# THE INNER EAR

INCLUDING

## OTONEUROLOGY, OTOSURGERY, AND PROBLEMS IN MODERN WARFARE

By

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# Authors' Prefaces

Through twenty years of postgraduate lecturing, I have constantly been asked the same question: "What book would you recommend for studies on the labyrinth and on otoneurologic problems?" I could never answer satisfactorily, because all that is available are either the ordinary textbooks that deal with the subject in a rather brief and elementary way, or the encyclopedias and handbooks of anatomy, physiology, neurology, etc., in which the respective chapters comprise large-scale dissertations delving into trifling details, so that the reader soon finds himself lost in the depths of science. One could also use the most important original articles on the labyrinth by authors of the various countries. However, this requires a certain knowledge of the subject, and would not give a satisfactory survey of the knowledge of the labyrinth.

Repeatedly I was asked by my students to write down my lectures on the labyrinth, which included instruction on anatomy, physiology, clinical practice, and operative technic on cadavers. I am fully aware of the great difference between the cold printed words of a book and the living lecture using anatomic specimens or patients, with demonstration of surgical procedures, and followed by a general discussion. Despite this, I have finally decided to bridge the gap between the usual textbooks and the voluminous encyclopedias and to write a treatise on the subject. Since the diseases of the middle ear are exhaustively discussed in the various textbooks, and since the function of the cochlear apparatus is generally known, the main attention is given in this book to the labyrinth and its central pathways.

Before I began on this difficult task, I scrutinized my registration book, which contains the signatures of all the students who have taken the various courses with me. I found two main groups of students. One group consisted of rather older otolaryngologists with great clinical experience and skill, who could not, however, keep pace with current progress in theory and research. The other group was represented by well educated young physicians with great theoretic knowledge (chiefly interns and residents) but without clinical experience.

This differentiation gave me the basis for the structure of my book. Almost every topic occurs twice—once in the chapters on physiology, where the various theories and hypotheses are discussed and references to the bibliographic material are brought out, and again in the chapter on functional tests, where indications, technic, clinical significance, and evaluation of the various tests are critically described In this manner each reader can find just what he is looking for without having to plow through pages of theory and literature.

In the discussion of the inflammatory diseases of the labyrinth, a new

principle of classification is used, as outlined by my former chief Alexander and myself. Particular stress has been placed on the pathologic anatomy of the various forms of labyrinthine disease; each is illustrated by photomicrographs. This emphasis stems from my conviction that correct diagnosis and proper treatment depend on an exact knowledge of the pathologic basis. The indications for surgical intervention, as used in our clinic, and the technics of the various operations have been outlined. In the complex matter of otoneurology, the important role played by the otologist in rendering diagnostic aid to the neurologist is exemplified in the differentiation between peripheral and central lesions.

The illustrations in the chapters on anatomy and physiology are, for didactic reasons, chiefly schematic and diagrammatic, while in the clinical sections photomicrographs are shown. The diagrams were made by Dr. Louis Bergmann, to whom I wish to express my appreciation for his artistic drawings. All macro and microphotographs used in this book are from specimens in my own collection.

JOSEPH FISCHER

As an otologist in active practice for more than twenty years, the various special problems of this field of medicine have been of particular interest to me. Every year or two I have managed to visit several of the special clinics either in this country or abroad. It has been especially interesting and instructive to see the various ways in which our present otologic problems are treated.

In this book, an effort has been made to review the important contributions in otology and also their relation to otoneurology. The subjects of Ménière's disease and otosclerosis are brought thoroughly up to date. In describing the intracranial complications, particular attention is given to the processes in the posterior cranial fossa, with special reference to their connection with the labyrinth. In the section on cerebellar abscess, the diversity of opinion between the otologist and neurosurgeon is critically discussed, and the interrelationship of their domains is shown in the discussion of the various diseases of the nervous system.

I wish to give grateful thanks to my many teachers and friends, many of whom are no longer with us: Neumann, Alexander, and Ruttin of Vienna; Jansen, Bruehl, and Passow of Berlin; Holmgren of Stockholm; Bárány of Upsala; Le Maître of Paris. And I wish to express my appreciation to Lempert in New York and Hughson in Abington, Pennsylvania, for their many kindnesses to me.

LOUIS E. WOLFSON

*Boston, February, 1943*

# I

# Clinical Anatomy

By *Joseph Fischer*

## 1. Nomenclature

The designation "clinical anatomy" is given to these studies to differentiate them from the descriptive treatment of anatomy found in most textbooks. The main attention is given to topographic relationships with respect to surgical procedures and to those anatomic details that have a clinical significance. Since it is often difficult to visualize anatomic relationships, illustrations are used chiefly in a schematic or half-schematic way to simplify matters, while for demonstration of pathology only photomicrographs are used.

The best way to get a proper conception of topographic relationships is to dissect petrous bones in various planes. Those who are interested in this subject are referred to the book about macroscopic and microscopic dissection of the ear by G. Alexander and J. Fischer.

In discussing the anatomy of the inner ear, I follow the nomenclature proposed by my former chief, Alexander. He divides the inner ear into cochlea and labyrinth. The latter consists of the vestibule and the semicircular canals. Hence the term "labyrinth" is applied to the noncochlear part of the inner ear. The nervus vestibularis is spoken of as nervus labyrinthicus. The same holds true for the ganglion vestibulare, which he calls ganglion labyrinthicum.

## 2. Topographic Relations within the Petrous Bone

If it were possible to visualize a transparent petrous bone, with the inner-ear spaces filled with stained fluids, three spaces would be outlined: (1) a perilabyrinthine space, between the surface of the petrous bone and the inner ear; (2) a perilymphatic space, between the bony and the membranous inner ear; (3) an endolymphatic space within the membranous inner ear (Fig. 1).

The *perilabyrinthine space* is not very definite and shows great anatomic variation. There may be a whole system of pneumatic cells extending from the mastoid, antrum, epitympanum, or eustachian tube down to the tip of the pyramid, or there may be very few cells or none at all—so that it cannot properly be called a space in the same sense as the perilymphatic and endolymphatic spaces, which are real, definitely outlined spaces filled

with fluid. The clinical importance of this region can best be seen in the fact that most of the anatomic studies in the various countries are made not by anatomists but by otologists.

Mouret and Portmann (France) often found on the tip of the pyramid large cells communicating with the perilabyrinthine space. They described two groups of cells in the apex: one, the external inferior group, originating from the eustachian tube; the other, the internal superior group, extending from the labyrinthine cells. A suppuration of the pyramidal tip can therefore come either directly from the eustachian

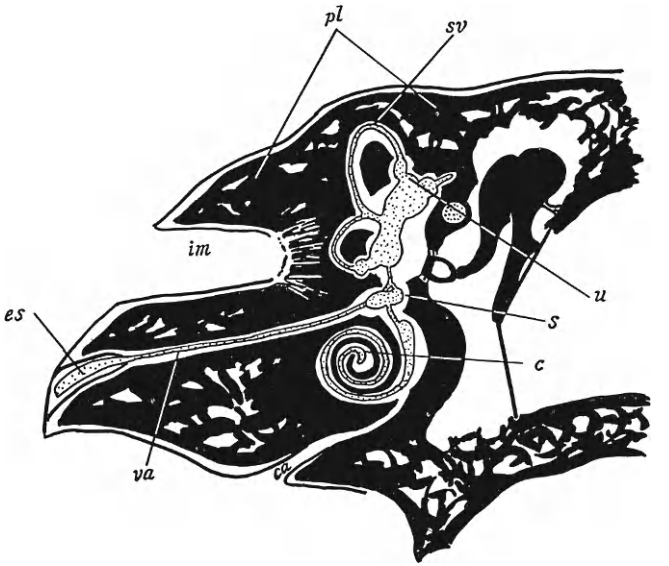


FIG. 1. SCHEMATIC ILLUSTRATION OF INNER EAR

black = endolymphatic space; white = perilymphatic space; *pl* = perilabyrinthine cells; *im* = internal auditory meatus; *es* = endolymphatic sac; *ca* = cochlear aqueduct; *va* = vestibular aqueduct; *s* = saccule; *u* = utricle; *c* = cochlea; *sv* = superior vertical canal.

tube or indirectly (over the perilabyrinthine cells) from the tympanoantral cells. Belinoff and Balan (Bulgaria) examined 40 petrous bones and found three different types of pyramidal structure, namely, the pneumatic, the diploetic, and the mixed type. In 62 per cent of these bones, the structure of the pyramidal tip did not correspond with the structure of the mastoid.

Myerson, Rubin, and Gilbert studied the anatomy of 200 petrous bones and found pneumatization of the apex in 11 per cent, while Hagens observed it in 34 per cent. Frenckner (Sweden) found in more than one-

fourth of his cases a pneumatized apex. He described the various cell passages, such as the posterosuperior, the inferior, and the tractus subarcuatus. The various cell systems joined and formed a common cell system within the pyramid.

J. R. Lindsay (Chicago) examined 100 petrous bones with the following results: pneumatization in the posterosuperior area (with cells from the epitympanum) in 36 per cent, pneumatization in the posteromedial area (with cells from the mastoid) in 25 per cent, pneumatization in the infralabyrinthine area (with cells from the mastoid to the jugular bulb or round window niche) in 25 per cent, pneumatization in the tip of the pyramid in 21 per cent.

The clinical importance of this region has been stressed by Eagleton, Friesner, Ramadier, Kopetzky, Brunner, etc. (p. 231).

The *perilymphatic space* lies between the bony and the membranous inner ear, and is filled with a fluid called the perilymph. Its communication outside the petrous pyramid is called the aquaeductus cochleae. It has two openings, one within the inner ear—apertura interna—in the scala tympani, or first turn of the cochlea, and the other—apertura externa—outside the pyramid on its inferior surface, as shown in FIGURE 1. There the duct debouches subdurally into the cerebrospinal spaces containing cerebrospinal fluid, and therefore communicates with the brain spaces. This anatomic fact is of great practical importance, since this is a pathway of infection from the inner ear to the cranial fossa in cases of meningitis and brain abscess. Conversely, pathologic conditions such as brain tumor, hydrocephalus, etc., which are accompanied by increased intracranial pressure, may likewise sooner or later produce changes in the inner ear (congestive inner ear or choked labyrinth), as I have shown in microscopic studies.

The *endolymphatic space* lies within the membranous inner ear and contains a fluid called the endolymph. Its communication is called the aquaeductus vestibuli. It has an internal aperture in the vestibule, where the canal begins from the ductus utriculosaccularis, and an external opening, located on the posterior surface of the petrous bone, halfway between the internal auditory meatus and the lateral sinus (FIG. 1). Studies on the anatomy of the endolymphatic duct and sac have been carried out recently by Guild, Anson, Bast, etc.

Bast described a valve that projects into the utricle and guards the utricular opening into the utriculo-endolymphatic duct. He believes that the valve prevents a sudden outflow of endolymph from the utricle. Perlman and Lindsay corroborate the presence of a utriculo-endolymphatic valve in man and in certain animals, which functions in maintaining the content and volume of the utricle and the semicircular canals.

The chemical composition of the peri- and endolymph fluids is the same, but anatomically they are separated, because the endolymphatic system is a closed system blindly intradural.

The dura in this region is very adherent and can easily be torn in the course of a labyrinth operation. It is therefore advisable to sever the

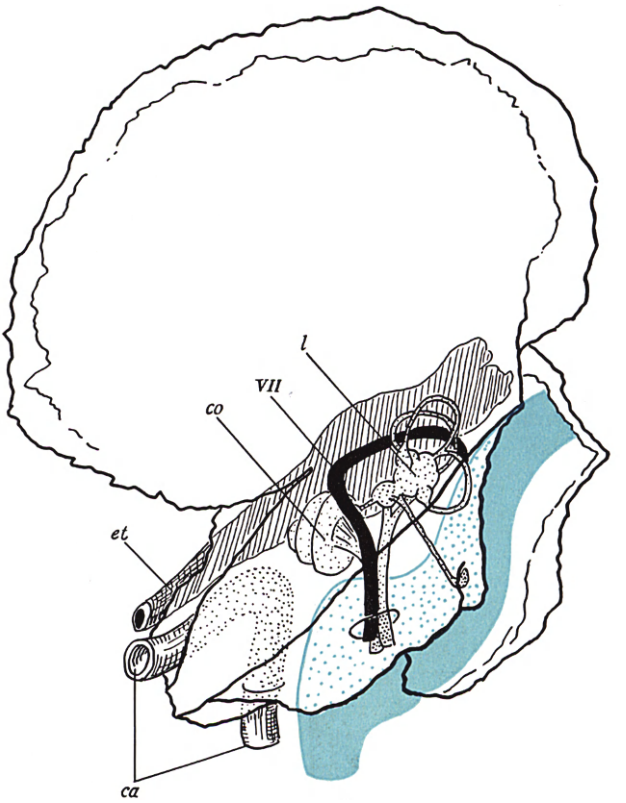


FIG. 2. DIAGRAM SHOWING TOPOGRAPHY OF INNER EAR

blue = sigmoid sinus; *ca* = carotic artery; *et* = eustachian tube; *VII* = facial nerve; *co* = cochlea; *l* = labyrinth.

attachment with a sharp instrument before chiseling on the posterior surface of the pyramid.

FIGURE 2 shows schematically the topographic relations of the various parts within an assumed transparent petrous bone.

## 3. THE BONY INNER EAR

The bony inner-ear structure consists of three parts, a central, an anterior, and a posterior part; these are, in order, the vestibule, the cochlea, and the semicircular canals.

The *vestibule* is oval-shaped. For purposes of demonstration, we shall assume a cube with six walls. The lateral wall constitutes also the medial wall of the middle ear, as seen in FIGURE 3, which shows the two windows—the fenestra ovalis, closed by the footplate of the stapes, and the fenestra rotunda, closed by the secondary membrane. Between these two windows

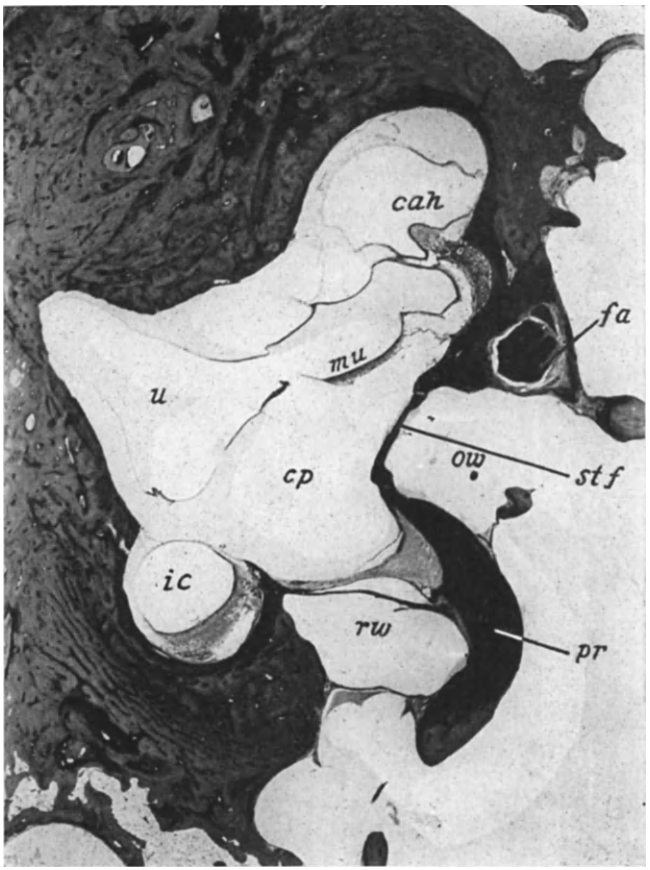


FIG. 3. VERTICAL SECTION THROUGH MIDDLE AND INNER EAR

*pr* = promontory; *rw* = round window; *ow* = oval window; *st f* = stapes footplate; *fa* = facial nerve; *cah* = crista ampullae horizontalis; *mu* = macula utriculi; *u* = utricle; *cp* = perilymphatic cistern; *ic* = inferior vertical canal.

is the promontory, which corresponds to the first turn of the cochlea. Above the oval window lies the fallopian canal, and above that, almost in the superior wall of the vestibule, the prominence of the horizontal semicircular canal is visible. The medial wall of the vestibule (FIG. 4) shows two depressions in the bone, one lying superoposteriorly, called the re-

cessus ellipticus, and the other in front and below, called the recessus sphericus. In the medial wall the mouth of the vestibular aqueduct and the openings for the nerves coming from the internal auditory meatus are seen. The latter are called the maculae cribrosae. On the anterior wall of the vestibule is the large opening for the cochlea, and on the posterior wall the opening for the semicircular canals can be seen.

The *cochlea*, which represents the anterior part of the middle ear, consists of two parts: one is called the vestibular portion, the other the cochlear

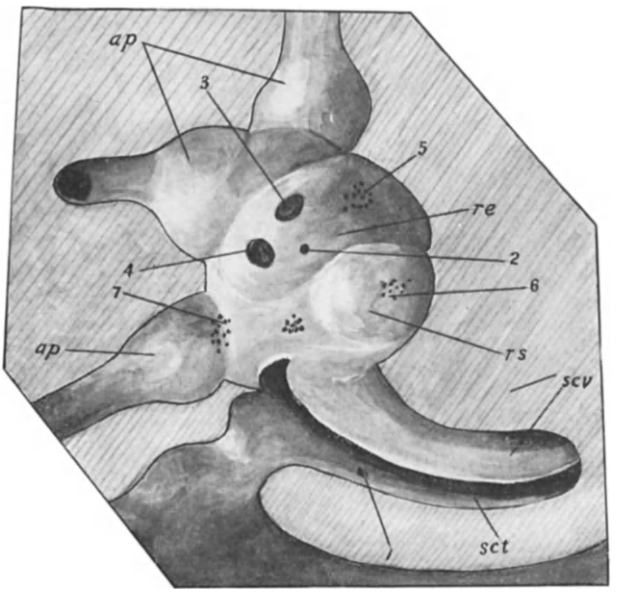


FIG. 4. DIAGRAMMATIC SECTION SHOWING MEDIAL WALL OF BONY LABYRINTH WITH TWO DEPRESSIONS

*re* = recessus ellipticus; *rs* = recessus sphericus; *sct* = scala tympani; *scv* = scala vestibuli; *ap* = ampullar ends of semicircular canals; *1* = cochlear aqueduct; *2* = vestibular aqueduct; *3* = horizontal canal; *4* = crus commune; *5* = macula cribrosa superior; *6* = macula cribrosa media; *7* = macula cribrosa inferior.

body. The latter shows two and a half turns, i.e., one basal whorl, one middle whorl, and one half whorl at the top. The blind end on top is called the cupula. The cochlea contains, on the inner bony wall, a bony septum following the same contour as the cochlea itself, and called the membrana spiralis ossea. This bony septum divides the space in the cochlea into two compartments. The lower compartment is called the scala tympani, and the upper the scala vestibuli. There is a large communication between the two scalae at the top of the cochlea, because of the absence of the bony membrane at this point; this passage is called the

helicotrema. This communication between the perilymphatic fluid in the two scalae plays a part in the physiology of hearing.

Topographically, (FIG. 2), the cochlea is located between the internal auditory meatus and the carotic canal in such a way that its base looks toward the internal canal, while its top is directed toward the middle ear in the region of the tensor tympani muscle. The first turn of the cochlea corresponds with the promontorium, while the top whorl is 5½ to 6 mm. distant from the anterior rim of the oval window, separated by a thin bony partition from the tympanic cavity. There the cochlea is in close contact with the carotic canal. In opening the cochlear spaces in the course of a labyrinth operation, these anatomic relations should be borne in mind. In the region of the tympanal ostium of the eustachian tube, a thin bony plate lies between the anterior wall of the tube and the ascending portion of the carotid artery. Great care must also be given to the region below the promontorium. On opening the cochlea in this area, injury to the jugular bulb may occur.

The three *semicircular canals* represent the posterior part of the inner ear. Each canal consists of a crus with two limbs (external and internal limb for the horizontal canals, ascending and descending limb for the vertical canals). There are, further, two endings on each canal: one is the enlarged or ampullar end, the other the smooth or sinus end. The diameter of the bony canals is 1.2 to 1.3 mm., while the lengths of the canals differ: the inferior vertical canal is the longest, the horizontal the shortest, the ratio being 6:5:4.

Their relationships correspond to the three main dimensions of space. There is much confusion in the nomenclature applied to them. The horizontal canal is frequently called horizontal, lateral, external, or medial; the upper vertical canal is frequently spoken of as frontal, superior, or anterior; the lower vertical canal is often called sagittal, inferior, or posterior. There is also a difference between the nomenclature used by the anatomists and that of the otologists. The anatomists refer to the upper vertical canal as sagittal, whereas the otologists call it frontal. Similarly, the anatomists differ in naming the lower vertical canal. The reason for calling attention to the differences in nomenclature is because in different countries the terms are differently applied, and this causes much confusion in the textbooks and literature. Further, the frontal canal is not really in the frontal plane, nor is the sagittal canal really sagittal. For this reason, we prefer the simple designations of horizontal, superior vertical, and inferior vertical for these canals.

In order to study the topographic relationships of the semicircular canals, the use of an infant skull, which offers easy dissection of the inner ear, is most practical. In the newborn the labyrinthine capsule can easily be

separated from the surrounding spongious bone of the pyramid. Such a skull shows best the normal and proper position of each canal. In such

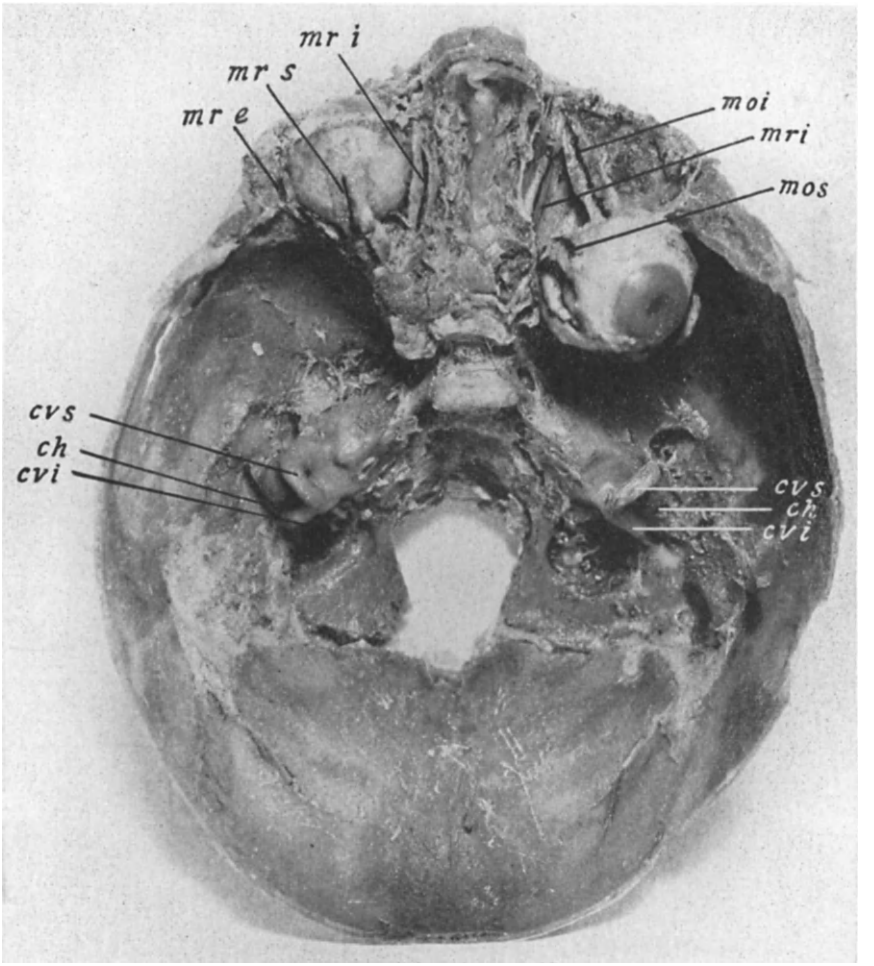


FIG. 5. PHOTOGRAPH OF BASE OF INFANT SKULL SHOWING EXPOSED SEMICIRCULAR CANALS AND EYE MUSCLES

For topographic studies, eye muscles are marked with black lines. Right eyeball is shown reflected. *ch* = canalis horizontalis; *cvs* = canalis verticalis superior; *cvi* = canalis verticalis inferior; *mr s* = musculus rectus superior; *mr i* = musculus rectus internus; *mr e* = musculus rectus externus; *mos* = musculus obliquus superior; *moi* = musculus obliquus inferior.

a specimen (FIG. 5), the inferior vertical canal forms a perfect 45° angle with the sagittal plane, with the open angle facing posteriorly; corre-

spondingly, the superior vertical canal makes a similar 45° angle with the sagittal plane, but the open angle faces anteriorly. In the normal erect position of the head, the horizontal canal deviates 30° from the horizontal plane, in the backward direction. Furthermore, other interesting observations may be made when the two sides are compared. The superior vertical canal on the right side will be found to be parallel with the inferior canal on the left side; and the superior vertical canal on the left side will be found to be parallel with the inferior canal on the right side. Both horizontal canals will be approximately in the same plane.

Such an infant skull can further be used to show the relationship of the semicircular canals to the eye muscles. According to anatomic-physiologic studies made in man by Ohm, the topographic relations are as follows:

1. When both eyeballs are elevated 30° above the horizontal plane, the two horizontal semicircular canals are parallel with the rectus muscles (external and internal) on each side (FIG. 5).

2. The superior vertical canal of the left ear and the inferior vertical canal of the right correspond with the sagittal rectus muscles (superior and inferior) of the left eye and with the oblique muscles (superior and inferior) of the right eye (FIG. 5).

3. The superior vertical canal of the right ear and the inferior vertical canal of the left correspond with the sagittal rectus muscles of the right eye and the oblique muscles of the left.

Exact knowledge of the topographic relations of the three canals is very important for operation in the labyrinth and will therefore be discussed in detail.

The *horizontal canal* has its ampulla just above the oval window. The external limb lies anteriorly in the antrum; the internal or deeper limb lies in the bony pyramid, with a course downward and backward. The arc between the external and the internal limb lies 5 mm. behind the upper rim of the oval window.

The *superior vertical canal* has its ampulla right in front of the horizontal ampulla. The ascending limb lies externally, and climbs straight up to the eminentia arcuata, while the descending limb is near the posterior surface of the petrous bone. The arc between the two limbs forms the eminentia arcuata, which is a thin bony plate (1 mm. thick) separating the middle cranial fossa.

The *inferior vertical canal* has its ampulla on the floor of the vestibule, near the jugular bulb. The ascending limb runs backward along the posterior surface of the pyramid, while the descending limb shows a course to the vestibule and in the direction of the jugular bulb. In labyrinth operations care must be taken not to injure the jugular bulb while attempting to open this canal.

The *internal auditory meatus* runs in a horizontal and frontal direction from the porus internus to the base of the cochlea. Its length is 10 mm., its width 4½ to 6 mm. The axis of the canal has, therefore, the same direction as the external auditory meatus. On X-ray pictures taken from a lateral view, the internal auditory meatus appears projected into the external canal as a small round opening within the big opening of the external canal. In the depth of the internal meatus, a transversal crest divides the canal into two parts, the upper giving passage for the facial nerve, the lower for the eighth nerve. Along with the nerves run the blood vessels (arteria and vena auditiva interna).

### 4. The Membranous Inner Ear

The membranous inner ear also shows three parts: the central part, comprising the utriculus and the sacculus; the anterior part, or cochlea; and the posterior part, comprising the semicircular canals (Fig. 6).

The *utriculus* lies in the posterosuperior part of the vestibule, in the recessus ellipticus; the *sacculus* lies in the infero-anterior position, in the recessus sphaericus. The two sacs communicate with each other by means of the ductus utriculosaccularis, which in its continuation forms the ductus endolymphaticus, as shown in Figure 1. Between the outer surfaces of the two sacs and the lateral wall of the vestibule there is a large space called the perilymphatic cistern (Figs. 1, 3). This space is of special importance because it is most frequently the part that first shows the spread of infection from the middle ear to the labyrinth.

The *membranous cochlea*, which forms the anterior part of the membranous inner ear, lies in the bony cochlea and adds a third scala media to the two already noted, the scala tympani and the scala vestibuli. The scala media contains endolymph and the organ of Corti. The communication between the cochlea and the sacculus is called the ductus reuniens.

The three *semicircular canals* represent the posterior part of the membranous inner ear. The ampulla of the inferior vertical canal is near the floor of the utriculus. Just beneath the enlarged end of the superior vertical canal is the ampulla of the horizontal canal, close to the medial wall of the antrum (Fig. 4).

The membranous inner ear is of the greatest importance because it is the bearer of the nerve endings that are the true sensory organs (sensulae). There are six such parts within the membranous inner ear: two within the sacs (the macula utriculi and the macula sacculi); three within the semicircular canals (the crista ampullaris horizontalis, the crista ampullaris verticalis inferior, and the crista ampullaris verticalis superior); and, finally, one within the cochlea (the organ of Corti or papilla basilaris).

The finer anatomic structure of these parts will be described in our discussion of the nervous apparatus.

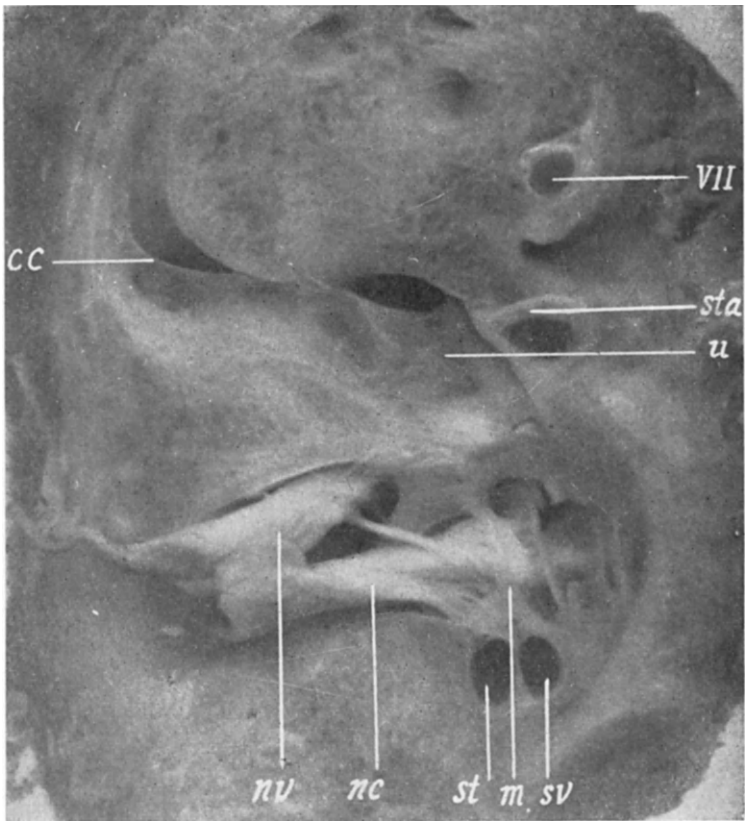


FIG. 6. ENLARGED PHOTOGRAPH OF SECTION THROUGH MEMBRANOUS INNER EAR SHOWING COCHLEA, UTRICLE, AND SEMICIRCULAR CANALS

*nc* = nervus cochlearis; *nv* = nervus vestibularis labyrinthicus; *sv* = scala vestibuli; *st* = scala tympani; *m* = modiolus; *sta* = stapes; *VII* = facial nerve; *u* = utricle; *cc* = crus commune of vertical semicircular canals.

## 5. THE NERVOUS APPARATUS OF THE INNER EAR

### THE FACIAL NERVE

The nerve enters the upper compartment of the internal auditory meatus and runs in the fallopian canal between the second turn of the cochlea and the ampulla of the superior vertical canal to the hiatus spurius, where the ganglion geniculi is located (FIG. 2). Two-thirds of the nerve fibers enter

the ganglion, while one-third pass close by. From there the nerve runs in an angle of 100° downward and backward (upper knee of the facial) between the horizontal semicircular canal and the oval window. The distance between the nerve and the upper rim of the oval window is 3 mm. In the antrum there is another sharp turn downward (lower knee) where the nerve runs behind the posterior wall of the external canal to the stylomastoid foramen.

The most important branches are the nervus petrosus superficialis major (coming from the geniculate ganglion), the nervus stapedius, and the chorda tympani.

### THE COCHLEAR NERVE

Since this book deals chiefly with the physiology, functional tests, and pathology of the labyrinthine diseases, the cochlear nerve will be discussed only briefly. For further details on this subject the reader is referred to the various textbooks.

The cochlear nerve lies in the internal auditory meatus in the anterior part of the lower compartment. Its ganglion (ganglion cochleare) consists of bipolar cells with a peripheral and a central processus. The former represents the nerve fibers coming from the organ of Corti through the bony spiral membrane and the modiolus. The central processus constitutes the nerve fibers that pass through the fine openings in the ground of the internal meatus (tractus spiralis foraminosus) and that form the nerve stem. The further course of the cochlear nerve is in a frontal direction from the internal auditory canal to the medulla oblongata.

The central pathways run caudally and externally from the restiform body to the tuberculum acusticum, to the superior olive, to the internal lemniscus, to the medial geniculate body, and finally to the centers of the temporal lobe.

### THE LABYRINTHINE NERVE AND ITS PERIPHERAL TERMINATION

The peripheral labyrinthine portion of the eighth nerve comprises the nerve endings in the membranous inner ear, the labyrinth nerve (vestibular nerve), and the labyrinth ganglion (ganglion of Scarpa)—in other words, the region of the first sensory neuron.

Within the internal auditory meatus the nervus labyrinthicus forms a superior and an inferior part. The nerve itself, approximately 12 to 15 mm. long, splits into two branches, called the nervus utriculo-ampullaris and the nervus sacculo-ampullaris. The former goes to the utriculus and to the ampullae of the superior vertical canal and of the horizontal canal, and the latter to the sacculus and ampulla of the inferior vertical canal (FIG. 7). Hardy describes small bundles of nerve fibers coming from the cochlear

ganglion and extending to the posterior tip of the macula sacculi. Since he considers this nerve bundle as a true branch of the cochlear nerve, he calls it nervus cochleosaccularis. Where the nerve terminates one finds the structure of a true sense organ.

The cristae ampullaris represent, as already stated, the nerve endings in the semicircular canals. The term crista refers to the prominent ridge in the transverse portion of the ampullated end that bulges into the endolymphatic lumen (FIG. 3). Histologically, there are three layers to be

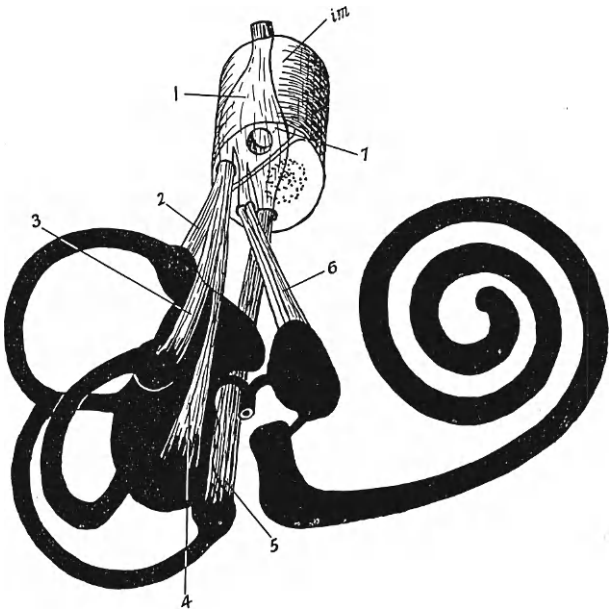


FIG. 7. HALF-SCHEMATIC ILLUSTRATION OF MEMBRANOUS INNER EAR WITH ITS NERVOUS APPARATUS

*im* = internal auditory meatus as transparent cylinder; *1* = nervus labyrinthicus; *2* = nervus ampullaris superior; *3* = nervus ampullaris lateralis; *4* = nervus utricularis; *5* = nervus ampullaris inferior; *6* = nervus saccularis; *7* = ganglion labyrinthicum (Scarpae).

noted: (1) a nerve layer, composed of myelin-sheathed nerve fibers (nervus ampullaris); (2) a cellular layer of two-typed cells, composed of hair or sensory cells and supporting cells—the hair cells are cylindric or bottle-shaped, and contain a round nucleus near the base of the cell; (3) a homogeneous gelatinous mass layer containing parallel canals in which the hairs of the sensory cells are imbedded, and called the cupula.

The histologic structure of the maculae (macula utriculi and macula sacculi) is the same as that of the cristae, but the macroscopic shape differs.

The maculae form a flat concave elevation (FIG. 3), but contain the same three layers—a nerve layer (nervus utriculi and nervus sacculi); a cell layer of hair or sensory cells and supporting cells, the hairs of the sensory cells being much shorter; and a third layer composed of a homogeneous gelatinous mass layer containing numerous chalk crystals (aragonite). This layer was formerly called the otolithic membrane, but is now called, according to its function, the statolithic membrane. The structure of the papilla basilaris (organ of Corti) will be discussed here only to the extent of directing attention to the anatomic fact that although this is the cochlear sense organ, its structure is essentially the same histologically as that of the maculae and cristae, because it contains a nerve layer (nervus cochlearis), a cell layer (three external and one internal hair cell), and also a homogeneous gelatinous mass layer, called the tectorial membrane. The reason for discussing the histologic structure of the nerve endings is that of their importance in the physiology and pathology of the labyrinth. For a thorough study of this important subject, we refer to the original work of Alexander, Gray, Held, Kolmer, Retzius, Shambaugh, Wittmaack, etc.

