\(^{15}\) reported a case of Primary Necrosis of the Labyrinth which he thought was due to the shutting off of the blood supply. In describing the pathology of the suppurative form he says - "When the inflammation reaches the enosteum, granulation tissue forms on the outer surface of that membrane and more clearly noticed when the labyrinth has become infected through the aqueducts of the cochlea from the meninges. Buds of granulation tissue sprout up in the vicinity of the internal aperture of the aqueduct and in a short time become transformed into connective tissue, which has a marked tendency to undergo ossification processes which eventually lead to a complete closure of the aqueduct".

The terminations of infective labyrinthitis from a pathological viewpoint may be as follows:–

(a)- Complete return to the normal. The drainage being sufficient to allow most of the infective process to drain away. These cases are rare.

(b)- The labyrinthine cavity may become filled by fibrous tissue. The numerous granulations present soon become
permeated by connective tissue structure, and the many fibrous bands present tend to make it a hard compact mass which soon contracts and, in time, by deposition of calcium salts, be transformed into bone. The function of the labyrinth is much diminished in such cases.

(c)- Extension of the infection to the meninges, through the aqueducts of the cochlea or vestibule; internal auditory meatus or by osteitis of the capsule from the superior semicircular canal; formation of extra dural abscess thence meningitis.

The pathological examination of the labyrinth should be made as early as possible following death. An autopsy should be made of the labyrinth whenever this structure has directly or indirectly been the cause of death, that more light may be cast on this subject.

The fistulous openings in the external capsule which are frequently found at operation or at postmortem, probably do not always represent real avenues of invasion from without inward, but at times avenues of escape through which pus is successfully discharged from the labyrinth into the tympanic cavity.
CHANNEL OF INFECTION FROM LABYRINTH TO THE BRAIN.

The cranial cavity frequently becomes infected by extension of the process along the acoustic nerve and the aqueducts of the cochlea and the vestibule. The infection may also extend through the internal auditory meatus, the latter being a means of common invasion. Hinsberg holds that a congenital dehiscence ought to be mentioned as a possible pathology of infection. Dunn in working on an autopsy accidentally found a dehiscence of the superior semicircular canal of a negro. In many cases of a severe suppuration, the infection may actually rupture through the bone as through the superior semicircular canal, the rupture taking place in the direction of least resistance and this is found toward the cranial cavity. Jansen thinks that most of such complications develop during the course of a neglected acute suppuration. Krisckle substantiates this view by demonstrating a case which had an acute otitis media. Dizziness. Radical operation. Vertigo relieved. Pyaemic symptoms ten days after operation; death eight days later from a jugular bulb thrombosis. Microscopical examination revealed no trace of stapes; there was much young vascular connective tissue invading the vestibule from the tympanum. All of the semicircular canals were filled with granulation tissue, the osseous wall at the apex of superior canal was eroded and only a minute layer of bone separating its lumen from the dura. The enosteum is a great limiting membrane for the
advancing infection which will work its way between this enosteum and the bone much easier than penetrating the membrane itself. Anatomically speaking, any infection of the labyrinth may take place easier from the cranial cavity through the aqueducts of the cochlea and vestibule and through the internal auditory meatus, than coming from the middle ear. For here the infection must invade and break down membranes and bone, in fact overcome anatomical barriers to reach the labyrinth, while an infection from the dura, simply has to follow well defined pathways which lead directly to the labyrinth.
DIVISIONS OF LABYRINTHITIS.

The inflammations set up in the different portions of the labyrinth are named according to their locations:

Paralabyrinthitis is an infection of the bony capsule.

Perilabyrinthitis is that condition where pus or an exudate is found in the perilymphatic sac.

Endolabyrinthitis is that condition where pus or exudates are found in the endolymphatic sac.

Panlabyrinthitis involves all parts of the labyrinth.
SYMPTOMS.

Usually the initial symptoms to appear in acute suppurative labyrinthitis are those occurring as a result of irritation of the end filaments of the vestibular and cochlear nerves. The former being attacked first, dizziness and vertigo appear along with disturbances in equilibrium. If the patient is not already confined to bed, this forces him to go. Examination of the eyes will reveal a rotary nystagmus most marked when eyes are looking toward the sound side or looking in the direction of the quick component, and least marked when looking toward the lesion or in the direction of the slow component. The patient complains of the peculiar vertigo in that all the objects tend to rotate before his eyes. In only one case was I able to gain that, when the patient was held in an upright posture these objects tended to rotate somewhat in a vertical axis while, when the patient was reclining on his back, the rotation appeared to be more in a horizontal manner. The direction of the rotation varied so much at different times that no definite result could be stated. Among our cases nausea and vomiting was nearly an invariable symptom and usually appeared early along with the vertigo and disturbances of equilibrium and subsiding in 2 - 3 days.

In a certain few cases the nystagmus may appear directed to the diseased side first, due to irritation and then in three or four days, it is directed to the sound side. The nystagmus to the sound side is explained upon the fact that when the
labyrinth has been destroyed on one side, there is an over
stimulation of Deiter's nucleus on the opposite side and
hence a nystagmus to the sound side follows.

Usually the first symptoms to appear are dizziness,
subjective noises and spontaneous nystagmus and these in
turn are the first to go, while the disturbance of equi­
librium, if present, will continue for a variable time.

The cases studied here show that the temperature rise
was somewhat gradual, only one case where it rose at all
suddenly.

One case showed facial paralysis early in the labyrinthine
involvement. Kopetzky\(^\text{16}\) holds that in labyrinthine infection
the onset is found to be marked with a distinct rise in tem­
peratura and sudden appearance of facial paralysis. On the
contrary Deuch\(^\text{17}\) holds that facial paralysis does not occur
early or is a common sign of purulent labyrinthitis. Facial
paralysis usually occurs in cases of long standing necrosis.
As to the frequency of facial nerve involvement many different
reports are given. Ballenger\(^\text{18}\) has found the facial nerve
involved in labyrinthine suppurations in 55 per cent of the
cases under his observation, while the report of Gerber\(^\text{19}\) and
Bezold\(^\text{20}\) varies from 77 per cent to 83 per cent. Blake\(^\text{21}\)
states that the most vulnerable points of the facial nerve
being the region above the oval window and the superior portion
of the posterior tympanic wall.

There is usually some disturbance in hearing in every
labyrinthine case. A gradual progressive loss of hearing
developing under close observation in a comparatively short time is characteristic of inner ear involvement. In diffuse infective labyrinthitis of long standing there is generally found a total loss of hearing. In two of our cases there was total loss of hearing and in the others the hearing is more or less partially cut down.

In the acute infective labyrinthitis, after three or four days, the acute symptoms disappear but the nystagmus remains most marked to the sound. There is now a condition known as the latent or chronic form of infective labyrinthitis. The nystagmus continues to the sound side for about three weeks and then becomes gradually weaker and finally disappears altogether.