### THE CENTRAL LABYRINTHINE APPARATUS

The study of the central pathways is very difficult, particularly for a non-neurologist. To simplify this complex matter, the most important pathways will be discussed briefly with the aid of a schematic picture. For more detailed discussion we refer to the original articles of Ramón y Cajal, Gray, Gehuchten, Jones, Leidler, Marburg, Muskens, Lorente de Nó, Ranson, Spiegel, Spitzer, etc.

The central apparatus comprises the area of the *labyrinth nuclei* in the medulla oblongata and the *secondary central pathways* from the nuclear region (FIG. 8).

In the area of the *labyrinth nuclei* five different portions must be distinguished:

1. *Nucleus magnocellularis* (Deiters) (FIG. 8). This lies in the lateral portion of the medulla, inward of the restiform body, and consists of exceptionally large cells analogous in structure to the motor ganglion cells. Some authors divide this structure into three portions, to which they give definite names. These are hypothetic and will not be discussed here.

2. *Nucleus parvicellularis* (Schwalbe) (FIG. 8). This nucleus lies on the floor of the rhomboid fossa, medially, and is called the dorsomedial main nucleus.

3. *Nucleus angularis* (Bechterew) (FIG. 8). This nucleus lies in the angle of the fourth ventricle at the anterior end of the spinal acoustic root, where the large cells of Deiters' nucleus fade away.

4. *Nucleus radicis descendentis* (Roller). This consists of cells in which the descending fibers of the vestibular root terminate.

5. *Nuclei in the cerebellum* (particularly in its flocculonodular part). According to some authors, there is a direct pathway from the labyrinth ganglion through the restiform body into the deep nuclei of the cerebellum.

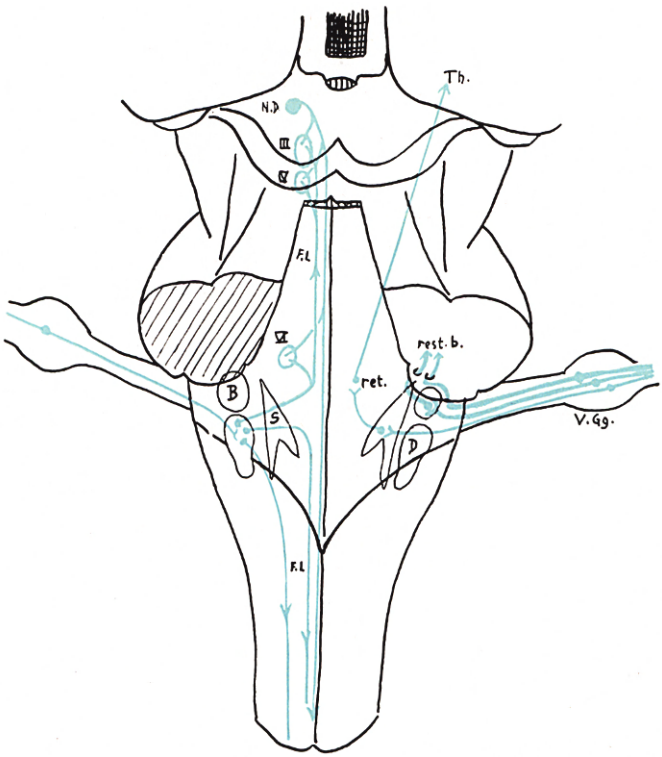


FIG. 8. DIAGRAM OF BRAIN STEM WITH CENTRAL LABYRINTHINE PATHWAYS

V.Gg. = vestibular ganglion; B = nucleus angularis of Bechterew; D = nucleus of Deiters; S = nucleus of Schwalbe; N.D. = nucleus of Darkshevich; Th. = thalamus; III = oculomotor nerve; IV = trochlear nerve; VI = abducent nerve; ret. = reticulate substance; rest.b. = restiform body; F.L. = fasciculus longitudinalis posterior. All connections run ipsi- and contralaterally except those through restiform body to cerebellum (thick lines).

The *secondary central pathways* originate in the region of the nuclei. For purposes of demonstration only the important pathways are here discussed:

1. *Pathways arising from the nucleus of Deiters.* These form a large portion of the posterior longitudinal bundle of the same and of the opposite side (FIG. 8), dividing into an ascending and a descending part; the former

serve as connections with the eye nuclei and other motor nuclei of the medulla, the latter form partly the vestibulocervical tract, partly the vestibulospinal tract.

2. *Pathways arising from the nucleus of Schwalbe.* The fibers of these run to the vegetative centers in the reticular substance and from there to the thalamus (FIG. 8).

3. *Pathways arising chiefly from the nucleus of Bechterew.* These form one portion of the vestibulocerebellar tracts going through the restiform body (FIG. 8). However, there are some other connections with the cerebellum.

From a clinical standpoint the most important role is played by the posterior longitudinal bundle. This consists of various parts (FIG. 8):

*a*) Fibers that run from the posterior commissure (nucleus of Darkshevich) close to the midline just underneath the central gray matter, down to the lowest cervical cord. This bundle sends connections to the motor nuclei along its course.

*b*) Ascending fibers arising from the labyrinth nuclei, going orad, and connecting the eye nuclei and other motor nuclei. The collaterals, sent also to the opposite side, are omitted in FIGURE 8.

*c*) Descending fibers, arising from the labyrinth nuclei and going caudally as far as the lower cervical cord, which connect the motor nuclei along their course.

Besides these pathways, many other tracts are assumed by the various authors on the basis of clinical symptoms or experimental examination, for which anatomic proof is still lacking.

Jones and Fisher, and some others, for instance, believe that fibers conveying impulses producing vertigo go through the cerebellum en route to the cerebrum, while fibers conveying impulses to the eyes run separate from these. They further believe that the fibers from the horizontal semicircular canal have a course distinct and separate from the course of the fibers from the vertical canals.

The central division of the labyrinthine nerve may be divided—according to Lorente de Nó—into five groups of fibers. The first and second groups contain fibers from the semicircular canals. Whether the third group contains fibers from the central parts of the cristae or from the region of the utricular macula has not been determined. The fourth group contains fibers from the utricular macula, and the fifth group has fibers from the saccular macula. This author therefore believes that the semicircular canals and the maculae have partly different and partly common central representations.

Aronson, Spiegel, and Alexander, on the basis of their experiments with

strychnine, came to the conclusion that there is a vestibular center in the cortex of the temporal lobe and probably also in the operculum. Each labyrinth is connected with the contralateral as well as with the homolateral portion of the cortex. The labyrinthine impulses travel, according to these authors, through the rubrothalamofrontal tract. However, there is no anatomic proof as yet that direct pathways from the labyrinth to the cerebrum really exist.

French authors, as Delmas-Marsalet, Bourguignon, and Verger, etc., assume direct connections between the vestibular apparatus and the striated body, because of the resemblance of certain clinical symptoms, such as compulsory involuntary movements and *manège* movements. They describe tracts from the vestibular nuclei to the lenticulate and caudate nuclei and from there to the cortex of the frontal lobe.

### 6. Blood Supply of the Inner Ear

Our knowledge of the blood supply of the inner ear must be attributed to the thorough work of Siebenmann, Shambaugh, and Schwalbe.

The arteries of the labyrinth arise from the basilar artery, which sends a branch, called the internal auditory artery, into the ear organ. As it enters through the internal auditory meatus, accompanying the eighth nerve, the vessel divides into three branches: (1) the vestibular artery; (2) the vestibulocochlear artery; (3) the cochlear artery. The vestibular artery supplies the anterosuperior half of the utriculus and the sacculus. The vestibulocochlear artery supplies the sacculus, the ampulla of the inferovertical semicircular canal, and the first turn of the cochlea. The cochlear artery supplies the entire cochlea.

The venous return has three paths of drainage: (1) the vestibular vein in the aquaeductus vestibuli, which drains the superior petrous sinus; (2) the vena auditoria interna, draining into the inferior petrous sinus; (3) the cochlear vein, draining into the jugular bulb.

Knowledge of the blood supply is especially important because by means of it many lesions can be explained with comparative ease. The internal auditory artery is a branch of the basilar artery, forming the circle of Willis. It is largely without branches and divides into end arteries. On this account, venous stasis and compression of the blood vessels of the inner ear are easily possible. Also, every disturbance in the blood circulation of the inner ear leads to changes in pressure, because the capillaries not only serve to nourish the tissue, but also secrete the endolymphatic fluid. This latter secretion is produced in the striae vasculares of the cochlea, so that the slightest disturbance in the vessels is quickly noticeable in the inner ear. It is well recognized that vasomotor changes in the labyrinth cause severe disturbances, such as are noted in Ménierè's symp-

tom complex, arteriosclerosis, hypertension, and other conditions. Ruskin, studying venous circulation of the petrous bone, came to the conclusion that the venous pathways of the temporal bone play a leading role in the dissemination of infections.

### 7. The Dura Mater and the Venous Sinuses

The dura consists of two lamellae—an outer, serving as a periost of the petrous bone, and an inner, covering the brain. Both layers show processes on certain parts; the outer layer covers the nerves on their way into the bone—for example, the eighth nerve in the internal meatus or the ninth in the cochlear aqueduct; the inner layer forms processes that subdivide the cranial cavity into various compartments. There are two sagittal processes (falx cerebri and falx cerebelli) and two transverse processes (diaphragma sellae and tentorium cerebelli).

The *falx cerebri* (Fig. 9) extends from the crista galli in front to the internal occipital protuberance. It has a convex superior and a concave inferior margin. Between the two layers of this dural process there is a space called the sinus sagittalis.

The *falx cerebelli* (Fig. 9) is the continuation of the cerebral falx in a backward and downward direction. Between the two layers the occipital sinus is imbedded.

The *diaphragma sellae* forms a bridge over the sella turcica and constitutes the free (inner) wall of the sinus cavernosus.

The *tentorium cerebelli* (Fig. 10) represents a transverse partition between the basal surface of the occipital region of the brain and the dorsal surface of the cerebellum. It therefore has two surfaces, one dorsal or cerebral, the other ventral or cerebellar. The tentorium is kept tense by the falx, thus preventing the cerebrum from exerting a pressure upon the underlying cerebellum. Between its two layers the transversal sinus lies posteriorly and the superior petrosal sinus laterally. Clinically, lesions of the brain are classified as supratentorial (cerebrum) and infratentorial (cerebellum and brain stem).

The *venous sinuses* that collect the blood from the cranial cavity form a communicating system of venous spaces. Their walls are fixed and independent of any pressure from the outside and have no valves. The blood is chiefly drained through the jugular foramen into the jugular bulb on each side. These sinuses comprise the following:

The *sinus sagittalis superior and inferior* (Fig. 9), the former running along the convex margin of the cerebral falx, i.e., from the foramen caecum to the occipital protuberance, the latter running along the concave margin.

The *sinus transversus* (Fig. 10), running along the posterior margin of the tentorium to the posterior margin of the pyramid. On the occipital protuberance the transverse sinus meets the sagittal and the *rectus* sinus

(confluens sinuum or torcular Herophili). This junction shows great anatomic variations.

Woodhall distinguishes four main types of the venous pattern in the region of the torcular: the common pool type, in which the sagittal and the rectus sinus meet in a common pool from which the blood flows to the lateral sinus; the plexiform type, in which the sagittal and rectus sinuses are divided into parts and the lateral sinuses are usually dissimilar; the ipsilateral type, in which the sagittal sinus runs to one side while the rectus goes to the other side; and, finally, the unilateral type, in which the sagittal and the rectus empty into one lateral sinus.

The *sinus sigmoideus* is a continuation of the transverse sinus, which curves downward and inward in order to reach the jugular foramen. The sinus is imbedded in a bony depression (sulcus sigmoideus), which often shows great differences as between the two sides. Koerner found equal anatomic conditions of the two sides in only 10 per cent of cases, while in 77 per cent the right sulcus was larger and extended deeper into the petrous bone than did the left sulcus; in 13 per cent it was vice versa. From a clinical standpoint, this shows that in cases of otitis media with mastoid symptoms the right sinus is more in danger of being infected than the left one. Other anatomic variations concern the position of the sinus within the mastoid and that of the jugular bulb. An anterior position of the sinus is often combined with lowered level of the middle fossa, thus increasing the technical difficulties during surgical procedures. Detailed studies on this subject will be found in the original articles.

The *sinus occipitalis* runs from the cerebellar falx to the occipital foramen.

The *sinus petrosus superior* (FIG. 10) lies at the attachment of the tentorium on the superior margin of the pyramid and connects the cavernous sinus with the sigmoid sinus. It therefore drains the blood from the sinus cavernosus to the jugular vein.

The *sinus petrosus inferior* (FIG. 10) runs along the inferior margin of the pyramid from the cavernous sinus in a backward, downward, and lateral direction to the jugular foramen (medial compartment) and debouches into the jugular vein.

The *sinus cavernosus* (FIG. 10), on each side of the sphenoid body, extends anteriorly to the superior orbital fissure and posteriorly to the tip of the pyramid. The two sides are connected by the *intercavernous* sinuses. The sinus communicates with a number of venous spaces—for example, with the *sphenoparietal* sinus and the eye veins in front, and with the *petrosal* sinuses in back. Through the cavernous sinus pass the abducent nerve and the carotid artery, while the sinus wall contains the oculomotor, the trochlear, and the first and second branches of the trigeminal nerve.

The close connections of the sinus sigmoideus with the sinus cavernosus

can easily lead to a phlebitis of the cavernous sinus. Although such complications seem to be rare, there is no doubt that infections exist more

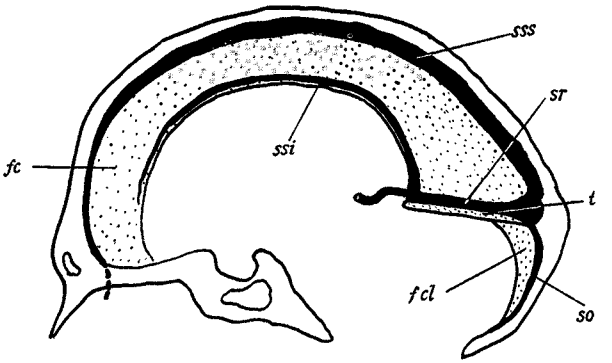


FIG. 9. SAGITTAL SECTION THROUGH SKULL SHOWING PROCESSES OF DURA MATER AND VENOUS SINUSES

*fc* = falx cerebri; *f cl* = falx cerebelli; *t* = tentorium cerebelli; *sss* = sinus sagittalis superior; *ssi* = sinus sagittalis inferior; *sr* = sinus rectus; *so* = sinus occipitalis.

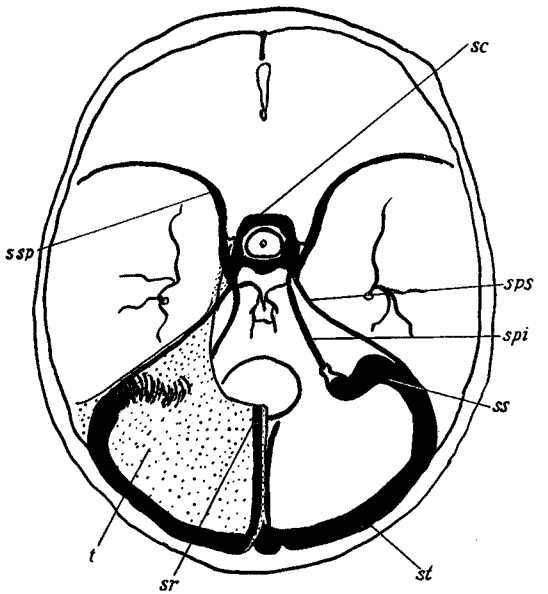


FIG. 10. DIAGRAM SHOWING CRANIAL FOSSAE WITH VENOUS SINUSES

*st* = sinus transversus; *ss* = sinus sigmoideus; *sps* = sinus petrosus superior; *spi* = sinus petrosus inferior; *sr* = sinus rectus; *sc* = sinus cavernosus; *s sp* = sinus sphenoparietalis; *t* = tentorium cerebelli.

often than is generally revealed in diagnosis. In all the cases in which septic symptoms occur in association with orbital and frontal headaches

and with a negative finding in the other sinuses, a disease of the cavernous sinus is present.

### 8. Communications of the Inner Ear with the Cranial Fossae

The knowledge of such communications is of great clinical importance. It shows on the one hand the various pathways of infection from the inner ear to the skull, and explains on the other the various changes in the inner ear in cases of brain lesions, particularly those associated with increased endocranial pressure.

One such communication is represented by the aquaeductus cochleae, which has partly been discussed above (p. 3). In spite of numerous experimental examinations for many years, the anatomy of the cochlear duct is still under dispute. Schwalbe injected methylene blue under constant pressure into the subarachnoid spaces and found the dye in the perilymphatic spaces of the labyrinth. Quincke found a communication between the subarachnoid spaces and the scala tympani. According to Hasse, the aqueduct plays a minor role in the flow of perilymph. Rejtoe denied the idea of a communication between the cerebrospinal and the perilymphatic fluid. He believed that the perilymphatic fluid is a product of the endolymphatic by osmosis. According to Karlefors, the flow of fluid is directed from the brain spaces toward the labyrinth.

Jampolsky called the perilymphatic space a system of sinuses of the subarachnoid space, functioning as outlets for drainage. Perlman and Lindsay assume that the probable flow of cerebrospinal fluid is from the subarachnoid space into the scala tympani. Ross and Hamilton injected mercurochrome into the middle ear in dogs and examined the petrous bones. The path of invasion of the dye was determined by the intensity of staining of the tissues.

In order to study the various communications between the inner ear and the endocranium, I made microscopic examinations on a large number of specimens, which can be summarized as follows. The eighth nerve does not completely fill the internal auditory canal; there are numerous perineural and perivascular spaces, which are especially large in the fundus of the internal meatus and in the region of the root of the lamina spiralis ossea. The wall separating the spaces of the scalae is thin and is formed in part by partitions of connective tissue, so that here also one must take into consideration a communication between the cerebrospinal fluid and the perilymphatic fluid of the inner ear. There is, furthermore, a connection between the ligamentum spirale and the lymph spaces of the modiolus by means of perivascular channels. Therefore, the following pathways communicate between the cerebrospinal fluid and the spaces of the inner ear: the aquaeductus cochleae, the internal auditory meatus, the peri-

neural and the perivascular spaces, the lymph channels of the bony labyrinthine capsule, the spiral ligament, and the pacchionian bodies.

### 9. Topographic Relation of the Inner Ear to the Brain

For the study of this topographic relationship, sections through the bony skull must be made in various planes. Special care must be taken

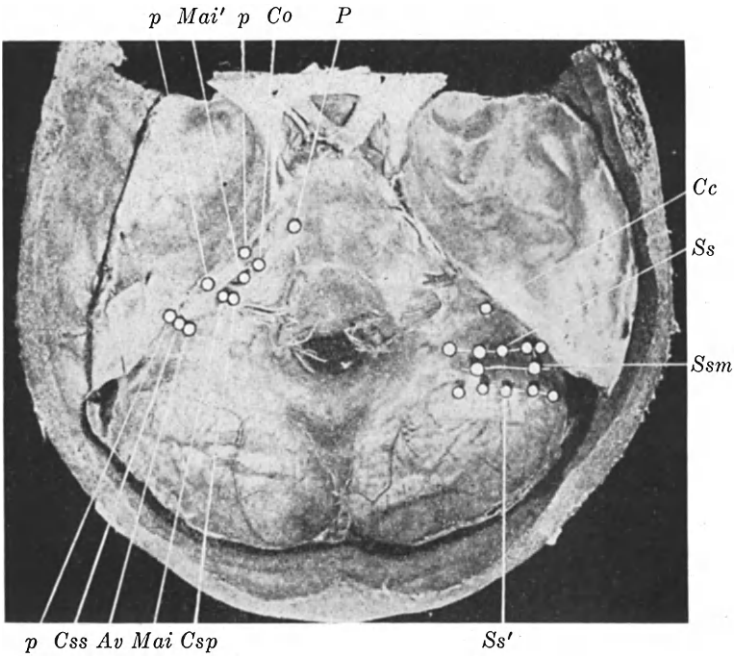


Fig. 11. Base of Skull with Markings of Various Parts of Inner Ear (*G. Alexander and J. Fischer*)

*Av* = aquaeductus vestibuli; *Cc* = crus commune; *Co* = anterior cochlea; *Csp* = canalis semicircularis posterior; *Css* = canalis semicircularis superior; *Mai* = meatus auditorius internus; *Mai'* = meatus auditorius internus (anterior margin); *p* = pyramid (superior margin); *P* = pyramid (tip); *Ss* = sinus sigmoideus (anterior border); *Ss'* = sinus sigmoideus (posterior border); *Ssm* = sinus sigmoideus (longitudinal axis).

to avoid any tugging or tearing during dissection, in order not to disturb the normal structures. In a book about the technic of macroscopic and microscopic dissection, G. Alexander and I described a new method for such purposes. The base of the skull, with the cerebellum left *in situ*, is taken out of the cadaver and put into 10 per cent formalin solution. After thorough fixation, the specimen must be decalcified in 5 per cent nitric acid, over a period of four to five weeks (daily change of solution).

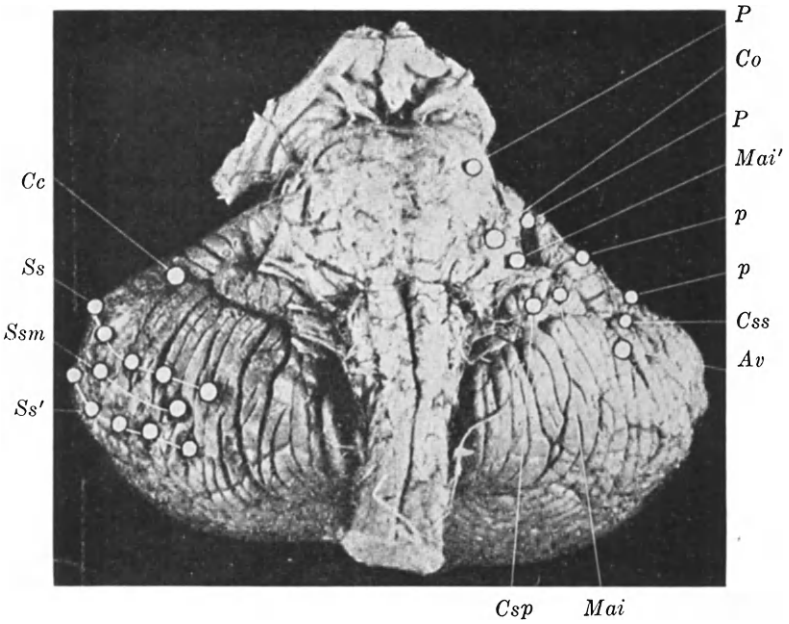


FIG. 12. ANTERIOR SURFACE OF BRAIN STEM AND CEREBELLUM WITH MARKINGS AT TOPOGRAPHIC GUIDE POINTS (*G. Alexander and J. Fischer*)

Key as for FIG. 11.

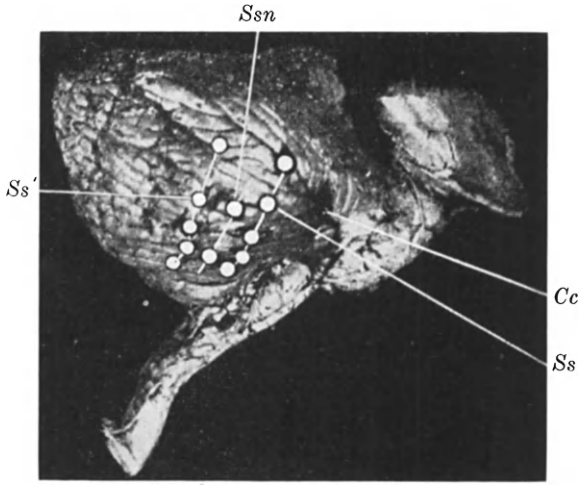


FIG. 13. BRAIN STEM AND CEREBELLUM, RIGHT LATERAL VIEW WITH MARKINGS AT TOPOGRAPHIC GUIDE POINTS

Key as for FIG. 11.

In this manner the bone is so softened that sections can be made with a knife instead of a saw. In order to remove the cerebellum, the tentorium must be incised and the cranial nerve severed. Pins are placed in the bone at the various topographically important points (FIG. 11). The heads of the pins are then painted with India ink and the cerebellum is returned to its former position. Shortly thereafter the cerebellum is removed, showing black points where there was contact with the painted pinheads. On these black points pins are now inserted into the cerebellum (FIGS. 12, 13, 14).

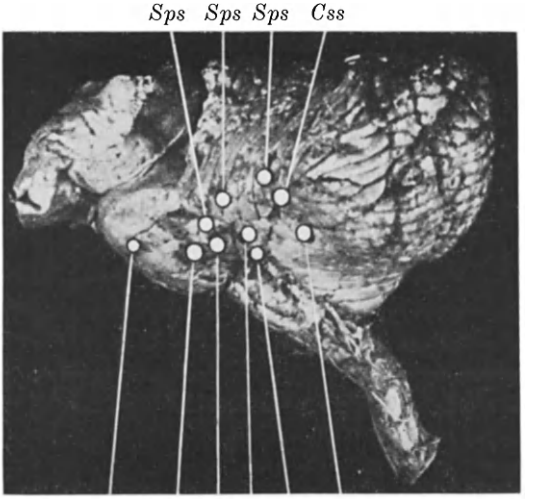


FIG. 14. BRAIN STEM AND CEREBELLUM, LEFT LATERAL VIEW, WITH MARKINGS AT TOPOGRAPHIC GUIDE POINTS

*Sps* = sinus petrosus superior. Key to other symbols as for FIG. 11.

## RELATION TO THE CEREBELLUM

Starting medially at the tip of the pyramid and continuing the dissections laterally to the mastoid, the relations of the various parts are as follows:

The *tip of the pyramid* (*P*, FIG. 11) faces the medial and anterior part of the bracchium pontis (*P*, FIGS. 12, 14).

The anterior pole of the *cochlea* (*Co*, FIG. 11) corresponds with the posterior part of the bracchium pontis (*Co*, FIGS. 12, 14) just behind the *root of the trigeminus.*

The *internal auditory meatus* (*Mai*, *Mai'*, FIG. 11) lies in the region of the *flocculus* near the *quadrangular lobe* (*Mai'*, *Mai'*, FIGS. 12, 14).

The *saccus endolymphaticus* (*Av*, FIG. 11) corresponds with the *lobulus biventer* (*Av*, FIGS. 12, 14).

The *commissure of the vertical semicircular canals* (*Css*, FIG. 11) has the level of the *sulcus horizontalis cerebelli* of the biventer lobe (*Css*, FIGS. 12, 14).

### RELATION TO THE CEREBRUM

The vestibule lies in the region of the *fusiform gyrus*.

The ampulla of the *superior vertical canal* corresponds with the lateral portion of the *occipitotemporal gyrus*.

The *geniculate ganglion* of the facial nerve is near the *third temporal gyrus*.

The *tegmen tympani* is in contact with the lateral part of the *fusiform gyrus* and the medial rim of the *third temporal gyrus*.

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# II

# General Physiology

By *Joseph Fischer*

## 1. Comparative Physiology

The physiology of the labyrinth in its simplest form is found in the lowest animals. With the phylogenetic ascent, its physiologic functions become more complicated by the addition of optic and perceptory organs. In man these functions are masked by the development of the central nervous system, hence the labyrinth seems to play a minor role. Some authors (Bárány), therefore, consider it a rudimentary organ, an opinion that lacks any substantial proof.

Some reasons why the importance of the apparatus of balance and the influence of tonus increase with the phylogenetic descent can be seen in the large size of these organs in relation to the body size and in the greater number of nerve endings (lagena, macula neglecta, crista neglecta). Even the plants have organs that orient them in the vertical direction by growing straight upward (Haberland). It is a known fact that destruction of the labyrinth in lower animals means annihilation of life. In man, however, destruction of the labyrinth is soon overcome by compensation of other mechanisms. For these reasons the physiology of the labyrinth is best studied in the lower animals.

The simplest sensory apparatus, as seen in the avertebrates, consists of a groovelike depression of the ectoderm (Chun, Hertwig, Buddenbrock). With phylogenetic ascent this groove becomes deeper and soon forms a vesicle (statocyst). The vesicular wall shows sensory hair cells, either scattered or arranged in plaques. Suspended above these hairs is a layer of chalk particles (statoliths) in a fluid (endolymph) that fills the vesicle. The function of this sensory organ is obvious: during every postural change of the animal, the endolymph moves faster than the solid particles, because of the higher specific gravity of the latter. Thus different pressure is exerted upon various hair cells, producing different degrees of stimulation (Engelmann, Delage). The same process occurs also when motion suddenly ceases. Owing to the law of inertia, these heavier statoliths persist in their course of motion for a certain time longer than the endolymph. The result again will be different pressure on various hair cells.

In summary, it can be stated that the statocysts represent sensory organs whose function depends on the laws of gravity and inertia, and that are

controlled by three factors: (1) the statolith as the stimulus; (2) the hair cells as the receptor; and (3) the nerve fiber for transmission to the brain.

The most primitive static organ (statocyst) is found in the coelenterates, e.g., the free-swimming hydromedusae. In vertebrates, the lower forms still show a statocyst similar to that of the avertebrates, with the addition of semicircular canals (Brock). These organs are always paired, one lying on each side of the medulla oblongata. With the phylogenetic ascent this vesicle elongates and forms the saccus communis or the vestibulum, to which is attached one semicircular canal with its ampulla.

In fish there already appears an invagination of the vestibule, thus forming a superior and inferior portion. The sensory organ is divided into a utricle, a saccule, and three semicircular canals, whose positions are more or less in three planes. The sensory organ resembles more and more the development found in man.

### 2. Physiology of the Human Labyrinth

Flourens laid the foundations for the study of the physiology of the labyrinth with his classic experiments on pigeons (1824). He sectioned the semicircular canals in pigeons and rabbits, and found that injury to the horizontal canals caused horizontal movements of the head and spinning of the body. Injury of the superior vertical canal caused vertical movements of the head and tumbling forward. Injury of the inferior canals likewise caused vertical movements of the head and tumbling, but in this instance backward. He also observed intensive movements of the eyeballs. However, his important discoveries were forgotten for almost half a century.

Goltz claimed that the function of the semicircular canals was the preservation of the sense of equilibrium. It was a great advance when a few years later three men, Breuer in Vienna, Mach in Prague, and A. Crum Brown in Boston almost simultaneously worked out a new theory of the labyrinthine functions. This theory was generally accepted and is still valid to a great extent today, in spite of much criticism and many new hypotheses. According to these authors, the three semicircular canals with their cristae represent a sensory organ for the perception of rotatory movements, while the two sacs with the maculae control the position of the head at rest. The labyrinth has, therefore, a twofold function: kinetic (semicircular canals) and static (sacs).

According to J. Tait, the subdivision of the labyrinth into a kinetic and a static portion is no longer valid. He believes that all labyrinthine interventions are kinetic. A similar view was taken by Maxwell, who assumed that not only static but also dynamic reaction originates in the otolithic apparatus. We shall later discuss the numerous theories.

Alexander explained the function of the inner ear as follows. The cochlea and the labyrinth are anatomically combined in one organ, because they both control orientation and respond physiologically to the movements of the endo- and perilymphatic fluid. The cochlear function is brought about by movements of the perilymph, whereas the labyrinth reacts to movements of the endolymph, caused by active or passive motions of the head or of the whole body. Hence he proposed the terms "organum perilymphaticum" for the cochlea and "organum endolymphaticum" for the labyrinth. This topographic difference in the elicitation of the movements of the fluid explains the fact that stimulation of the labyrinth can never lead to sound perception, and, vice versa, an acoustic stimulus can never produce nystagmus or vertigo.

Wittmaack considers the vestibular apparatus as a sensory organ of the "sixth sense." The organ, however, differs from other sensory organs in one point, i.e., the stimulation process does not come to consciousness. He therefore calls it an inner organ. The sensulae of the labyrinth receive their stimuli not through the influence of the surroundings but through the relation of the organism itself to the surroundings. A person becomes aware of the existence of his labyrinth only when its function is disturbed. The great importance of the labyrinth can be seen in the fact that slight disturbances usually cause severe clinical symptoms.

### 3. Histophysiologic Factors

In order to understand the complex facts concerning the physiology of the labyrinth, a thorough knowledge of the histologic structure is absolutely necessary. There are three factors essential to a proper function of the inner-ear organs: (1) stimulation—it is obvious that stimulation does not necessarily depend on a highly developed tissue, because any structureless homogeneous body could serve this purpose; (2) perception—here only cells modified to perceive sensations (true sensory cells) are required; (3) conduction—certain tissues capable of transmission of the stimulus to the brain (nerve fibers) are necessary.

Histologic examination of the inner ear reveals the presence of the following structures in the nerve ending places (p. 13): (1) a homogeneous jelly-like mass in the crista, macula, and papilla basilaris, called in these sites respectively cupula, statolith membrane, and tectorial membrane (Figs. 15 and 17); (2) ciliated hair cells (Figs. 15, 17); (3) nerve fibers (Figs. 15, 17). The ampullar nerve divides into two branches: one to the utricular and the other to the canal end of the crista (Poljak, McNally). Histologic examinations by Lorente de Nó further show the distribution of the nerve fibers on each crista. There are three kinds; thick fibers supplying the central part of the crista, medium-size fibers for the lateral parts, and fine fibers running to the basal portion. According to Ramón y Cajal,

all the fibers end at the sensory cells of the crista and macula in such a way that the fine endings embrace the hair cells (FIG. 16).

Wittmaack opposes the theory that the sensory cells represent the actual elements of perception. He believes that the neuro-epithelium functions mainly as a physical conducting apparatus without exercising a pure sensory biologic function. He considers the peripheral nerve fibers (the nonmedullated nerve endings) as the true elements of perception. This theory, however, has not been proved as yet.

### 4. FUNCTION OF THE CRISTAE

According to Breuer, Brown, and Ewald, the semicircular canals react only to movements with angular accelerations, but do not respond to

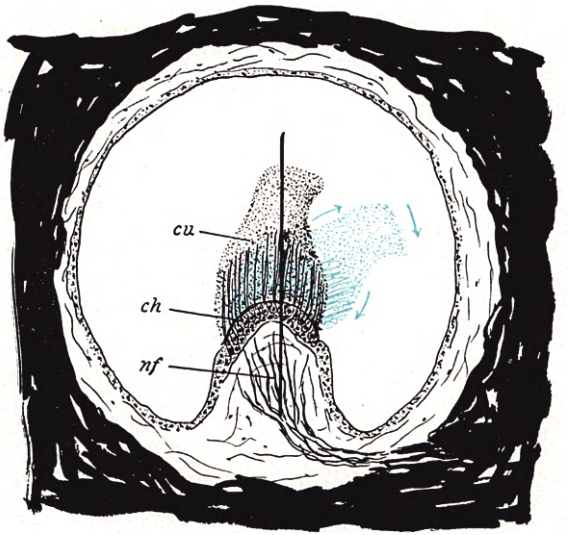


FIG. 15. DIAGRAM OF CRISTA AMPULLARIS

*cu* = cupula; *ch* = hair cells; *nf* = nerve fibers; blue = deflected cupula; black = cupula at rest.

movements with permanent velocity. If, therefore, the head or the body is rotated, only the beginning or the end of this movement, or a change of speed, will cause a sudden push on the endolymphatic fluid, which in turn leads to deflection of the cupula and the hairs of the sensory cells. However, the cupula returns to its original position after a certain time because of its elasticity.

FIGURE 15 demonstrates the physiology of the semicircular canals in a cross section through the ampulla, according to the old theory of Breuer. It shows the crista with the cupula at rest (black). The new position of the crista after turning of the head is shown in blue. The deviation of the cupula is to the right, or clockwise. The hairs of the sensory cells should

be noted. For better understanding, a line divides the crista into a right and a left half. Another point to note is how the hairs on the right side are bent and pushed down by the new position of the cupula, while the hairs on the left side are pulled away or torn. Pushing and pulling of the hairs act as the stimulus. The stimulation is received by the sensory cells and transmitted by the nerve fibers to the brain (FIG. 16). It is obvious that a rotatory movement of the head in the opposite direction will cause a push on the hairs of the left half, and a pull on the right will reverse the effect.

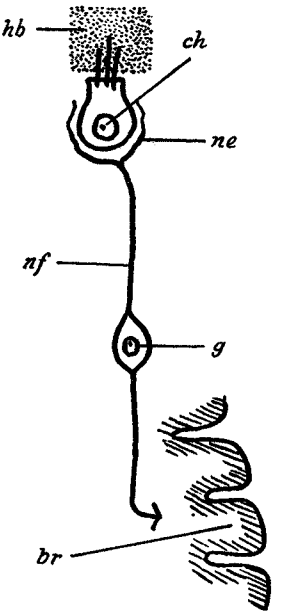


FIG. 16. DIAGRAM ILLUSTRATING TRANSMISSION OF STIMULI

*hb* = homogeneous body (cupula or statolith membrane); *ch* = hair cell; *ne* = nerve ending; *nf* = nerve fiber; *g* = ganglion; *br* = brain.