Cott calls attention to the fact that during the first few days of a labyrinthine suppuration there is a spontaneous nystagmus to both sides, after which they enter upon the second stage, and the nystagmus is towards the diseased side only. Dizziness continues, irritability of the semicircular canals is increased and hearing is plus. If resolution does not take place the disease enters on the third stage. Nystagmus is then toward the sound or healthy side, vertigo is still absent, irritability decreased and hearing lost. After a few days nystagmus again becomes of equal degree on both sides in the fourth stage but dizziness is absent, irritability of the semicircular canals and hearing gone. When nystagmus stops, the fifth stage is entered on, in which nystagmus, vertigo, irritability and hearing are all absent. (The normal ear has no nystagmus or dizziness,
irritability normal and hearing plus). If the third stage shows a negative caloric, it substantiates the diagnosis of destruction of the labyrinth. He further states that the first two stages, which show increased irritability to diseased side are perilabyrinthitis and when the nystagmus changes to the sound side and there is lessened excitability of the semicircular canals, the process is distinctly endolabyrinthine. Headache is a common accompaniment of purulent labyrinthitis. The character is that of a dull heavy pain referred to the temporal region on the side involved.

Tinnitus aurium occurs rarely in the course of a labyrinthine suppuration and only an occasional record is found among the many cases published.

Disturbances of equilibrium are fairly constant symptoms in the acute and subacute forms. In the chronic forms they are not so constant. Such disturbances include giddiness, standing or walking, with closed or open eyes and vertigo when even in a position of rest. The subjective vertigo complained of by the patient is a sensation of falling toward the healthy side and a tendency to walk in a circle.

Kerrison sums up his investigations by stating: "There are two distinct forms of labyrinthine vertigo and associated ataxia,—one the familiar spontaneous type, due to vestibular irritation and occurring only during the acute stage of suppurative labyrinthitis; and the other not spontaneous, not constant, not necessarily accompanied by nystagmus, characteristic only
of the lateral, or quiescent, stage of the disease, and induced solely by sudden and unexpected calls upon the lost or defective orientation - sense, in the maintenance of which the intact vestibular organs are normally so important a factor".

An intratympanic examination in certain cases may reveal evidences of necrosis of the inner wall of the tympanic cavity, if pus is found exuding from the oral window or from a fistulous track through the promontory, a positive diagnosis could be made of a labyrinthine suppuration.

The daily examination of the blood should be made in every case. In the acute form a high leucocytosis and increase in polymorphonuclear percentage are found. The large mononuclears are at times increased beyond their relative count. Frequently when a case commences to subside, an examination of the blood will detect this condition several hours prior to the time the symptoms manifest themselves. In the chronic form, there is little to be learned from the blood count before the operation.
The preoperative diagnosis of labyrinthine suppuration can seldom be made even where the typical symptoms such as vertigo, deafness, nystagmus, nausea, vomiting and headache are present. Impaired hearing, developing while the case is under observation and later having Webber's test localized in the sound ear, is generally evidence of invasion of the labyrinth. Accompanying this, the bone conduction is much impaired, and the range of audition greatly reduced; the upper tone limit lowered and the lower tone limit elevated. Barany\textsuperscript{1} states that disturbance of equilibrium is characteristic only of the acute stage of labyrinthine diseases, and that it regularly disappears as the lesion advances either toward resolution or destruction of the vestibular structures. VonStein\textsuperscript{1} however, holds that disturbances of equilibrium are present in all stages of suppurative labyrinthitis. When such disturbances of equilibrium are found in chronic suppurative labyrinthitis they are probably due to the over stimulation of the sound labyrinth. The caloric tests are negative. Rotary nystagmus to opposite side and the direction in which the patient would fall would always be influenced by the position of the head.

In a differential diagnosis, cerebellar abscess must be excluded. The nystagmus in a case of cerebellar abscess increases from day to day while in suppurative labyrinthitis it becomes less and less marked and may finally disappear.
owing to the extension of the suppurative process. In cerebellar abscess the quick component of the nystagmus is directed to the sound side and later it is directed to the diseased side. In labyrinthian suppuration the quick component may be directed for the first two or three days to the diseased side and then it is directed to the sound side. In cerebellar abscess the caloric tests are positive; no deafness present. Patient always tends to fall to the diseased side irrespective of the position of the head. Choked disc is present whereas it seldom appears in labyrinthine suppuration. In some of those cases where the diagnosis before the mastoid has been opened are vague, they will become clear on opening into the tympanic cavity and at this stage, then and only then are some cases diagnosticated rightly. Politzer considers the Schwabach test of special diagnostic value.

The diagnosis of acute labyrinthine suppuration can be made at times where the labyrinthian symptoms have developed during a few days while the patient has been under full observation. The gradual progressive deafness coming on, along with the other symptoms makes the diagnosis almost a certainty.

Having excluded, by a differential diagnosis, other diseases, the diagnosis of chronic suppurative labyrinthitis can be made almost positive, when functional examination reveals total loss of hearing, Webber's test localized to opposite side, along with a shortened Schwabach and in
addition the subjective signs of dizziness and spontaneous nystagmus. The diagnosis is confirmed when a fistula is found extending into a semicircular canal or any portion of the labyrinth.

In some few cases of localized purulent meningitis, the differential diagnosis may be difficult. A sudden rise in temperature, marked headache, vomiting, rigidity of neck muscles and in finding the Diplococcus intracellularis meningitidis or Pneumococcus in the spinal fluid points to a meningeal infection.

Dench\textsuperscript{17} states that in cases of beginning meningitis involving the vestibular nerve where a nystagmus toward the sound side, indicating a paralysis of the affected labyrinth, may later diminish and disappear, and a nystagmus toward the affected side make its appearance. This, he holds, is a positive evidence of a retrolabyrinthine lesion, which may be either in the cerebellar substance or in the meninges in the immediate vicinity of the vestibular nerve.

Reik\textsuperscript{23} mentions that occasionally during the operative treatment that the semi-circular canal wall presents a bluish, or brownish discoloration; this he states as being due to the appearance of granulations or blood-clot seen through the thin bony covering.

While vertigo, vomiting, nystagmus and disturbances of equilibrium of labyrinthine origin are characteristic of an invasion of the labyrinth yet upon these alone, a positive diagnosis cannot safely be made. With the above symptoms should pus be found coming through the oval window, then a
positive diagnosis could be made. If these were found at the time of the operation, the diagnosis would be that much more certain.
COMPLICATIONS.

The complications most liable to occur in suppurative labyrinthitis are, Meningitis, Cerebellar abscess, and septic thrombus of the jugular vein. Extension to the dura takes place through:

(1) - Peri-neural and peri-vascular sheaths of the seventh and eighth nerves.

(2) - The aqueductus vestibuli with consequent formation of an empyema of the saccus endolymphaticus and subsequent cerebellar abscess.

(3) - The aqueductus cochlea.

(4) - Erosion of superior and posterior semicircular canals.
PROGNOSIS.

It is difficult to state the prognosis of a labyrinthian suppuration. The acute form may subside gradually and healing take place, however, this may only be temporary and sooner or later the condition returns. Every labyrinthian suppuration should be regarded as a dangerous process for the elimination of which we should use every possible means. We are not exactly able to distinguish between the favorable and the unfavorable forms. The prognosis is unfavorable in the chronic forms, perforation of the promontory wall; pressure of cholesteatoma extending into the interior of a semicircular canal.

Politzer holds that the prognosis is very unfavorable in an established perforation of the promontory wall, which, with a simultaneous deafness, must be looked upon as a sure sign of a diffuse labyrinthine suppuration and in which, as already mentioned, the destructive process not infrequently extends to the peripheral part of the internal meatus and to the auditory nerve.

An infection of the labyrinth of tuberculor or exanthematosus origin is generally held to be more destructive than due to other pyogenic causes.
TERMINATIONS.

According to Milligan and Wingrave the terminations of a labyrinthine suppuration may be as follows:

The acute diffuse pyolabyrinthitis may terminate in:
1. Total destruction of the auditory function.
2. Sequestration of part or the whole of the labyrinth.
3. General blood infection.
4. Chronic disease.
5. Death.

The chronic pyolabyrinthitis may terminate in:
1. Ossification of the affected area.
2. Caries of part or the whole of the labyrinth.
3. Erosion of bone and infection of the contents of the middle or posterior fossa.
4. Destruction of the static or acoustic segments of the internal ear.
5. Septicaemia or pyaemia.
6. Death.
TREATMENT.

There are many different views held now regarding the treatment of Labyrinthine suppuration. Much confusion exists as to indications and contraindications of a labyrinthine operation. The treatment should only be considered after a most careful study of the existing conditions which are present in each individual case.

The purpose of the labyrinth operation according to Ballenger are

1- To prevent the extension of the purulent infection to the meninges and brain, and to check and cure an incipient meningitis having its origin in the labyrinth.

2- The second purpose is to cure an offensive purulent otorrhoea which is perpetuated by a labyrinth fistula, remaining after the destruction of the labyrinth by a diffused suppurative labyrinthitis.

3- The third purpose is to relieve the patient of a distressing giddiness which occasionally persists after the cochlear nervous apparatus is destroyed by a suppurative process, while the vestibular nervous apparatus is but partially destroyed and gives rise to distressing and disabling giddiness.

The indications which are most generally accepted at the present time for opening the labyrinth are-

1- With the labyrinthine symptoms, complete deafness and non-irritability of the vestibular nerve of a chronic process
or an acute exacerbation of a chronic process indicate immediate opening of the labyrinth.

2- Necrosis of the labyrinth as induced by cholesteatomatous pieces discharging from the wound and if associated with facial paralysis indicate opening of labyrinth and removal of all necrotic tissue.

3- Following a radical mastoid operation, at the first sign of meningeal irritation open and drain labyrinth.

4- According to Jansen any symptoms of cerebral complication makes a positive indication to at once open labyrinth.

Generally speaking there is no indication to open the labyrinth in an acute labyrinthine suppuration. However, the case must be kept under most careful observation and at the first sign of meningeal irritation the complete labyrinth operation should be performed. In the acute type it is better to keep the patient at absolute rest, by that I mean, no movement whatever of the head or body. Ordinarily it will not require much will power to keep such a case at rest, for the patient will remain very quiet to avoid the most distressing symptoms that arise on movement. In certain cases, were with a marked onset and pronounced symptoms of the process becoming diffuse, one is obliged to operate at once. The latent stage of a labyrinthine suppuration is the period in which it can be operated upon with the best prognosis. Kerrison states that vomiting, headache and elevation of temperature are distinctly characteristic of vestibular irritation and are not necessarily indicative of meningeal disease and where these
are not excessive or too prolonged, surgical intervention should be delayed.

In cases of chronic middle-ear suppuration where there has been evidences of past suppurative labyrinthitis, it is best and safer to open and drain the labyrinth at the same time performing the radical mastoid operation.

Scheibe in reporting five cases of acute purulent labyrinthitis stated that the therapy consists in absolute rest, after the eradication of the primary foci in the middle ear spaces. He employed this method in four cases. The patients were kept almost immovable in bed for at least three weeks. He had one fatal termination, and in this case absolute bed rest was not enforced. He reports that in comparison with these uniformly good results, his results, when he still treated labyrinthine suppuration as ambulatory cases, were a uniformly fatal termination.

According to such men as Hinsberg, Jansen, Neumann, Barany and Richards it is most essential that the vestibule should always be opened and drained in the chronic labyrinthine suppuration because-

1- The operation secures free drainage from the vestibule which is always involved in suppurative labyrinthitis and

2- Enables operator to destroy the membranous structure of the vestibular apparatus which is a main point in controlling permanently vestibular symptoms.

The route posterior to the facial canal from the manipulative standpoint appears to be the most convenient, because
of least danger to the important structures and allows the operator to fully expose the limits of the disease.

Richards⁶ states that it is preferable to enter the vestibule posterior to the facial nerve for a number of reasons, namely,

1- The semicircular canals are usually involved along with the vestibule and this allows of their exploration.

2- If the canal system be only involved, there is not so much danger of intra-cranial complications by way of the labyrinth.

3- The maximum amount of working room is obtained in this manner. It also allows of working down in the petrous pyramid in an axis which is the least dangerous.

4- The ampullary areas of the different canals can be examined.

5- The route anterior to the facial nerve does not allow of a complete exploration of the canal system which we know is usually involved along with the vestibule. It does not allow of a complete exploration of the inner wall of the vestibule where it is most essential to remove all diseased structure.

6- The anterior route can only allow of drainage and this is in part an erroneous idea, for in suppurative labyrinthitis of longstanding, the interior of the labyrinth and especially the capsule will be found very badly diseased.

7- The anterior route is more dangerous, speaking from a manipulative standpoint. The greatest dangers here
are that the jugular bulb may rise high up under the outer vestibular wall. An injury to the internal auditory meatus is very liable to occur while working in this location.

Much discussion has arisen during the past as to the nature and extent of the operation. Neumann advocates the complete exposure of the cochlear and vestibular apparatus while Alexander and others believe in only partial exposure. Operating during the latent stage of the disease we are inclined to hold that a complete labyrinth operation should be performed. It will be found safer for the patients and the results are better. Rut tin has performed one hundred complete labyrinth operations with one death resulting from the operation.

The primary step in every labyrinth operation is the complete exposure of the tympanic cavity and the horizontal semicircular canal by the radical mastoid operation. The internal wall of the tympanic cavity and the horizontal canal should be carefully examined for any fistulous tracks leading into the labyrinth. Should a fistulous opening be found in any of the canals, they should be thoroughly opened and removal of all granulation tissue and necrotic bone. In many cases it will be necessary to chisel away the canals to their base, thus opening the vestibule above and behind. Where fistulae are found the labyrinth is better exposed behind the facial ridge. The posterior crus of the horizontal canal is opened and the anterior crus is chiselled away to the labyrinth. Then the bone lying below the oval window and above the round window is removed, thus exposing the vestibule and small portion of
the cochlea. The opening is now enlarged so as to freely drain the first and second turns of the cochlea. The posterior cranial fossa is not opened as a routine measure in the labyrinth operation.

The Jansen\textsuperscript{23} Neumann\textsuperscript{29} method opens the posterior cranial fossa as part of the labyrinth operation. They claim for it the easy exploration of the posterior and middle cranial fossa.

The method of Hinsberg\textsuperscript{5}, Bourguet\textsuperscript{25} and Batey\textsuperscript{25} are very much similar. They all reach the vestibule from in front of the facial ridge and then open and drain the promontory, etc.