A rotatory movement of the head or body also produces another reaction, namely, movements of the eyeballs. The flow of the endolymph causes nystagmus, the direction of which depends on the direction of the endolymphatic stream. These facts were proved experimentally by Ewald. He opened a semicircular canal near its simple end and plugged it. A second opening was made between this plug and the ampulla. He then in turn compressed and aspirated the canal, thus producing an ampullofugal and an ampullopetal flow of the endolymphatic fluid. By this procedure he proved that direction of the nystagmus depends on the direction of the endolymphatic stream.

Tait and McNally corroborated this theory. They cut the fibers of the ampullar nerve in a frog and showed that the animal could not maintain its position in the plane of the destroyed canal. On the basis of this experiment they assumed that the crista could be stimulated by the endolymph in only *one* direction of flow. This has not been proved in man. These authors made further studies of the function of the vertical canals, using their method of severance of the ampullar nerve externally to the membranous labyrinth. By these various operations they could examine all combinations of the functions of the four vertical canals (p. 81).

A number of authors do not recognize the Mach-Breuer-Ewald theory any longer. They are opposed in particular to the assumption of endolymphatic circulation. Gray questions whether, in such minute tubules as the membranous semicircular canals, endolymphatic fluid could circulate at all. Maxwell believes that traction and pressure rather than endolymphatic circulation act as the stimulus. He made an experimental study of the transmission of stimuli by sectioning the horizontal canal near its utricular end. He came to the conclusion that the stimulation of the crista depends on changes in tension of the utricular membrane according to the laws of inertia acting in the large spaces of the utricle (endolymph) and the vestibule (perilymph).

Shambaugh believes that the cupula remains fixed, while the perilymph is the important factor for the movement. Maier and Lion studied capillary circulation and came to the conclusion that endolymphatic flow is possible in the minute semicircular canals, even in man. However, they suggest that bending of the hairs of the crista produces chemical changes similar to those occurring in the retina. According to Schmalz, changes in the ionic concentration around the cupula are the effective factors. Although Ivy believes in an endolymphatic circulation, he does not consider it as the stimulating factor.

Wittmaack states that *in vivo* the cupula is so rigid because of turgor that it cannot be moved at all by the endolymphatic current. He believes rather in direct stimulation of the cupula by hydrostatic currents and inertia pressure.

Steinhausen opposes Wittmaack's theory on the basis of his own experimental studies. He injected india ink into the semicircular canals and then stimulated them by rotation and calorization. He could observe *in vivo* not only the current of the endolymph but also the deflection of the supravitally stained cupula. Biehl rejects the idea of any circulation in the entire labyrinth, but believes that pressure is the only effective factor in the vestibular apparatus.

Werner assumes that the shifts in the endolymph do not originate in the canal itself, where they are transmitted to the ampulla and crista, but

instead take place on the ampulla, and that the canals serve merely as shunting places. Lowenstein and Sand used action currents for their examinations and found the action potentials increased by homolateral rotation and decreased by contralateral rotation. Sand, experimenting on fishes, could further show that there is a continuous discharge of action currents and that this discharge is increased by stimulation and diminished by inhibition.

All these numerous theories neither invalidate nor replace the old Mach-Breuer theory. On the contrary, there is a tendency to revert to it again. This tendency is fully justified. Although the Mach-Breuer theory is not completely correct, it is the only theory that explains most of the phenomena of the physiology of the labyrinth.

### 5. Function of the Maculae (Otolithic Organ)

Analogous to the three cristae in three different planes, one should expect also three maculae in corresponding planes. In animals there is a suggestion of such an analogy (Breuer). Analogous to the crista of the horizontal canal, there is the macula of the utricle; for the sagittal crista there is the saccular macula, and for the frontal crista there is the lagena. Magnus and De Kleyn tried to establish a similar analogy. They assumed in corresponding planes the crista of the horizontal canal and the utricular macula; the crista of the sagittal canal and the main portion of the saccular macula; the crista of the third plane and the dorsal lobe of the saccular macula. Whether such analogies occur in animals is questionable, but in man they usually do not exist.

I described years ago a third macula in man that I called "macula utriculi accessoria" (Fig. 67). In a topographic sense its position is perpendicular to that of the macula utriculi as well as to that of the macula sacculi. I denoted this third nerve ending place as an atavistic formation.

According to the Breuer theory, the statolith exerts a certain pressure on the macula. This pressure is due to the weight of the statoliths and changes with the position of the macula. Figure 17 illustrates the physiologic mechanism according to the old theory of Breuer.

When the head is upright, the macula utriculi lies approximately in a horizontal plane. The weight of the statolith (black portion) presses with equal force at all points on the sensory hair cells. It acts as a stimulus that is received by the hair cells and transmitted to the brain (Fig. 16), thus informing the individual of the position of his head in relation to space and to his own body. If the head is now turned 180°, that is, hanging down, the macula utriculi will again be in a horizontal plane, as it was in the former position. The only difference is that the statoliths now pull on the hairs of the sensory cells. This again acts as a stimulus that is

received by the cells and transmitted to the brain. Any position of the head between these two extremes could be assumed. The blue portion of FIGURE 17 represents the position of the head when tilted toward the shoulder. The oblique position of the macula causes the statolith to slide down, thus stimulating the hair cells.

Kreidel tried to prove the Breuer theory in a rather interesting manner. He based his experiment on the observation that crayfish, when shedding their skins, also loose their statoliths. With the development of the new skin they introduce into their ears any particle of matter from their environment; this acts as a new statolith. Kreidel obtained some crayfish immediately after shedding of their old skins, when the new skin was

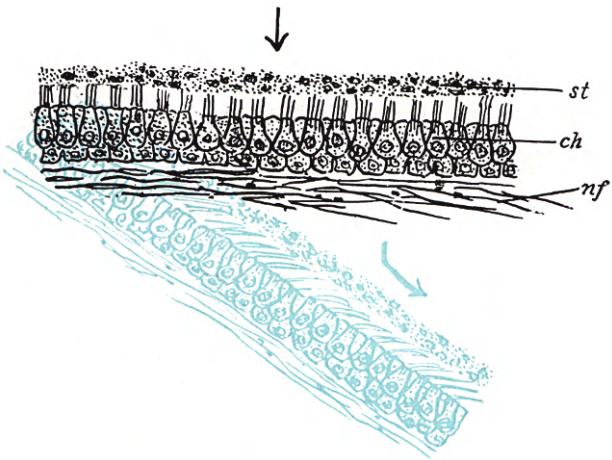


FIG. 17. DIAGRAM OF MACULA

*st* = statolith membrane; *ch* = hair cells; *nf* = nerve fibers; blue = position of macula after tilting of head; black = position of macula at rest.

beginning to grow. He kept them in a jar of filtered sea water to which he had added fine particles of reduced ferric oxide. As soon as he observed that the animal put some of these iron particles into its ears, he approached it with a magnet. Immediately the crayfish assumed an oblique position in the direction *away* from the magnet. Kreidel explained this phenomenon as follows. In the normal position of head and body, the statoliths exert a certain pressure, thus informing the animal of its position. If a magnet approaches these iron statoliths, the direction of the pressure exerted is changed. This gives the animal the impression of being in an oblique position (tilted *toward* the magnet) and the effort to overcome this leads to overcorrection in the other direction.

Modern researchers deny the assumption of older workers that animals

have a sense of position that they can control by conscious efforts. They believe rather that changes of the head release certain postural reflexes independent of consciousness (p. 92).

Another point of argument is as to whether pressure or traction, analogous to these factors in the semicircular canals, is the effective stimulus. Bárány opposes the Mach-Breuer sliding theory and believes that the higher specific gravity of the otolith exerts different traction upon the sensory cells in different positions of the head, and thus produces different stimuli. According to Magnus and De Kleyn, traction is the determining factor and pressure secondary. Tait and McNally also reject the sliding theory and believe that each otoconium "is itself gravity-oriented much as an anchored buoy is gravity-oriented. Stimulation occurs when the tiny otoconial buoys are, by simple slow tilt of the head, by centrifugal force, or by appropriate linear acceleration of the head, made to alter the direction of their (normally vertical) axis with respect to the underlying macula surface." The authors arrive at the conclusion that there are two kinds of labyrinthine reactions: one elicited by slow movements of the head, originated in the utricle; the other elicited by fast movements, originated in the utricle and the vertical semicircular canals.

According to Quix, the otoliths are static organs that not only control the posture of the head, but also produce a certain tonus of the muscles of the eye, of the extremities, and of the trunk (p. 92). Versteegh destroyed the nerves of the macula utriculi in rabbits and could demonstrate that the utriculus was the seat of the labyrinthine righting reflexes for the head, for the compensatory position of the eye, and of the tonic labyrinthine reflexes of neck and trunk.

Ulrich exerted a certain pressure upon the otoliths, using a pike hair. By pressure in an anterior and outward direction, a deviation of the ipsilateral eyeball upward and backward was elicited. Pressure outward produced vertical deviation upward, while pressure inward did not produce any change of the eyeballs. De Kleyn accepts neither the pulling nor the pressing theory.

### 6. Linear Movements

Breuer differentiates sharply between the reflexes of the semicircular canals, which control motion, and the reactions of the otoliths, which control postural as well as rectilinear (progressive) movements. While rotatory movements are motions with an angular acceleration, the progressive movements are linear, i.e., forward, backward, upward, and downward. According to Breuer, the nerve terminals of the ampulla are inadequate because of physical reasons for the transmission of progression movements. However, bodies of different specific gravity, such as the

otoliths, which exert a steady pressure upon the sensory hair cells, and which even during rest produce the sensation of equilibrium, are particularly suited for this function.

According to De Kleyn, progressive movements are produced by a combination of crista and otolithic reflexes. Mach believes that movements of progression with regular acceleration cannot be felt. On a train, acceleration of movement is perceptible only during the period of starting and the period of stopping. According to Delage, the labyrinth is not at all concerned with perception of progressive motion. Such perception is due to changes of pressure in the large vessels and viscera.

Magnus and De Kleyn believe that linear accelerations may stimulate the otolith as well as the semicircular canals. According to Lorente de Nó, rectilinear movements lead to a displacement of the semicircular canals, this stimulating the cristae. Ter Braak assumes that the differences between the specific gravity of the cupula and of the endolymph produce a deflection of the crista in movements with linear acceleration.

### 7. Function of the Saccule

Great dissension prevails over the function of the saccule. According to Mach, it serves for the perception of progressive movements. Many authors (Laudenbach, McNally and Tait, Versteegh, etc.) deny entirely that the saccule has a vestibular or equilibration function. McNally and Tait destroyed the saccule experimentally and did not observe any disturbance of equilibrium. According to Magnus and De Kleyn, the saccule elicits asymmetric righting reflexes, while the utricle elicits symmetric righting reflexes. Benjamins and Huizinga assume that rotary motion of the eye in animals with laterally placed eyes originates in the saccule. Maxwell maintains that otolith and semicircular canals have mutual influence in spite of different functions. Lorente de Nó also assumes that the membranes of the semicircular canals are movable, so that the cristae can be stimulated even by changes of the position of the head.

Hasegowa corroborates the experimental findings of Tait and McNally to a certain extent. He also finds reflexes still elicitable after destruction of the sacculus, but only when he examines the progression movements in the direction of the long axis of the animal. On examination of movements in the direction of the dorsoventral axis no reflexes occur. He therefore comes to the conclusion that the sacculus controls rectilinear movements in the direction of the dorsoventral axis of the animal.

Many authors believe in an *acoustic function* of the sacculus. According to Benjamins, the pars superior of the labyrinth serves for static function, while the pars inferior is concerned with hearing function. Parker believes

that in fish the sacculus serves for the perception of mechanical oscillations; this is in agreement with the findings of Frisch and Stratter. Similar observations were made by Versteegh for rabbits and by Tait and McNally for frogs and snakes. Tait stated that the sacculus in vertebrates (living in the air) reacts to acoustic stimuli transmitted by bone induction. Ashcroft and Hallpike corroborated the theory of Tait by their electrophysical experiments. Undritz and Sassanow take a similar view. According to Grahe, the saccular otolith produces reflexes that influence certain muscular reactions when swing music is played. He therefore assumes that the saccule is the seat of our sense for rhythmic music.

### 8. Separation of Macula and Crista Functions

From this discussion it is evident that diversity of opinion is much greater with regard to the functions of the otolithic organ than with regard to those of the semicircular canals. The reason for the difference of opinion on the function of the otolithic organs lies in the fact that there is abundant proof for the functions of the semicircular canals, such as vertigo, nystagmus, and turning sensations depending on the position of the head, while such effects are lacking after stimulation of the maculae.

In order to test the function of the static labyrinth (utricle and saccule) apart from the function of the dynamic labyrinth (semicircular canals), Wittmaack destroyed the otoliths. He rotated guinea pigs on a centrifuge at 2,000 revolutions per minute. On histologic examination he demonstrated that the otoliths were thrown off completely, while the cupula on the semicircular canals remained intact.

Magnus and De Kleyn based their experiments on the method of Wittmaack. After the otoliths were thrown off, the function for rotatory movement remained intact, while the functions controlling position of head and body were lost. The authors came to the conclusion that the maculae are sensory organs producing reflexes that are elicited by change of position of head or body.

Their experimental findings can be briefly summarized as follows. In any given position of the head, a definite constant stimulus is produced by the otolithic organ, eliciting a reflex that persists as long as this position is maintained (postural reflex). Traction and pressure exerted upon the otolithic membrane are the forces eliciting this postural reflex. With maximal or minimal traction or pressure, maximal or minimal reflexes occur. Hence there is only one position of the head eliciting maximal and only one position eliciting minimal reflexes. The maximal effect is obtained when the otolith lies below the macula, pulling on the hairs, while the minimal effect is assumed when the otolith lies above the macula pressing on the hairs.

One can therefore see the tendency of these newer authors, on the basis of their experiments, to revert to the old Mach-Breuer theory. The only differences lie in their assumption that traction exerts the maximal and pressure the minimal stimulation, and in the fact that they consider the reflexes for rectilinear movements a function of the cristae and not of the maculae as Breuer believed.

Other methods of separating the crista and macula functions consist in severance of the supplying nerve fibers or in operative destruction of the terminating places in the labyrinth.

### 9. Influence of the Labyrinth on the Striated Musculature (Tonus)

Ewald already maintained that both labyrinths produce continuous stimulations that under normal conditions maintain the balance of the organism. Disturbance of the labyrinth either by increase or decrease of its irritability will release certain muscular reactions. With unilateral destruction of the labyrinth, he could observe atonia of the extensor and abductor muscles of the extremity of the affected side. On the other side there occurred hypertonia of the flexors and adductors. He came to the conclusion that each labyrinth controls the flexors and adductors of the contralateral side, and the extensors and adductors of the homolateral side.

The problem of the tonic effects of the labyrinth upon the entire musculature has become more and more important in recent studies. Particularly Magnus and De Kleyn and their students have developed the theory of the various reflexes, such as the tonic labyrinthine reflexes, the neck reflexes, and the righting reflexes. Tonic labyrinthine reflexes are those based upon changes of the position of the head in respect to space. Neck reflexes are based upon changes of the position of the head in respect to the body. Labyrinthine righting reflexes permit the organism to bring the head from an abnormal position back into a normal one and maintain it there. Owing to the great physiologic importance of the entire problem, special treatment is devoted to it in chapters iii and iv.

### 10. Influence of the Labyrinth on the Autonomous System

The influence of the labyrinth upon the autonomous system was already known to earlier workers. Graham and Brown could demonstrate changes of the respiratory curve in a frog whose labyrinth had been destroyed. Allers and Leidler confirmed these observations. They assumed that stimulation of the vestibular nerve leads to inhibition of the central mechanism of the respiratory regulators. Byrne considered as autonomous reflex actions such symptoms as nausea, vomiting, palpitation of the heart, perspiration, and vasomotor changes. Spiegel and Demetriades observed

lowering of blood pressure after stimulation of the labyrinth, and explained it as a vascular dilation in the region of the splanchnic nerves. Cantele found on *irritation* of the vestibular apparatus a lowering of blood pressure, whereas a *destruction* of the labyrinth was followed by irregularities in the respiratory curve. He considered the anastomotic fibers of the intermedius nerve as the route of transmission of these stimuli. Wotzilka examined blood pressure and pulse after turning of the patient. Neumann found a bradycardia in cases of labyrinthitis, which he explained on the basis of an irritation of the labyrinth. However, Brunner and Kauf could not corroborate this theory in their experimental examinations. Byrne described the influence of the labyrinth on the gastro-intestinal tract (Kremer, Spiegel, and Demetriades).

Another mechanism controls the pupils of the eyes. Wodak and Fischer found during rotation a slowly increasing miosis, after rotation a sudden mydriasis. Spiegel assumes that the labyrinthine reflex travels over the posterior longitudinal bundle and the oculomotor nerve. Each labyrinth controls the pupillary sphincters of both sides. Muck succeeded in eliciting a rhythmic horizontal nystagmus after mechanical, thermal, or electric stimulation of the vertebral or radial artery.

According to Portmann, the normal circulation of the ear is regulated by the periarterial and sympathetic cervical nerves. Stimulation leads to vasoconstriction and ischemia, while section of the sympathetic nerves produces vasodilatation with hyperemia.

### 11. Kinetovisual Function

This term has recently been applied by Arellano to the function of the vestibular apparatus, which cooperates with the visual apparatus in vision of moving objects during movements of the head. There are two movements during rotation: one is the movement of the external objects (movement of the panorama). The other is a rhythmic movement of the eyes, the perrotatory nystagmus, elicited by stimulation of the semicircular apparatus. This nystagmus is directed around the axis of rotation of the head with its slow component in an opposite direction and the rapid phase in the same direction. These nystagmic movements oblige the eyes to follow the movements of the different sectors of the panorama; thus the eye and the panorama are displaced in the same direction, and this in turn leads to an immobilization of the optic image on the retina, so that the vision is accomplished in the same manner as vision of still objects. The immobilization of the optic image, an essential factor for producing clear visual perception, is carried out, according to Arellano, by several mechanisms, as follows:

When the movement of the optic image is produced by *rotation* of the

head, the stimulation of the semicircular apparatus produces a perrotatory nystagmus, which plays a great role in the immobilization of the optic image.

When the movement of the optic image is produced by *inclination* of the head, it is the otolithic apparatus that is stimulated, eliciting compensatory movements of the eyes (*Gegenrollung*), and causing an immobilization of the optic image.

When the optic image is produced by a *transfer movement* of the organism or by a *movement of the external object*, the vestibular apparatus is not stimulated. The eyes follow the moving panorama or object (cf. optokinetic nystagmus, p. 69).

## 12. Summary

The labyrinth is concerned with the following functions:

1. Control of movements of angular acceleration of the body.
2. Control of rectilinear (progressive) movements of the body.
3. Informing the central organ of the position of the head in relation to space, to the neck, and to the body.
4. Maintaining a certain tonus of the entire musculature.
5. Preserving a normal position of the eyes, which assume a special position corresponding to each change of the head.
6. Producing reflexes that bring about a certain position.
7. Immobilizing optic images during movements of the head ("kinetovisual function"—Arellano).
8. Controlling equilibrium within the frame of the rest of the centripetal mechanism (skin, muscles, joints). The labyrinth is the most important control mechanism and is closely associated with the central equilibrium mechanism (cortex, cerebellum, and brain stem).
9. Influence upon the autonomic system (respiratory, cardiac, and gastro-intestinal mechanisms).

The underlying principles of the various labyrinthine functions are the forces of gravity, inertia, and centrifugence.

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# III

# Applied Physiology

By *Joseph Fischer*

## SPONTANEOUS MANIFESTATIONS

In contrast to the reactions and reflexes elicited by the various stimulations (irrigation, turning, etc.), we may observe certain symptoms in patients without having applied any manipulations. These are spoken of as spontaneous manifestations. Since such symptoms do not occur in normal persons, they must be regarded as pathologic signs. Their occurrence usually has great clinical importance for localization of the site of the disease. Such manifestations are vertigo, disturbance of equilibrium, spontaneous nystagmus, and disturbances of the associated eye movements.

### 1. Vertigo

#### DEFINITIONS

The study of the phenomenon of vertigo reveals very soon that the subject is much more complicated than at first believed. This is due to the fact that we are concerned not with objective symptoms that can be ascertained clinically or measured scientifically, but rather with subjective symptoms. The physician is entirely dependent on the history given by the patient. The patient calls the most variegated sensations vertigo. He describes sensations of being turned around, or of objects turning around; he has the feeling of losing the ground or of being lifted up; some have feelings of being pulled to the side (lateropulsion), others notice a sham movement of stationary objects. Many describe a general unsteadiness and darkening of vision; others again complain of double vision or of jumbling of letters, or of vertical lines appearing oblique. Still others complain of a disturbed sensation of gravity, such as lightness or heaviness of the head.

This variety in description is also evident in the nomenclature, which is equally confusing. Terms are used such as the following: turning, ocular, tactile, spatial, labyrinthine, neurasthenic, hysteric, epileptic, cardiac, intestinal, or height vertigo, etc. According to Purkinje (1820), vertigo consists of sham movements of the patient's own body and of the surroundings (ocular vertigo), and also of disturbed tactile sensations (tactile vertigo). This author based all phenomena of vertigo on the visual and the vascular system, and believed that the blood supply of these organs,

especially of the brain, was not equally distributed. Since that time numerous definitions of vertigo have been given, but none can as yet be regarded as satisfactory. Some consider vertigo as a purely physiologic process, while others believe it to be entirely psychologic. Hitzig considers vertigo as the perception of disturbances of normal conception of the spatial relations of the body. He differentiates between systematic (directional) and asystematic (nondirectional) vertigo. Ebbinghaus regards vertigo as a conflict between sensations of movement and posture arising from different sources. Lewandowsky defines vertigo as insecurity or error in our spatial consciousness. Hence, all organs concerned with spatial consciousness can elicit it (labyrinth, eyes, central pathways, proprioceptive pathways). Leidler considers it as an emotion of specific character that is always associated with perception of movement and that in most instances shows characteristics of aversion.

From this it is evident that neither definition nor nomenclature shows uniformity. Otologists are particularly interested in the following problems: (1) Can labyrinthine and nonlabyrinthine vertigo be differentiated on the basis of history and clinical examination? (2) Within the labyrinthine vertigo, can the central and peripheral types be differentiated?

According to Leidler, most vertigoes are produced by way of the labyrinthine reflex tracts. Not only the peripheral and central but also the ocular vertigo is produced by regressive changes in the central vestibular apparatus. A similar explanation pertains to the vertigo elicited by the tactile system. It is produced by the close relations of the labyrinth to the tonus of the body musculature. It occurs, therefore, only when some lesion affects the vestibular system. According to Leidler, increased cranial pressure alone does not produce vertigo unless the labyrinth becomes involved by way of the internal canal and the vestibular nerve.

### ANATOMIC PATHWAYS

Efforts to find any specific anatomic tract concerned with the elicitation of vertigo date many years back. Such researches have been carried out especially by American authors. Jones and Fisher described such pathways for vestibular vertigo. The vestibular portion of the eighth nerve enters the medulla oblongata and then divides into two parts. The pathway to the eye muscles is considered as the vestibulo-ocular tract, and impulses along this tract result in nystagmus. The tract to the cerebellum is the vestibulocerebellar pathway. Impulses traveling along this tract and its continuation to the cerebral cortex elicit vertigo. However, histologic proof of the existence of such a pathway has not as yet been produced. Kato in particular studied series of brain tumors with symptoms of increased cranial pressure and with pronounced vertigo. He

found changes in the small cell portion of the vestibular nuclei and also in the small cell portion of the dorsal vagus nuclei, as well as in the nuclei of the substantia reticularis. However, neither his material (brain tumors with increased cranial pressure) nor his conclusions can be regarded as proof for the existence of such pathways for vertigo. Marburg localizes vertigo without nystagmus in the oral portions of the vestibular nuclei. On the other hand, Brunner and Bleier found normal conditions in the vestibular nuclei in a case of encephalomalacia of the cerebellar cortex that had been characterized by extreme turning vertigo.

According to Hitzig, vertigo is particularly marked when the basal portions of the vermis are involved. Leidler believes that vertigo in cerebral lesions often has the same origin as in cerebellar diseases, namely, the loss of inhibitory fibers between the cerebellum and the vestibular nuclei. Spiegel and Alexander, who assume a vestibular center in the cortex of the temporal lobe or the operculum, find vertigo in lesions near the sylvian fissure.

All these contradictory theories show that no scientific basis for localization of vertigo exists. Hence any clinical localization must be considered as a tentative diagnosis.

### DIFFERENTIATION OF PERIPHERAL AND CENTRAL VERTIGO

Peripheral labyrinthine vertigo is almost always a typical turning and only rarely a tactile vertigo. Sham movement of the surroundings is again more prevalent than turning of the body, and acute attacks predominate. The more intensive the vertigo, and the more it takes the form of short attacks, the greater the impression on the patient and the more accurate his description. In such cases the patient is usually able to state the direction of the sham movements. If nystagmus coexists, then the direction of the sham movement is the same as that of the fast component of the nystagmus.

Central labyrinthine vertigo can also be either turning or tactile (Purkinje). In the latter instance there is sham movement of touched stationary objects. The sensation of being pulled to the side (lateropulsion) is a symptom of chronic nonsuppurative diseases of the central vestibular region (Brunner). Spontaneous nystagmus is usually absent.

In peripheral labyrinthine vertigo, an attack is usually brought about by a sudden change of the position of the head. In central labyrinthine affections the attack is not dependent on any change of the position of the head.

In peripheral labyrinthine vertigo, an attack lasts usually only a few seconds or minutes. Only in rare instances of a sudden break into the labyrinth (suppuration or hemorrhage) does the attack persist for hours

or days. Yet, in gradually progressing lesions, such as chronic circumscribed labyrinthitis or degenerative atrophy, the symptoms are not so marked. This is due to the fact that the central compensatory processes have sufficient time to adjust themselves to the disturbed functions of the peripheral organ. Central labyrinthine vertigo develops gradually, has a slow course, and a long duration.

For further differentiation it often becomes necessary to take into consideration the results of the various functional tests (caloric, turning, galvanic, etc.).

## 2. Spontaneous Disturbances of Equilibrium and Coordination

### Comparative Physiologic Considerations

Goltz, on the basis of his experiments, was the first to advance the opinion that the semicircular canals are the sensory organs concerned with the equilibrium of the head and the entire body. Maintenance of balance by a static organ is necessary only in such animals as move either temporarily or permanently in a labile equilibrium.

These functions are particularly evident in those lower animals that live either in water or in the air. The bodily equilibrium of fish and water amphibia is controlled solely by the labyrinth. With phylogenetic ascent, this mechanism becomes increasingly complicated. With the development of appendages, the superficial proprioceptive pathways become more and more effective in the spinocerebellar tracts. Higher animals, therefore, show additional optic and sensory mechanisms.

These mechanisms are most complicated in man, who has assumed an erect posture. The perception of the vertical line and the presentation of the gravity line of his erect posture is very important. However, the function of the human labyrinth is less evident, and does not seem to play such an eminent role. A destruction of the labyrinth is soon overcome by compensation in the other equilibrial mechanisms, such as the sense of sight and the proprioceptive factors (skin, muscles, joints). The peripheral sense organs send out impulses that maintain the equilibrium automatically. This action is brought about partly by reflex arcs or partly by elicitation of certain sensations (posture, motion, resistance).

According to Hitzig, the statics of the head, the eyes, and the body are controlled by the mechanisms that react to active motion. They function congruently only when the intentions of movement are congruent. While the body is in the position of rest, the "physical vertical" is sufficient; in active movement, the "resultants of mass accelerations" are necessary for equilibrium (Mach). This resultant is the product of gravity and all the accelerations resulting from the complicated muscular coordination. Abels calls the physical vertical the static, and the resultant the dynamic

gravity line. The direction of the dynamic line of gravity changes with every change of movement in order to maintain equilibrium during every phase.

### SENSE OF STATICS

Kobrak defines as the "sense of statics" the functions of all static organs. This sense of statics is, therefore, not a function of one sensory organ, but rather a combination of perceptive and associative functions. The vestibular apparatus is the most important of the equilibrium control mechanisms, and is connected with all other central portions that are concerned with equilibration (cerebellum, cerebrum, and brain stem). The cerebellum is the main organ for maintenance of the muscular equilibrium. Every directional muscular activity is represented in the cerebellum, but is determined by the vestibular apparatus.

### INTERPRETATION OF THE VARIOUS MECHANISMS

From the preceding discussion it becomes evident that the function of human equilibrium represents a complicated muscular activity. It is concerned not only with the maintenance of the static equilibrium but also with the ability to perform skilfully complicated action. This process requires special exact cooperative action of the individual muscles (coordination). All these coordination mechanisms are rooted in the eye, the labyrinth, and in the sum total of proprioceptive factors.

Yet the role of the individual components in this coordination is still the subject of controversy. Wallenberg believes that the vestibular apparatus is the most important of the three factors. The slightest disturbance is sufficient to upset the statics, while ocular lesions are less effective. The kinesthetic component is the least important, because even severe disturbances do not produce dizziness. According to Foerster, the eye influences only the cerebrum. The labyrinth influences cerebrum and cerebellum, but the proprioceptive influence extends to the cerebrum, the cerebellum, and the spinal cord. Therefore the proprioceptive functions are the most important and the ocular functions the least important factors, while the labyrinthine functions lie in between.

The existence of a cortical center of the sense of equilibrium is still under dispute. Some authors assume the frontal lobe to be the seat (Bruns, Hitzig and Wollenberg, etc.). Others place it in the parietal lobe (Bechterew, Foerster, M. H. Fischer, etc.), while still others consider the temporal lobe as the cortical center (Mills, Knapp, Spiegel, Aronson, etc.). Poljak describes a bundle of corticipetal fibers ending on the posterior part of the sylvian fissure that may play a role in the function of equilibrium. Van Gehuchten studied the anatomic pathways between labyrinth, vestibular nuclei, cerebellum, and spinal cord. He came to the conclusion that the

vestibular system is the chief center for the maintenance of equilibrium. For a thorough study of this complex matter the reader is referred to the interesting original article.

## 3. Spontaneous Nystagmus

### PRINCIPLES AND DEFINITIONS

The first studies of nystagmus were made by ophthalmologists, who ascribed nystagmus to disturbances of the ocular muscles. They were succeeded by the neurologists, who considered it as disturbed function of the central parts of the brain. Finally came the otologists, who produced nystagmus by certain manipulations of the ear. Jansen was the first to observe spontaneous nystagmus in inflammatory diseases of the inner ear and in otitic intracranial complications.

Before delving into this complex matter, it is advisable to discuss a few principles, the nomenclature, and the clinical aspects of the problem.

Nystagmus denotes a rhythmic movement of the eyes, i.e., two successive movements occurring at regular intervals, to and fro. If these movements are equal, we speak of an oscillating or undulating nystagmus; if they are unequal, it is called a jerking nystagmus. In the latter case the inequality lies in the fact that there is one fast and one slow movement (fast and slow component or phase). The nystagmus is denoted by the direction of the fast component. If a nystagmus is designated as a right nystagmus, it means that the fast component is to the right, and the slow component to the left. The nystagmus is increased if the patient looks in the direction of the fast component. This is particularly true of the labyrinthine nystagmus. Undulating nystagmus can be only ocular, but this is not true of the converse. Ocular nystagmus may be undulating as well as jerking. In other words, while undulating nystagmus can be only ocular, jerking nystagmus may be either labyrinthine, central, or ocular.

Furthermore, we must differentiate between spontaneous and induced nystagmus. The former is present in the patient without interference on the part of the examiner, while the latter must be elicited by certain manipulations, either of the ear or of the eye (induced labyrinthine or induced ocular nystagmus). Since nystagmus is almost never present under normal conditions, its occurrence is usually a pathologic sign. On the other hand, the induced nystagmus must be considered as a physiologic reaction, which may be either typical or atypical.

### NYSTAGMOGRAPHY AND CINEMATOGRAPHY

In order to study the phenomenon of nystagmus, numerous procedures have been devised. Ohm constructed an ophthalmoscope (Zeiss) that

enabled him to project both images separately and to study them by comparison. The introduction of nystagmography was a definite step forward, because it permitted kymographic recordings of the movements of the eyeballs. However, the transmission of the ocular movements was not very exact, particularly in the apparatus used by Ohm at that time. In recent years the technic has improved to a certain extent.

Later, cinematography was applied to the study of nystagmus. In 1923, I demonstrated at the Otolaryngologic Congress at Kissingen records taken with slow motion after exposure of 60 frames per second. I could demonstrate an analysis of the phases of the nystagmus. By means of skin markings it was possible to superimpose the individual pictures and to plot graphically the deviations of the eyeball.

The most modern methods employ an electro-encephalograph. The underlying principle is based on changes in the electric field produced by the movements of the eyeball. Perlman and Case constructed an apparatus based on the electric potential between the cornea and retina. Jung also described such an electric nystagmograph. All these methods have not been employed clinically for obvious reasons, and serve exclusively for laboratory investigations.

### GENESIS OF THE SLOW PHASE

The mechanism of the slow component is more easily comprehensible than that of the fast phase. If, for instance, a person turns his head around a vertical axis, the following observation can be made. The eyeballs lag behind because they do not move at first. Then they turn slowly in the opposite direction, toward the orbital margin, and at their maximal excursion snap suddenly back. This slow movement of the eyeballs in the opposite direction is designated as the conjugate deviation. The purpose of this slow movement is to preserve the image of the fixed object on the retina. The conjugate deviation is therefore the basis of the slow component of the nystagmus, and is of labyrinthine origin. The reflex arc runs from the labyrinth to the central vestibular nuclei, then to the eye muscle nuclei, and finally to the eye muscles (FIG. 18). According to Bartels, this leads to a slow contraction of certain eye muscles (external homolateral rectus and internal contralateral rectus) and to relaxation of the antagonists (internal homolateral rectus and external contralateral rectus). The slow component (conjugate deviation) is succeeded by the fast phase of the nystagmus and leads to the opposite processes, namely, rapid contraction of the previously relaxed group of eye muscles, and fast relaxation of the previously contracted group of eye muscles.

### GENESIS OF THE FAST PHASE

The mechanism of the fast component of nystagmus is more complicated. The numerous contradictory and confusing theories can best be classified into four groups:

*Peripheral labyrinthine theory.* Several authors consider the peripheral sense organs as the point of origin of the fast component. Breuer and Maupetit ascribed it to the cristae, and Rejtoe to the maculae. These theories are disproved by the experimental destruction of the labyrinthine functions, and likewise by the phenomenon of the "Bechterew compensation." This phenomenon consists of the fact that if one labyrinth is destroyed first, and the other labyrinth some time later, a nystagmus toward the side of the first operation will appear.

*Peripheral ocular theory.* The orbit is regarded as the place of origin of the fast component. During the slow phase the eye muscles contract. This in turn stimulates the nerve endings of the trigeminus and releases a reflex that constitutes the fast phase (Ewald). Bartels, Brunner, and Marburg assume a stimulation of the proprioceptive nerve endings in the eye muscles. This reflex arc follows the Sherrington-Tozer pathway to the ocular nuclei. This theory was disproved by the experiments of De Kleyn. He injected procaine into the eye muscles in order to eliminate the proprioceptive perception and still succeeded in producing nystagmus.

*Cortical theory.* This theory is based on clinical observations that the fast component of nystagmus is absent in patients who are unconscious or under general anesthesia (Bartels, Rosenfeld, Jones, Wilson and Pike, etc.). Bartels assumed two centers: one infranuclear coordination center for the slow component and one supranuclear center for the fast component. The stimuli that elicit the fast component originate in the eye muscles and travel to the cortex. According to Rosenfeld, the location of the center for the fast component is in the frontal lobes.

Jones and Fisher propounded the following theory. The labyrinth causes the slow movement of the eyeballs. The cerebrum becomes aware of the contraction of certain eye muscles and also of the relaxation of others. It knows that the eyes are moved from the normal position, and in consequence sends impulses to the antagonistic muscle set, which then effect the rapid return of the eyeball. The pathways run through the internal capsule and the crus cerebri into the posterior longitudinal bundle. The right cerebral center controls the quick movements of both eyes to the left and the left cerebral center controls the movements to the right side.

Wilson and Pike state that the quick component is dependent upon the integrity of a cerebral reflex arc. According to their view, the center of the fast component is cephalad to the corpora quadrigemina. This

theory was disproved by the experimental work of Hoegyes, Bauer and Leidler, Magnus and De Kleyn, etc. These workers could show that the fast component could be elicited even in decerebrate animals.

*Subcortical theory.* The theory of a subcortical center has many followers. However, there is great dissension concerning the exact location of this center, which has been ascribed by various authors respectively to the following parts: reticulate substance (Lorente de Nó); tegmentum quadrigemini (Adamek); diencephalon (Spitzer); nucleus abducens (Wernicke); aqueduct of Sylvius (Monakow), etc. Bárány believed that there is a supranuclear center in the region between the vestibular and the abducens nuclei. Both the cortical impulses (fast component) and the vestibular impulses (slow component) are joined in this area. Klestadt, like Magnus and De Kleyn, believes that a rhythmic center is necessary for the origin of the two phases, but they do not mention a location for this center.