Many operations have been advocated for purulent labyrinthitis but the truth is even made more evident that we must make the surgical technique meet the desired condition in each individual case.
Miss J. L., school girl, age seventeen, contracted scarlet fever nine years ago which was followed by nearly a constant discharge from the ears. At times under treatment ears will become dry and then seemingly without any cause will discharge again. Three months ago patient developed measles following which there was an increased purulent discharge from the right ear with dizziness and some nausea. This condition continued up to her admittance to the hospital. On examination of right ear bone conduction is better than air. High notes cut down markedly. Drum head absent. Hears loud noise at one inch. Has slight stricture of right tube. A remnant of the hammer is present but the bony process is gone. The inner wall of the middle ear is covered with a red polypoid mucous membrane. Left ear hears loud noise at four feet, bone conduction better than air. No nystagmus present. Bacteriological examination of right ear, cultures yielded colon bacilli and staphylococcus albus. Patient did not tend to fall in different directions, though this test was not satisfactory. Blood revealed, Reds, 4,120,000; whites 9,900; D.C.(100 cells) Polys 74%; large lymphocytes 11%; small lymphocytes 15%. HC 32%. It was decided in this case to do a radical mastoid combined with labyrinthectomy.

Operation June 17, 1910. The preliminary radical
operation was performed, much necrosis being found. A complete labyrinthectomy was performed. Pus was under pressure at junction of posterior semi-circular canal with horizontal semi-circular canal. Wall of cochlea was necrotic. Internal to additus and facial canal there was extensive necrosis. Vestibule was curetted of many granulations. First and second turns of the cochlea were opened and drained. Patient for the next six days had a slight rotary nystagmus which was most marked on looking to the left. Patient was a little dizzy and objects seemed to move from right to left, this however, cleared up three days after the operation. July 25, blood showed whites 19,800; polys 83; large mononuclears 1%; lymphocytes 16%; July 28, patient was feeling better, blood, whites 18,200; polys 78%; lymphocytes 22%. August 3, patient was doing nicely, dizziness is clearing up. Patient gradually grew stronger in every way and with no return of symptoms, she was discharged Aug. 18th. Saw patient again Oct. 31st and she was feeling better than she had for a number of years.

Case II.

Mr. W. F., farmer, age thirty-three, came to the hospital complaining of a discharge from his left ear and frequent dizzy spells. Patient says there has been a discharge for the last fifteen years. As a child always was troubled with ear-ache. At times complains of sharp shooting pains which radiate from the left ear to the fore-head. Patient says these attacks occur periodically usually ever four to
six weeks. During one of these attacks, patient has intense pain in the ear and over the mastoid, accompanied by nausea, vomiting and dizzy spells. On ear discharging there is relief of these symptoms. The patient states that he is compelled to lie right down by these attacks. They usually last an hour or more. The discharge is composed of much blood and very foul pus. The last attack came on about three weeks ago, patient being compelled to go to bed until the spell was over. The attacks were becoming more severe each time. Examination of left ear revealed that most of drum head was gone. Bone conduction better than air. With alarm apparatus is unable to hear whispered or spoken voice. Right normal. Romberg present. Spontaneous nystagmus present, with quick component directed to the right. Caloric test of left ear was negative. Water temperature 79°F and 110°F producing no nystagmus to the opposite or same side respectively. Right ear, caloric test was positive. Rotating the patient from left to right, causes vertigo and objects seem to move from right to left. Nystagmus, rotary in character, with quick component to the left was seen when the rotations were suddenly stopped and the eyes directed toward the left. Reflexes are normal. Fundi normal. With feet together, eyes closed and head tilted to the left, there is a tendency to fall to the left. With head inclined to the right, patient tends to fall backward and to the right. With head erect, patient tends to fall backward. Blood reveals reds 4,310,000; whites 9700; D.C. (100 cells) Polys 71%; large lymphocytes 10%; small lymphocytes 19%. Cultures and smears from left ear revealed
staphylococcus albus and a few pneumococci.

Diagnosis - otorrhoea chronica with involvement of the labyrinth. Operation. The tympano-mastoidectomy was first performed. There was much necrosis throughout all the mastoid region. The external canal was opened and drained. It contained a few granulations. The vestibule was curetted for here was found many old granulations; the first and second turns of the cochlea were opened and drained. Only a few granulations were present here. Patient recovered nicely from the operation. During the next five days, nystagmus was more marked to the right, no nystagmus on looking to the left. Patient dizzy on moving head quickly, no nausea, no vomiting. Three days later, caloric test of right ear was positive. In left ear negative. Rotation from right to left causes vertigo and objects move from right to left. During the next week the nystagmus gradually improved. Dizziness is gradually disappearing. Patient has a slight oscillatory nystagmus on looking to the right. With seigles speculum pressure seemed to inhibit the nystagmus to the right. Rarification of air seemed to start a slight vertical nystagmus. Caloric test of left ear; water, temperature 78° stopped nystagmus completely in ten seconds. In twenty seconds, started it again with quick component directed to the right. Water, temperature 110° caused no nystagmus on looking to the left, nystagmus was slightly increased on looking to the right. Patient gradually grew better in every way and was discharged with wound dry and healed. Saw patient three months later,
had gained some thirty pounds and was enjoying very good health with no return of any of the former symptoms.

---

Acute Purulent Labyrinthitis followed by septic meningitis and death.

Case III.

Mrs. M. H., thirty-four years of age had suffered from a chronic right-middle-ear suppuration for the last two years. The family history is negative. The patient had scarlet fever and mumps as a child. Patient stated that during a slight cold she jerked her head suddenly one day, a severe headache followed and in about two hours afterwards the right ear commenced to discharge. Ear discharges at intervals from two to six weeks apart and lasts from a few days to five or six weeks. During some of these attacks the pain has been very severe, patient becoming unconscious. When she awakens she is very dizzy, nauseated and vomiting follows. The dizziness is becoming more severe at each attack. When patient feels these spells coming on she usually calls for help and runs and lies down. During some of the attacks there is much pain in the right eye and the right side of the face becomes swollen. Following these attacks the discharge is usually increased and the odor becomes most offensive. Examination revealed a fairly well nourished individual. Pupils equal, and reacting to light and accommodation. Fundi normal. No nystagmus present. Right ear drum head is reddened and bulging. Bone conduction better than air and high notes not cut down. Hearing with alarm apparatus, whispered voice four feet and spoken voice ten feet. Caloric test using water temperature
110°F produced a nystagmus to the right, directing the eyes to the right. Blood revealed Hb 90%; Reds 4,860,000; whites 76%; LL 8%; S.L. 16%; Culture showed Staphylococcus albus. Diagnosis - Otorrhoea chronica right. April 21, patient had radical mastoid operation. There was much necrosis in the tip of the mastoid. The roof of the attic was extremely necrotic. Dura or lateral sinus not exposed. The next five days following the operation, the patient was free from pain and was resting very easy. On April 27th patient for the first time complained a severe headache in right temporal region. Urine negative - temperature 100.2. Pulse 90. On April 28 performed plastic on mastoid, following this slight operation there was considerable vomiting, much more so than is common after an ordinary anaesthetic case. During the next five days, patient had an occasional severe headache and much emesis present. On May 7th temp. 102.3 and pulse 122. Patient is complaining of a severe pain in right side of head. Much emesis. On May 8th temperature 106.8, pulse 96, still has severe headaches and emesis continues. Urine negative. There is a spontaneous rotary nystagmus to the left with quick component to the left. Fundi normal. Blood count reveals whites 12,800; reds 4,740,000; Hb. 90%; polys 88%; large lymphocytes 2%; small lymphocytes 10%. In the evening patient complains of pain in the back and the neck. The neck muscles are somewhat rigid. On May 9th temp. 100.4, pulse 100. Patient had a good night, but complains of pain in neck and top of head. Nystagmus the same. Fundi normal. Urine negative. Patient seems nauseated all the time. Blood count shows
whites 10,400; polys 86%; large lym. 4%; small lym. 8%.