Other authors deny the existence of a supranuclear center. According to Spiegel and Marburg, the central vestibular nuclei proper are also the site of origin of the fast component. Dohlmann places the site of origin of the fast component in Deiters' nucleus and certain cell groups of the reticulate substance, while the triangular and Bechterew nuclei are considered the point of origin of the slow component. Ohm believes it futile to search for a site of either component because nystagmus is an integrated process of oscillation (oscillation theory).

Hyndman gives a rather interesting explanation of the origin of the quick component. He draws a close analogy between nystagmus and the mechanism of the ankle clonus, which he considers also a rhythmic reflex. He assumes two reflex arcs, "one subserving the contraction phase and one the release phase. It appears that the latter is more cephalad that the former and can be eliminated as an independent reflex."

This discussion makes it evident that the whole problem is not yet solved satisfactorily. The most logical theory would be to assume that the fast component originates somewhere between the vestibular and abducens nuclei without necessity of assuming a supranuclear center.

### CENTRAL LOCALIZATION

Another problem concerns the central localization of the various forms of nystagmus. The experimental work of Leidler laid the foundations for all further studies. His studies in animals were later to a large extent corroborated by clinical observations. He found that injury of the caudal portion of Deiters' nucleus caused horizontal nystagmus toward the injured side but no conjugate deviation. Injury of the caudal portion of the spinal root of the acoustic nerve produced rotatory nystagmus, while

injury of the cephalad portion of the abducens nucleus resulted in vertical nystagmus. If, however, the cephalad portions of Deiters' nucleus or the arcuate fibers (leading from the nucleus to the longitudinal bundle) were injured, there occurred also conjugate deviation of the eyeballs. Injury still more cephalad to the abducens nucleus, where the arcuate fibers begin to fade gradually, produced a nystagmus in the opposite direction and a vertical deviation of the eyeballs.

The various portions of the central nuclear area not only control the type of nystagmus but also its direction. Injury of the most cephalad portion produces nystagmus in the opposite direction. On the basis of his experiments, Leidler came to the conclusion that slight interruption of connection between the vestibular and the ocular nuclei causes only homolateral nystagmus, while severe injury leads to contralateral nystagmus. Lorente de Nó believed that the reticulate substance determines the direction of the nystagmus.

Recently Buchanan produced electrolytic injuries in the various regions of the central vestibular nuclei of the guinea pig. Upon destruction of the area of Deiters, he observed deviation of the eyes in the vertical plane, while destruction of the medial nuclei produced deviation of the eyes in a horizontal plane. In both instances there occurred spontaneous nystagmus in the direction opposite to that of the deviation. Hidesi Matusita studied the various types of nystagmus as well as the position of the eyes and of the body after injury of the vestibular nuclei, particularly those of Deiters and the triangular nuclei.

### INFLUENCE OF THE CEREBRUM

The preceding discussion indicates that the *brain stem* is primarily concerned in the production of nystagmus. With regard to the role played by the other portions of the brain, particularly the cerebrum and the cerebellum, there are a number of diverging opinions. Some ascribe to them an inhibitory influence while others believe that they promote nystagmus. Still others deny any influence of these parts upon the nystagmus.

The earliest workers, such as Hoegyes and Ewald, recognized that the labyrinthine reactions were not at all influenced by the cerebrum. Korány and Loew, on the basis of their experiments on rabbits, found that removal of one cerebral hemisphere caused a unilateral hyperirritability of the labyrinth in relation to turning stimuli. Hence they concluded that the cerebrum exerts an inhibitory influence. Bauer and Leidler observed the same phenomenon after extirpation of one hemisphere but noted that this hyperirritability was of a transient nature. However, if both hemispheres were removed, no inhibitory action whatever occurred.

Some authors believe in direct connections between the vestibular nuclei

and the cerebrum, such as a vestibuloreticular tract (Obersteiner), a vestibulothalamic tract (Held), a vestibulorubral tract (Rademaker), and, finally, fibers from the posterior longitudinal bundle to the nucleus ruber and other nuclei in the diencephalon (Spiegel, Whitaker, and Alexander). However, definite anatomic proof of such direct connections is still lacking.

Other pathways that conduct labyrinthine impulses to the cortex of the cerebrum run through the cerebellum (Monakow, Spitzer, etc.). Spiegel believes that the vestibular nuclei receive impulses not only from the peripheral labyrinth and from the cerebellum but also from the higher parts of the central nervous system. Magnus and De Kleyn removed the cerebrum gradually part by part. Yet, even after complete decerebration, nystagmus could still be elicited. They concluded, therefore, that the cerebrum does not play any part in the origin of nystagmus. Contrary to all these observations, Wilson and Pike found that extirpation of the cerebrum caused disappearance of the fast component.

### INFLUENCE OF THE CEREBELLUM

With regard to the cerebellum, the older workers denied any influence of the cerebellum upon the labyrinthine reactions (Hoegyes, Loewenberg, Lange). Leidler also, on the basis of his experiments, stated that the cerebellum could not produce any nystagmus. On the other hand, Spiegel and Demetriades claimed that they succeeded in eliciting nystagmus experimentally from the cerebellum. The majority of authors are of the opinion that nystagmus cannot be elicited from the cerebellum. However, under normal conditions the pathways through the cerebellum serve in regulating nystagmus. But the mechanism of this regulation is subject to controversy. Most authors believe in an inhibitory effect of the cerebellum (Ruttin, Wilson and Pike, Grahe, etc.). Opposed to this view is Spitzer, who believes in the promoting influence of the cerebellum. He believes it to be a connection between the vestibular nuclei and the cerebrum. The impulse travels from the cerebellum to the red nucleus and from there over the rubrothalamic pathway to the cortex. This theory is weakened by experimental evidence that stimuli may reach the cortex even after extirpation of the cerebellum (Spiegel, Price and Spiegel). Bauer and Leidler showed that duration and intensity of the nystagmus increased for five to six days after removal of the cerebellum, but only if the vermis was also removed at the same time.

Camis made galvanometric studies on this subject. On stimulation of the labyrinth he found changes in the electric potentials of the cerebellar nuclei, but could not reveal any changes of the cortical cerebellar potentials. Price and Spiegel, however, using a string galvanometer with amplifiers, succeeded in finding distinct changes in the potentials of the

cerebellar cortex after stimulation of the labyrinth by rotation. In clinical work, hyperirritability of the labyrinth is often observed in cases of cerebellar abscess. Ruttin explains this fact on the basis of loss of the inhibitory fibers of the cerebellum.

### LABYRINTHINE REFLEX ARC

Experimental examinations and clinical observations yield the fact that elicitation of rhythmic eye movements—i.e., nystagmus—involves: afferent fibers from the labyrinth, efferent nerves to the eye muscles, and a certain connection between them. The exact anatomic pathways

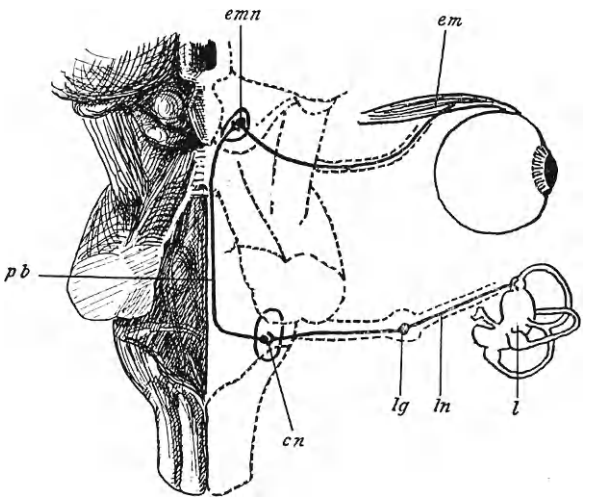


FIG. 18. DIAGRAM OF LABYRINTHINE REFLEX ARC

*l* = labyrinth; *ln* = labyrinth nerve; *lg* = labyrinth ganglion; *cn* = central nuclei; *pb* = posterior longitudinal bundle; *emn* = eye muscle nuclei; *em* = eye muscle.

of the so-called labyrinthine reflex arc are still in dispute. For purposes of theoretic discussion, we assume the following course (FIG. 18): peripheral labyrinth, vestibular nuclei, arcuate fibers to the longitudinal bundle, posterior longitudinal bundle, reticulate substance, eye nuclei, eye muscles.

Magnus and De Kleyn could show that a single eye muscle and its nerve (external rectus muscle and abducens nerve) are sufficient to produce nystagmus toward both sides by way of this reflex arc. According to Lorente de Nó, it is the reticulate substance that plays the main role, while the posterior longitudinal bundle is not as important as is generally believed. He made experimental examinations in rabbits and after

severance of the longitudinal tracts (posterior longitudinal bundle, mesencephalic tract) found the labyrinthine reflexes still elicitable. This led him to the conclusion that the labyrinth reflex arc, when interrupted in the long tracts, can be restored by the reticulate substance. A similar view is taken by Spiegel, who states that the posterior longitudinal bundle does not represent the only extracerebellar system carrying vestibular impulses in a cranial direction.

Godlowski distinguishes two pathways for the vestibular impulses, one laterodorsal (vestibulomesencephalic tract), the other ventral. The former consists of fibers to the eye nuclei and to the nuclei of the posterior commissure and the thalamus; the latter conveys impulses to the central parts of the reticulate substance. Ohm assumes a coordination center representing the junction of three different pathways: one from the frontal lobes (voluntary eye movements), another from the occipital lobes (optic impulses), and the third from the labyrinth. This center, which, according to Ohm, lies in the region of the vestibular nuclei, collects the various impulses and transmits them by way of the posterior longitudinal bundle and the reticulate substance to the eye muscles.

### PRINCIPLES OF CLINICAL CLASSIFICATION

In correspondence with the reflex pathways, we can classify nystagmus clinically as peripheral labyrinthine, central, and ocular. The differential diagnosis of the various types is discussed in chapter iv.

Another principle of classification is applied by Kestenbaum. On the assumption that the movements of the eye are controlled by three factors, namely, vestibular apparatus, fixation mechanism, and correction mechanism, he divided nystagmus into three groups:

Vestibular nystagmus—this is elicited by stimulation or irritation of the vestibular apparatus anywhere from the peripheral labyrinth to the termination of the reflex tracts in the so-called "oculomotor center";

Fixation nystagmus—this is produced by a faulty development of the fixation apparatus;

Correction nystagmus—this form is caused by a weakness of correction due to motor lesions.

### MECHANISM OF END POSITION NYSTAGMUS

A number of nystagmus types can be grouped as a clinical entity under the designation of "end position" nystagmus. This type occurs in lateral vision, often only in the end position of the bulbi, that is, the nystagmus will be to the right when the subject is looking to the extreme right, and correspondingly to the left under reverse conditions. The clinical im-

portance of these types lies in the fact that they are the ones that occur most often in the practice of the otologist. They are far more frequent than the nystagmus of higher degrees of intensity (second and third degree). Furthermore, they are the types that are more difficult to diagnose.

Sommer and I, in a study of the mechanism of the end position nystagmus, included the following five types in this group:

*Correction and fixation nystagmus.* This occurs chiefly in the lateral end position of the eyeballs and is a gradually decreasing nystagmus. The extreme looking to one side puts more strain on the equilibrium of the ocular muscles, because one agonist is more strongly contracted and one antagonist more strongly relaxed (cf. also chap. iv).

*Fatigue nystagmus* (Alexander, Uffenorde). Continuous fixation in a lateral position of the eyeballs produces a nystagmus that gradually increases. It can be compared to the tremor of a muscle that occurs when, for instance, an object is held for a long time in the extended hand (cf. also chap. iv).

*Labyrinthine end position nystagmus.* This is usually a mixed type of nystagmus (horizontal-rotatory, or rotatory-horizontal), of medium amplitude and frequency. It also persists unchanged for longer periods of time and is often associated with labyrinthine symptoms such as vertigo, nausea, disturbed equilibrium, etc.

*Musculoparetic nystagmus.* This type is found in patients with paresis of the eye muscles and occurs when the patient is looking in the direction of the diseased muscle. It can be explained on the basis of increased strain of the eye muscles (cf. also chap. iv).

*Conjugate-paretic nystagmus* (Bartels). When a patient with conjugate paresis is requested to fix upon the examiner's finger moved in the direction of his paresis, slight movements may occur, followed by fast jerks in the opposite direction.

Our studies of the end position nystagmus were based upon various clinical observations. We saw, for instance, in patients who originally had no spontaneous nystagmus, the development of an end position nystagmus that persisted for a long time after the induction of experimental nystagmus (by rotation or calorization). Furthermore, we found on stimulation by the minimal test that the end position nystagmus was dependent upon fixation, position of the patient, etc. The fact that, on minimal calorization, nystagmus in the end positions of the eyeballs often occurred as the first reaction, suggested the idea of referring all these phenomena to the same genesis.

Our studies led us to the conclusion that end position nystagmus is the

expression of a competition between two components. One component is the tonus of the eye muscles (irrespective of labyrinthine or cortical origin) and the other is the fixation and correction reflex. Extreme lateral position of the eyes involves a marked contraction of the agonists and a marked relaxation of the antagonists, as compared with the position of rest (looking straight ahead). Maintenance of extreme lateral fixation is tolerable only for a short time. Furthermore, to this uncomfortable position is added the effort of fixation, consisting of smaller or larger oscillations around a fixation point. This would explain the occurrence of certain nystagmic movements of the eyeballs (end position nystagmus). It is incompatible with physiologic functions to move the eyes only in lateral fixation; this is accomplished instead by turning the head. Extreme lateral position of the eyeballs without corresponding turning of the head creates a feeling of unpleasantness. This is particularly pronounced in nervous patients (neurasthenics). If the labyrinthine tonus of the muscles (Ewald) is disturbed, either by caloric or turning stimulations, or by changes of posture, nystagmic movements of the eyeball can be observed (end position nystagmus). These movements may occur even before the labyrinthine stimulus with its specific directional determination becomes effective.

The above-mentioned five types of end position nystagmus can therefore be differentiated as follows: the fixation and fatigue nystagmus are within the realm of physiology, while the labyrinthine, musculoparetic, and conjugate-paretic types are of a pathologic nature.

### CHARACTER AND SIGNIFICANCE OF POSITIONAL NYSTAGMUS

Bárány described cases of nystagmus and vertigo that occurred only with certain positions of the head. He observed these symptoms only when the patient was in the dorsal recumbent position with the head turned to the right side, so that the left labyrinth was on top and the right labyrinth beneath. The same patient also showed nystagmus and dizziness in the sitting position whenever the head was tilted to the right shoulder, so that the same situation prevailed, namely, the right labyrinth was on top and the left labyrinth beneath. Since this nystagmus was dependent on a definite position of the head, Bárány designated it as "positional" nystagmus, and assumed that it was elicited by the otolithic apparatus (otolithic reflex). Similar observations were made by many other authors, who, however, differed in their interpretations. While Bárány, Voss, Ruttin, etc. assumed a disturbance of the otolithic apparatus, Brunner and Stein, Grahe, and other authors believed that central lesions, as of the neck reflexes (Magnus and De Kleyn), were responsible.

Voss described a syndrome of isolated otolithic disease. It consisted of

vertigo and positional nystagmus, anomaly of the counterrolling of the eyes, and differences in the tonus of the body musculature of each side.

Brunner and Stein rejected the assumption of an otolithic origin of these symptoms. They based their conclusion on their own material and on a critical survey of the literature. They believed rather that the neck reflexes were the eliciting factors. These reflex arcs apparently begin in the cervical roots and reach the cerebellum by the spinocerebellar tracts. According to Grahe, vertigo and positional nystagmus are signs of abnormal central irritation. The positional stimulus is transferred to the vagus center and to the nystagmus tracts respectively.

The differences of opinion arise from the fact that positional nystagmus and dizziness are not symptoms of one particular disease but may be the expression of various pathologic processes. It is rather difficult to comprehend why positional nystagmus should be an otolithic reflex, since we know that nystagmus cannot be elicited by the otolithic apparatus. On the other hand, it has not been proved thus far that the neck reflexes do produce it. Yet there are cases where positional nystagmus and vertigo are caused by diseases of the peripheral sense organ. Whether there is in such cases isolated disease of the otolith organ only, is difficult to prove. In fact, it is difficult to comprehend how such a lesion could be confined only to the utricular macula without involving either the terminal endings of the semicircular canals or the endolymphatic fluid. Furthermore, there may also be cases where vascular processes in the inner ear (similar to the Ménière syndrome) are concerned. Finally it could be considered that the nerve endings may be so sensitive as to react to the slightest stimulation, particularly in neurotic persons.

While the occurrence of positional nystagmus and vertigo was formerly a relatively rare incident, in recent years an increasing number of such observations have been reported on the basis of various pathologic conditions. Experimental approach has been shedding further light upon this problem. Rothfeld succeeded in eliciting positional nystagmus in animals intoxicated by alcohol. De Kleyn could demonstrate that experimentally induced nystagmus disappeared upon extirpation of both labyrinths. Experimental injection of certain solutions into the tympanic cavity or through the fenestrae also produced positional nystagmus. Hasegowa injected heterotonic saline solutions and diluted toxins into the oval window and elicited positional nystagmus. Under the influence of hypotonic solutions, its direction was opposite to that produced by hypertonic solutions, while isotonic saline had no effect at all.

Clinically an attempt has been made to utilize positional nystagmus for diagnostic purposes, particularly in diseases of the central nervous system. Ruttin differentiated three groups of positional nystagmus:

Positional nystagmus elicited by changes of body position, or inclination or rotation of the head, and lasting only until the new position is reached (probably motion reflexes are concerned).

Positional nystagmus following certain laws. The nystagmus is directed either to the side to which the position was changed, or to the opposite side (here an otolithic reflex is most probably involved). However, the site of the disease may also be either the nerve or the central nuclei.

Positional nystagmus with no regularity of direction (here intracranial processes are most probably involved).

Borries differentiates between a kinetic and a static positional nystagmus. Nylen studied patients with brain tumors for positional nystagmus and classified them into two groups: (1) where direction of the nystagmus was dependent on the position of the head; (2) where direction of the nystagmus was independent of the position of the head, but where this influenced the intensity of the nystagmus.

Frenzel believes that the majority of cases with positional nystagmus have a central affliction. According to Klestadt, positional nystagmus is of peripheral origin if it is the only symptom in association with otitis media. Kelemen described 2 cases with "mirror-like" positional nystagmus: with the head to the right the nystagmus was to the left, and with the head to the left the nystagmus was to the right. He assumed a lesion in the supranuclear center.

Seiferth studied 200 cases of positional nystagmus and classified them into three groups: (1) nystagmus of changing direction (this group corresponds to group 3 of Ruttin and group 1 of Nylen); (2) irregular positional nystagmus; (3) directional positional nystagmus. While the first two classifications indicate brain lesions, the third is diagnostic of peripheral diseases. This author regards positional nystagmus as a latent form of spontaneous nystagmus that manifests itself only after positional irritations and that indicates disease either in the labyrinth or in the labyrinthine reflex arcs.

Ruttin, on the basis of recent studies, propounded an entirely new theory concerning positional nystagmus. Under normal conditions, physiologic stimulation of the macula will not elicit a nystagmus. On the other hand, a pathologically irritated macula will produce it. However, this otolithic nystagmus differs essentially from that originating in the semicircular canals. The otolithic nystagmus is characterized by a "wheel-like" movement of the eyeballs, i.e., inclination of the meridian of the eye without displacement. The rotatory (semicircular canal) nystagmus is the result of the action of two rectus muscles and one oblique, while the wheel-like

nystagmus (otolithic) is produced by the synergistic action of the two oblique muscles. Ruttin believes that the occurrence of the wheel-like nystagmus is pathognomonic of diseases of the "position receptors"—a term used by Lorente de Nó—which comprise utricle, saccule, nerve, and central pathways. He believes furthermore that each otolithic apparatus can produce nystagmus toward either side.

### 4. Conjugate Paresis and Conjugate Deviation

During the normal act of vision, there are required certain movements of the eye whose purpose is to fix the attracting object on that portion of the retina that permits optimal acuity of vision. The eye movements may be voluntary or reflex associated. The latter are elicited by passive movements of the head or by labyrinthine stimulation (caloric, rotatory, etc.). Each movement impulse affects equally the motor apparatus of both eyes, and during every change of the direction of vision there takes place a change of the tonus of the entire ocular musculature.

*Conjugate paresis* denotes a condition that makes impossible a turning of both eyes in a definite direction (lateral and vertical conjugate paresis).

*Conjugate deviation* is the associated deviation of both eyes, which are moved in the same direction.

Frequently conjugate paresis in one direction is combined with a conjugate deviation in the other direction. Consequently a conjugate paresis affecting movement to the right corresponds to a conjugate deviation of the eyes to the left. It is due to the fact that traction of the antagonists deviates the eyes in the opposite direction. The conjugate pareses are produced by lesions of the pathways or of the optic centers. They differ from peripheral lesions in that instead of isolated ocular muscles being paralyzed, an entire group of muscles for a definite function is involved. Therefore, a group of muscles paralyzed for a given function can still be functioning in a different combination. For example, in a conjugate paresis affecting movement to the right the left medial rectus and the right lateral rectus muscle are involved, but for convergence they are still functioning because they act in a different combination. However, even in conjugate paresis some voluntary movement can occasionally still be performed with the aid of reflex stimulation. If, for instance, the head of a patient while fixating an object is passively jerked to one side, then to the voluntary impulses are added the reflex impulses, with the result that the eyeballs are deviated farther to the side than could be accomplished with volutary intentions only (Roth-Bielschowsky symptom). Muskens believes that conjugate paresis is not a true paralysis at all but only a part of the complex syndrome of compulsive movements and compulsive posture.

### OPTIC CENTERS

There are a number of cortical areas in the cerebrum that are regarded as the optic centers:

A scouting center in the lower portion of the second frontal gyrus (Tschermack). Muskens denies the existence of such a supravestibular center in the frontal cortex;

Visual centers located on each side of the calcarine fissure and the occipital lobe;

An acoustic visual center located in the posterior and temporal gyrus of the insula and the insular part facing the first temporal gyrus.

Each hemisphere has a center for right and for left ocular movement. If these cortical centers are stimulated, e.g., that of the left hemisphere, there occurs not only a contraction of the right external rectus and the left internal rectus muscle, but also a relaxation of the right internal rectus and of the left external rectus (Sherrington).

The conjugate deviation may be a symptom of either irritation or paralysis. The destruction of the center in one hemisphere produces a reaction similar to that of irritation of the center in the other hemisphere. Spiegel and Sommer, therefore, assume that continuous impulses flow from the cortical centers to the periphery even during rest.

### CENTRAL PATHWAYS

The opinions on the further course of the pathways from the cortical centers vary. Bárány believes that there is a supranuclear center between the vestibular and the ocular nuclei. Uhthoff believes that the tracts run from the frontal cortex to the internal capsule, while Bernheimer assumes that the tracts run to the cerebral peduncle. According to Spiegel and Sommer, the pathways run from the rhombencephalon together with the vestibular tracts to the eye nuclei. Therefore in the region of the vestibular nuclei the corticifugal impulses use the same pathways as the vestibular impulses. Spitzer maintains that the posterior longitudinal bundles are particularly connected with the symptom of conjugate paresis and conjugate deviation. Each posterior longitudinal bundle conducts the impulse for a conjugated deviation of the eyeballs to its own side. Stimulation of the posterior longitudinal bundle of one side produces conjugate deviation to the same side, while destruction of the bundle has the opposite effect (deviation to the other side).

Brunner rejects the idea of any influence of the cortical centers upon the vestibular nuclei, and that the pathways terminate in the vestibular nuclei, as Spiegel and Sommer maintained on the basis of their experiments. He believes rather that the site where the agonistic labyrinthine and the

antagonistic cortical impulses meet is in the region of the eye nuclei. He also objects to denoting the conjugate paresis produced by destruction of the posterior longitudinal bundle as a labyrinthine paresis.

Wilson and Pike stimulated the labyrinth in monkeys by the caloric method, after removal of one temporal lobe, and observed a conjugate deviation of the eyeballs instead of an induced nystagmus. In testing the ear of one sound side, they obtained the expected nystagmus.

The conjugate movements of the eyeballs are impaired in lesions either of the cerebrum or of the rhombencephalon, particularly the pons. Lesions of the former involve the cortical centers; in lesions of the latter the central pathways in the region of the posterior longitudinal bundle and the abducens nucleus are involved. Since in the pontine type of lesion the labyrinthine reflex arc (p. 56) is also involved, one may observe disturbances in the elicitation of labyrinthine reflexes (caloric, rotatory, etc.).

TABLE 1.—*Classification of Bielschowsky*

| Movements | Cortical Lesions | Pontine Lesions |
|---|---|---|
| Conjugate paresis | contralateral | homolateral |
| Conjugate deviation | homolateral | contralateral |
| Pursuit movements | positive | negative |
| Roth-Bielschowsky symptom | positive | negative |
| Rotation of head | toward affected side | toward either side |
| Paralysis of facial nerve | contralateral | homolateral |
| Paralysis of extremities | contralateral | contralateral |
| Labyrinth stimulation | normal nystagmus | abnormal nystagmus or no nystagmus |

For the differential diagnosis of the *cortical* and the *pontine* type of associated eyeball movements it is practical to follow the classification of Bielschowsky (Table 1).

### VERTICAL CONJUGATE PARESIS

The cortical centers and pathways for vertical movement of the eyeballs in man have not as yet been fully investigated. Each labyrinth is said to control the upward movement of the homolateral and the downward movement of the contralateral eye. Disturbances of the vertical movement occur in connection with lesions in the region of the sylvian aqueduct.

## 5. CONVERGENCE SPASM

If a patient is requested to fix his eyeballs in an uncomfortable or forced position, e.g., extreme lateral position or extreme upward rotation, certain phenomena can occasionally be observed. The extremely abducted eye

can be seen to perform a few rapid jerking movements and then return gradually toward the midline, and the pupil becomes miotic (convergence spasm). Distraction of attention will eliminate this phenomenon.

The physiologic basis of this manifestation is still subject to dispute. Some authors (Binswanger, Parrinaud) believe that it is caused by a tonic spasm of the rectus internus, the sphincter pupillae, and the ciliary muscles in persons who show a predisposition to muscular spasms in general. Sachs assumes an organic lesion of the ocular muscles. He maintains that if this lesion is labyrinthine the convergence will persist. However, there is no proof as yet of a labyrinthogenic origin. It is well known that the labyrinth can produce only associated movements of the eyeballs (conjugation, deviation, nystagmus), but not dissociated movements. Several years ago I described a number of cases that exhibited convergence nystagmus not only with the usual forced ocular movements but also after labyrinthine stimulation, such as calorization, rotation, or pressure upon the tragus. In one particularly severe case of hysteria I could produce convergence spasms not only by compression of the tragus but also by stimulation of the hysteric pressure points (nipples, ovaries). I came to the conclusion that in certain instances the convergence spasm is a functional phenomenon and that the labyrinth acts as a hysterogenic center.

The symptom of convergence assumes clinical importance for the otologist if it occurs simultaneously with an inflammatory lesion of the ear (acute or chronic otitis media). If in such patients a convergence nystagmus can be elicited upon pressure on the tragus, then it can easily be confused with a positive fistula symptom. This may lead to errors in diagnosis and indications for surgery. Pertinent cases of this kind are discussed in chapter iv.

### 6. Labyrinthine Strabismus

Several authors maintain that the labyrinth plays an essential role in the elicitation of strabismus. Bartels believes that defects of the auditory apparatus either before occurrence or after loss of the fusion tendency of the eye cause strabismus reflexly. He bases his contention upon experiments that proved that the labyrinth exerts a greater influence upon the homolateral eye. He assumes that in disturbance of the fusion tendency a concomitant strabismus is produced by the labyrinth, because it is only this fusion tendency that opposes the strong influence of the labyrinth upon the eye muscle tonus. Ohm compares the various possibilities of strabismus with experimentally produced strabismus. The latter occurs after stimulation or severance of the vestibular nerve or the semicircular canals. But stimulation of the labyrinth produces strabismus in man also. Ohm describes cases with amblyopic nystagmus and vertical

strabismus in which the labyrinthine irritability was markedly increased. He believes that the labyrinthine influence is particularly evident in internal strabismus with vertical deviation. This influence may be sufficient to overcome a normal fusion tendency. Any disturbance of the fusion, such as poor vision, poor refraction, etc., facilitates the development of labyrinthine confusion of binocular harmony.

Several years ago Sommer and I studied the so-called labyrinthine strabismus in inmates of the Institute for the Deaf-mute of Vienna. We selected deaf-mute children because in these subjects the defects dated back to an age at which, according to most authors, the fusion tendency of the eye is not as yet developed. Furthermore, they offered all degrees of pathologic change of the labyrinth, from slight impairment to complete loss of labyrinthine function. Among 100 children, 14 exhibited strabismus. This group included cases with various forms of pathology, such as retinitis pigmentosa, coloboma of the optic nerve, persistent hyaloid artery, high degree of refractory anomaly, etc. In none of these children did we succeed in proving beyond reasonable doubt the labyrinthogenic basis of strabismus. Although we did not rule out the possibility that labyrinthine disturbances may produce strabismus, we concluded that labyrinthine strabismus is rarer than most authors believe.

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## INDUCED REACTIONS

Stimulation of the labyrinth elicits certain reactions and reflexes that can be classified as primary and secondary. In the first classification are the various forms of induced nystagmus, in the latter the subjective sensations (vertigo, turning sensation, nausea) and the objective reactions such as falling, past-pointing, arm tonus, etc.

The primary reaction (nystagmus) will be discussed according to its elicitation, while the secondary manifestations are treated in a separate section below.

### 1. OPTOKINETIC REACTION

Bárány observed that when people viewed landscapes with telegraph poles, while riding on trains, they experienced rhythmic movements of the eyeballs; this he termed "railroad nystagmus." Because of the fact that this optic nystagmus has an important clinical significance, more and more studies were made by various authors and different names were given it, such as optokinetic, optomotor, induced optic nystagmus, etc. The nystagmus consists of two components, one slow and one fast (jerking nystagmus). For its elicitation three impulses are necessary (Bárány): the impulse of fixation, the impulse to pursue moving objects, and the impulse to jerk back as soon as the next object for fixation appears. The concentration of attention upon the moving objects plays a great role in the elicitation of this form of nystagmus.

Another type of induced optic nystagmus, described by Bartels and others, is independent of the consciousness of the subject, as in the newborn, in idiots, and in unconscious persons.

The first is called the cortical type, the second the subcortical type, or, according to Scala and Spiegel, the active and the passive type.

### MECHANISM OF THE NYSTAGMUS

The eyes pursuing the object remain for a certain period of time fixing the object (slow component). Then they give up fixation and turn suddenly to a new object (fast component). According to Kestenbaum, the slow component is due to fixation; the fast component is caused by the tendency of the eye to relax and by the voluntary intention to look in a certain direction (directional impulse). Because the excursions of the eyes pursuing the objects are always the same, the nystagmus is more or less regular in type.

The fact that attention plays a great role in the fixation of objects led Bárány to the belief that this optic nystagmus is of cortical origin. The center for the slow component lies in the angular gyrus and that for the fast component in the frontal lobe. Brunner ascribes a cortical origin to the slow component, while the fast component is controlled by the Sherrington-Tozer fibers, which go to the eye muscles.

A number of authors assume a reflex arc for elicitation of the optokinetic nystagmus, with the following pathways: retina → optic nerve → optical radiation → calcarine region (afferent portion of the arc); visual centers in the cortex → subcortical region → eye muscles (efferent portion). Cords rejects the supposition that any influence is exerted upon the eye movements by the afferent part of the reflex arc, which he calls optosensory tract, while he holds that only the efferent portion (optomotor tract) can be responsible for the optokinetic nystagmus.

Spiegel, who has shown experimentally that impulses from the occipital lobes can be transmitted to the eye muscle nuclei, states that the vestibular nuclei are of importance in the production of the optokinetic nystagmus, particularly of the subcortical or passive form (Scala and Spiegel).

### CLINICAL SIGNIFICANCE

The optokinetic nystagmus is of great interest to the ophthalmologist, the neurologist, and the otologist.

For clinical purposes it was first used by Wirths and Bielschowsky for the diagnosis of conjugate paresis of the eyeballs, and later for the differentiation of the various forms of hemianopsia.

The clinical importance of the optokinetic nystagmus for neurology has been stressed by many authors like Fox and Holmes, Stenvers, Strauss,

Cords, etc. Stenvers believes that a lesion damaging either of the two centers (occipital lobe, frontal lobe), or the pathways between them, leads to a loss of the optokinetic reaction toward the opposite side. Fox and Holmes deny the existence of a frontal center. According to Cords, any lesion in the optomotor pathway (corticifugal part) causes an elimination of the fast component of the optokinetic nystagmus toward the opposite side.

Guettich considers the weakening of the optokinetic reaction in cases of conjugate paresis as a sign of cortical lesion. According to Brunner, any anomaly of the optokinetic reaction associated with conjugate paresis of the eyeballs must be considered as a sign of a beginning increased endocranial pressure.

In the otologic clinic the optokinetic nystagmus was introduced by Bárány and by Brunner, particularly for localization of a spontaneous nystagmus. They studied the influence of the induced optic nystagmus upon a spontaneous nystagmus, using a turning optical wheel (cf. functional tests, p. 193). Independently of each other, they both came to the same conclusion: if a patient with spontaneous nystagmus showed a typical optokinetic reaction, the test could not be used for the localization of the spontaneous nystagmus. If, however, the optokinetic reaction was atypical (p. 194), the conclusion could be drawn that the spontaneous nystagmus was of a nonlabyrinthine origin. Brunner called this atypical reaction "inversion phenomenon."

I. Sommer studied the interference of spontaneous nystagmus with induced ocular and induced labyrinthine nystagmus. He found that any spontaneous ocular nystagmus can easily be influenced by an induced ocular nystagmus, but not so easily by a labyrinthine (caloric) nystagmus. On the other hand, a spontaneous nystagmus of central labyrinthine origin can easily be influenced by a caloric nystagmus, but not so easily by an induced optic nystagmus. He explains the normal optokinetic reaction as follows. The eye nuclei region can be compared to a central railroad junction, where trains from all directions meet (from the cerebrum, from the eye, from the central labyrinth), in order to converge at the eyeballs. If trains go from one direction (e.g., impulses from the labyrinth), the line is blocked for trains coming from the opposite direction (e g., optic impulses from the eyeballs).

## 2. Caloric Reaction

### Historical résumé

The fact that thermic stimuli could elicit labyrinthine symptoms was already known to Brown-Sequard (1860), Hitzig, Loewenberg, and others. Bonnhard (1876) was the first to apply this knowledge experimentally.

When the semicircular canals of pigeons are cooled with ice, the birds behave as they would if the membranous canals had been destroyed. These interesting physiologic observations shared the fate of the classic experiments of Flourens, since both were forgotten for more than a generation. It is the merit of Bárány to have rediscovered the caloric reactions (1905) and to have introduced them into clinical practice. He observed that patients with chronic discharge from the ear showed nystagmus and dizziness after irrigation of the ear. One patient complained of dizziness only after syringing with cold water. When Bárány substituted warm water the patient complained again, and on examination a nystagmus in the direction opposite to the previous one was noted. This observation led him to a systematic study of this phenomenon. He found a certain regularity of the eye movements, depending upon the temperature of the water. Hot water elicited a nystagmus toward the syringed side, while cold water reversed its direction. Furthermore, a change of the position of the head by 180° also reversed the nystagmus.

### THEORIES OF CALORIC REACTION

The numerous theories concerning the physiology of the caloric reaction can be classified into four main groups: (*a*) nerve theory, (*b*) vascular theory, (*c*) physical theory, (*d*) otolith theory.

*Nerve theory* (Bartels-Ewald). The thermic stimulus acts directly upon the nervous apparatus. While heat increases the physiologic action of the nerve, cold has the contrary effect, namely, that of decreasing the function of and paralyzing the nerve. The cold causes a nystagmus toward the opposite side as a result of hyperfunction of the opposite labyrinth. Many arguments can be presented to disprove this theory. Even an exposed nerve requires for caloric stimulation great differences in temperature, which in turn would lead to damages of the nerve tissue. Furthermore, this theory cannot explain why, in the case of a unilateral destruction of a labyrinth after calorization of the normal ear, a nystagmus toward either side can be elicited. It also affords no explanation as to why nystagmus can be influenced by position of the head, etc.

*Vascular theory* (Kobrak). Thermic stimulations produce vasomotor changes that in turn elicit a circulation of the lymphatic fluid. Heat produces a superficial hyperemia and a deep ischemia, while cold has the opposite effect. This theory was disproved by Grahe, who swabbed the lateral wall of the labyrinth with adrenalin (at body temperature). In spite of vasomotor changes, he was not able to elicit a labyrinthine reaction.

*Physical theory* (Bárány). This theory is based on physical laws. Bárány compared the labyrinth with a vessel filled with water at 98.6 F.

If one wall is sprinkled with cold water from the outside, the fluid inside near the wall cools. This leads to a higher specific weight, with the effect that the cold fluid sinks to the floor of the vessel, while the fluid near the opposite wall rises. If, on the other hand, the wall is irrigated with warm water, a contrary effect will be obtained. This same reversed effect can also be achieved with cold water when the vessel is turned upside down. Applied to the labyrinth, this would mean that a change in the position of the head by 180° reverses the caloric nystagmus. As a matter of fact, Bárány was able to corroborate his physical theory by demonstration on patients.