P.M. specimen of urine shows a slight trace of albumin. May 10th temp. 102.2, pulse 110. Patient is very restless and when awakes from a nap, immediately complains of pain in her neck. Blood shows whites 12,800; polys 89%; marked photophobia present. May 11th, temp. 98.6 and pulse 96. Has many shooting pains from right ear to top of head. Blood shows whites 11,200; polys 83%; myelocytes 3%; small lym. 9%; large lym. 3%; basophiles 1%; eosinophiles 1%. Nystagmus still rotary in character to the left. Slight horizontal nystagmus on looking straight ahead. Eyes seem to converge when concentrating gaze. Patient says she feels dizzy; objects do not remain stationary. Complains of entire head throbbing. May 12th s complete labyrintectomy was performed. The vestibule was filled with pus. Temperature 101. ax. Pulse 100. Patient appear drowsy and does not seem to complain of much pain. Urine negative. Blood shows whites 14,400; polys 94%; myelocytes 1%; small lymphocytes 3%; large lymphocytes 2%. Nystagmus the same. Right eye deviates outward on convergence. Lumbar puncture made and culture from cerebrospinal fluid and the tympanic cavity both revealed Streptococcus pyogenes. May 13th, temp. 103.8; pulse 100. Resp. 26. Some complaint of pain in top of head. No facial paralysis. Blood revealed reds 5,120,000; whites 11,750; D.C. (200 cells) polys 91; large lym. 1%; small lym 8%; hb. 80%. May 14th, temp 104°. Pulse 100. Resp. 26. There is a definite facial paralysis. A typical babinski of both feet, more marked on left. Both pupils react to light. Neck is moderately stiff in all motions, but
not retracted. Patient is mentally clear. Babinski's continue. Kernig test shows more resistance than yesterday. May 15th temp. 103.8. Pulse 108. Respiration 24. Patient is very restless and cannot be awakened, with respirations becoming irregular; neck is markedly rigid. Facial nerve is completely paralyzed. Knee jerks are sluggish and babinski indifferent. Blood shows Reds 5,220,000; whites 26,400; Hb 80%. D.C.(200 cells); Polys 93%; small lym. 6%; large lym. 1%. May 16th, temperature 101. Pulse 96. Respiration 28. Patient in much pain all the time, is very noisy and irrational. Much serous discharge coming from wound. Blood shows reds 4,200,000; whites 28,000; D.C.(300 cells); Polys 97%; small lym. 2%; large lym. 1%. Patient gradually sank and died. No autopsy could be obtained.

CASE IV.

Miss B.P., clerk, twenty-three years of age, was admitted to the University Hospital complaining of a discharge from the left ear. Patient gave history of having scarlet fever when three years old and that the left ear has discharged continually since that time. The right ear has discharged at intervals, usually following a cold but would clear up in a few days. About eight weeks ago patient noticed that the odor was becoming quite offensive and the discharge much more purulent in character. At no time can there be elicited any pain, either in the ear or occurring in the region of the mastoid. On examination only remnants of the drum head could be found. Canal was filled with foul smelling pus. Bone conduction better
than air. Webers test not localized to either ear. High
otes not cut down. Hearing in left ear, whispered voice
was 6 inches; spoken voice 1-1/2 feet. Right ear normal.
No nystagmus present. Caloric test of both ears were
positive. No nausea or vomiting. Blood count showed
reds 4,220,000; whites 9,700; Polymorphonuclears 74%; large
lym. 10%; small lym. 16%. Bacteriological examination re­
vealed an abundance of diplococci, a few resembling pneumo­
occi, and a few groups of staphylococcus albus. Urine
negative.

Diagnosis - Otorrhoea chronica left.

Operation Nov. 2, 1911,- Tympano mastoid exenteration
with labyrinthectomy. Mastoidectomy was performed. The
whole of the mastoid was diseased. The lateral sinus was
not exposed. The petrosal sinus was, owing to the fact that
the necrosis extended deep into the petrous portion of the
temporal bone. The petrosal sinus appeared normal. The
tympanic cavity was found filled with granulations and there
was no remnant of the incus or stapes to be found. About
one-third of the malleus was left as a rough diseased piece
of bone. Pus was coming both from the round and oval windows.
A labyrinthectomy was performed. The semicircular canals
seemed normal. The vestibule was filled with granulations
as was also the first turn of the cochlea. At the completion
of the operation there was an abundant discharge of clear
cerebrospinal fluid into the vestibule. Nov. 3, patient has
a marked rotary nystagmus, with the quick component directed
to the right, on looking to the right. At times a slight
horizontal nystagmus on looking to the left. When patient moves her head, she becomes at once very dizzy and objects in the room seem to move from left to right. Patient tries to keep head perfectly still on this account. There is no headache. Blood count shows reds 4,380,000; whites 11,600; polys. 79%; large, lym. 2% and small lym. 19%. Temp. 99.2. Pulse 110. Nov. 4, patient feeling good, no headache, has rotary nystagmus to the right on looking to the right. No nystagmus to the left on looking to the left. Becomes very dizzy on any attempt to move the head. There is much serum draining from the wound. Patient had several vomiting spells during the night. Does not feel nauseated now. On looking straight ahead, patient has a horizontal nystagmus, with the quick motion directed to the right. Temp. 98.8. Pulse 108. Nov. 5, patient not feeling very good. Has slight headache. Rotary nystagmus to the right which disappears on looking to the left. Nystagmus is disappearing in amount. Patient was slightly delirious last night for two hours. Temp. 98.8. Pulse 92. Whites 16,800; polys 94%; large lymphocytes 5%; small lymphocytes 11%. Nov. 10, patient doing nicely, has a slight rotary nystagmus to the right, disappearing on looking to the left. Nystagmus is fast disappearing. Wound is healing nicely. No nausea or vomiting. Patient sitting up in chair. No dizziness present. Nov. 20, wound is healing nicely, a very slight rotary nystagmus to the right on directing the eyes to the right. Cerebro-spinal fluid is still draining from the wound. Patient is not dizzy when moving about. Nov. 24th, patient doing nicely in all respects. Caloric test was tried
on the left ear. Water whose temperature was 110°F was used. No nystagmus appeared to the left or same side, when directing the eyes to the left, thus demonstrating that the function of the semicircular canals on this left side have been destroyed. Plastic performed on left mastoid. Dec. 2nd, patient has a horizontal nystagmus when directing eyes either to left or right. Jan. 16th, 1912, no nystagmus on directing the eyes to either right or left. Wound is practically all healed. Patient is in the best of general health. No headache, nausea or vomiting, no dizziness whatever.

Case V.