Further proof of Bárány's theory can be seen in the experimental examination by Maier and Lion and by Meurmann. The former authors studied in pigeons, under the microscope, the circulation of the lymphatic fluid during calorization; the latter determined differences of temperature of the semicircular canals during caloric stimulation.

Bruenings, who made large-scale examinations corroborating the physical theory, came to the following conclusions. Caloric stimulation leads to circulation of the endolymphatic fluid in the same way as the turning stimulation. Each semicircular canal can be brought into such a position that conditions for the endolymphatic flow are particularly good (optimum position) or into a position in which they are extremely bad (pessimum position).

He found as optimum positions for the various forms of nystagmus the following: for horizontal nystagmus, head bent backward at 60° and tilted slightly toward the shoulder of the irrigated side; for rotatory nystagmus, head flexed forward at 90° and slightly tilted toward the opposite shoulder; for vertical nystagmus, no position found. Any nystagmus elicited in the optimum position can be suppressed when the canal is brought into its pessimum position.

Hofer tried to find the proper position of the head for eliciting the various forms and directions of caloric nystagmus. He could show a change of the direction of the nystagmus by a change in the position of the head. For example, he irrigated the right ear with cold water and elicited a nystagmus toward the left side. He then tilted the head of the patient toward the shoulder on the side of the nonsyringed ear and observed a nystagmus toward the right side. However, he found that not only the direction but also the form of the caloric nystagmus could be influenced. He produced, for example, a caloric nystagmus of a rotatory-horizontal type. When he then tilted the head of the patient toward the shoulder on the side of the irrigated ear, the nystagmus became a pure horizontal one.

*Otolith theory* (Borries). Borries made experimental examinations in pigeons and came to the following conclusions. The semicircular canals

are not necessary for elicitation of a caloric nystagmus. The otolith apparatus is the site of the caloric nystagmus. The change of direction of caloric nystagmus is an expression of the otolithic reflex. While Mygind believes that the caloric nystagmus is the result of a stimulation of the semicircular canals plus the otolith apparatus, Borries' view is that the semicircular canals are responsible for the turning reaction, whereas the otolith apparatus controls the caloric stimulation.

### QUANTITATIVE DETERMINATIONS

The various methods of testing the caloric reaction, and the modifications of these, can be classified into two groups: i.e., qualitative and quantitative tests. The former are made only to decide whether the caloric function is present or not, while the latter determine exactly the degree of excitability.

Quantitative determinations of the caloric irritability are subject to many difficulties. As a matter of fact, we do not even know as yet the exact figures of the normal labyrinthine excitability, the reason being that, with all the various methods, it is not the peripheral sense organ that is directly examined but rather a reflex (nystagmus) resulting from the stimulation. This reflex depends not only on conditions in the peripheral sense organ but also on those in the central vestibular apparatus as well as in the eye nuclei and eye muscles. Therefore we can state that the factors involved in the caloric reaction are the following: *extralabyrinthine factors* (external meatus, drum membrane, structure of the mastoid, granulations, cholesteatoma, etc.); *labyrinthine factors* (sensory apparatus, lymphokinetic apparatus); *retrolabyrinthine factors* (conductivity of vestibular nerve); *central vestibular factors* (excitability of central pathways, emotional strain, anxiety, fatigue, etc.); *ocular factors* (muscular apparatus of the eyes). All these explain the difficulties of an exact determination of the physiologic excitability.

The first quantitative method for the caloric reaction was used thirty years ago by Bruenings. He constructed an apparatus (otocalorimeter) that enabled him to measure the volume of the water used and the speed of irrigation. This apparatus was provided with a device for measuring the angles of the various positions of the head (otogoniometer).

*The various methods of minimum tests.* It was a great step in progress when Kobrak introduced his minimum test (2–5 cc. of cold water). According to his view, the test represents an exact method for determining the labyrinthine threshold for caloric stimuli. It further facilitates diagnosis of the so-called "nystagmus preparedness" (*Nystagmusbereitschaft*). Kobrak applied this term to the latent stage of nystagmus that either precedes or succeeds the manifest nystagmus. In such a stage

no nystagmus at all is noticeable, but it can easily be elicited even with stimuli so weak that they could not produce any nystagmus without the nystagmus preparedness. For elicitation of such a nystagmus preparedness, he exerts a pressure on the tragus for ten seconds before the caloric stimulation is applied. For a thorough study of the caloric reaction, he divides the nystagmus into the following stages: nystagmus preparedness (preceding phase); occurrence of slow component; development of jerking component; rhythmic course; nystagmus preparedness (succeeding phase).

On the basis of these theoretic assumptions, the minimum test (original Kobrak method) requires the following technic. The tragus of the ear to be examined is compressed for ten seconds. If no reaction occurs, pressure is exerted on the tragus of the other side. If there is no response, irrigation with 2 to 5 cc. of water at 95 F. is applied. If still no reaction is seen, water should be used at a lower temperature, first at 93 F., then at 85 F. If all these procedures fail, the volume of water must be raised to 10 cc. It must be borne in mind that between each examination and the next a period of five minutes must elapse.

It is obvious that such a method—although very exact from a scientific point of view—cannot be used for practical purposes. The determination of the caloric excitability for both ears would require more than an hour.

*Clinical procedure.* Mayer and Demetriades modified the minimum test for clinical purposes. Following a suggestion of Brunner, they examined many hundreds of patients presenting the varying etiologies, such as external otitis, otitis media, catarrhal conditions, labyrinthitis, intracranial complications, central lesions, trauma, etc.; even normal persons were included in the examinations. Using always the same volume of water at the same temperature (5 cc. of water at 55 F.), they determined the "latent time" and the duration of nystagmus. They came to the following conclusions. The normal excitability of the labyrinth to caloric stimuli shows a wide range, the average figures based on clinical examinations being fifteen to thirty seconds for latent time, and sixty to one hundred and twenty seconds for duration of the caloric nystagmus. Compression of the tragus failed to produce a nystagmus preparedness.

The reason why the authors could not find any nystagmus preparedness can best be understood by a study of the end position nystagmus (p. 57). In collaboration with Sommer, I have shown that the nystagmus often preceding or succeeding a minimum stimulation is of a nonvestibular origin and has nothing to do with the caloric nystagmus.

### PERIPHERAL AND CENTRAL EXCITABILITY

Efforts have been made to differentiate the peripheral from the central excitability of the labyrinth. Abels and Bárány considered the *duration*

of the caloric nystagmus as an expression of the central excitability, while according to Kiproff, Beck, Brunner, etc., the *latent period* of the nystagmus is particularly related to the peripheral excitability. Brunner therefore applies to changes in the peripheral part the term "hyper- (hypo-) irritability," and to changes in the central part the term "hyper- (hypo-) sensitivity." The former designation includes all changes from those in the external auditory meatus up to those at the entrance of the vestibular nerve into the brain, whereas the latter includes all lesions in the central pathways.

### DETERMINATION OF LABYRINTHINE THRESHOLD

It has been stated above that an exact determination of the physiologic threshold of the labyrinth for caloric stimuli is very difficult. Too many unknown elements are involved. One such unknown factor—the extralabyrinthine—relates to the cooling of the various tissues; another—the labyrinthine—to the stimulation; and a third—the retrolabyrinthine—to the conductivity. Since the peripheral excitability is expressed by the latent period, it appears clear that the latent period is constituted by certain components (Frenzel, Lorente de Nó, etc.). One component is called the physical, the other the physiologic latent time.

Efforts have been made to separate these two components from each other in order to improve the accuracy of the quantitative tests. This should be accomplished in such a way that irrigation is performed with the canal in the *pessimum position.* After a waiting period of one and a half to two minutes, e.g., the time required for cooling of the various tissues (Veits), the head of the patient is brought into *optimum position.* This is done to elicit nystagmus and to determine the latent time. The resulting figures express the true or physiologic latent period. If for instance the head is bent forward at an angle of 30°, the horizontal canal is in such a position that no circulation of endolymphatic fluid occurs during calorization. This is called "first pessimum position" (Bruenings) or "anterior indifferent position" (M. H. Fischer). Any change in this position leads to a stimulation of the horizontal canal. The reaction will be more intense the more the head departs from the indifferent position. In bending the head backward, the climax is reached after an inclination of 90° (optimum position). An irrigation with cold water with the head between pessimum and optimum positions leads to an ampullofugal flow that in turn elicits a nystagmus to the side of the nonirrigated ear. When bending the head is continued beyond the optimum position, the caloric reaction becomes less and less extensive and disappears entirely after an inclination of 90° is reached again ("second pessimum position" or "posterior indifferent position"). An irrigation with cold water with the

head between the optimum and the second pessimum position leads to an ampullopetal flow that in turn elicits a nystagmus to the side of the same ear. The distance between the two pessimum positions amounts therefore to 180°.

### WARM AND COLD STIMULATIONS

Bárány was the first to stress the fact that examinations for caloric reaction must include warm as well as cold stimuli. When Ruttin examined patients under general anesthesia or patients with loss of consciousness, he demanded repeated tests with cold and with warm water before a diagnosis of the labyrinthine excitability was made. In the literature many cases are described in which erroneous diagnoses were made because only cold stimuli were employed.

Experimental examinations in rabbits (Dusser de Barenne) have revealed that after extirpation of a cerebral hemisphere the caloric nystagmus toward the operated side was much more intensive than that toward the other side—regardless of whether cold stimuli were employed on one side or warm stimuli on the other. If for instance the right hemisphere of the cerebrum had been resected, a caloric nystagmus toward the right side was much more easily elicited either by cold irrigation of the left ear or by warm irrigation of the right ear. It was, therefore, not due to a hyperirritability of one labyrinth with respect to cold and warm stimuli but rather to a preparedness of nystagmus in a certain direction. Similar experiments were carried out by Wilson and Pike, De Kleyn and Versteegh, etc.

The experimental studies were proved by clinical observations. This phenomenon was found by Eagleton, Grant and Fisher, Ruttin, etc., in cases of brain tumor, by Veits, Guettich, etc., in diseases of the frontal lobe and in trauma, by Brunner in connection with supratentorial tumors, and by Werner in cases of disseminate sclerosis, vascular lesions, tumor of the medulla, etc.

Vogel called attention to systematic examinations for this phenomenon, which he considered as an expression of central tonus differences and which he called "warm-cold contrast reaction." According to Brunner, this symptom indicates a lesion of the centers or pathways of the conjugate eye movements. He applied the term "oculomotor hyperirritability" (p. 78). This explanation appears to me very likely, since all these cases show normal excitability of the peripheral as well as the central vestibular organ.

The phenomenon has attracted the attention of numerous clinicians in the various countries, as can be seen from the varying nomenclature applied to it, including such terms as warm-cold contrast; central tonus

difference; nystagmus preparedness; caloric crossed asymmetry; oculomotor hyperirritability; and *hemiavestibulie lateral homonym* (Barré).

### 3. Bilateral Calorization

#### CLINICAL IMPORTANCE

When Ruttin first performed the bilateral calorization, he had the following considerations in mind. In the overwhelming majority of cases showing disturbances of the labyrinthine excitability, only one vestibular apparatus is involved. But even when both vestibular apparatus are affected, one side is usually more involved than the other, this in turn leading to a difference in excitability on the two sides. He found cases in which after bilateral calorization with cold water the nystagmus was directed toward the labyrinth with the lesser irritability. In normal conditions, and in cases of lesion with symmetric excitability of the labyrinths, usually no nystagmus at all occurred.

Bárány explains these facts in the following manner. When both labyrinths are stimulated in the same degree, the innervation in the central vestibular nuclei is neutralized, so that no impulses at all can be sent to the eye muscles. Such an interpretation sounds very logical but cannot explain the cases in which often nystagmus cannot be elicited by bilateral calorization when the patient looks straight ahead, but does occur when the subject looks to either side (nystagmus of the first degree). This would mean that, in spite of the counteracting stimuli, impulses are sent from the central nuclei to the eye muscles.

Brunner introduced bilateral calorization as a clinical test into otoneurology. He uses it to differentiate lesions in the posterior cranial fossa from lesions in the anterior and middle fossa. The method gives valuable results, particularly in connection with processes accompanied by increased endocranial pressure (supra- and infratentorial tumors).

#### THEORIES OF THE METHOD

*Labyrinthine hyperirritability.* It is extremely rare for any lesion in the posterior fossa to irritate both central nuclei in corresponding regions and in the same degree. Therefore, in an overwhelming majority of cases, bilateral calorization must lead to a different stimulation of the vestibular nuclei. There cannot be a neutralization of the stimuli as in normal cases. The vestibular nuclei therefore will send different impulses to the eye muscles, resulting in a nystagmus in a certain direction (positive reaction).

*Oculomotor hyperirritability.* The theory just mentioned can be used to explain the positive reaction in lesions of the posterior cranial fossa. But why do processes in the anterior or middle fossa yield a negative reaction, in spite of the existing hyperirritability?

Brunner made the following hypothesis. In cases of supratentorial tumors the labyrinthine function is usually normal. The so-called hyperirritability is not labyrinthogenic but due to lesions in the cortical optical centers. He therefore proposes the term oculomotor hyperirritability. An irritation, e.g., of the cortical center in the left hemisphere (center for voluntary movements of the eyeballs to the right side) causes a contraction of the right external and the left internal rectus muscle. At the same time a relaxation of the left external and the right internal rectus takes place. On irrigation of the left ear with cold water, the quick component of the nystagmus to the right would be increased, and the slow component to the left decreased. This would lead to the diagnosis of a hyperirritability of the left labyrinth. On irrigation of the right ear with cold water, the contrary effect would be seen, namely, a decrease of the quick component of the nystagmus to the left, which in turn would be considered as a hypoirritability of the right labyrinth. As a matter of fact, it is neither a hyper- nor a hypo-irritability, but is due to an affection of the cortical innervation for the eye movements. The labyrinthine function is usually normal. If therefore a bilateral calorization is performed, the stimuli from both sides become neutralized in the central vestibular nuclei and no impulses at all are sent to the eye muscles (negative reaction). Brunner cites in support of his theory the experimental examinations of Wilson and Pike, Bauer and Leidler, Dusser de Barenne, and De Kleyn. These authors observed in rabbits, after extirpation of one hemisphere of the cerebrum, a nystagmus toward the operated side, regardless of the manner of stimulation. In all these cases the bilateral calorization yielded a negative reaction. Brunner also refers to the clinical observations made by Eagleton, Ruttin, Grant and Fisher, etc.

*Elicitation of vertical nystagmus.* Some authors (Bárány, Ruttin, Brunner, M. Fischer) observed vertical nystagmus when the bilateral calorization was performed. According to Fischer, an upward nystagmus is elicited when the head of the patient is bent backward, while a downward nystagmus is produced when the head is bent forward. Bilateral calorization with the head in the indifferent position does not reveal any nystagmus at all. Bárány explained the vertical nystagmus in the following way. On bilateral calorization, the two components (horizontal and rotatory) become neutralized, while the third component (vertical) remains intact. Ruttin described a case of aneurysm of the internal carotid with vertical nystagmus after bilateral calorization, and attributed it to a hyperirritability of the sagittal semicircular canals. These contradictory reports show that a satisfactory explanation of the vertical nystagmus has not yet been found. Further studies are necessary.

## 4. Turning Reaction

### ETIOLOGY

Turning of the head or body represents an adequate stimulus for the peripheral sense organ (labyrinth). As effective factors we must assume the positive angular acceleration in the beginning of turning (acceleration at start) and the negative at the moment of stopping (retardation at stop). For elicitation of the reaction the deflection of the cupula is responsible. If therefore the peripheral sense organ is destroyed, a reaction cannot occur. In contrast to this, inadequate stimuli, e.g., galvanic current, do not require an intact sense organ, since such stimuli may act directly upon the nerve stem.

The various reactions elicited on turning a subject can be classified into subjective reactions such as turning sensation, vertigo, nausea, etc., and objective reflexes such as nystagmus, falling, past-pointing, etc. Since these various reactions are all elicited by movements of the endolymph, the general physiologic laws (Mach-Breuer, Ewald, Purkinje, Flourens, p. 29) should be applied. Five main rules are here briefly presented:

1. *Mach-Breuer:* The semicircular canal, which lies at an angle of 90° to the turning axis, is the one most likely to be stimulated.
2. *Flourens:* Each semicircular canal elicits a nystagmus in its own plane. Hence, horizontal nystagmus is produced by the horizontal canal, rotatory nystagmus by the frontal canal, vertical nystagmus by the sagittal canal.
3. *Ewald,* law 1: For the horizontal canal, ampullopetal flow of the endolymph is much more effective than ampullofugal, while for the vertical canals the opposite is true.
4. *Ewald,* law 2: The effective endolymphatic flow produces a nystagmus to the same side.
5. *Purkinje-Bárány:* The postrotatory nystagmus is dependent solely upon the position of the head *during* rotation and is not influenced if the subject *after* turning brings his head back to the normal position.

### ANATOMIC-PHYSIOLOGIC FACTORS

Although most of the reactions on turning can easily be explained by the physiologic laws outlined above, some problems remain still unsolved. One difficulty can be attributed to the fact that the real anatomic conditions in man do not correspond entirely with those assumed theoretically. Thus the horizontal canals do not lie in a true horizontal plane, neither does the frontal canal lie in a frontal nor the sagittal in a true sagittal plane. Between the horizontal canals and the horizontal plane there is an angle of 23° to 25°, opening downward and backward. Hence the head of the patient should be bent forward at 20° to 30° for testing the hori-

zontal semicircular canals. But even in such a position the canals are not brought entirely into the horizontal plane, since their axis does not represent a straight horizontal line, but is deflected downward. The two vertical canals lie just between the sagittal and the frontal axis, thus forming an angle of 45° with the horizontal plane.

The three semicircular canals, although they correspond to the three dimensions, do not form an angle of exactly 90° in relation to one another. The average figures are as follows: between the superior vertical (frontal) canal and the inferior vertical (sagittal) canal there is an angle of from 83° to 85°; between the superior vertical canal and the horizontal canal there is an angle of from 83° to 85°; between the inferior vertical canal and the horizontal canal there is an angle of from 80° to 92°. While it is generally accepted that the horizontal canals produce a horizontal nystagmus and the frontal canals a rotatory nystagmus, great dissension exists as to which canals are responsible for the origin of a vertical nystagmus. The various theories can be classified into three groups, according to the following views:

1. There is a cooperation between the frontal canal of one side with the sagittal canal of the other side, resulting in a vertical nystagmus (Breuer, Wittmaack, Ohm, Fischer).

2. There is a cooperation between the two frontal canals or the two sagittal canals. The former leads to a rotatory nystagmus, the latter to a vertical nystagmus (Ruttin, Schilling).

3. For a vertical nystagmus, only the direction of the endolymphatic flow of the two vertical canals of one side is responsible. An ampullopetal flow in one canal and an ampullofugal flow in the other produces a vertical nystagmus upward (E. R. Lewis, Fisher, Jones, Favill). Tait and McNally differentiate functionally between the vertical canals (gravity set) and the horizontal (nongravity set). The former are responsive to tipping displacements of the head and work in close association with the utricular maculae; the latter respond to rotation about a vertical axis. This theory, however, is not corroborated by the experiments of Sand, who arrived at the following conclusions. The activity of the horizontal canals is confined to planes near the horizontal. The vertical canals are active in all planes, and work together in antagonistic pairs, but the pairs are different and specific for each of the three primary axes of rotation.

The various phenomena of the turning reaction become more understandable on the basis of the five main laws set forth above. For example:

A subject with the head bent forward 30° is turned around a vertical axis. In this position both horizontal canals lie approximately in the horizontal plane. FIGURE 19 illustrates a turning to the right (clockwise). Owing to the physiologic law of inertia, the endolymph does not move as

fast as the solid walls of the semicircular canal; through this lag, a regressive movement of the endolymphatic flow will result (line arrows). In this figure we can compare the direction of flow of the endolymphatic fluid on each side of the labyrinth, noting on the right side the ampullopetal flow, on the left side the ampullofugal. According to Ewald's law for the horizontal canals, the ampullopetal flow is the really effective one. Therefore during turning the nystagmus goes to the right side. If turning is then suddenly stopped, everything is inverted (dotted arrow), the ampullopetal flow is in the left ear, the ampullofugal in the right; this results in a nystagmus to the left side. In other words, the perrotatory (during turning) nystagmus goes to the same side as the turning, the postrotatory (after turning) nystagmus goes to the opposite side.

In spite of the fact that both labyrinths are irritated by turning, the more effective stimulation is employed only on the side where an ampullo-

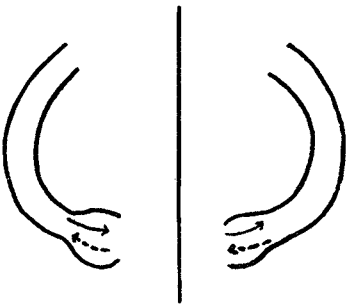

FIG. 19. DIAGRAM OF LEFT AND RIGHT HORIZONTAL SEMICIRCULAR CANALS

Line arrows = direction of endolymphatic fluid during rotation to right. Dotted arrows = direction of endolymphatic fluid after cessation of right turning.

petal flow takes place—that is, *during* turning toward the side of the same ear, *after* turning toward the opposite side. As we use for clinical examination only the after-turning reaction, we can put it in this way: After having turned a subject to the right, the left labyrinth is particularly tested. After turning to the left, the right labyrinth is particularly tested.

A very interesting experiment on this subject was described by Dryfuss. Four guinea pigs were put on a turning apparatus. One animal was normal, one had a labyrinth destruction of the right side, one of the left, and the last one a bilateral loss of labyrinthine function. For all four guinea pigs, fodder was placed on the turning apparatus. First they were turned to the right side (clockwise). The normal animal stopped eating, the one with bilateral loss of function tried to eat but could not, the one with the loss of the right labyrinth kept on eating, while the one with the left labyrinth loss stopped eating. On turning to the left, the first two animals showed the same reaction, while the one with the right labyrinth

loss stopped eating, and the other kept on. This experiment illustrates the fact that during turning the labyrinth of the side corresponding to the direction of turning is the one that is stimulated most. Any single pair of the three semicircular canals can be tested as above described if it is brought into a horizontal plane in order to get the most effective stimulation.

### RHYTHMIC PHASES OF POSTROTATORY NYSTAGMUS

It is generally assumed that the stronger the stimulus, the stronger will be its effect. Applied to the turning reaction, this would mean that with increase in number and speed of revolutions, the intensity and duration of the rotatory nystagmus should also be increased. However, Bárány observed that the rotatory nystagmus has its greatest duration after 10 revolutions within 20 seconds. Any stimulus above or below this number leads to a nystagmus of shorter duration. Buys found that the height of stimulation is reached after 16 to 17 revolutions. If the subject is then still turned steadily, the nystagmus will subside entirely after the seventeenth revolution. If steady turning is further continued, a nystagmus to the opposite side is elicited which may last as long as 22 degree seconds.

Buys called this "inverted turning" nystagmus. A similar observation was made by Bárány. He called the nystagmus observed after stopping of such prolonged turning "post-postrotatory nystagmus." Buys interpreted the inverted nystagmus as being of a central origin. This phenomenon of Buys remained unnoticed for a long time, until M. H. Fischer and Wodak made their thorough studies of the physiology of the turning reaction. They found that the course of the turning reaction is characterized by rhythmic phases. For example, when the turning is stopped, a nystagmus in the direction opposite to the direction of turning is elicited (first phase). This nystagmus gradually subsides (first intermission). Shortly thereafter a nystagmus in the direction of turning will occur (second phase), followed by another intermission (second intermission). There may be a third, fourth, or fifth phase. In fact, Veits observed six such rhythmic phases in some cases. Woletz made quantitative determinations of the rhythmic phases, the average figures being: for the first phase, 28 seconds and 48 movements, i.e., a frequency of 1.6; for the first intermission, 15 seconds; for the second phase, 60 seconds and 37 movements. Not only the nystagmus but also the other reactions, such as turning sensation, show such a rhythmic course.

### PHENOMENON OF INTERFERENCE

M. H. Fischer and Wodak do not consider the turning test, performed in the usual manner, as being an exact physiologic method. Their argu-

ments are the following. On turning a subject, the angular acceleration acts as a stimulus; on stopping, the retardation represents a counterstimulus. In the usual test of turning reaction, the first stimulus is still present when the second stimulus (counterstimulus) is applied by the stopping. This in turn leads to an interference of the stimuli with each other. In order to get exact results, particular care has to be taken to separate strictly the effects of one from those of the other. This can be accomplished (*a*) if turning is begun with the lowest possible speed, and (*b*) if steady turning (without acceleration) is continued for a certain period of time (approximately 3 minutes). Under such circumstances the first stimulus will be eliminated when the counterstimulus is applied; thus no interference can take place.

### THEORIES OF THE SHORT-TURNING METHODS

The turning test as introduced clinically by Bárány employs stimuli that far exceed the physiologic threshold of the labyrinth (maximum stimuli). Efforts have been made to find a method to make the test more physiologic by using stimuli less intensive (minimum stimuli). This can be accomplished either by lowering the speed of turnings or by decreasing the number of revolutions. Bruenings observed nystagmus after two revolutions, while Bárány was able to elicit nystagmus even after one or half a revolution, examining behind opaque eyeglasses. Grahe worked out a method of palpating nystagmus through the closed eyelids, after turning a patient only 90° (p. 136). Finally, Buys has reported eliciting a nystagmus by turning a patient only 20°, which means the eighteenth part of one full revolution.

However, it becomes more and more unlikely that a nystagmus observed with such methods has anything to do with a vestibular reaction. Cemach and Kestenbaum made large-scale examinations on normal persons and on deaf-mutes. They first employed the same technic as the other authors, namely, stimulation with a minimum effect and examinations for nystagmus behind opaque or convex eyeglasses. With this method they succeeded in getting a nystagmus not only in normal persons but also in deaf-mutes, when no labyrinthine function at all was present. This is proof that the nystagmus observed was of a nonlabyrinthine origin.

The authors then repeated the examinations, but with a change in technic. In order to eliminate the fixation, they used, instead of opaque eyeglasses, a dark room with a dim red light coming from a screened lamp placed laterally. The results thus obtained were quite different from those obtained before. Such "short turnings" did not cause any eye movements at all. This was another proof that the nystagmus observed by various authors after short turnings, has nothing to do with a vestibular

reaction. The nystagmus must be considered as due to the mechanism of fixation.

### QUANTITATIVE DETERMINATION

Since the short-turning methods have failed to fulfil the expectations of an exact physiologic test, efforts have been made to improve the long-turning methods for quantitative determinations. In order to prevent technical errors, an electric turning chair was constructed, which has the following advantages. Turning can be started with a stimulus below the physiologic threshold and can gradually be increased, thus permitting determination of the degree of stimulus. In the same way, turning can be stopped at any given speed—for example, at 20, 30, 60 degree seconds, etc. Such chairs were constructed by Buys-Reylant, Fischer and Toenius, Guettich, etc. The use of them permitted quantitative determinations of the physiologic threshold for turning stimuli, of the duration of the nystagmus, and of the number of jerkings, and, finally, determination of the rhythmic phases of the postrotatory nystagmus. Allard studied the question of what amount of stimulus of negative retardation of 60 seconds is required to elicit nystagmus. He started turning with a positive acceleration of 0.3 degree seconds and stopped with a negative retardation of 6 degree seconds. He found a reaction time of 0.5 to 1.75 seconds for the horizontal semicircular canals, and a reaction time of 1.75 to 2 seconds for the vertical canals. Recently, investigations on the same question were carried out by Baxter, Bent, and C. Travis with similar results, the average being between 0.196 and 1.79 seconds. They also made comparative examinations between right turnings and left turnings. The average figures for the reaction time amounted to 1.002 seconds less for left turns than for right turns. This shows that for practical purposes such a difference is negligible.

As far as the *duration* of the postrotatory nystagmus is concerned, the average figures show less variations with the long-turning methods, as compared with the figures obtained with the old Bárány method. The duration of the nystagmus represents the conditions in the central vestibular organ (cf. caloric reaction, p. 75).

Examination of the *rhythmic phases* of the postrotatory nystagmus can be used for diagnosis of the central excitability. The first phase is usually the result of two stimuli, namely, the positive acceleration and the negative retardation. The former can be eliminated as indicated above. Thus the first phase represents only the stimulation by retardation. Before the second phase occurs, no other stimulus has been employed. Hence the second phase represents the excitability of the *central vestibular* organ. Applying the terminology of Alexander and Brunner (cf. caloric reaction,

p. 76), it could be said that the first phase expresses the irritability and the second phase expresses the sensitivity in relation to turning stimuli.

## 5. Mechanical Reaction (Fistula Symptom)

### Definition and Pathology

The term "mechanical reaction" is applied because the reaction is a result of mechanical stimuli, such as compression or aspiration, which respectively lead to increase or decrease of the endolabyrinthine pressure. In order to elicit this reaction, certain pathologic changes must be present. The reaction does not occur in normal persons.

The most common changes involve the bony labyrinthine capsule when there is a circumscribed defect of its wall—fistula symptom. Such a fistula creates conditions similar to those Ewald produced experimentally in pigeons, using his pneumatic hammer. It is therefore generally assumed that the mechanical stimuli produce certain movements of the endolymphatic fluid that in turn elicit the eye movements (mechanical nystagmus). According to the law of Ewald, the direction of this nystagmus is dependent upon the direction of the endolymphatic flow. For the horizontal semicircular canals, the ampullopetal flow represents the effective stimulus, thus eliciting a nystagmus in the same direction, while an ampullofugal flow produces a nystagmus in the opposite direction. It can therefore be said, in other words, that compression causes a nystagmus in the direction of the compressed side, while aspiration leads to a nystagmus in the direction of the opposite side.

However, it must be admitted that a great number of clinical cases do not run according to the theoretic laws of Ewald. If the nystagmus follows the laws, the reaction is called "typical positive reaction"; if the nystagmus is of opposite nature, it is called "inverted positive reaction" or "paradoxic positive reaction." Between these two extremes numerous combinations are possible: the compression may run typically while aspiration is inverted, or vice versa; or compression only may produce nystagmus while aspiration has no effect, or vice versa; finally, the nystagmus may have only a slow component in the one or the other direction, etc. Ruttin described twenty-six different reactions and applied a name to each one. Neumann tried to locate the site of the fistula by the direction of the mechanical nystagmus. Reports in the literature concerning the percentages of typical and paradoxic reactions differ to a great extent: the highest average figure quoted for typical reaction is 65 per cent, that for paradoxic 35 per cent.

I think that the whole question is too much overemphasized. It is not the direction of the nystagmus that is important for the diagnosis, but only the fact as to whether or not eye movements can be elicited. If they can

be produced the reaction is positive, otherwise it is negative. No other conclusion should be drawn.

Mygind, Collet, Mayoux, and others, consider the fistula symptom an otolithic reaction to an adequate irritation. According to Lund, the fistula sign is caused partly by a displacement of the cristae and partly by the general pressure effect on the static sensory epithelium of the labyrinth. The mechanical stimuli may also in certain cases cause other vestibular symptoms, such as vertigo, falling, etc.

#### PSEUDO FISTULA SYMPTOM

It has been stated above that a fistula in the bony capsule of the labyrinth is responsible for the elicitation of a mechanical reaction. However, there are certain cases presenting a positive fistula symptom without presence of a fistula. This phenomenon is called pseudo fistula symptom and is explained in different ways. One theory is that there is a decrease of the threshold for mechanical stimuli (Alexander-Lassalle), or an increase of the motility of the content of the labyrinthine windows (Alexander-Hennebert). In cases of inner-ear lues with normal drum findings, this pseudo fistula symptom may be caused by the luetic inflammation of the eighth nerve, which leads to increased conductivity for mechanical stimuli (Alexander).

There are some other rare forms of the pseudo fistula symptom; for instance, pressure upon the carotid of the diseased side elicits nystagmus toward the opposite side (vascular symptom of Mygind). Pressure on the neck veins by means of a tourniquet may give a positive fistula reaction (Borries). The same phenomenon can sometimes be observed after inhalation of amyl nitrite (Borries), occasionally even with deep inspiration (Alexander-Brown). Experimental examination with hyperventilation, by Adelsberg and Forschner, revealed an increase of the labyrinth excitability. This phenomenon belongs to the same group as those described by Alexander and Brown.

### 6. Galvanic Reaction

#### HISTORY

It was already known to Hitzig (1871) that an electric current sent through the head of a subject elicits eye movements (nystagmus). However, it was Breuer who first recognized the vestibular apparatus as the site of origin of this phenomenon. Ewald made experimental examinations in pigeons and came to the conclusion that the galvanic current stimulates the fine endings of the vestibular nerve but does not act upon the vestibular sense organ proper.

### THE THEORIES OF GALVANIC REACTION

According to Bárány, the cathode applied on the mastoid produces a catelectrotonus in the vestibular nerve, which in turn leads to an increase of its excitability. On the other hand, the anode applied on the mastoid produces an anelectrotonus, thus decreasing the excitability of the vestibular nerve.

Bruenings opposes any theory of a stimulation of the nerve on the basis of the following arguments. The galvanic current is a constant stimulus, and according to physiologic law a nerve can be stimulated only by an alternating electric stimulus. Furthermore, the direction of the galvanic nystagmus depends upon the direction of the current, a fact which could not be explained by the theory of Bárány.

Bruenings expounded a physical theory to the effect that the galvanic current causes a cataphoresis of the mobile particles, this in turn leading to a stimulation of the sensory cells of the labyrinth. Cataphoresis or electro-endosmosis is a process in which, in an electric field, free mobile particles (blood cells, bacteria, spermatozoa, etc.) move, according to the electrical charge, either with the positive or the negative current.

The clinical significance of the galvanic reaction lies in the fact that it facilitates differentiation between lesions of the lymphokinetic apparatus and lesions of the nerve apparatus. Examples of lymphokinetic lesions are: traumatic and operative injuries of the labyrinth associated with a drainage of endolymphatic fluid; deformities of the semicircular canals; stenosis of the canal lumen due to diseases of the walls, or to scars, etc. Examples of nerve lesions are affections involving either the fine nerve endings of the cristae or the maculae of the labyrinth or the nerve stem.

Marx made large-scale examinations in guinea pigs. He first plugged the various semicircular canals and obtained a normal galvanic reaction. Then he destroyed all the canals and still secured the same positive reaction. Finally he destroyed the entire labyrinth and even then was able to show a positive galvanic reaction. He therefore opposes the theory of Bruenings that a galvanic current acts upon the sensory cells of the labyrinth. These results obtained by Marx in animals were not surprising to the clinicians, since Neumann, Ruttin, and Bárány long ago made similar observations in man. Neumann has reported a case in which a complete resection of the labyrinth was performed. He found on functional testing that the caloric, turning, and mechanical reactions were negative, while the galvanic test yielded a positive result. Such observations in patients led the clinicians to the assumption that the galvanic reaction represents an important test for differentiation between labyrinthine and retrolabyrinthine (vestibular nerve) diseases.

Mackenzie found in cases of unilateral destruction of the labyrinth a

decrease of the galvanic nystagmus toward the affected side. He could show further that the direction of the nystagmus can be influenced by the electric current in spite of a complete destruction of the peripheral sense organ. Brunner believes that anode as well as cathode can stimulate the nervous apparatus of the ear. He tried to prove it by use of the combined (galvanic and caloric) test (p. 143).

A few other theories concerning the site of galvanic stimulation may briefly be mentioned. It has been held that the electric current acts upon the vestibular ganglion in the internal auditory meatus (Dohlman); that the galvanic reaction originates in the terminating fibers of the ampullar nerve (Shapiro); that there is a direct stimulation of the peripheral nerve and its central pathways (Blonder). Northington and Barrera made experimental examinations in monkeys, severing the eighth nerve and also destroying the labyrinth. In the very beginning the galvanic reaction was elicitable, but it gradually decreased until no reaction at all was present. On histologic examination they found a degeneration of the nerve stem.

The numerous theories mentioned above reveal the fact that a definite solution of the problem of the galvanic reaction has not as yet been found. It seems likely that the peripheral sense organ as well as the nerve stem can be considered as the site of the galvanic stimulation. However, there still remain unknown some factors influencing the galvanic reaction, such as the conducting power of various tissues (skin, subcutaneous tissue, bone, etc.), resistance, distribution of current, change in current, polarization, etc.

### CHRONAXIA

In recent years many authors (especially in France) have become more and more interested in quantitative determinations of the electric stimulation. Particular attention has been given to the determination of the so-called "vestibular chronaxia." It would go far beyond the scope of this book if all the electrophysical problems involved were here discussed. The reader is referred to the original articles. Only some definitions will be given. The threshold after the closing of an electric current is called the *rheobasis*. The period of time required for an electric current with an intensity of two rheobases to pass through a circuit is called the *chronaxia*. It represents therefore the period of time required for the same reaction with double rheobasis. Such determinations were carried out for the various nerves of the central nervous system. Bourguignon, examining the vestibular nerve, found that the chronaxia of the vestibular nerve was very high as compared with that of the other nerves. The average figures were between 12 and 22 $\sigma$, while Altenburger obtained an average of from 2 to 10 $\sigma$. The latter author also found that caloric and turning stimuli lead to a transient decrease of the vestibular chronaxia.

He explains it on the basis of a "preparing" influence of these stimuli upon the galvanic reaction.

Ferreri and Medolesi used the determination of chronaxia for differentiation between central and peripheral lesions. If the peripheral sense organ was diseased, the chronaxia was decreased by one-fifth, while if the central vestibular pathways were involved, normal chronaxia was found.