Mr. H. S., farmer, eighteen years of age, was admitted to the hospital Feb. 2, 1911, complaining of a discharging sinus behind the right ear. Patient gave history of having a discharge from right ear when seven years old which at times would completely stop and then would start again to discharge without any apparent cause. Patient states that five operations were performed on right ear while in Sweden, but does not recall the nature of any of them. At times patient has dizzy spells, becomes nauseated and vomits. About one week ago, right ear and mastoid became very tender and there was intense pain present over mastoid region. Examination revealed a red swollen area over mastoid, at the tip of which there was a sinus discharging a white, foul smelling pus. Drum membrane red and bulging. Bone conduction better than air. Low notes markedly cut down. High notes not cut down, cannot hear spoken voice with right ear. No nystagmus present. Fundi normal. Blood showed Reds 4,140,000; whites,11,300; polys 72%; large lymphocytes 9%
small lymphocytes 19%; Myringotomy was performed and irrigations ordered. Mastoid region painted with tincture of iodine. With no improvement, one week later, Feb. 17th, a tympano-mastoidectomy with labyrinthectomy was performed. The mastoid was very necrotic. A sinus was present leading into the horizontal semicircular canal. All of the three canals were opened and drained. The superior canal being curetted thoroughly, this proved to be the most nearly normal of any of the canals. All of the canals were filled with granulations. The first two turns of the cochlea were removed. Lateral sinus was exposed at the knee. The next day patient had a marked rotary nystagmus to the left (sound side) on looking to the left, quick component being directed to the left. On looking to the right no nystagmus. Patient was nauseated and vomited at times. Objects in the room appeared to be moving all the time, making the patient feel dizzy and very uncomfortable. Temp. 99.3. Pulse 92. Blood count shows whites 11,600; Polys 81%; large lym. 5%; small lym. 14%. Feb. 19, there is a rotary nystagmus to the right and left on directing the eyes in both of these directions respectively. Rotary nystagmus is most marked to the left. No headache. Some nausea and vomiting. Feb. 22, patient has a rotary nystagmus to the left on looking to the left. The quick component is directed to the left. Nystagmus on looking to the right, there is a slight rotary nystagmus, with quick component to the right. Some headache this morning. Feb. 24, patient feeling fairly well. On moving about there is at first some dizziness which clears up soon. Rotary
nystagmus is most marked to the left side. No headache, nausea or vomiting. Plastic on mastoid performed. March 5, nystagmus is gradually disappearing. No dizziness apparent. No headache. Wound nearly healed. Patient in good general condition. March 28, nystagmus has disappeared. No headache present. No nausea, no vomiting. Wound practically healed. Discharged from hospital. Aug. 2, had occasion to see patient at this time. There was no recurrence of any symptoms. Patient was feeling fine; had gained many pounds in weight.

Case VI.

W.F., farmer, thirty-five years of age, had suffered from a chronic suppuration of his left ear since he was thirteen years of age. At times the discharge would cease and then the pain would become very severe and on discharging, the pain would be relieved. Any cold always aggravates the condition. Three weeks ago had severe pain in left ear which was followed by a purulent discharge with very bad odor. Patient was confined to his bed. Whenever he attempted to gain a sitting posture in bed he became very dizzy and nauseated and vomiting followed. This condition lasted about one week, after which the attacks became less severe and the patient managed to get about but was always somewhat dizzy. When he was sitting still this dizziness was not apparent. Patient stated that when in bed and moving his head from side to side he had the sensation that the bed was going to turn over. Any quick motion of the head causes dizziness. Changing the position of the head always changed the direction
in which the patient would fall. Caloric test was very slightly positive. Water, temperature 78°F caused a slight rotary nystagmus after sixty seconds. It lasted twelve seconds. The quick component being directed to the right and the slow component to the left. Water temperature 110°F produces no nystagmus. Functional tests showed absolute deafness in the left ear, normal in the right. Fistula test-air condensation gives a slight nystagmus to the opposite side - this test is rather unsatisfactory owing to the fact that the external auditory canal was over half filled with granulations. Temperature 98; pulse 70; respiration 18. Blood count revealed Reds, 4,600,000; whites 9,800; polys. 76%; large mononuclears 6%; small lym. 18%. Operation May 1, 1911, left radical mastoid was first performed. Dura exposed in the roof of the antrum. Tympanic cavity covered with granulations. Tension of dura very low; was opened. Opened horizontal semicircular canal which was followed down to the vestibule. The cochlea was curetted by opening the first and second turns. May 2nd, patient doing nicely. Temp. 99.2. Resp. 18. Blood count, whites 12,900; polys. 81. large lym. 7%; small lym. 12%. Slight nystagmus with quick component directed to the right. During the next two weeks patient improved rapidly. May 25, blood count showed, whites 9,200; polys.78%; largelym. 8%; small lym. 14%; nystagmus has practically all disappeared and patient in good shape. Saw patient again in August 2 and there was no recurrence of any of the preceding symptoms and patient was in the
best of general health.

**Case VII.**

Mr. E. P., minister, twenty-four years of age had suffered from a discharge from his left ear since early childhood and the last month noticed that attacks of dizziness have been appearing. Family history negative. Had measles, mumps, whooping cough and chicken pox as a child. Gave history of having much otitic trouble as a child with frequent attacks of pain in mastoid region. Discharge has always been of a very bad odor, yellowish white in color. For the last years has suffered with intense headaches, usually localized in the left frontal-temporal region. About one month ago these dizzy spells came on and have gradually become more severe in character. Dizziness always worse when patient first gets on his feet and then gradually diminishes somewhat. During the past month he has lost some twenty-five pounds. In these attacks of dizziness patient states that all objects in front of him tend to move from the left to the right. An examination revealed a poorly nourished male, pupils equal, reacting to light and accommodation. Vision OD =6/4. OS =6/5. Vision fields not cut down or overlapping. Has rotary nystagmus to the right on looking to the right, rotary nystagmus to left on looking to the left, though less marked than on looking to the right. Caloric test, water temp. 78°F, rotary nystagmus to the right increased and to the left is stopped. Water temp. 110°F, no change in the nystagmus. Left
ear revealed canal partially filled with a yellow odorous discharge, with perforation of drum head. Bone conduction better than air, high notes partially cut down. Hearing with alarm apparatus, loud voice eighteen inches, cannot hear whispered voice. Fistula symptom negative. With eyes closed, head erect patient tends to fall to the right. With head way back, falls to left and posterior. With head tilted to left falls to left; with head tilted to right, falls to the right. Fundus oculi are negative. Urine negative. Blood shows Reds 4,940,000; Whites 8,900. D.C. (100 cells); polys 68%; small lym. 25%; large lym. 7%; temp. 99, pulse 60, resp. 18.