Determination of vestibular chronaxia may also be of clinical value in relation to lesions of the vestibulospinal tract (syndrome of Barré), for differentiation of the various cerebrospinal reflexes, and, finally, for differentiation of vestibular from nonvestibular reflexes.

### 7. Tonic Reflexes and Reactions

It is very difficult to give an exact definition of "tonus," since a uniform version of this term does not exist. According to Spiegel, tonus represents a permanent stage of tension of the body musculature, maintained without voluntary innervation, and serving the purpose of controlling posture and the mutual positions of the various parts of the body.

Ewald must be considered the founder of the theory that the labyrinth controls the tonus of the body musculature; in this connection he applied the term "tonic labyrinth." He assumed that from both labyrinths impulses emanate to the musculature of the body. He proved experimentally that each labyrinth is concerned with the tonus of the homolateral extensors and abductors and with that of the contralateral flexors and adductors. Resection of one labyrinth has a crossed effect upon the muscles that move head and spine. Impulses emanating from the labyrinths are transmitted to the centers and central pathways, thus reaching the muscles.

Bickel, on the basis of experimental examinations, came to the conclusion that the labyrinths maintain a certain tonus of the musculature. In opposition to Ewald, he observed that this tonus exists also during the stage of rest. Marikovszky found a connection between the labyrinth and the musculature of the neck; for instance, the right labyrinth influences the muscles that move the neck to the left, and the left labyrinth controls neck movement to the right. E. Pollack differentiates between physiologic and clinical tone and between static and kinetic tone. Bárány explained the decrease of muscle tonus after resection of a labyrinth as analogous to the symptoms found after severing any sensory nerve.

Hoegyes (1881) first described the eye muscle tonus. On the basis of his interesting experimental work he came to the following conclusions. Equal impulses are sent from both labyrinths to the eyeballs, with the result that under normal conditions a certain balanced position of the eyeballs is maintained. A unilateral destruction either of the centripetal pathways (labyrinth, vestibular nerve) or of the conjugate centers causes

a disturbance of balance so that the eyeballs lose their primary position. Owing to the fact that the tonus of one side predominates over that of the other, a deviation of the eyes occurs.

Bartels came to similar conclusions. He also believed that the semicircular apparatus exerts an influence upon the tonus of the eye muscles. Each labyrinth shows a continuous preparedness to elicit nystagmus toward its own side.

The compensatory positions of the eyes were already known to the older otologists (Breuer, Ewald, Bárány, etc.). However, a proper explanation for this phenomenon could not be given in their time. Arellano has recently made studies of the kinetovisual functions of the labyrinth. He found that the compensatory eye movements (*Gegenrollung*) play a great role in the immobilization of the optic image during inclination of the head (p. 40).

The classic experiments of Sherrington brought about great progress in respect to the entire problem of tonus. He severed the brain stem caudally from the anterior parts of the quadrigeminal bodies and observed the following phenomenon. All the extensor muscles were in an extreme state of rigidity, with the result that the animal stood in a peculiar posture. The slightest push was sufficient to overthrow the animal. Any change in position, as for instance of the head, caused a change in the tonus of certain groups of muscles. Sherrington called this phenomenon "decerebrate rigidity."

The importance of the phenomenon lies in the fact that with elimination of the voluntary movements it is much easier to study the various problems of tonus, such as distribution, mutual influence, posture, etc. Magnus and De Kleyn based many experiments on this phenomenon in their study of tonic labyrinthine reflexes, of righting reflexes, and of neck reflexes (see below). One of the reasons why knowledge of the tonus problem is farther advanced in relation to animals than it is in relation to man may lie in the fact that in the case of man decerebration cannot be used for such studies.

The anatomy of the central pathways is still in dispute. According to Magnus and De Kleyn, there are two different centers, one in the medulla oblongata (extensor center), and one in the nucleus ruber (flexor center). Impulses from both centers to the spinal cord act like two reins controlling the tonus of the musculature. Other authors consider the central vestibular region, particularly the nucleus of Deiters, as the center of tonus. Ingram and Ranson have shown that the nucleus ruber is not the only center of the labyrinthine righting reflexes. Bernis and Spiegel believe that the substantia reticularis is also responsible for the decerebrate rigidity. It is impossible to discuss the numerous theories about the

central pathways. Although an exact localization of the centers has not as yet been achieved, one conclusion can be drawn from all these experiments, namely, that there are close connections between decerebrate rigidity and the function of the central vestibular apparatus.

Intensive research work on the part of Magnus and De Kleyn and their followers shed new light upon the problem of tonus. Their interesting and instructive experiments revealed new reactions and new points of view. The numerous reflexes and reactions have been classified by these authors in the following groups:

A. *Postural reflexes (otolith reflexes)*
   1. Tonic labyrinthine reflexes
   2. Labyrinthine righting reflexes
   3. Compensatory eye positions

B. *Movement reflexes (semicircular reflexes)*
   1. Angular movement reflexes
   2. Linear movement reflexes

C. *Reflexes upon inadequate stimuli*
   1. Reflexes upon thermic stimuli
   2. Reflexes upon galvanic stimuli

The tonic reflexes and reactions are here discussed on the basis of this classification.

### A. POSTURAL REFLEXES (OTOLITH REFLEXES)

These reflexes are produced by the otolithic apparatus and depend upon the position of the otoliths with respect to the horizontal plane. Hence they represent true labyrinthine reflexes. From the otolithic maculae constant impulses are sent out, so that the reflexes persist as long as the given position of the head is maintained. The fact that all these reflexes in animals are elicitable even when the cerebrum has been completely removed shows that they are independent of conscious sensations.

1. *Tonic labyrinthine reflexes.* These reflexes are identical with the "postural reflexes" of Sherrington. They enable the individual to bring parts of his body into harmonious positions and also to maintain them in these. These reflexes can best be studied in decerebrate animals. The labyrinths have, according to Magnus and De Kleyn, a direct influence not only upon the tonus of the musculature of the extremities but also upon that of the neck and trunk muscles. Besides the primary influence of the labyrinth upon the musculature of the neck (tonic labyrinthine reflex), there is also a secondary influence of the neck muscles upon the muscles of the extremities ("neck reflex").

It must be borne in mind that each labyrinth influences the musculature

of the neck unilaterally, while the musculature of the extremities is bilaterally influenced. The tonic labyrinthine reflexes are elicited by a change in the position of the head with respect to space, while the neck reflexes are caused by changes in the position of the head with respect to the body. The tonic labyrinthine reflexes in conjunction with the neck reflexes are responsible for the fact that to any given position of the head there corresponds a certain position and posture of the rest of the body.

In order to test the tonic labyrinthine reflexes, the neck reflexes must first be eliminated. Some authors (Spiegel) advise severing of the posterior roots of those sensory nerves that supply the muscles of the neck. Others put a bandage with plaster of paris on the head and neck of the animal to prevent a change of position.

To test the tonic labyrinthine reflexes upon the extremities, the animal is held by the hind legs, the body and head swinging freely. In this position the tonus of the fore legs is very slight. Now the animal is turned over a frontal axis back to the floor so that the mouth lies 55° above the horizontal plane. After a period of ten to twelve seconds, the tonus of the extensor muscles of the fore legs has reached its maximum.

In the tests for neck reflexes, the animal is first placed on a table, lying on its belly. When now the head is raised (dorsoflexion), the tonus of the extensor muscles increases in the fore legs and decreases in the hind legs, i.e., the animal gets up. Lowering of the head (ventroflexion) leads to the opposite effect.

The animal is next placed in the same position. On turning the head to the right, the tonus of the extensor muscles decreases on the same side (right fore and right hind leg); while on the left side (fore and hind leg) an increase of the tonus can be noticed.

2. *Labyrinthine righting reflexes.* All those reflexes that enable the animal actively to regain its former normal position and to maintain it are called labyrinthine righting reflexes. These reflexes can no longer occur either when both labyrinths are destroyed or when the brain stem is severed just in front of the anterior part of the quadrigeminal bodies. Hence, for elicitation of the labyrinthine righting reflexes, the labyrinths (otolithic membrane) as well as the nucleus ruber (Rademaker) must be intact. In case of unilateral destruction of a labyrinth, the righting reflexes act, according to Magnus and De Kleyn, in the following manner. The reflexes tend to bring the head of the animal into such a position that the intact labyrinth lies on top, while the destroyed one is underneath. This is the position in which the labyrinthine righting reflexes are at their minimum, while with the opposite position their maximum is reached. In the latter event, the animal tries with all its efforts to regain the "minimum position," which is identical with the position at rest. Magnus and

De Kleyn differentiate as between symmetric and asymmetric righting reflexes. The latter are considered to be sacculus reflexes, the former utriculus reflexes, since they are elicitable even after experimental destruction of the sacculus. Recent examiners, however, do not agree with this theory.

The maximum effect of the otolithic function is seen when the otolithic membrane—lying in a horizontal plane—is hanging down from the macula, while the minimum effect occurs when the otolithic membrane presses upon the macula.

In testing for labyrinthine righting reflexes, the animal is first lifted up, held by its haunches. When then the trunk is turned to the side, the animal brings its head into normal position.

The animal is next placed on a table in lateral position. When the head is held by the examiner's hand, the animal tries to get up by means of its trunk.

The animal is then lifted up and held on its side. In this position the head also remains in lateral position. When the animal in this same lateral position is placed on the table, it immediately tries to bring its head into normal position.

Another postural reflex has recently been described by Hyndman under the term "prepared posture reflex." This reflex "prepares the person to meet an expected change in position or acceleration." The author uses as an example a person standing on the platform of a streetcar and fully aware that the car is going to move. This reflex, which elicits countermovements for the maintenance of balance, may use corticocerebellar pathways.

3. *Compensatory eye positions.* Two types of eye reflexes are elicited by the labyrinth, i.e., vertical deviations and counterrolling. The former are caused by changes in the position of the head over a nasooccipital axis, the latter by changes over a bitemporal axis. Magnus and De Kleyn state that to any given position of the head in space, a certain tonus of the eye muscle corresponds, resulting in a certain position of the eyeballs.

*Vertical deviations* are usually due to action of the superior and inferior rectus muscles. When, for instance, the head of the animal is turned to the right, a maximum contraction of the right superior rectus and the left inferior rectus will result, while at the same time a relaxation occurs in the left superior rectus and right inferior rectus. This in turn leads to a maximum deviation of the right eye upward and of the left eye downward. Vertical deviation cannot occur either when both labyrinths are resected or when the otolithic membranes are thrown off. It is, therefore, assumed that the compensatory eye deviations are otolithic reflexes. Magnus and De Kleyn consider them as sacculus reflexes.

*Counterrolling* is usually effected by both oblique muscles. When the head of the animal is turned over a bitemporal axis, for instance upward, a maximum contraction occurs in the right and left superior oblique muscles, while at the same time both inferior oblique muscles become maximally relaxed. These reflexes cannot be elicited either when both labyrinths are resected or when the otolithic membranes are thrown off. According to Magnus and De Kleyn, the counterrolling represents neither utricular nor saccular reflexes. Its origin is not known to date.

In order to test for compensatory eye positions, the animal is first turned over an occipitonasal axis, for instance to the right. This results in a maximum deviation upward of the right eye and a maximum deviation downward of the left eye. Turning the head to the left has an opposite effect.

The animal is then turned over a bitemporal axis, for instance with the snout upward. Both eyeballs now roll forward. Turning the head downward leads to an opposite effect, i.e., rolling of the eyeballs backward.

For evaluation of the significance of the compensatory eye positions, a differentiation has to be made between animals with laterally placed eyes and those with frontally placed eyes. Further details may be found in the original articles on this subject.

### B. MOVEMENT REFLEXES (SEMICIRCULAR REFLEXES)

1. *Angular movement reflexes.* Reflexes upon the neck are tested as follows. When an animal in normal position is turned over a vertical axis in space, a deviation of its head in the horizontal plane will occur. Occasionally a head nystagmus can be noted. During turning, the deviation is in the opposite direction, while the head nystagmus shows the same direction as the turning. When turning is stopped all reactions become reversed. The deviation of the head is due to the fact that the animal tries during turning to maintain the former position of the head.

Reflexes upon the eyes are noted as follows. When an animal in normal position is turned over a vertical axis in space, deviation of the eyeballs in a horizontal plane and nystagmus will occur. During turning the eye deviation is in the opposite direction, while the nystagmus has the same direction as the turning. After turning is stopped, all reactions become reversed. The deviation of the eyeballs is due to the fact that the animal during turning tries to keep its eyes in the former position.

Reflexes upon the pelvis and extremities may be observed when an animal (monkey) is held by the head with its body swinging freely, and is turned over a vertical axis, for instance to the right. The left arm is abducted, the elbows extended, and the hand shows a grasping motion. The right arm is adducted. This leads to a grasping motion of both arms

toward the left side. The left hind leg is abducted, the right one adducted. Simultaneously a turning of 90° on the pelvis can be noted (girdle reaction).

2. *Linear movement reflexes.* Among the various reactions upon linear movements are the following.

As a test of reaction to lifting, an animal is placed in normal position on a horizontal board. When the board is lifted upward, an extreme flexion of the fore legs can be noticed. At the same time the head goes down, approaching the base of the board. When the lifting is stopped, a maximum extension of the fore legs and raising of the head occurs. Lowering of the board has the opposite effect.

The test of springing-poise reaction (*Sprungbereitschaft*) consists of holding the animal on its pelvis with the head swinging down. When the animal is lowered, the fore legs move forward and are stretched far out.

### C. REFLEXES UPON INADEQUATE STIMULI

Reactions to thermal and galvanic stimuli are discussed in chapter iv (pp. 71, 87).

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# IV

# Functional Tests

By *Joseph Fischer*

## SPONTANEOUS MANIFESTATIONS

### 1. Vertigo

#### TYPES OF LABYRINTHINE VERTIGO

Labyrinthine vertigo can be classified, according to Alexander, into three kinds, namely, physiologic, experimental, and pathologic.

*Physiologic vertigo* represents a protective measure, designed to maintain equilibrium. A sudden movement of head or body resulting in a shift of the gravity line causes vertigo. This symptom warns the body of the imminent danger of falling, thus eliciting certain reactive movements for counteraction.

*Experimental vertigo* is produced by the various labyrinthine stimuli, such as turning, caloric, galvanic, etc.

*Pathologic vertigo* is due to diseases of the labyrinth or the labyrinthine pathways. In contrast to the physiologic types of vertigo, these lesions do not produce any counteracting reactions but rather favor the falling of the subject. Pathologic vertigo can be of either a peripheral or a central origin.

#### CLINICAL HISTORY

Any functional test of the labyrinth must start with a detailed history of vertigo. As described in our chapter on the physiology, the patient reports as vertigo many sensations that from an otologic point of view often have no connection with the labyrinth at all. On the other hand, there may be true symptoms of vertigo that may be not at all related to the ear condition, but may be rather of an ocular origin. It is the task of the examiner to determine whether such a vertigo is of labyrinthogenic or nonlabyrinthogenic nature. For this purpose certain specific points should be noted in taking the history.

Reports of true *turning sensations* usually indicate a labyrinthine origin. Peripheral labyrinthine vertigo is expressed almost always by a turning sensation (semicircular canal vertigo). The central labyrinthine vertigo may be of either the turning or the tactile type. In the latter instance the patient will complain of a sensation of being pulled to one side (latero-

pulsion). In taking the history, all leading questions must be strictly avoided, but if the patient mentions turning sensations spontaneously, his statement is of great clinical importance.

Sensations of being turned around are less frequent than reports of turning of surrounding objects. A *sham movement* of the object to the right, for example, corresponds to a sham movement of the subject to the left.

Patients are often able to state the *direction* of the sham movement (clockwise or counterclockwise). Occasionally there is at the same time a spontaneous nystagmus. In such cases the sham movement of objects is in the direction of the quick component of the nystagmus.

The *influence of head movements* is disclosed in the effects of changes of position. Patients very often report occurrence of the vertigo early in the morning on getting out of bed or while dressing (flexion of the head). This is a characteristic symptom in peripheral labyrinthine lesions.

The *duration and intensity* of the vertigo may also be of diagnostic value. Peripheral labyrinthine dizziness occurs usually in the form of attacks that may last from a few seconds to some minutes. In especially acute and severe instances (rupture of the windows, hemorrhages, and trauma) these symptoms may persist for hours and even days. Furthermore, the duration of peripheral labyrinthine vertigo is always self-limited. As soon as the stage of irritation is over and the stage of destruction begins, the spells of dizziness gradually subside. Peripheral labyrinthine vertigo usually lasts for one or two weeks. If it persists longer than that, and if its intensity does not decrease, a central labyrinthine origin is indicated. The form and intensity of the attack depend on the underlying pathologic processes—whether these are of slow and gradual or rapid or sudden onset. Central labyrinthine vertigo usually starts more slowly, develops less rapidly, and often persists for a very long time. Dandy observed that a peripheral labyrinthine vertigo that occurred on sectioning of the eighth nerve disappeared rapidly in spite of its great intensity.

Disturbances of *consciousness* or even loss of consciousness do not belong in the picture of peripheral labyrinthine vertigo. In fact, they suggest instead dizziness of central (epileptic) origin.

### RELATION OF SPONTANEOUS TO INDUCED VERTIGO

In some cases it is necessary to stimulate the peripheral labyrinth and to observe how the *induced* vertigo interferes with the *spontaneous* vertigo. Such a procedure may be helpful in the localization of the dizziness. Concerning this the following theories have been advanced:

Absence of the induced vertigo bespeaks primarily a central origin (Leidler). Guettich, however, believes that the absence of induced

vertigo may also be due to habituation to spontaneous chronic vertigo. Although such habituation may be possible theoretically, I doubt its practical occurrence, and assume instead a central origin.

A disproportion between induced vertigo and induced nystagmus indicates an irritation of the brain stem (Grant and Fisher). However, in my brain tumor material I have found this disproportion not only in diseases of the brain stem, but also in about 10 per cent of cerebral tumors. Absence of induced vertigo associated with hyperirritability of the labyrinth indicates cerebellar lesions (Bárány). Intensive spontaneous vertigo without spontaneous nystagmus is more indicative of a central origin.

### DISEASES ACCOMPANIED BY VERTIGO

*Peripheral labyrinthine lesions.* This includes all infectious inflammatory diseases, secondary degenerative processes, and all vascular diseases of the labyrinth such as ischemia, hyperemia, and arteriosclerosis, Ménière's syndrome, and, finally, trauma of the labyrinth. According to Brunner, the vertigo that follows concussion of the brain accompanied by ear symptoms usually persists for a long time. This group includes also all inflammatory, infectious, and toxic lesions as well as trauma and neoplasms of the labyrinth nerve.

*Central labyrinthine lesions.* This group includes all diseases in the region of the pons and the medulla oblongata (inflammations, hemorrhages, encephalomalacia, and tumors). The observation that vertigo is a frequent symptom of multiple sclerosis cannot be confirmed by experienced otoneurologists. In fact, true turning vertigo in these lesions is a rather rare phenomenon. In some cases we even find well marked spontaneous nystagmus without dizziness. On the other hand, in diseases of the cerebellum vertigo is a common symptom. It is often very intensive and may lead to the so-called persistent vertigo. This is particularly observed in tumors of the cerebellum. The question whether this vertigo is a true focal or rather a remote symptom due to pressure on the central vestibular region is difficult to decide. Nylen believes that early occurrence of vertigo indicates brain stem involvement, while late occurrence suggests cerebellar lesion.

Vertigo associated with lesions of the cerebrum is still more difficult to explain except in those cases where increased endocranial pressure influences the vestibular region.

*Seasickness.* The vertigo of seasickness is very pronounced and is associated with marked subjective symptoms such as nausea, vomiting, and perspiration. The similarity of this dizziness to that produced by lesions of the peripheral labyrinth leads to the belief that the vestibular apparatus is chiefly involved also in the vertigo of seasickness. This

theory is supported by the observations of W. James that deaf-mutes without labyrinthine functions are not subject to seasickness. Kreidl showed experimentally that the symptoms of seasickness were alleviated after section of the eighth nerve or extirpation of the labyrinth. However, there is one main symptom missing in seasickness, namely, spontaneous nystagmus, which is characteristic for labyrinthine lesions.

Seasickness is apparently caused by the various movements of a ship (pitching, rolling, tossing, etc.), unusual motions that are imparted to the passenger. These movements are unnatural to man and elicit not the normal counterreactions for the maintenance of equilibrium, but rather bizarre and unusual reactions. The combined motions of pitching and rolling of the ship are the main factors responsible for these symptoms. In this connection the observations of Byrne are of particular interest. He reproduced the exact movements of a ship and was able to produce seasickness experimentally. Another important factor in the development of seasickness is ocular vertigo. Fixation on certain objects of the surroundings increases the symptoms of seasickness.

*Ocular vertigo.* Ocular vertigo is almost never of the turning type. It is produced by the visual and muscular apparatus of the eye. Double vision due to muscular paralysis produces disturbances of orientation that the patient may describe as vertigo. Ordinary refraction disturbances may elicit similar symptoms. Height and mountain dizziness can also be considered as ocular vertigo. When looking down from a high mountain or tower, neurotic individuals may experience attacks of dizziness, owing to the absence of the usual objects for fixation. Such persons have the feeling of losing the ground and of falling down, but on closing of the eyes the vertigo subsides gradually.

The remaining types of vertigo can be explained on the basis of either systemic organic diseases or functional disorders.

*Vertigo with organic diseases.* In diseases of the cardiovascular system, tactile symptoms or vague symptoms of dizziness are usually described, while the true turning type is rare. Most of these conditions are associated with either mitral stenosis or arrhythmias, and the vertigo is apparently due to anemia of the brain or the labyrinth.

The vertigo associated with gastro-intestinal disorders is usually described as vague dizziness or a feeling of uncertainty. This vertigo is believed to be due to auto-intoxication, infections, or reflex action of the gastro-intestinal system. In this connection the close anatomic relationship of the central vestibular and vagus nuclei should be pointed out.

Vertigo due to diseases of the brain may be of different origin. In lesions of the vestibular nuclei, the dizziness represents a focal symptom. In diseases in which there is increased endocranial pressure, vertigo is

mainly a remote symptom. Finally, disturbances of the circulation within the brain may also lead to vertigo.

*Vertigo with functional diseases.* This classification includes the symptoms of vertigo occurring in neurasthenia, hysteria, migraine, climacterium, and in the various forms of the Ménière syndrome. In all of these conditions the dizziness has more or less the same character. It may be of the turning or tactile type, or may be merely a vague feeling of uncertainty. In addition, Leidler enumerates a number of vagovegetative symptoms, such as tachycardia, increased blood pressure, cardiac disturbances, headaches, nausea, anxiety, and tremor. Among the many explanations attempted, the theory of vasomotor disturbances in the region of the labyrinthine vessels seems the most probable. This brings us into the wide field of diseases grouped as an entity under the term "morbus Ménière." Its clinical importance makes it necessary to devote a whole chapter to it.

## 2. Spontaneous Disturbances of Equilibrium and Coordination

### Examination of the Static Labyrinth

In our section on the physiology of equilibrium, it was pointed out that the maintenance of equilibrium depends on three components: the eye, the labyrinth, and the proprioceptive impulses. The labyrinth and the proprioceptive impulses are of greatest importance, while the eye plays a less important role. Diseases of the labyrinth, especially acute ones, lead therefore to severe disturbances of the equilibrium. If such a disease causes complete loss of the labyrinthine function, the disturbances soon subside, owing to compensatory mechanisms. It is therefore evident that equilibrium can be maintained with only two components. Examinations of a large number of deaf-mute children that I made together with Alexander did not reveal any differences in the performance of gymnastic exercises by such children as compared with normal children. Only in complicated gymnastics did disturbances of equilibrium become evident.

Clinical functional tests must take into consideration static and kinetic equilibrium.

*Romberg test.* Examination of the static equilibrium should be initiated with the Romberg test. The patient is required to stand with both feet close together in order to reduce the proprioceptive element. He must also close his eyes in order to eliminate the ocular component. He is now carefully observed not only for direction of falling, but also for tendency to fall, and even for the slightest swaying.

*Mann test* (Fig. 25). The Romberg position may be replaced by the Mann position. The patient is placed so that one foot is in front of the

other, with the toe of the posterior foot touching the heel of the anterior foot, and the eyes are shut. This is a more sensitive test, but it has the disadvantage that some patients cannot normally maintain equilibrium in this position.

*One-leg test.* The patient is asked to stand alternatingly first on one leg and then on the other. While the supporting leg must be kept extended, the raised leg must be maximally flexed at the knee. Here again the eyes are closed.

*Static adaptation test* (Rademaker and Garcin). The patient is placed on a table in such a position that he rests on his knees and his hands, with his eyes shut. The table is then tilted over a frontal or longitudinal axis and the reactions of the patient are watched. Normal persons, and likewise patients with cerebellar ataxia or with tabes, maintain their equilibrium by shifting the trunk toward the raised part of the table, while patients with loss of the labyrinthine function on both sides will lose their balance and fall.

*Goniometer test* (von Stein). Von Stein constructed a device (the goniometer) that measures the horizontal angle. The patient is placed on a platform that is gradually inclined. The equilibrium is considered normal if the patient can sustain a forward inclination of the platform of 35° to 40° before falling forward, and a backward elevation of 26° to 30° before falling backward. The great variation within the physiologic range gives this test little practical value.

### EXAMINATION OF THE KINETIC EQUILIBRIUM

The following tests are used:

Walking forward and backward with eyes closed.

Jumping on one leg with the eyes closed, first forward, then backward. These movements are performed first on one and then on the other leg.

Walking on a straight line. A straight line is chalked on the floor and the patient is requested to walk along this line with his eyes closed.

Side-stepping (Alexander). The patient is asked to walk sideward with overstepping while the eyes are kept closed (FIG. 20).

### EXAMINATION FOR COORDINATION DISTURBANCES

*Asynergie cérébelleuse* (Babinski). The patient, standing in an erect posture, is asked to bend backward with his eyes closed. A normal person will subconsciously flex the knees during this maneuver in order to maintain the line of gravity. If coordination is disturbed (cerebellar disease) flexion of the knee will not occur and the patient will fall backward.

A similar test requires the patient to move forward with the eyes closed.

If coordination is impaired (cerebellar disease), the feet will advance while the trunk lags behind and the patient will fall backward.

A third test for asynergism is to place the patient in a recumbent position and then to ask him to sit up. A normal person will raise the trunk first, but a patient with a cerebellar disease will raise the feet instead.

*Dysmetria.* This means loss of the ability to measure movements. The movement may be excessive and overshoot the target, and is then designated as hypermetria. This is tested by asking the patient to touch

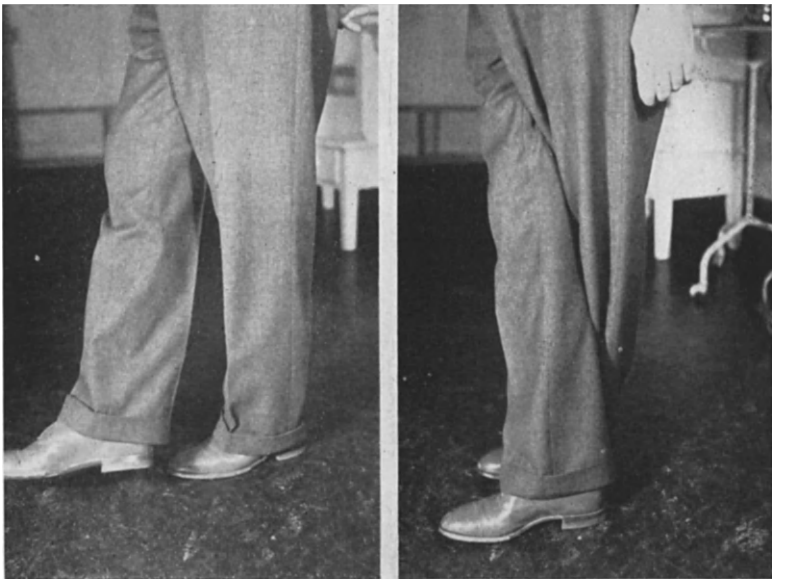

FIG. 20. SIDE-STEPPING TEST

the tip of the nose with his index finger. If the cerebellum is diseased, the patient will not stop this movement when he reaches the nose, but will violently overshoot the target.

*Adiadokokinesis.* The patient is directed to pronate and supinate the forearm or to flex and extend the fingers in rapid succession. If there is a disturbance of coordination (cerebellum), the function of the affected side will be decreased.

*Disturbance of tone.* This is tested by the rebound phenomenon of Stewart-Holmes (FIG. 21). The patient is asked to flex the arm at the elbow while the examiner with a firm hold on his forearm tries to hinder flexion of the arm. On sudden release, a normal person will continue this flexion for a short distance, but then stop. With disturbed coordination (cerebellum) there is a lack of inhibition and the flexion will continue until

the patient hits his shoulder. Another test for disturbances of tonus is the past-pointing test (Bárány), which is discussed on page 150.

*Treading test* (Unterberger). The patient is requested to perform alternating stepping movements while remaining in the same place and keeping the eyes closed. If a disease of the labyrinth is present, it will be observed that the patient is turning during the treading without being conscious of it. This symptom is due to disturbances of the muscular tonus influenced by the labyrinth.

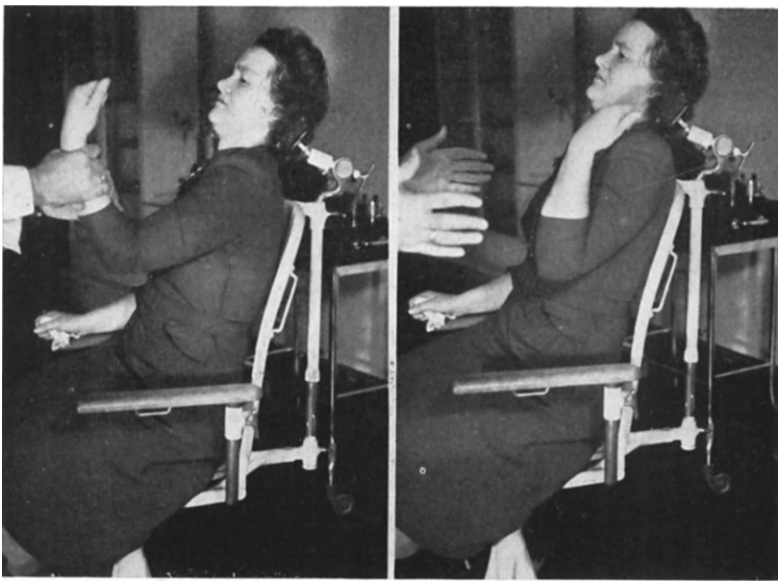

FIG. 21. REBOUND TEST FOR TONE DISTURBANCE

## EVALUATION OF RESULTS OF EXAMINATION

To prevent diagnostic errors, great caution is necessary, especially if the disturbances of equilibrium are only slight. The wide physiologic variations in the performance of these tests by normal individuals must be kept in mind. There are skilled and awkward subjects, especially older and obese persons, who cannot satisfactorily perform the various tests of balancing, such as jumping on one leg, etc. It is therefore necessary to differentiate between natural awkwardness and pathologic disturbances. Hence it is advisable in such borderline cases to repeat the tests several times, first with the patient keeping his eyes open and then with eyes shut. If the patient improves his performance with repetition of the tests, the verdict is more in favor of natural awkwardness. In the presence of pathologic processes, repetition will not change the results. If a diagnosis

of pathologic changes of equilibrium is made, it becomes necessary to localize the site of the lesion.

### DIFFERENTIAL DIAGNOSIS OF DISTURBANCES OF EQUILIBRIUM

In discussing the various diseases associated with disturbances of equilibrium, an attempt will be made here also to define the characteristics of the various types of diseases.

*Diseases of the peripheral labyrinth.* This group includes all diseases of the labyrinth, in particular those with an acute onset, such as serous or purulent labyrinthitis, trauma, intoxication, etc. The disturbances of equilibrium show certain peculiarities. Spontaneous falling takes place in the direction of the slow component of the nystagmus. The direction of the fall is dependent on the position of the head. For instance, if in a patient facing forward a nystagmus toward the right is present, he will fall to the left. If, however, the head is turned to the right, the nystagmus is directed backward, and therefore the patient will fall forward. The characteristics of a peripheral labyrinthine disturbance of equilibrium are: falling in the direction of the slow component of nystagmus, and dependence of the fall on the position of the head.

*Diseases of the central labyrinth.* This group includes syringobulbia, to a certain extent also multiple sclerosis, vascular diseases of the medulla oblongata, increased cranial pressure, particularly upon the vestibular nuclei, etc. The spontaneous falling takes place in the direction of the quick component of the nystagmus. It is not influenced by the position of the head, as compared with the peripheral labyrinthine type. The patient reports that he is being pulled to one side (lateropulsion).

*Diseases of the cerebellum.* Disturbances of equilibrium depend upon the type, site, extension, and duration of the pathologic process. In instances where there are symptoms of pressure upon the vestibular nuclei, the disturbances of equilibrium will be of the central type. In other cases the spontaneous falling will be in the direction of the affected side, independent of the position of the head. In general in cerebellar diseases the disturbances of the kinetic equilibrium are more marked than those of the static equilibrium. The patient with a cerebellar affection will stand with his feet spread apart. When placed in the Romberg position, with his eyes closed, he will often sway. Swaying motions are due to the alternating contractions of the flexors and extensors of the leg and the toes. The gait of such a patient resembles that of a drunk, and when walking along a straight line he will deviate to the side of the affection. Sidestepping in the direction of the involved side is impossible.

Further forms of disturbance of coordination include the various types of asynergy, adiadokokinesis, dysmetria, atonia, ataxia, etc. (p. 105).

*Tabes.* This is a lesion of the posterior roots of the spinal cord. If a tabetic is placed in the Romberg position, swaying will start first in the legs, while in cases of peripheral labyrinthine disease there will be observed a flexion of the upper trunk. Another differential test was described by Erben. If a tabetic is permitted to touch a fixed object with his finger, his disturbances of equilibrium will be relieved. On the other hand, this test will not work in a patient with a labyrinthine disease, and he will always fall in the direction of the slow component of the nystagmus. A further differential test is the gait. A patient with a cerebellar disease walks like a drunk, while the tabetic walks by flinging his legs.

*Disseminating sclerosis.* The disturbances of equilibrium are dependent on the site of the lesions. If the lesion is located in the region of the vestibular nuclei, the disturbances will have the character of the central type. Some authors, however, believe that the swaying of the patient in the Romberg position is nothing but an intention tremor of the entire body. The disturbance of balance shows in a characteristic swaying. It starts with fine oscillations gradually increasing in amplitude until the patient loses his balance.

*Neurasthenia.* The neurasthenic in the Romberg position sways without having a definite falling direction. Occasionally, however, ataxic movements can be observed. The disturbances in neurasthenics have one common peculiarity, namely, dysharmony. They do not fit into the pattern of any organic disease, and there is often a disproportion between the disturbances of equilibrium and the other neurologic findings (O. Mauthner). The neurotic is very susceptible to suggestion, which can often influence his falling.

*Hysteria.* The hysteric often shows inability to stand (astasia) or to walk (abasia). Sometimes there is a definite direction of falling, e.g., sideward and backward. In contrast to the labyrinthine fall, which sometimes is very precipitate, the hysteric slides down rather carefully in order not to get hurt. Furthermore, the hysteric makes no efforts to counterreact and maintain his balance.

### 3. Spontaneous Nystagmus

#### CLINICAL IMPORTANCE OF NYSTAGMUS

The occurrence of spontaneous nystagmus in patients is of the greatest diagnostic importance for the otologist. In many inflammatory diseases of the ear it may indicate not only involvement of the inner ear but also the beginning of intracranial complications. Furthermore, even in relation to nonotogenic diseases the otologist may be in a position to be of diagnostic aid to the neurologist. This is particularly true in cases of

brain tumors, hydrocephalus, brain abscesses, multiple sclerosis, encephalitis, cerebral arteriosclerosis, etc.

The occurrence of nystagmus is almost always a pathologic sign, because it does not occur in the normal individual. It indicates a disease in a certain part of the reflex arc (FIG. 18). This lesion may be located either in the peripheral labyrinth with its nerve terminals, in the vestibular nerve, in the central vestibular nuclear region, in the eye muscle nuclei region, or finally in the peripheral ocular apparatus. It is therefore necessary to localize its origin exactly.

In otologic practice those cases are of particular importance in which purulent otitis media exists simultaneously with spontaneous nystagmus. The proper diagnosis and treatment of such cases depend on recognition of the labyrinthine or nonlabyrinthine origin of the nystagmus. Even the experienced otologist may sometimes have difficulty in diagnosing the exact origin of the nystagmus, since many factors influence the elicitation of this symptom. For example, jerking nystagmus may be of labyrinthine, central, or ocular origin. Again, each labyrinth may produce nystagmus toward either side, or even the form of the nystagmus may not be characteristic. In this connection one might assume a vertical nystagmus to be caused by a lesion of the vertical semicircular canal. This, however, is not true in practice, because isolated disease of a single semicircular canal does not occur, since all semicircular canals communicate freely through the utricle. The result, therefore, is a mixed nystagmus. Entirely different, however, are the conditions in induced nystagmus, where the head can be placed in such a position that any given pair of canals can be stimulated separately, thus producing any kind of nystagmus (horizontal, frontal, vertical).