Diagnosis - chronic otorrhoea with recent involvement of the labyrinth. Patient advised to have tympano-mastoid operation with partial exenteration of the labyrinth. Operation July 12, 1911. The cortex of the mastoid was very hard. The lateral sinus was far forward, being exposed at the knee it appeared normal. The necrosis in the floor of the additus and posterior and superior to it was marked, especially in the latter situation. There was no sinus visible in the outer wall of the horizontal canal. The bone covering it was very soft and was removed readily with a curette with one scraping, giving the impression it was a small sequestrum that was removed. The interior of the canal seemed perhaps congested otherwise normal. There was no excess of fluid, no pus, no granulations in the canal. The horizontal canal was opened to the vestibule. This was curetted. There were no granulations or other evidence of marked inflammation. A slight remnant
of the incus and hammer was found.
The first and second turns of the cochlea were opened.
Light iodoform packing was used. During the next two days, there was no nystagmus, no facial paralysis, no vertigo, no vomiting. Temp. 99.3. Pulse 63. Resp. 20. Blood, - Reds, 4,300,000; whites 9,300; polys 75%; large lym. 6%; small lym. 25%. On July 15th, a rotary nystagmus appeared, more marked to the right than to the left. Quick component is directed to the right and slow component to the left. Blood shows, Reds, 4,280,000; whites 9,900; Polys 76%; large lym 4%; small lym. 20%. During the next two days, patient complained of some severe frontal headache. Temp. 99.4, Pulse 72. Resp. 20. On looking to the left there is a slight horizontal nystagmus, with quick component to the left and on looking to the right, there is a rotary nystagmus, quick component to the right. Blood shows Reds 4,560,000; whites 9,400; Polys 74%; large lym 3%; small lym 23%. On July 19, Temp. 98.4, Pulse 72. Resp. 18. Nystagmus rotary to the right and a very slight horizontal nystagmus to the left. Patient complains of intense frontal headaches. Blood shows Reds 5,150,000; whites 8,900; D.C.(100 cells) Polys 76%; large lym. 3%; small lym. 21%. Fundus oculi are normal. With ophthalmoscope slight horizontal nystagmus detected when looking straight ahead, with quick component to the right. Pupils react to light and are equal. Does not wake up from headaches. During the next four days patient remained the same. Nystagmus to the left is very slow, there being only three to five oscillations and then the nystagmic movements
Patient is up walking around, sleeps good and has a good appetite. On July 24th temp. 93; pulse 76; resp. 20. No dizziness at all. All objects remain stationary now.

Rhomberg negative. Blood shows Reds 4,930,000; whites 9300. D.C. (100 cells); Polys 71%; small lym. 23%; large lym. 6%; Had plastic on left ear. On July 27th, Temp. 98.2; pulse 80; resp 20. Nystagmus practically the same, rotary to the right, with quick component to the right and slow horizontal nystagmus on looking to the left. No Rhomberg present. No headache. On placing the head to the left, to the right, backwards and forwards, there is no tendency to fall in any direction. There appeared just a little general dizziness following these tests. Blood count shows Reds 4,930,000; Whites 9,600; D.C. (100 cells) Polys 70%; L.L. 6%; small lym. 24%; wound is clearing up rapidly. During the next three days patient the same. July 31st, Temp. 99; Pulse 80; Resp. 20; Patient in very good condition. No headache, no vertigo, nausea or vomiting. Nystagmus is gradually diminishing in character. Station tests are all negative. Blood shows Reds 4,630,000; whites 9700; D.C. (100 cells); Polys 70%; s.l. 24%; l.l. 6%. Sleeps well and has good appetite. Wound healing nicely. Discharged from hospital and receives daily treatment at the office. Discharged to allow home doctor to take the case. Saw patient again Aug. 15, was doing fine in every way. Nystagmus is clearing up. On Nov. 21, saw patient again. Wound all healed. On looking to the right there is a rapid horizontal nystagmus, with quick component to the right. No nystagmus on looking straight
ahead. On looking to the left there is a less rapid horizontal nystagmus, with quick component to the left. No dizziness at any time. Objects do not tend to move before him at all as was formerly the case. All station tests negative. Has gained some twelve pounds in weight, now appearing very strong and healthy.

Case VIII.

Diffuse suppurative labyrinthitis complicating measles. This case was reported by Kerrison and it presents in regular sequence all the classical symptoms of the disease from onset to recovery following a labyrinthine operation.

Miss A. W., trained nurse, 21 years of age, was admitted to hospital in December, 1908 suffering from a severe type of hemorrhagic measles. Ears examined on admission were reported to have been normal. On or about Jan. 5, 1909, she developed acute purulent otitis media of right ear with some mastoid tenderness. Myringotomy by the resident physician. Two days later, when I first saw her, a mastoid operation was clearly indicated. Removal of the cortex revealed pus in the antrum and extensive inflammatory changes throughout the mastoid cells.

Five days after the operation, when still very ill from the systemic infection, she developed the following additional symptoms, viz., marked rotary nystagmus to the left, which was most pronounced when the eyes were voluntarily turned to the left. She complained much of subjective vertigo. She was
nauseated and vomited several times during the day. These symptoms underwent gradual but progressive diminution. During the dressing of the wound as she lay with the left ear buried in the pillow, the deafness of the right ear was very noticeable. During the month following the operation, the patient gained strength very slowly and the wound showed hardly any signs of repair. The bone remained comparatively bare of granulations, and such granulations as did form were of the type characteristic of bone disease rather than of repair. To correct this condition, a secondary operation was performed on Feb. 17th. This operation, which was essentially a curettage of the original wound, showed extension of bone necrosis in various directions. Following this the wound took on a healthy appearance except at the aditus where pus continued to collect. During the month following, the patient rapidly gained in weight and strength, and was soon the picture of health. She now experiences absolutely no vertigo and there was no disturbance of equilibrium. There was no nystagmus. On March 22, the condition of the right ear was as follows; a large perforation was seen in the upper posterior quadrant of the membrane tensa. Into the mastoid wound pus still escaped through the unclosed aditus. Functional examination showed complete deafness of right ear. Caloric reaction absolutely negative. On March 24, a radical labyrinth operation was performed. No trace of the malleus, incus or stapes was found. The oval window was therefore opened. A fistula leading into the horizontal semicircular
canal was present. The labyrinth operation included opening of the vestibule, the lower wall of the horizontal canal being used as the surgical guide to its position, removal of the promontory and careful curettage of the cochlear space thus laid bare. The patient recovered with a dry tympano-labyrinth cavity, but, of course, with absolute unilateral deafness.
CONCLUSIONS.

In order to study labyrinthine suppurations rightly each case must be given the most careful observance.

The time most favorable to operate is during the latent stage of the suppuration.

A fistula once found should be traced down to its origin and every vestige of necrotic tissue surrounding it should be removed.

The characteristic rotary nystagmus to the sound side caused by labyrinthine suppuration does not tend to disappear in a few days but rather keeps up for weeks and in some cases months. Case VII on returning three months following the operation had a rotary nystagmus to the right and a slight quick horizontal nystagmus to the left.

With regard to Case III in which, with evidence of past suppurative labyrinthitis, a radical mastoid operation was only performed and then at a later date when the first signs meningeal irritation set in, the complete labyrinth operation was performed. From the study of this case and of others reported in the literature, where there is definite past evidences of labyrinthine suppuration, I am satisfied that the labyrinth operation should be performed at the same time as the radical mastoid is done.

As to the dangers of the labyrinth operation, I feel that when a case is taken during a latent stage and in such competent hands where not only a thorough knowledge of the anatomical features are known but also the mechanical results
to be attained, the labyrinth operation is not particularly dangerous. The labyrinth operation does not only mean good drainage but takes into consideration that all diseased membranes and bony tissue must be removed.

Attacks of dizziness without nystagmus are not characteristic of labyrinthitis and vomiting usually a symptom of the onset, continues fairly constant for a day or two and then finally disappears altogether.

It is to be hoped in all autopsies in the future where the labyrinth has been involved by an infective agent, that a thorough macroscopical and microscopical examination be made, in order that new light may be shed on this most important organ, the labyrinth.

I wish to thank Dr. L. W. Dean for the many helpful suggestions and valuable assistance rendered in the working out of this paper.

I am also indebted to Dr. H. J. Prentiss through whose kindness a microscopical study of the inner ear could be made.
BIBLIOGRAPHY.