### AIDS IN LOCAL DIAGNOSIS

As has been pointed out in the preceding discussion, there are no scientific rules for determining the origin of spontaneous nystagmus. Yet, on the basis of clinical experience, we suggest a number of aids, as used in our clinic, that will help in the localization of the nystagmus.

If spontaneous nystagmus is noted in a patient, the examiner should observe the following seven points.

1. The nystagmus is designated as *associated* nystagmus if both eyeballs show identical movements, and as *dissociated* nystagmus if the movements are not identical. The dissociation may be qualitative or quantitative. An example of the former would be horizontal movement of one eye and rotatory movement of the other eye. An example of the latter would be fine quick movements of one eye and coarse slow movements of the other eye.

2. With regard to the *forms* of nystagmus, we differentiate simple forms such as horizontal (→), vertical (⇅), rotatory (↷), or oblique (↗) movements, and mixed forms such as rotatory-horizontal (⇄) and horizontal-rotatory (⇄), depending on which component predominates.

3. The *direction* of the nystagmus may be to the right, to the left, or to both sides, as well as up or down.

4. There are three *degrees* of nystagmus. A nystagmus of the *first* degree means that it occurs only when the patient looks in the direction of the quick component. If nystagmus also occurs when the patient is looking straight ahead, it is of the *second* degree. If a patient shows nystagmus not only in the direction of the quick component and when looking straight ahead, but also when looking in the opposite direction, then it is a nystagmus of the *third* degree. An example will best illustrate the degree of nystagmus. Let us assume a patient with spontaneous nystagmus toward the right while looking to the right (first degree). Another patient may show nystagmus toward the right when looking to the right and also when looking straight ahead (second degree). Finally, there may be a patient with nystagmus toward the right when looking to the right, also when looking straight ahead, and even when looking to the left (third degree).

An examiner not very familiar with the subject may easily confuse a nystagmus of the third degree toward one side with a nystagmus of the first degree toward both sides. For example, a patient may have a nystagmus toward the right while looking to the right and also toward the left while looking to the left. This is a nystagmus of the first degree toward both sides. Another patient may show nystagmus toward the right while looking to the right, and also nystagmus toward the right while looking to the left. This is a nystagmus of the third degree to one side.

5. The *amplitude* is denoted by the excursions of the movement. It is called coarse if the excursions are more than 3 mm., medium if between 1 and 2 mm., and fine if less than 1 mm.

6. The *frequency* denotes the number of movements per minute. Here again we speak of high frequency (more than 100 jerks per minute), medium (50 to 100 jerks per minute), and low (less than 50 jerks per minute).

7. As regards *duration*, the nystagmus may be either permanent or transitory.

With the aid of these seven points, the examiner can proceed to the local diagnosis of spontaneous nystagmus within the reflex arc. How can these various points be utilized for such a purpose?

1. *Association, dissociation.* The presence of a dissociated nystagmus always excludes the possibility of a peripheral labyrinthine origin. Theo-

retically, only the possibility of a central or ocular origin remains. However, a central origin of a dissociated nystagmus is rare, and for practical purposes any dissociated nystagmus can be regarded as of ocular origin.

2. *Form.* Spontaneous nystagmus of the simple forms (→ ↑ ↗) points rather against the likelihood of a peripheral labyrinthine origin. Again, possibility of either a central or an ocular origin remains. If the nystagmus is oblique (↗), it is only of ocular origin. If it is vertical (⇵) a central lesion is usually indicated. This lesion may be in the central vestibular organ, at about the level of the abducens nucleus (Marburg). Usually the region between the pons and the corpora quadrigemina is involved, either by tumors in the anterior regions of the cerebellum or by temporal lobe abscesses rupturing into the ventricle. On the basis of experimental studies, Leidler located the origin of rotatory nystagmus in the caudal region of Deiters' nucleus, horizontal nystagmus in its medial, and vertical nystagmus in its cephalad portion.

The mixed types of spontaneous nystagmus indicate a peripheral labyrinthine genesis, since, as previously stated, isolated lesions of individual semicircular canals practically never occur. Therefore, the result of any peripheral labyrinthine disease will be a mixed type of spontaneous nystagmus (↻⇉).

3. *Direction.* For diagnostic utilization of the direction of the nystagmus, the relationships are not simple. Each labyrinth endeavors to elicit a nystagmus in its own direction, and the labyrinths of the two sides have antagonistic functions. This means that the elimination of one labyrinth has the same effects as stimulation of the other. Every inflammatory labyrinthine disease produces in the early stages a nystagmus toward the affected side (irritation effect) but later, with the destruction of the labyrinth, a nystagmus toward the healthy side occurs (elimination effect).

Diseases of the central labyrinth (vestibular nuclei and fibers to the posterior longitudinal bundle) usually produce a nystagmus toward the same side. As has been explained above, a patient with a labyrinthine disease leading to complete destruction of function will show a nystagmus toward the sound side. When in the course of the disease the nystagmus changes back toward the affected side, it is evident that this nystagmus could not have arisen from the destroyed labyrinth. Therefore the direction of the nystagmus is pathognomonic of a central extension of the disease (Neumann).

4. *Degree.* The intensity of spontaneous nystagmus is also of diagnostic value, especially if it is of the second or third degree. If such a nystagmus is of a peripheral labyrinthine origin, it is always associated with severe subjective symptoms such as vertigo, turning sensations, nausea, vomiting, etc. If, therefore, a patient with a second or third degree nystagmus does

not show these associated symptoms, a peripheral labyrinthine origin is not probable. As an example, we might cite the spontaneous nystagmus of third degree in cases of syringobulbia, where the patient does not complain of any of these symptoms. If such a nystagmus were of a peripheral labyrinthine origin, such a patient would be confined to bed and avoid even the slightest movements, because these would produce the most intense vertigo, nausea, etc.

5, 6. *Amplitude and frequency.* Amplitude and frequency run usually hand in hand. Since amplitude and frequency are controlled by unknown factors, e.g., light, psychic excitement, fatigue, etc., it is obvious that their diagnostic value is not of great importance. Here again clinical experience is the criterion. Diseases of the peripheral labyrinth usually show nystagmus with medium frequency and amplitude, while central lesions show a rather coarse and slow nystagmus. Nystagmus of ocular origin may be of any type.

7. *Duration.* The diagnostic value of the duration of nystagmus depends either on a reliable history or on observation for a longer period of time. The nystagmus of peripheral labyrinthine origin is self-limited. It usually regresses gradually and disappears after days or a few weeks. Occasionally it may occur as repeated attacks of short duration.

The central labyrinthine nystagmus persists for many months or years and may even increase in intensity. Very often there is a disproportion between the intensity of the nystagmus and the subjective symptoms. The existence of a nystagmus of third degree for many months or years rules out the assumption of a peripheral labyrinthine origin.

Ocular nystagmus persists usually for the duration of life and never changes.

The following paragraphs present a discussion of all diseases of the labyrinthine reflex arc that are associated with spontaneous nystagmus.

### DISEASES OF THE PERIPHERAL LABYRINTH ASSOCIATED WITH SPONTANEOUS NYSTAGMUS

This group includes labyrinthine as well as retrolabyrinthine lesions.

*Labyrinthine lesions.* These comprise the various infectious inflammatory diseases of the labyrinth, such as serous and purulent labyrinthitis, circumscribed or diffuse labyrinthitis, etc., and, in addition, secondary degenerative processes of the labyrinth due to chronic middle-ear affections (adhesive processes, catarrhal conditions, etc.). Also included are the vascular lesions of the inner ear, such as essential hypertension, arteriosclerosis of the labyrinthine vessels, particularly of the internal auditory artery—which as an end artery supplies the labyrinth—neurocirculatory

spasms of the labyrinthine vessels, Ménière's syndrome. Other causes may be diseases of the hemopoietic apparatus (lymphatic and myeloid leucemia), which may lead to hemorrhages into the labyrinth, and, finally, traumata such as fractures or fissures of the base of the skull involving the inner ear, or post-traumatic neuroses.

*Retrolabyrinthine lesions.* Here again we must differentiate between primary diseases of the vestibular nerve and secondary involvement of the nerve extending either from labyrinthine or meningeal affections. The former include all those rare cases of isolated neuritis of the eighth nerve such as luetic neuritis (O. Beck), rheumatic neuritis (Ruttin), influenza (J. Fischer), and drug intoxications. Furthermore, they include all neoplasms (acoustic tumors). To the secondary diseases belong all those lesions that spread either from the inner ear to the cranial fossa or from the cranial fossa to the inner ear (otogenic intracranial complications). The spontaneous nystagmus of the majority of cases will, therefore, be of peripheral labyrinthine origin. However, in certain cases, e.g., cerebellar abscess, pressure upon the central vestibular nuclei may elicit a spontaneous nystagmus of central origin.

### DISEASES OF THE CENTRAL VESTIBULAR ORGAN ASSOCIATED WITH SPONTANEOUS NYSTAGMUS

Here we have to differentiate between direct diseases within the course of the reflex arc and indirect diseases exerting pressure upon the vestibular nuclei. The spontaneous nystagmus in the former cases would constitute a focal symptom, while that in the latter cases would be a remote symptom.

*Direct effects.* Inflammatory diseases of the pons and the medulla oblongata, such as the encephalitides (lethargic and polio-encephalitis), disseminated sclerosis, thrombosis, embolization, and hemorrhages belong to this group. Here thrombosis of the inferior posterior cerebellar artery near its origin from the vertebral artery is of particular importance. Such a thrombosis leads frequently to an involvement of the central vestibular pathways (spinal-acoustic route). Other diseases of this group include syringobulbia, which involves particularly the arcuate fibers (Leidler). The spontaneous nystagmus associated with it is rotatory and shows the highest intensity (third degree). Furthermore, intrapontine tumors, such as cysticerci and solitary tubercles (Brunner), may also affect the vestibular pathways. Finally, toxic infections such as pneumonia, streptococcus sore throat, typhoid fever, etc., may also produce direct effects.

*Indirect effects.* All processes associated with increase of intracranial pressure may produce spontaneous nystagmus if they exert pressure upon the central vestibular pathways. To this group belong the various brain tumors, particularly those of infratentorial location, hydrocephalus, otitic

intracranial complications such as meningitis of the posterior fossa or cerebellar abscess. Occasionally abscesses of the temporal lobe may also produce spontaneous nystagmus of central origin, especially if they break into the ventricle. This nystagmus is predominantly vertical (Ruttin). The sudden occurrence of a vertical nystagmus in the course of a temporal lobe abscess indicates a grave prognosis.

### DISEASES OF THE OCULAR ORGAN ASSOCIATED WITH SPONTANEOUS NYSTAGMUS

*Amblyopic nystagmus.* Spontaneous nystagmus occurs in cases of either congenital or early acquired amblyopia. It is usually associated with such gross pathologic conditions as cataracts, micro-ophthalmos, coloboma of the iris or optic nerve, corneal astigmatism, extensive scars, etc. Occasionally, however, these signs may be absent. The nystagmus is either of a jerking or of an undulating type. In the former case it is predominantly of the horizontal associated bilateral type and independent of the position and movement of the body.

*Blindness nystagmus.* The nystagmus of blind people is usually completely irregular, coarse, and in all directions, and cannot be classified as either jerking or undulating.

*Albinotic nystagmus.* This type occurs in persons who have little or no pigmentation of the eyes. The nystagmus is very intense and of an undulating character.

*Miner's nystagmus.* This occurs in about 5 per cent of all miners, and may be of any type—horizontal, rotatory, vertical, oblique, and occasionally even circular and elliptic. It may be jerking or undulating, fine or coarse, associated or dissociated, fast or slow. It is influenced by light and convergence, i.e., with good light and convergence it is regular, fine, and fast. In contrast to amblyopic nystagmus, it is dependent on position and motion of the body.

Ohm considers miner's nystagmus as a reflex disturbance of the ocular and vestibular tonus of the ocular and palpebral muscles. Causes of this nystagmus are generally believed to be the deficient illumination of mines, the toxic effects of mine gases, and the unnatural posture of the miner, who in either flexed or recumbent position overstrains his eyes during fixation.

*Darkness nystagmus.* Similar to miner's nystagmus, this was produced experimentally (Raudnitz) in young animals kept for a certain period of time in a dark room.

*Spasmus nutans nystagmus.* This is of the same type as miner's and darkness nystagmus. It is a disease that occurs between the sixth and twelfth months of life and is characterized by a head nystagmus associated

with torticollis. Raudnitz believes it related to darkness nystagmus, because it occurs chiefly among poor children who are being kept in dark rooms. In contrast to the theory of Raudnitz, a study of many cases has led me to the conclusion that the balancing of the head, which becomes perfect only in the second half of the first year, is the essential factor, and for this reason I assume an anomaly of the tonus of the neck muscles as the cause for the head nystagmus.

*Latent nystagmus.* This is characterized by the fact that it occurs only when one eye is closed, but does not occur with binocular vision. It is a jerking nystagmus, either horizontal or rotatory, and of coarse or medium amplitude, in the direction of the open eye. It is usually found either in diseases of one eye only, or in diseases that affect one eye more than the other. The nystagmus is often associated with strabismus (particularly of diverging type). However, the mechanism of this latent nystagmus is not as yet fully understood. Ohm assumed as cause the difference of the volume of light perceived, while Kestenbaum believed that the two impulses that direct the right eye to the right and the left eye to the left are less developed than the opposite impulses.

*Correction and fixation nystagmus.* This type can best be observed when the patient is requested to fix upon the examiner's finger held in an extremely lateral position at a certain distance (approximately 2 feet). It is usually a jerking nystagmus, associated, fine or medium, and plain horizontal. Occasionally it may be difficult to differentiate this form from the labyrinthine type. The correction nystagmus is usually of short duration, gradually decreasing, but reappears when the patient again is requested to fix upon the finger. The nystagmus is unaccompanied by labyrinthine symptoms.

*Fatigue nystagmus.* When the patient is requested to fix upon the examiner's finger, held in a lateral position, for a period of one to two minutes, a nystagmus may occur that gradually increases the longer the patient keeps his eyes fixed. The form is usually plain horizontal.

Although this nystagmus may be elicited even in normal people, it is obvious that persons with functional weaknesses (hysteria, neurasthenia) will show this fatigue symptom to a greater extent. In neurasthenia the nystagmus can be increased if the patient is asked to look repeatedly to one and then to the other side, and after several such trials is requested to fix upon the examiner's finger for one to two minutes.

*Musculoparetic nystagmus.* When a patient with a paresis of an eye muscle, for instance the external rectus, is ordered to look in the direction of the paretic muscle, a nystagmus can be observed. It is horizontal, fine, fast, and often dissociated. In the case of a complete paralysis of an eye muscle, there is often no nystagmus at all.

### RARE FORMS OF NYSTAGMUS

*Hereditary nystagmus.* Nystagmus occurs occasionally in several members of the same family. In the literature there can be found genealogies of four to five generations in which such nystagmus occurred. It is generally of the undulating type, but occasionally also jerking, and depends on the direction of vision.

*Retractory nystagmus* (Koerner). The eyeballs show jerking movements into and out of the orbit. This rare form was observed in tumors of the quadrigeminal bodies.

*Cortical nystagmus* (Bartels). Patients with conjugate paresis of the eyes show this type of nystagmus when requested to look in the direction of the paresis.

*Reflexive nystagmus* (Baer). A reflex nystagmus can be produced by sensory stimulation of the trigeminal nerve, for instance by irritation of the cornea or of the nasal mucosa.

### TESTS FOR POSITIONAL NYSTAGMUS

When positional nystagmus is tested, care must be taken to avoid sudden jerking movements of the head, because a nystagmus thus elicited may be the result of a movement reflex rather than of a position reflex (p. 148).

It is best to request the patient to assume that position in which he usually experiences vertigo and nausea. He can often find this position very easily. If he fails, the following systematic procedure is recommended. The patient is examined first in dorsal recumbent position, then in either lateral or ventral position, and finally again in dorsal position with the head overextended. The positional nystagmus must be examined in every one of these positions in all directions of vision. While the nystagmus caused by the jerking movements of the head (head movement nystagmus) is usually of short duration, the true positional nystagmus lasts longer, often as long as the new position is maintained.

Furthermore, observations should be made on the following points: form, intensity, duration, and direction of the nystagmus. The dependence of the nystagmus on the position of the head and the body should be noted, because only in this way are we able to use this test diagnostically.

The results of these examinations can be classified into three groups (Ruttin, Seiferth):

1. The direction of the nystagmus is always the same, regardless of the position of the patient. It occurs in association with diseases of the peripheral labyrinth, for instance in labyrinthitis.

2. The nystagmus is dependent upon the position of the patient, and changes with a change of position. It is found in cases with tumor and trauma.

3. The nystagmus changes its direction even though the patient maintains the same position constantly. It occurs in connection with diseases of a central origin.

Interesting and instructive as these classifications may be, it must be remembered that they are arbitrary classifications, and that the pathology is far more complicated. In fact, the authors themselves admit that some cases may fit into one or more groups, while other cases do not fit into any group.

Finally, I wish to stress a point that is not emphasized enough. In numbers of cases we deal with neurotics with a labile psychic equilibrium. In contrast to Klestadt, who maintains that positional nystagmus is never of psychogenic origin, I observed a number of cases of positional nystagmus in neurotics.

### 4. Conjugate Paresis and Conjugate Deviation of the Eyeballs

#### CLINICAL IMPORTANCE

Disturbances of the associated eye movements (conjugate paresis and conjugate deviation) are due to lesions of the cortical centers and the central pathways. They are, therefore, of great diagnostic significance in diseases of the central nervous system. For the clinical otologist their importance lies in the following facts:

The vestibular reflexes exert a certain influence upon the associated eye movements.

Lesions of the cerebrum and of the pons are frequently associated with disturbances of the conjugate eye movements.

Simultaneous involvement of the labyrinthine reflexes causes disturbances of the induced nystagmus. Turning or caloric stimulation of the labyrinth may lead to a decrease or elimination of the quick component, with the result that either a slow deviation of the eyeballs or no eye movements at all occur.

The experienced otologist may therefore make valuable suggestions to the neurologist and thus aid him in diagnosis. If a patient shows disturbances of the eye movements, it becomes necessary to determine whether a nuclear or supranuclear lesion is the cause. This may be done by examination of the labyrinthine functions. For example, caloric stimulation of the left labyrinth (cold) should normally produce a nystagmus with the quick component toward the right and the slow component toward the left. However, if the slow component can be observed only in the left eye, a nuclear lesion (left abducens) is present. But if this stimulation produces a conjugate deviation to the left (which means that the slow labyrinthine component is elicitable), it can be concluded that the lesion is not in the labyrinthine pathways but in the supranuclear region.

### THE VARIOUS TYPES OF CONJUGATE PARESIS AND DEVIATION

Bielschowsky distinguishes the following types of associated conjugate deviation:

In the type called pseudo-ophthalmoplegia, the patient cannot look in the direction of the paresis either voluntarily or upon request. If, however, the eyes of the patient are slowly led by the finger of the examiner in the direction of the paresis, they may be able to move in this direction to a certain extent (pursuit movement). The same eye movement can be elicited upon stimulation of the labyrinth. In this example the site of the lesion would be in the cortical center and the central pathways respectively.

The patient can look in the direction of the paresis neither voluntarily, nor upon request, nor by means of pursuit movements. However, labyrinthine stimulation will still elicit these movements. In this instance the lesion is located either in the pons or in the corpora quadrigemina in the proximity of the ocular nuclei.

The patient is able to look in the direction of the paresis neither voluntarily, nor upon request, nor by means of pursuit movements, and not even upon stimulation of the labyrinth. Such a lesion would be located in the posterior longitudinal bundle (uni- or bilateral). If the conjugate deviation is vertical, then a nuclear ophthalmoplegia may be present.

Supranuclear and nuclear conjugate paresis may occur in combination.

### DISEASES ASSOCIATED WITH DISTURBANCES OF THE CONJUGATE EYE MOVEMENTS

In otologic practice, the following diseases may show disturbance of the conjugate eye movements: supratentorial tumors; temporosphenoidal abscesses; cerebellar abscesses occasionally; diseases of the brain stem and especially of the pons. Grant and Fisher observed conjugate deviation upon cold stimulation in about 30 per cent of cases with supratentorial tumors. Conjugate deviation instead of normal induced nystagmus was also described by Eagleton in cases of temporosphenoidal abscesses and by Ruttin and Neumann in cases of tumors of the cerebrum. True labyrinthine conjugate deviation occurs according to Brunner only in connection with acute lesions—especially of a vascular nature—of the vestibular nuclei. In chronic diseases of the brain stem the lesions are so extensive that the associated conjugate deviation of the eye is due to pressure effects.

### DIFFERENTIAL DIAGNOSIS OF CORTICAL AND PONTINE LESIONS

For differentiation between cortical and pontine lesions the reader is referred to the chart of Bielschowsky (p. 64). Only the most important points will be discussed here.

In *cortical* conjugate deviation, the eyes are deviated homolaterally, while in *pontine* lesions the deviation is contralateral. In the former there is usually also a simultaneous rotation of the head in the direction of the conjugate deviation, while in the latter this deviation is either in the opposite direction or completely absent. *Cortical* paresis is transient and leads only to decrease of the conjugate eye movements toward the contralateral side, while in *pontine* paresis the conjugate movements toward the homolateral side are completely abolished. In *cortical* paresis the paralysis of the facial nerve and that of the extremities are homolateral, while in *pontine* paresis the facial paralysis is homolateral whereas that of the extremities is crossed.

To these distinctions it can be added that in *cortical* paresis stimulation of the labyrinth produces a normal nystagmus, while in *pontine* paresis the nystagmus elicited is of a pathologic nature. There is either a decrease or loss of the quick component or a deviation of the eyeballs toward the opposite side. However, the conjugate deviation must not be confused with the Bell phenomenon. The latter term denotes the fact that if the eye is opened against the resistance of the patient, the eyeball will roll upward and outward.

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## INDUCED REACTIONS

When it has been ascertained that spontaneous manifestations either are not present or have previously been checked, the various functional tests can be performed. The reader may wonder how all the numerous tests can be made on one patient. Fortunately, in the majority of cases it will not be necessary to perform all these tests. For example, if a patient shows a spontaneous nystagmus of a definite origin, the optokinetic reaction can be omitted. The same is true for some other reactions, such as bilateral calorization, mechanical reaction, and galvanic reaction, which have a limited indication.

Even though in many cases only certain tests have to be performed, the reactions and reflexes resulting from them are so numerous that they often

cannot be analyzed in one examination. For example, the examiner must note the latent time and duration of the induced nystagmus, and further its form, direction, intensity, frequency, rhythm, etc. He must also observe the secondary reactions, such as falling, past-pointing, arm tonus reactions, etc. Considering that these reactions last only a short time (seconds or a few minutes) the difficulties with which the examiner is faced become more understandable. It is therefore fortunate that in the majority of cases it is quite sufficient to determine only the latent time, the duration, and the intensity of the nystagmus, and to observe the falling reaction. However, certain otoneurologic cases, or cases with otogenic complications, or cases that are significant for scientific reasons, do require a thorough study of labyrinthine reactions.

Attempts to solve all the problems in one examination can only lead to erroneous results. Some authors use certain devices that the patient has to carry along so that stimulation as well as examination can be accomplished simultaneously. Other authors require the patient to sit on the edge of a chair in readiness for examination of the reflexes of the musculature of body and extremities, with an assistant in readiness to check the induced nystagmus at the same time.

I do not consider such methods highly satisfactory, and I cannot see any advantages in forcing all the tests into one examination. It is much more advisable to focus the main attention on those reactions that in the given case are particularly of clinical importance, and to have these determined exactly. If the duration of reaction is over before all the observations are finished, a second and if necessary even a third stimulation should be instituted. However, it should be borne in mind that a certain period of time must elapse before the test can be repeated, if erroneous results are to be avoided. It is a fact that repeated examinations, although tranquilly carried out, as well as emotional strain and overfatigue can influence the outcome of the tests. Since most of the cases requiring a thorough study of the reactions are hospitalized, ample time is provided for repeated tests.

### 1. Optokinetic Tests

If the otologist observes a spontaneous nystagmus in a patient and cannot determine whether it is of a labyrinthine or nonlabyrinthine origin, he should employ the optokinetic test. In addition, there may be some otoneurologic cases, e.g., with conjugate paresis of the eyeballs, where the optokinetic reaction should be tested.

#### TECHNIC

For examination of the optokinetic reaction certain devices are used, such as an optical wheel, optical cylinder, optical umbrella, etc. It is advisable to use the optical umbrella of Brunner (Fig. 22). It is made

of pasteboard in the shape of a low cylinder, 70 cm. in diameter and 30 cm. high. There are six vertical black stripes, 9 cm. in width and 30 cm. apart. It can be placed on a tripod by means of a cross or it can be hung from the ceiling.

Demetriades (1923), following a suggestion of Brunner, made large-scale examinations at the Policlinic of Vienna, where I had opportunity of observing them. Since then this test has been introduced in our routine labyrinth examinations. The technic is as follows. The patient is seated in such a way that the lower margin of the cylinder is in the same plane as the upper orbital margin. He is requested to look at the black stripes. The examiner sits in front of the patient and turns the cylinder with an

FIG. 22. OPTICAL UMBRELLA

approximate speed of ten revolutions in twenty seconds. The eyes of the patient look straight ahead, fixating the black stripes.

Demetriades found in normal persons an optokinetic nystagmus in the direction opposite that of turning, depending upon the plane of rotation. If therefore the optical cylinder was turned in a horizontal plane as shown in FIGURE 22, say to the right, an optokinetic nystagmus to the left was elicited; on turning the cylinder to the left, a nystagmus to the right occurred. If, however, the cylinder was turned in a sagittal plane, a vertical nystagmus could be observed; but the examiner did not succeed in eliciting a rotatory nystagmus with any position of the cylinder.

The frequency of the optokinetic nystagmus depends upon the speed of the revolutions and upon the spacing of the black stripes. The closer the stripes, the greater is the frequency of the nystagmus.

The optokinetic nystagmus follows the same rules as the labyrinthine.

Hence it will be increased by looking in the direction of the fast component and decreased by looking in the direction of the slow component. However, there is a certain influence of the attention upon the optokinetic nystagmus. Distraction of the fixating attention exerts an inhibitory influence upon the optokinetic nystagmus.

#### DIFFERENTIATION OF LABYRINTHINE AND NONLABYRINTHINE NYSTAGMUS (INVERSION)

On examination of a patient with spontaneous nystagmus, the optokinetic reaction may occur in any of three possible ways:

*a*) On turning the optical cylinder, a nystagmus is elicited in the direction opposite to that of turning (normal optokinetic reaction).

*b*) On turning the optical cylinder, a nystagmus is observed in the same direction as that of the turning (atypical reaction or inverse phenomenon).

*c*) On turning the optical cylinder, no eye movement at all occurs (atypical reaction or inverse phenomenon).

Thus the normal optokinetic reaction is of no help in diagnosing the origin of the spontaneous nystagmus of the patient.

The atypical reaction (inverse phenomenon) indicates a nonlabyrinthine origin of the spontaneous nystagmus of the patient.

The test for optokinetic reaction is very simple, does not require complicated apparatus, and can be performed even on patients with very poor vision.

Many years ago, in collaboration with I. Sommer, I examined the inmates of the deaf-mute institute in Vienna. We were able to elicit optokinetic nystagmus in children with such poor vision that they were just able to see the finger of the examiner at a distance of 20 inches. We also succeeded in producing optokinetic nystagmus in imbeciles on whom an ordinary test for vision could not be performed.

### 2. Caloric Tests

Thermal stimulations of the ear produce certain effects that can be divided into objective and subjective reactions. The former include caloric nystagmus, falling reaction, past-pointing reaction, arm tonus reaction, etc. The latter include various sensations such as turning sensations, nausea, vertigo, etc. If the temperature of the stimulus is higher than the body temperature, nystagmus to the same side occurs (with upright position of the head), whereas a stimulus of less than body temperature produces nystagmus to the opposite side. Stimuli at body temperature elicit no effects whatever. Nystagmus produced by heat can be suppressed by cold stimuli.

The advantage of caloric testing lies in the fact that each labyrinth can

be tested separately, while in the turning test the two labyrinths are always stimulated simultaneously. In irrigation of the ear with cold or warm water, it should be borne in mind that it is not the water but the difference in temperature that constitutes the stimulus. Consequently, in cases where irrigation with water is contra-indicated (dry perforation, trauma), other thermal stimuli such as cold air, ether, ethyl chloride, etc., can be used. The latter produces freezing, which may lead to severe labyrinthine reactions. It must, therefore, be sprayed carefully into the external canal, and one should not wait until nystagmus occurs; it should be employed rather in repeated fractionated doses.

In testing for caloric nystagmus, care must be taken to avoid faulty technic as well as faulty observation. For example, some jerking movements of the eyeballs in the beginning of the test may easily be mistaken for true vestibular nystagmus, while on the other hand true vestibular nystagmus may be masked by ocular stimuli (fixation, convergence, accommodation). This ocular interference was already known to Bárány, who attempted to eliminate it by having the patient fix the examiner's finger at a distance of not less than 20 inches. In order to eliminate fixation, Bárány and Abels used opaque eyeglasses. For the same purpose Bartels employed glasses of 20 diopters. This had the advantage that their magnifying effects made observation easier.

Unfortunately the use of these devices did not entirely eliminate the fixation of the eyes, nor could it suppress the tendency of the patient to fixate. On the contrary, since it caused a decrease of vision and blurring, it led to ocular movements that were caused by the fixation mechanism (p. 57). Consequently these eye movements make the interpretation of the true vestibular nystagmus still more difficult. In order to eliminate fixation completely, examination must be carried out in a dark room with a dim red light. These requirements were most closely approximated by the device of Frenzel called *Leuchtbrille*. It consisted of a goggle frame with two lenses of 15 diopters each. It contained a small electric bulb whose light blinded the patient's eyes and thus prevented fixation. Its use requires a dark room.

For practical purposes it is sufficient to have the patient fix a finger held 20 to 30 inches from the eyes. The finger should be held laterally and slightly higher than eye level to obtain full opening of the eyelids. Passive separation of the eyelids by the examiner must be strictly avoided, since the pressure exerted upon the eyelids will elicit certain eye movements (associated nystagmus of Stransky). Furthermore, it is essential that the patient be examined for a spontaneous nystagmus before any caloric tests are applied.

It may easily happen that after calorization the nystagmus observed is

of a spontaneous nature, unless its existence was ruled out beforehand. If such a spontaneous nystagmus is observed, then the following technic is advisable. If the nystagmus is of the first degree, e.g. toward the right, the patient is requested first to fix toward the right side. The finger is then gradually moved to the midline, i.e., in the direction of the slow component, which decreases the nystagmus. When this point is determined, the caloric stimulation of the labyrinth can be applied. It is obvious, therefore, that in the presence of spontaneous nystagmus of the highest degree (third) this technic is not applicable. In such instances, it is difficult, if not impossible, to influence the spontaneous nystagmus by a caloric nystagmus, and the diagnosis of labyrinthine excitability to caloric stimulation must be made from other reactions, such as falling reaction, past-pointing, turning sensations, vertigo, etc. In order to study the earliest phase of the caloric nystagmus, it is advisable to observe the blood vessels of the sclera most carefully for any signs of movement.

In examining for reaction to caloric stimulation, differentiation must be made between the volume method (150–200 cc. of water) and the minimal method (2–10 cc.).

### MASS OR VOLUME METHOD (BÁRÁNY)

The volume method is indicated in the following conditions:

*a*) Markedly decreased labyrinthine excitability.

*b*) Existence of spontaneous nystagmus of higher degree, or the presence of certain types of ocular nystagmus, e.g., blindness nystagmus, amblyopic nystagmus, albinotic nystagmus, etc. Experience has shown that these forms of nystagmus are difficult to influence by caloric stimulation. In such cases it is necessary to apply 200 cc. of water or more in order to get an intense reaction. Particular attention should also be paid to the other vestibular reactions, such as falling or past-pointing.

*c*) Where minimal calorization fails. Bárány's method uses 100 to 200 cc. of water at 68 F. Irrigation is carried out with the head in upright position, and the nystagmus is observed from a distance of 20 inches. The nystagmus is horizontal-rotatory. It usually becomes increasingly rotatory if the patient looks in the same direction, and increasingly horizontal if he looks in the opposite direction. If the head is tilted toward one shoulder, then on cold application there will be a horizontal nystagmus in the direction of the higher ear, while the rotatory nystagmus will be in the direction of the lower ear.

### MINIMAL METHODS (KOBRAK)

Minimum stimulation is held to have the following advantages: the physiologic threshold for caloric stimuli can be determined; inhibition of

the reaction by overstimulation is prevented; the specific volume of water used (5 cc.) permits good average results; the specific temperature used likewise permits good results; the small volume of water used prevents such unpleasant subjective reactions as nausea, vertigo, vomiting, etc., yet produces the desired nystagmus.

Since the clinical introduction of the minimal method by Kobrak, many modifications have been attempted in order to reduce the technical errors. It is impossible to instance in detail all the variations and modifications, some of which are discussed in chapter iii. Four of these methods will be presented in detail here.

1. *Demetriades-Mayer modification.* With the head of the patient in upright position, 5 cc. of water at 55 F. is injected slowly into the external auditory canal in the direction of the eustachian tube. If no reaction occurs within 3 to 4 minutes after injection, the head is brought into the optimum position, i.e., inclined 60° backward and tilted slightly toward the syringed side. Now 10 cc. of water of the same temperature is injected, and the patient is requested to fix the finger. The latent time is determined, i.e., the period elapsing between the end of irrigation and the occurrence of the earliest sign of nystagmus, which usually starts with the slow component. Then the duration of the nystagmus must be ascertained. Clinical observation of more than 1,000 cases has established an average value of 15 to 30 seconds for the *latent time* and 60 to 120 seconds for the *duration.* Since uniformly the same volume of water is used at the same temperature, the recording is simple. The results are expressed in two figures: the latent time first, followed by the duration in parentheses, thus: AD–18(92); AS–20(100). Additional attention should be directed to the following features of the nystagmus:

Intensity (first, second, or third degree)
Form (horizontal, rotatory, mixed)
Direction (right, left)
Amplitude (coarse, fine, medium)
Frequency (slow, medium, fast)
Quality (associated, dissociated)
Rhythm (relation of slow to fast component)

2. *Veits-German modification.* The head of the patient is bent back 20° (indifferent position). Irrigation is carried out by injection of 10 cc. of water at 65 F. in 7 to 10 seconds, with a calibrated syringe. The stream of water is directed to the superoposterior wall of the external canal. The nystagmus is observed through the Frenzel *Leuchtbrille* in a dark room. With this position of the head (indifferent position) no nystagmus should occur. However, if it does occur, the head must be

corrected; for instance, if the nystagmus occurs in the direction of the syringed side, the head must be advanced slightly forward; if the nystagmus occurs in the opposite direction, the head must be bent a little more backward. After the actual indifferent position for the horizontal canal has been determined, then 1 to 1½ minutes must be allowed to elapse, after which the head is suddenly jerked backward through 90° (optimum position). From this moment on the latent time and the duration of the nystagmus are determined. The average values are given as 5 seconds for the latent time and 70 to 120 seconds for the duration, if the nystagmus is observed with the eyes in the lateral position, but only 60 to 90 seconds if the nystagmus is observed with the eyeballs in the midposition.

The purpose of this method is to separate the physiologic component of the latent time from the physical component (see p. 76). This is accomplished by irrigation with the head in the indifferent position and determination of the reaction for the optimum position, the two being separated by a period of 1 to 1½ minutes.

3. *Atkinson modification.* The head of the patient is placed in such a position that the horizontal canal occupies a vertical plane. This is done by leaning the head and body toward the horizontal plane so that the ear to be syringed is above the other, and tilting the chin upward through 45° by rotating the neck. Then 1 cc. of water, with ice floating in it, is instilled into the external canal with a special syringe. The time of instillation is noted by a stop watch. The patient is requested to fix upon the examiner's finger held at a certain distance. The moment at which the first flicker of nystagmus occurs is noted. At once the ear is emptied of water. The patient is tested for nystagmus first with the head inclined forward 30°, and then with the head bent back 60°. The time when the nystagmus disappears is noted. The average of the latent time lies between 15 to 60 seconds, while the duration is normally about four times as great as the latent time.

4. *Warm and cold stimulation* (Vogel contrast reaction). The ears are irrigated alternately first with cold and then with warm water, and the results are compared. By this method it can be shown that it is sometimes much easier to elicit nystagmus toward one side than to the other (nystagmus disposition). For example, the right ear may react more easily to cold stimulation, and the left ear more easily to warm stimulation. The result will be a marked nystagmus to the left but a moderate nystagmus to the right. This indicates a central lesion (brain tumor, head trauma, etc.) If there is such a nystagmus disposition toward the affected side, then a central irritation is assumed, while a disposition toward the healthy side indicates a destructive lesion in the central nuclei (Koch).

## SIGNIFICANCE OF THE VARIOUS TESTS

In studying the results of the minimal tests, we are impressed by the exact figures and the great diagnostic value of these tests. It seems that they enable us not only to determine quantitatively the physiologic threshold of the labyrinth for caloric stimuli, but also to differentiate between the excitability of the peripheral and the central labyrinth. Still more, even within the central vestibular organ it seems possible to determine slight tonus differences between the right and the left side.