1- Kerrison - "The Phenomena of Vestibular Irritation in Acute Suppurative Labyrinthitis, with special reference to the studies of Dr. Barany of Vienna." (Reprint).

2- Dan McKenzie - Archives of Otology, Vol. XXII.

3- Politzer - Labyrinthine Suppurations. Diseases of the ear.

4- Shambaugh - "Physiology of the Cochlea". Arc. of Otology, Rhinology and Laryngology, Vol. XIX.

5- Hinsburg - Suppurations of the Labyrinth. Archives of Otology, Vol. XXXI.

6- Richards - Surgery of the Labyrinth. Trans. of Amer. Laryn. Rhinol. and Otol. Soc. 1907.

7- Habermann - Uber Erkankungen des Felsenheils und des Ohrlabyrinthes bei den acuten Mittellahrentzundungen. Arch. f. Ohrenheilk., Bd. XIII, p.123.

8- Jansen - Surgical treatment of Infective Labyrinthitis after fifteen years experience. Trans. of Amer. Laryn. Rhin. and Otol. Soc. 1908.

9- Koener, Der eitrigen Erkrankungen des Schlafenbeins, p. 64.
10- Hinsberg- Zeitschrift f. Ohrenheilk. Vol. VIII.
11- Dunn, "Spontane Dehiscenzam oberen halbzurkelformigen Canal". Zeitschr. f. Ohrenheilk., Bd. XXVIII, p.139.
12- Jansen "Ueber Hirnsinusthrombose nach Mittelohreiterungen". Arch. f. Ohrenheilk., Bd. XXXVI, p. 1. Fall 35.
13- Krishke. "Ein Beitrag zur Lehre von den Todtlichen Ohrerkrankungen". Inaug. Dessert., Breslau, 1901.
14- Gorke, The Pathology of Inflammatory diseases of the Labyrinth. Journal of Laryn. Rhinol. and Otology, Vol. XXVI, 1911.
15- Healso,- Archives of Otology, Vol. XXXI, 1902
16- Kopetsky- The Present Status of Labyrinthine Surgery, Annals of Otol. Rhin. and Laryngology, Vol. XIX, No.4.
17- Deuch- The Symptomatology and Diagnosis of Meningitis of Otitic origin. A.M.A. Aug. 27, 1910.
18- Ballenger,- Diseases of ear, nose and throat.
19- Gerber- Archiv. fur Ohrenheilkunde, Band LX.
20- Bezold- Zeitschrift fur Ohrenheilkunde, Band XVI.
21- Blake- Purulent affections of the labyrinth consecutive to disease of the middle ear Pathology. (Reprint).
21- Cott- Infectious Labyrinthitis - A.M.A. July 31, 1909.

22- Kerrison- Vertigo of Vestibular Paralysis - Laryngoscope, Vol. XXI.

23- Reik- The Symptomatology and Diagnosis of Labyrinthitis, consecutive to Purulent Otitis Media. Trans. Am. Laryn. Rhinol. and Otol. Soc. 1907.

24- Milligan and Wingrave, Diseases of the Ear.

25- Ballenger, Indications and contra-indications for the labyrinth operation. Trans. Amer. Academy of Oph. and Otolaryngology, 1911.

26- Scheibe (Discussion) Verhand. Dtch. Otol. Gesell. 1909.

27- Ruttin- One hundred complete Labyrinth Operations, Arch. f. Ohrenh. 1 u, 2 Heft, Bd. 76, 1908.

28- Jansen- Surgical treatment of Infective Labyrinthitis after fifteen years experience. Trans. Amer. Laryn. Rhinol. and Otol. Soc. 1908.

29- Neumann; Arch. f. Ohrenheilk, Vol. LXVI,

30- Kerrison, Clinical Studies of Five Cases of Suppurative Labyrinthitis, Reprinted from the Am. of Otol. Rhinol. and Laryn. March 1911.

31- Guggenheim - The anatomic explanation of vestibular nystagmus. (Reprint).
Semicircular Canals of pigeon.

Enlarged diagram after Ewald.
Enlarged diagram after Ewald, showing the semicircular canals of a pigeon.

1- Horizontal canal.

2- Posterior vertical canal.

3- Anterior vertical canal.
Frontal section through the external auditory meatus tympanic cavity, mastoid antrum, labyrinth and internal auditory meatus of a normal adult temporal bone.

1- Prominence of the horizontal semicircular canal.
2- Anterior vertical semicircular canal.
3- Facial nerve.
4- Fenestra ovalis.
5- Promontory.
6- Cellulae tympanicae.
7- Cochlea, beginning.
8- Cysterna perilymphatica.
9- Utriculus.
10- Internal auditory meatus.
Illustrating the different forms of nystagmus.
Illustrating the different forms of nystagmus.

a- Horizontal nystagmus.
b- Oblique nystagmus.
c- Vertical nystagmus.
d- Rotary nystagmus.
e- Rotary nystagmus.
The Membranous Labyrinth.
The membranous labyrinth.

1- Ampullae.
2- Superior semicircular canal.
3- Posterior " "
4- External " "
5- Common canal.
6- Utricle
7- Saccule
8- Scala media of cochlea.
9- Canalis Reunieus.
10- Ductus Endolympathicus.
Diagram to show the entire central mechanism of vestibular nystagmus. V.A. represents the vestibular apparatus of the left side. The vestibular nerve fibers are first interrupted at V.G., the vestibular ganglion in the internal auditory meatus. From here they pass directly to the medulla, some ending in the small cell vestibular nucleus, V.N., others in Deiter’s nucleus D, and still others in the nucleus angularis A. From Deiter’s nucleus and the nucleus angularis pass fibers directly into the cerebellum C. From Deiter’s nucleus fibers pass also to the fasiculus longitudinalis posterior F.L.P. The fibers of the fasiculus longitudinalis posterior are seen to communicate with, first, the abducens nucleus Ab. and superiorly with the oculomotor nucleus OC, both of the right side. From these two nuclei of the right side the two nerve trunks are traced to the eyes. Although Deiter’s nucleus of each side is in communication with the abducens and oculomotor nuclei of both sides, the crossed fibers are the more important. For this reason the latter alone are given in the diagram. At G.A., in the cortical center for voluntary movement of the eyes to the left. This center in gyrus angularis, gives rise to the rapid component of vestibular nystagmus.

The diagram illustrates how vestibular nystagmus may be caused to the left from, for instance, an increase in the normal stimulation from the left vestibular apparatus. The increase in the normal stimulation passes from V.A.-V.G. directly to D. of the left side. From D. most of the stimulation passes through F.L.P. of the right side to the abducens and oculomotor
nuclei, Ab. and Oc. From these two nuclei the stimulus finally reaches the eye muscles through the abducens and oculomotor nerves. The result is a slow movement of the eyes to the right. As soon as the eyes have reached extreme vision to the right from vestibular action, they are jerked back toward the left by voluntary action originating in the gyrus angularis of the right cortex. The center for voluntary vision in the right gyrus angularis acts first upon the cells of Monakow (M in the diagram) of the left side (center for vision to the left). From here the stimulation passes through a supranuclear tract to the two nuclei Ab. and Oc. of the left, and thus finally to the eyes. The action then of the gyrus angularis is to cause the rapid component of vestibular nystagmus.