However, from a clinical point of view the problem is not as simple as it looks. It is evident that as complicated a physiologic process as caloric testing cannot be interpreted as a mathematical problem. There are too many unknown factors involved that diminish the clinical value of the resulting figures. A few examples will illustrate this point. In some cases, the form and direction of the nystagmus cannot be influenced by a change of posture of the head, as is generally assumed. Bárány explains this failure of effect by assuming that the reaction, once elicited centrally, cannot be inhibited. This theory as well as several others does not negate the fact that numerous clinical cases do not run according to theoretic considerations. Furthermore, it is generally assumed that a stronger stimulus elicits the stronger effect. Applied to the caloric reaction, this would indicate that with an increase in the difference of temperature and volume of the stimulus the reaction should also be greater.

Judging the results of the various methods from this physiologic point of view, we arrive at startling conclusions. Comparison, for instance, of the latent time under the method of Demetriades, using water at 55 F., with that under the method of Grahe, using water at 68 F., shows the same results (15 to 30 seconds). The same is true of the duration of the nystagmus; regardless of the stimulation, the average figures are between 60 and 120 seconds. In this connection the results of Fleischmann are very instructive. He made large-scale examinations, varying the temperature, volume, and duration of irrigation, and found almost no variations in the results in spite of the difference in technics. His results could be criticized on the basis of the fact that he used the minimal methods, which provide such small stimuli that great variations could not be expected. This argument loses its validity when the results of the volume method are studied. Ruttin, using 150 to 200 cc. of cold water, obtained an average latent time of 15 to 30 seconds, and an average duration of 90 seconds—results that are identical with those of the minimal method.

Many other arguments could be brought forward to show that the problem of the caloric reaction is more complicated than is indicated by the theories. The method of German, e.g., where the head is jerked backward,

is the same technic as is used for the elicitation of the "head-moving" nystagmus (see p. 148). However, even with a gradual retroflexion of the head, it is difficult to rule out the neck reflexes (Magnus and De Kleyn) or other sensory stimuli.

Examples of other unknown factors are: various types of pneumatization of the petrous bone; condition of the blood vessels; psychic states such as excitement, anxiety, fatigue, etc. All these unknown elements tend to affect the accuracy of the figures.

In evaluating all these methods, we come to the conclusion that for practical reasons those methods are best that can be applied easily and that are based on clinical comparison. Such a method can be evolved only by averaging the clinical results of examination of thousands of normal as well as pathologic cases. I came to the conclusion that it is not the method that matters, but that it is important that the same method be always used, and that all conclusions be based upon clinical comparison.

### EVALUATION OF CALORIC EXCITABILITY

While the tests of the cochlear apparatus by means of voice, tuning fork, and audiometer yield accurate results, the same is not true in regard to the labyrinthine apparatus. In spite of the relative precision of the minimal tests, standard norms of the excitability of the labyrinth under caloric stimuli have not yet been established.

Clinically it is rather easy to diagnose loss of irritability, hypo-irritability, and extreme hyperirritability. This is particularly easy if there is a great difference between the right and the left side. However, this task may become very difficult if a so-called "slight" hyperirritability has to be differentiated, which appears to be the most frequent diagnosis. We cannot caution too much against making this hasty diagnosis simply because a patient complains of dizziness and nausea after irrigation, or shows a slightly decreased latent time. We made the diagnosis of hyperirritability (in Brunner's clinic) when three factors showed proportional changes as follows: a decrease of the latent time below 10 seconds; an increase of the duration above 2 minutes; and the occurrence of a nystagmus of second or third degree when the method of Demetriades and Mayer was used. The subjective reactions (vertigo, turning sensations, nausea), although usually proportional to the degree of hyperirritability, cannot be used for diagnosis, since they may or may not occur.

In the evaluation of the caloric excitability, not only these quantitative but also the qualitative changes of the caloric nystagmus must be considered. The latter include changes of rhythm, in ratio between quick and slow components, dissociation of the eye movements, abnormal direction of the nystagmus, etc. All these qualitative changes point to a lesion in the central part of the labyrinthine reflex arc.

### 3. Bilateral Calorization

The bilateral calorization represents an exact method for disclosing slight differences in the excitability of the two labyrinths, even in cases where the unilateral method has failed. The indication for the test is given, according to Brunner, when the decision has to be made as to whether the posterior cranial fossa or the other fossae are involved. This concerns particular processes with increased endocranial pressure.

#### TECHNIC

For the examination, certain devices such as Ruttin's apparatus or Brunner's modification are used (Fig. 23). The latter consists of a head

Fig. 23. Bilateral Calorization

set that has on each end an ear speculum movable in all directions. In the center of the ear speculum a cannula is fixed, with a rubber hose inserted. The two rubber hoses are connected by a Y-shaped glass tube. On the third end of the glass tube a rubber hose is in connection with a syringe for irrigation. Particular attention must be paid in order to prevent technical errors. Under control of the eyes, the two ear specula must be inserted in corresponding parts of the external meatus. It may happen that after one ear speculum is focused, efforts to focus the other bring the first out of the proper position. Therefore, before applying the stimulus, we check the positions of both ear specula. It is advisable to insert the two ear specula

in such a position that the water is directed toward the inferior anterior quadrant of the drum. The head of the patient is in upright position, the temperature of the water should be 55 F., the amount 200 cc.

### EVALUATION OF RESULTS

If after bilateral calorization a nystagmus of second or third degree is observed, the reaction is called positive. If there is no nystagmus at all, or if there is a nystagmus of first degree, or a vertical nystagmus, it is spoken of as a negative reaction. Brunner, who made large-scale examinations, found a positive reaction in the majority of cases having lesions in the posterior cranial fossa, while cases with such processes in the anterior or middle fossa gave a negative reaction.

The direction of nystagmus in positive results was usually toward the affected side, which corresponds to greater excitability of the labyrinth of the sound side. In spite of this fact, definite conclusions concerning the side of the affection cannot be drawn.

The occurrence of a nystagmus of second or third degree indicates only the location of a lesion in the posterior cranial fossa.

Summarizing, it may be said that bilateral calorization represents a useful aid in diagnosing processes accompanied by increased endocranial pressure. Yet it should be borne in mind that this test is not a 100 per cent proof. If, therefore, a positive reaction is found, care must be taken to eliminate technical errors, such as asymmetric irrigation. In addition, however, local processes in the canal, such as scars in the drum or chronic adhesive processes, may influence the results of the test. Large-scale examinations should be made in order to overcome all the errors and to develop the method for clinical use.

In the field of otoneurology the test should be more often employed, particularly in cases in which the differentiation between frontal tumor and cerebellar tumor can hardly be made. In 1920, when I studied the brain material of the surgical clinic in Vienna, I described 5 cases in which the diagnosis of a cerebellar tumor was made on the basis of symptoms such as disturbance of equilibrium, spontaneous nystagmus, spontaneous falling, past-pointing, hyperirritability of the labyrinth, atypical falling reaction, ataxia, etc. In all 5 cases autopsy was later performed and revealed a tumor of the frontal portion of the brain on the opposite side. These cases dated back to a time when the method of bilateral calorization was not known. In subsequent years I had plenty of opportunity, as consulting physician of the neurologic clinic, to examine such cases and to employ the bilateral caloric test. There were some cases in which I was able to render useful aid to the neurologist.

### 4. Turning Tests

From a physiologic point of view, the turning reaction has much more significance in relation to the labyrinthine function than any other test—since turning acts as the only adequate stimulus for the semicircular canals. Another advantage lies in the fact that with certain positions of the head the various pairs of canals can be separately stimulated (horizontal, rotatory, vertical nystagmus). On the other hand, it must be considered as a disadvantage in that both ear sides are involved in the reaction. This is in contrast to the advantage of the caloric stimulation, by which each side can be tested separately.

The turning test was originally performed in such a way that the nystagmus was observed during rotation (perrotatory nystagmus). For such a purpose special turning chairs were used in which both the patient as well as the examiner were rotated. Later on various apparatus and devices with mirrors were constructed in order to control the perrotatory nystagmus.

At the present time methods are used in which the nystagmus after the turning stops (postrotatory nystagmus) is observed. Its direction is opposite to that of the perrotatory nystagmus. Aside from this there is no difference, since the postrotatory nystagmus follows the same laws of physiology (p. 80).

Here a brief summary of relevant facts of applied physiology is in order.

*Each semicircular canal* produces a nystagmus in its *own plane*, i.e., the horizontal canal a horizontal nystagmus, the superior vertical canal a rotatory nystagmus, and the inferior vertical canal a vertical nystagmus. The type of nystagmus elicited by turning depends solely upon what position of the head was maintained *during* spinning—regardless of later change. When, for instance, a patient is turned with the head tilted toward the shoulder, a vertical perrotatory nystagmus is elicited. When the turning is stopped and the head is brought into normal upright position, the postrotatory nystagmus will still be of a vertical type—but the direction is now opposite, i.e., instead of vertical up it will be vertical down. The *perrotatory* nystagmus is chiefly produced by the labyrinth of *the same side*, while the *postrotatory* type is produced by the *opposite* labyrinth. Since only the latter nystagmus is examined with our present methods, it can be said in other words: After *stopping* a *right* turn the *left labyrinth* chiefly is tested, while after *stopping* a *left* turn the *right ear* function chiefly is examined.

The various types of turning tests can be classified into three groups: forced-turning methods (Bárány); short-turning methods; long-turning methods.

In our discussion of the caloric reaction, it has been shown that certain factors such as fixation, convergence, accommodation, etc., may influence the induced nystagmus and lead to erroneous results. The same holds true for the turning reaction, where an additional factor—optokinetic nystagmus—may occur. This nystagmus is due to fixation of moving objects (p. 69). During turning, both types of nystagmus—the optokinetic as well as the turning type—have the same direction. This leads to an increase of the induced nystagmus. After turning has been stopped, the two types of nystagmus tend in opposite directions, with the result of a decrease in the induced nystagmus. Efforts have been made to eliminate the optokinetic factor by means of opaque eyeglasses or convex lenses. M. H. Fischer uses a black cowl that covers the entire head and face of the patient. This may be a good method from a scientific point of view, but it cannot be employed on clinic patients.

I still believe that the simplest and most satisfactory method of eliminating the optokinetic nystagmus is to order the patient to keep his eyes shut during turning. After turning is stopped, he must open his eyes and look straight ahead or in the direction of the expected postrotatory nystagmus. When, for instance, a patient is turned to the right (clockwise), a postrotatory nystagmus toward the left will be elicited. Therefore the patient is ordered to look to the left side at the finger of the examiner, which is held from 20 to 25 inches above the horizontal plane of vision and to the side. It must be borne in mind that before testing the turning reaction, an examination for spontaneous nystagmus must be made.

### FORCED-TURNING METHODS

These methods employ at least ten revolutions within 20 seconds. Some otologists use turning with steady velocity, while others turn with increasing acceleration. The most common test at the present time is the original Bárány method. The patient is placed in a turning chair (Fig. 24). Since the chair is already turned around a vertical axis, those semicircular canals are particularly stimulated that lie in a horizontal plane. Therefore the head of the patient must be placed in a definite position before turning. The more exactly this is done, the purer will be the type of the nystagmus elicited (horizontal, rotatory, vertical). Otherwise mixed forms will result, such as horizontal-rotatory or rotatory-horizontal. According to Bárány, the turning nystagmus is determined by the angle formed by the crossing of the spatial horizontal line and the line between the eyeballs. After the head of the patient is brought into proper position and his eyes shut, he is turned ten times with a steady velocity of 2 seconds per revolution. When turning is suddenly stopped, the postrotatory nystagmus must be controlled. Before examination of the other ear, a

period of a few minutes should elapse. It is advisable for the examiner to turn always toward the same side in starting his routine work, in order not to be confused later as to which labyrinth has already been tested. I always start by turning to the right (clockwise), so that I am sure that when the patient has been turned once it has been a right turn.

*Test for horizontal nystagmus.* In order to elicit horizontal nystagmus, the head of the patient is bent forward 30°. On turning to the right, a postrotatory nystagmus to the left can be observed, and vice versa. The duration of the nystagmus shows an average of 15 to 35 seconds. The

FIG. 24. TURNING CHAIR

difference in duration as between right and left side in normal cases is practically negligible.

*Test for rotatory nystagmus.* In order to elicit rotatory nystagmus, the head of the patient is bent forward 90°, so that the chin touches the chest. After stopping the right turn, rotatory nystagmus to the left can be observed, and vice versa. The duration of nystagmus is shorter than with the horizontal canals, averaging 10 to 20 seconds.

*Test for vertical nystagmus.* In order to elicit vertical nystagmus, the head of the patient is tilted 90° toward the shoulder. The direction of the nystagmus depends upon the side of the tilting and upon the direction of turning. Since it is almost impossible for the clinician to remember

immediately under which conditions and in which of the vertical canals an ampullofugal or an ampullopetal flow occurs, the following practical advice is given. When the tilting of the head and the turning of the patient have the same direction, the vertical nystagmus (postrotatory) is downward. For example, tilting of the head toward the *right* shoulder and turning to the *right* results, after stopping, in a vertical nystagmus downward. The same effect is obtained when the head is tilted toward the left shoulder and the turning is to the left. On the other hand, the nystagmus is upward when the head is tilted to the right and the patient turned to the left and suddenly stopped. The duration of the vertical nystagmus is very short, the average time being between 5 and 10 seconds.

### SHORT-TURNING METHODS

For examination of the turning reaction, only one revolution (360°) or less (60°) is employed. Various methods exist, but only one test (Grahe's method) will be discussed here.

The patient is placed in a turning chair and is well balanced, with the legs flexed. The examiner stands behind him and puts two fingers (ring and middle fingers) loosely on his closed eyelids. Now the patient is turned either 90° or 180°. During this procedure the examiner palpates with his fingers through the closed eyelids the movements of the eyeballs (nystagmus) and determines the number of jerks. After a few minutes' pause, the patient is turned to the other side and the eye movements are checked as before.

### LONG-TURNING METHODS

Turning is begun very slowly (below threshold) and is continued over a period of 3 to 4 minutes. Among the various methods (p. 85) the following two tests may be discussed.

*Veits-Dodge method.* The patient is placed in an ordinary turning chair and is spun very slowly at first, then with increasing acceleration. As soon as the speed of one revolution every 2 seconds is reached, turning is continued with a steady velocity (without acceleration). Turning is stopped 1 to 2 minutes after the patient no longer feels the turning sensation. This stage can be diagnosed either by the report of the patient that the turning sensation has subsided or by the following method of Dodge. A tuning fork is struck and the patient is asked to locate the tone. As long as turning sensations persist, the patient has the impression that the tone is moving around while he himself is sitting still. After the turning is suddenly stopped, the eye jerks are counted and the so-called *Schlagdichte* determined, i.e., the number of jerks per the time unit.

*Fischer (M. H.) method.* The patient is placed in an electrically driven

turning chair with the head bent 15° forward. To keep this position fixed, the patient bites on a device that acts as a brace. To eliminate the optokinetic nystagmus a black cowl is used. Turning is begun with a speed of 0.3 degree seconds and is slowly increased to 1–2 degree seconds, which is considered the physiologic threshold for turning stimuli. From then on, turning is continued with a steady speed for 3 minutes, after which turning is suddenly stopped. The postrotatory nystagmus (examined with the patient looking straight ahead) is then exactly determined. Before testing the other side a period of one hour must elapse.

### COMMENT ON THE VARIOUS METHODS

The forced-turning methods have often been the subject of criticism, the contention being made that the test is too rough, the stimuli far exceed the physiologic threshold, or there is interference between the positive acceleration on turning and the negative retardation on stopping (p. 83), etc. However, the fact is that despite all these arguments the forced-turning still represents the most common clinical turning test. As far as the so-called *exact* or *quantitative* methods are concerned, the same can be said as in regard to the quantitative test for caloric stimuli: all the alleged exactness has its limit in the various unknown factors.

The short-turning methods have not yielded the expected results. On the contrary, it has been shown that the nystagmus after short turns is of a nonlabyrinthine character, hence it has nothing to do with a vestibular reaction. The method of Grahe is said to require a certain experience in palpating the eye movements. I could not succeed in achieving this even after many years.

The long-turning methods are not introduced into the routine examination, since they require complicated apparatus, such as electrically driven turning chairs and other scientific equipment. What are the advantages of these methods? First, the results are more exact, the latent time represents the true latent period, the duration time of the nystagmus does not differ as much as under the other methods, and, second, differentiation between peripheral and central excitability can be made by controlling the rhythmic phases of the postrotatory nystagmus (p. 83). The first phase expresses the peripheral excitability as well as the central one, while the second, third, or following phases relate only to the central vestibular organ. Hence a thorough determination of the various phases permits of differentiation between *hyper-* (*hypo-*) *irritability* (peripheral) and *hyper-* (*hypo-*) *sensitivity* (central). Furthermore, determination of the postrotatory phases may reveal tonus differences between the right and the left side. Such a differentiation can be made when the second phase of one side and the first phase of the other side are markedly in-

creased (Arslan, Mittermaier). This corresponds to the warm-cold contrast reaction of the caloric test.

In summary, the following can be said. For practical routine examination the forced-turning methods are quite satisfactory. However, for certain purposes, as in otoneurologic cases or in scientific research, the long-turning methods may render useful diagnostic aid. If an electrically driven turning chair is not available, the method of Veits-Dodge, using the hand-driven chair, can be employed. I would not recommend the use of short-turning methods.

### EVALUATION OF LABYRINTHINE EXCITABILITY

*Unilateral loss of irritability.* The diagnosis of pathologic changes in the labyrinthine excitability under turning stimuli may often be quite difficult. Some reasons for this have already been given in our discussion of the caloric reaction (p. 129). However, another factor must be added, since turning stimuli involve both ears, while with caloric reaction each labyrinth is separately tested. It is obvious that the figures resulting from turning stimulation must be evaluated differently from those resulting from caloric stimulation. An example will illustrate the facts.

A diffuse purulent inflammation of the right inner ear led to destruction of the right labyrinth with complete loss of its excitability. The figures for the functional tests of caloric and of turning reactions are:

| | AD | AS |
|---|---|---|
| Caloric | 0 | 20 (90) sec. |
| Turning | 6 sec. | 20 sec. |

The caloric test shows plainly the loss of function, while on turning the right labyrinth still seems to be functioning, although much less (6 seconds). Such a conclusion would be wrong, since the 6-second duration of nystagmus is due to the left labyrinth (cf. chap. iii). Only a brief explanation will be given here. When the patient, with head bent forward 30°, is turned to the right and suddenly stopped, a horizontal nystagmus to the left can be observed. This nystagmus is elicited chiefly by an ampullopetal flow of the endolymph of the left horizontal canal. Since our patient has a normal left labyrinth, the duration will be approximately 20 seconds. When now the patient is turned to the left and suddenly stopped, an ampullopetal flow of the right horizontal canal should elicit a nystagmus to the left. Since the right labyrinth is completely destroyed this cannot happen. However, we know that stopping a left turn also causes an ampullofugal flow of the left horizontal canal, which in turn produces a very weak nystagmus (in this case lasting 6 seconds). Hence the resulting figures of 6 seconds to 20 seconds reveal a complete loss of function of the right labyrinth.

*Phenomenon of compensation* (Ruttin). When after loss of excitability of one labyrinth a certain period of time has elapsed (weeks or months), and a turning test is performed again, the resulting figures will be quite different. Using as an example the same patient as above (6 seconds to 20 seconds) the reaction will now give 12 seconds to 14 seconds. This shows that the figures as between right and left ear do not differ very much, since the direction of the turning nystagmus of the right ear has increased, while that of the left ear has decreased. Ruttin, who first described this observation, called it "phenomenon of compensation." It is obvious that this phenomenon does not express a normal or a slightly decreased function of the right labyrinth, since function can never be regained after a complete destruction of the sense organ. The compensation must therefore be considered to be a shift in the central impulses under turning stimuli. The average figures for the phenomenon of compensation (according to Ruttin) are: for the horizontal canals, 10 to 15 seconds; for the frontal canals, 6 to 8 seconds. For the vertical canals no compensation occurs. It is interesting to note that the phenomenon of compensation is usually observed in cases where a complete destruction of the sense organ has taken place, but does not occur if only a part of the labyrinth is destroyed.

### 5. Mechanical Tests (Fistula Symptom)

Compression or aspiration of the air of the external auditory meatus or of the tympanic cavity can under certain circumstances produce eye movements (mechanical nystagmus). Occasionally some other vestibular reactions may also be associated with eye movements, such as vertigo, falling, etc. The indication for the tests is given in cases of purulent otitis media, in which the question arises as to whether the inflammation is going to overstep the normal landmarks of the middle ear. In the majority of cases, defects of the bony labyrinthine capsule are present, particularly in the predilectory place, i.e., the bony wall of the horizontal semicircular canal. Owing to the narrowing of the region of the antrum, retention of pus favors development of a circumscribed fistula in this area. Another indication is given in cases of inner-ear lues, particularly of the hereditary type. In such cases the drum membranes usually appear normal.

#### TECHNIC

The external auditory meatus is tightly closed with a cannula that is connected by means of a rubber hose with a Politzer bag. The latter is provided with a fistula opening. Before the test is performed, it is important to check whether every connection is tight, so that air cannot

escape either through the insertion of the cannula or at the junction of the hose with the bag or at the fistula opening. First, compression is instituted and the eyeballs of the patient are observed. The aspiration test follows, and the eyeballs are again observed carefully. In the presence of a fistula of the bony labyrinthine capsule or in some cases of inner-ear lues, certain eye movements will be elicited (mechanical nystagmus). When on compression the nystagmus is toward the same side or on aspiration toward the opposite side, it is spoken of as a typical positive reaction; otherwise the result is called atypical positive, or paradoxic positive, or inverted positive.

### EVALUATION OF RESULTS

In discussing the physiology of the mechanical reaction, it was stated that the mechanical nystagmus follows the laws of Ewald; however, the clinical experiences do not always accord with this theory. This is not surprising, since we know that there is quite a difference between the physiologic experiment and the various pathologic occurrences observed in patients. A fistula may exist, e.g., on the bony wall of the horizontal semicircular canal, which is closed by cholesteatomatous masses (Fig. 37). It is obvious that in this case the mechanical test will yield a negative reaction in spite of the presence of a bony defect. On the other hand, there are cases in which granulation tissue (a pedunculated polyp) may form a solid bridge between the labyrinth and the external auditory meatus. When such a patient touches his tragus slightly, as in washing his face, he can produce the fistula reaction by himself, with intensive symptoms such as nystagmus, vertigo, falling, etc. The underlying pathology varies so greatly that no regularity of the direction of the mechanical nystagmus can be seen.

There are so many combinations possible that Ruttin, for example, described twenty-six different types, all of which he named by different terms. If the examiner directs all his attention to the form and direction of the nystagmus, he may easily overlook the few jerks that occasionally occur, because he expects a well marked nystagmus in a certain direction. I therefore used to inform the students in all my courses to watch the eyeballs carefully and to note whether a movement occurs or whether the eyes remain quiet. In the former case the reaction is called positive, in the latter negative. No other conclusions should be drawn. The mechanical reaction can be produced either with the technic described above or in an unusual manner, as by pressure on the tragus (true fistula symptom) or pressure on the carotid or neck veins (vascular fistula symptom). Further, there are cases in which a positive reaction seems to occur though neither a true nor a pseudo fistula symptom is present. Years ago I published a

number of such cases that had led to erroneous diagnosis. One of the most instructive cases will be discussed briefly here.

A white, 22-year-old female patient complained of vertigo and disturbance of balance. She had had radical mastoidectomy on the right side four years previously, and was now advised to undergo another emergency operation on the same ear. She refused to be operated on, and came into the out-patient department of our hospital. On examination, I found in the right ear a radical mastoid cavity partly epithelialized and partly granulating; the left ear was normal. On pressure upon the right tragus, I noticed that the left eyeball moved first toward the inner angle of the eye; then the right eyeball made an opposite movement, and the pupils became very miotic. Simultaneously the patient complained of intensive vertigo and fell to the right and backward. Pressure upon the left tragus had no effect at all. However, on pressure of the right carotid the same symptoms were elicited as with that on the right tragus.

My first impression was that a labyrinthine fistula was present. On further examination I had to change my opinion, since aspiration, compression, turning tests, and caloric tests all had exactly the same results as far as the eye movements were concerned. These movements did not have the character of a true nystagmus but were rather like the convergence spasm I have described in relation to patients with severe hysteria (p. 65). Therefore I tried to produce the reaction by pressure upon the hysteric pressure points. In this I was successful. Pressure on the nipple, as well as over the ovaries, led to eye movements, intensive vertigo, and to falling backward and to the right. The further course of the disease proved it to be of functional origin and not anatomic (i.e., no fistula was present).

### 6. Galvanic Test

The galvanic reaction was introduced as a clinical test by Neumann, Bárány, Mackenzie, Ruttin, Bruenings, etc. Since there is great dissension as to the physiologic effect of the galvanic reaction (p. 88), it is obvious that the pathologic significance is also still a matter of dispute. We use the galvanic test in order to differentiate diseases of the labyrinth from affections of the vestibular nerve (retrolabyrinthine lesions). The indication for the test will therefore be given in a case in which, for example, the labyrinthine excitability under caloric, mechanical, and turning stimulation is absent, so that it becomes important whether or not the galvanic reaction can be elicited. A positive response indicates a labyrinthine disease, a negative reaction points to the possibility of a retrolabyrinthine lesion.

In recent years attempts have been made to differentiate diseases of the nerve stem from lesions of its central pathway by determination of the vestibular chronaxia (p. 89).

#### TECHNIC

The various methods of testing the galvanic reaction can be classified into the following groups: (*a*) unipolar test, (*b*) bipolar test, (*c*) double electrode test, (*d*) combined (galvanic-caloric) test, (*e*) chronaxia test.

The galvanic nystagmus usually shows a horizontal-rotatory or a rotatory form and has the same direction as the galvanic current. Since the electric current flows from the anode to the cathode, the galvanic nystagmus is directed toward the side of the cathode. The position of the head has no influence upon the form and direction of the nystagmus, a fact that clearly shows that the galvanic current does not act upon the various semicircular canals but rather upon the vestibular nerve.

FIG. 25. BIPOLAR TEST IN MANN'S POSITION

For the galvanic test electrodes are used that have the form either of a small bulb or of a large square. Before use they should be soaked in a saline solution. If there is a doubt as to which is the cathode and which the anode, it is best to put both electrodes into a basin of water, close the electric circuit, and observe air bubbles. The latter indicate the cathode.

*Unipolar test.* One electrode (stimulating) is applied on the tragus or on the mastoid, the other (indifferent) is placed on the neck or forehead or is held in the patient's hand. On closing the electric circuit, a nystagmus will be elicited at 10 to 16 milliampere intensity. If the cathode has been used as a stimulating electrode, the galvanic nystagmus is directed toward the ear of the same side; if the anode has been used, the galvanic nystagmus is directed to the opposite side. Mackenzie studied the nystagmus not only after closing the electric circuit but also after opening it. Further details may be found in the original article.

*Bipolar test.* Two bulbar electrodes connected like a radio set (FIG. 25) are placed on either the tragus or the mastoid so that the electric current is sent through the head. A galvanic nystagmus toward the cathode can be observed at 2 to 5 milliampere intensity of current. In the case of persons who are very sensitive even to such small charges of electricity, the intensity of the current can be further reduced if it is not the nystagmus but the falling reaction that is examined. Falling or tendency to fall, tested in Mann's position (FIG. 25) can usually be observed by the use of galvanic currents of 1 to 1½ milliamperes, while the patient does not show any other uncomfortable sensations.

*Double galvanic test.* The indifferent electrode has the usual square shape and is placed on the forehead or neck. The stimulating electrode is split into two bulbar electrodes, which are applied on each side of the tragus or mastoid. If the vestibular excitability of the two sides is equal, the stimuli to the two labyrinths become neutralized and no nystagmus can occur. If, however, there is a difference between the two sides, a galvanic nystagmus will be elicited. When the stimulating electrode has been used as cathode, the nystagmus goes to the side on which there is greater irritability of the labyrinth; when the stimulating electrode has served as anode, the nystagmus is directed toward the side of the labyrinth with the lesser irritability.

*Combined caloric and galvanic test.* The arrangement is the same as for the double galvanization, but a caloric irrigation is applied at the same time.

*Chronaxia test.* The indication for this test is given in cases in which a differentiation should be made between lesions of the peripheral neuron and supranuclear affections. Cases of the latter kind usually show normal responses, while those of the former type respond with a marked decrease in the vestibular chronaxia. For the determination of chronaxia, electric discharges of condensers with varying capacities are used. The rheobasis is determined by means of a potentiometer. The electrodes are the same as those used for the double galvanic test. In France and Germany, special apparatus are constructed, equipped with all the necessary features. The apparatus are very complicated and very expensive, which in turn explains why the test has not yet found its way into the routine clinical examinations. Further details on this test will be found in the original articles.

### COMMENT ON THE VARIOUS METHODS

The unipolar test, as worked out by Mackenzie, is not very often used now. The method requires intensive electric current, occasionally as high as 20 milliamperes. Some patients are very sensitive and complain of burning pain even before a galvanic nystagmus can be noted. It is

claimed that the method gives particularly good results in cases of unilateral hyperirritability (Mackenzie). This author also states that the galvanic reaction is better qualified to give exact determinations than the turning reaction.

Admitting that the galvanic reaction represents a very good test for clinical purposes, I do not believe that any of the various methods can be used for exact quantitative determinations. Too many elements as yet undetermined are involved in the reaction (p. 89). Such factors are, for instance, the conducting power of the various tissues (skin, bone, etc.), resistance, distribution, polarization. Even the site of the stimulation in the labyrinth is still not definitely known.

We prefer the bipolar method, which is very simple and does not require more than 2 to 4 milliamperes of current to elicit a nystagmus. Even a lower intensity, 1 to 2 milliamperes, may produce a galvanic reaction (vertigo, falling reaction) before nystagmus can be noticed.

### 7. Tonic Reflexes and Reactions

Magnus and De Kleyn and their followers have worked out completely the old Ewald theory of the tonic labyrinth. Although their classic experiments have shed new light upon the tonus problem and revealed new reactions (p. 92), it cannot be denied that there is a great gap between instructive experiments on animals and pathology in man. It is not possible to apply the results of the animal experiments to man. Some of the reasons why it cannot be done are the erect gait in man, the position of the eyeballs, the voluntary looking, the high development of the cerebrum with its inhibitory influence, the complicated structure of the sense organs, etc.

From a practical point of view two questions arise: (1) Which of the labyrinthine reflexes found in animals can be noticed in man? (2) What is their clinical significance? The answers are not very satisfactory. In the overwhelming majority of cases, the tonic labyrinthine reflexes in man cannot be separated from other superposing reflexes. In animals they can best be studied in decerebrated subjects. Further, in order to study the labyrinthine reflexes, the neck reflexes must be eliminated. However, there are cases in which tonic labyrinthine reflexes can be noticed, as in sucklings, or in cases of disturbance in development, particularly of the central nervous system. Neck reflexes are more easily observed in man than the other reflexes.

The tonic influence of the labyrinth in man can best be seen in physiologic vertigo (p. 100), where a number of counteracting movements are elicited in order to prevent falling of the patient.

In describing the functional tests for the various reflexes and reactions, the classification of Magnus and De Kleyn is here again used (p. 92).

## POSTURAL REFLEXES AND REACTIONS (OTOLITHIC REFLEXES)

On change of the position of the head, various reactions and reflexes occur that are partly subjective (postural sensations) and partly objective. The latter affect the eyes (compensatory eye positions) or the neck, trunk, and extremities. Some of these reflexes originate directly in the labyrinth (true otolithic reflexes). Some others are neck reflexes, according to Magnus and De Kleyn. The latter reflexes are elicited when the position of the head is changed with respect to the position of the body, while the tonic labyrinthine reflexes occur when the position of the head is changed with respect to space. In order to test the function of these otolithic reflexes, it is obvious that the disturbing neck reflexes must be eliminated. This can only be accomplished when the head and neck are fixed, so that no change of position can occur. Various methods are used for this purpose, such as fixation of the head and neck in a plaster cast, or use of a device on which the patient bites, and which acts as a brace. Some authors have constructed tilting tables on which the patient is fixed in a certain position (Grahe, McNeil and Tait).

*Tests for postural sensations.* The older authors determined the error the patient made as to the optic vertical line when his head was tilted over a sagittal axis. Aubert examined patients in a dark room with an illuminated vertical line. On tilting the head toward one shoulder, the subject saw the vertical line moved in the opposite direction. According to Delage, a person with his eyes shut is able to orientate perfectly when he grasps a pole with both arms extended and holds it symmetrically.

Other tests were concerned with the subject's estimation of his position when the body was bent forward or backward. The examinations revealed the interesting fact that the errors in judging position were very slight when the body was tilted 60°. When tilting was less, the patient usually underestimated, while tilting more than 60° led to overestimation.

Great progress in functional testing of the otolithic reflexes was achieved when Grahe introduced his systematic examinations on his tilting table. He tested the vertical sensations, the head-righting reflexes, the spontaneous position of the head, and the compensatory eye positions.

In these tests, the patient is fixed upon the tilting table (the arms are free, the eyes are shut, head and body form a straight line). The table is first tilted backward 25° to 30°, then slowly lifted straight up. The patient is asked to tell when he thinks he has reached exactly the vertical position. Now the angle between his alleged vertical position and the true vertical plane is measured. The patient is then tilted forward 20°, then slowly lifted straight up until he again thinks he has reached a vertical plane. The same maneuver is performed over a frontal axis, i.e., tilting to the right and to the left side, and the angle between alleged vertical position and true vertical line is determined.

*Tests for head-righting reflexes* (Grahe). The patient is fixed on the tilting table (the head is free, the eyes are shut). The table is first tilted forward and backward, then to the right and to the left side. In each position the head of the patient is carefully watched. A normal person when tilted in any direction always has the tendency to bring the head into normal position (righting reflex). For example, a person tilted to the right side turns his head to the left.

*Tests for spontaneous position of the head* (Grahe). The patient is fixed on the tilting table and brought into a vertical position. Under normal conditions, head and body form a straight line. When a disease of the otolithic apparatus is present, a deviation or deflection of the head can be noticed. The head is either turned or tilted to one or the other side.

*Tests for compensatory eye positions.* To any given position of the head there corresponds a certain position of the eyeballs. Hence a change in the position of the head leads to deviation of the eyeballs. Since these deviations represent true otolithic reflexes (tonic labyrinthine reflexes) they cannot occur when the labyrinths are destroyed, as in certain cases of deaf-mutism. However, these reflexes play a much greater role in animals than in man. The deviation of the eyeballs may occur either in the frontal plane (counterrolling) or in the vertical plane (vertical deviation) (p. 94).

Bárány was the first to introduce this reaction into clinical tests. He constructed a certain apparatus that enabled him to measure the angle of deviation. Average figures on tilting the head 60° toward one shoulder are from 4° to 16°. Many authors did not consider the Bárány test an exact method and constructed other devices. Some use the ophthalmoscope to measure the deviations on the eye ground. Reference is suggested to the original articles on this subject. Since all the methods are more or less complicated, and since the results vary to a great extent, it must be stated that to date a clinical test that can be used for routine examination does not exist.

Recently G. Schubert and G. Brecher described a method of determining the vertical deviations on tilting the head forward and backward. In order to eliminate the neck reflexes, they prevent changes of the position of the head; in order to eliminate the fixation factor, they use a homogeneous opalescent glass with diffuse illumination (cf. original article).

*Tests for positional nystagmus.* There are cases in which nystagmus can be observed only when the patient assumes a certain position of the head or body (p. 59). Since nystagmus is usually associated with vertigo, nausea, etc., the patient himself knows best how to find this position. In testing for positional nystagmus, it should not be overlooked that the patient himself can render valuable help. When such efforts do not

succeed, a systematic examination must be made. The patient is first tested in dorsal recumbent position, then in either lateral or vertical position, and finally again in dorsal position with the head overextended (p. 117).

*Practical limitations.* It is a striking fact that none of the numerous tests concerned with the function of the static labyrinth has found its way into the routine clinical examinations. There are many reasons, such as the following.

In spite of important and instructive experimental work on animals, it is still not proved satisfactorily what role the otolithic organ plays in man. Further, there are a number of reflexes in animals that cannot be observed in man, either because they are superposed and masked by other reflexes or because they may not exist at all. For example, it is very difficult to determine the head-righting reflexes of the body. The same holds true for the tonic labyrinthine reflexes in man. Similar difficulties apply in relation to the various methods of testing postural estimation. It appears to be very difficult to analyze the influence of the labyrinth, the role of the kinesthetic sensations, and the part played by the optic factor in the testing errors. No wonder that the resulting figures of the respective authors vary. Thus, for example, Alexander and Bárány found, in opposition to others, that the errors in postural estimation as between normal persons and deaf-mutes (without labyrinthine function) do not differ. The same divergence can be noticed in the results of the test for compensatory eye positions.

To all these various arguments it must further be added that most of the tests require more or less complicated apparatus. However, these critical arguments do not permit the conclusion that the function of the static labyrinth should not be tested. On the contrary, all effort must be made to improve the various methods and to convert them from the present laboratory stage into practical functional tests. This aim can be achieved only when clinical examinations on a large scale are systematically performed and average figures are obtained. I agree with Grahe and with Tait and McNeil, who demand much more use of the tilting table. It would also be of great diagnostic value if a simple clinical test for the compensatory eye positions could be found.

### MOVEMENT REACTIONS AND REFLEXES

*Rotatory and postrotatory sensations.* These sensations occur only in movements with angular acceleration, while they are absent in motions with steady velocity. The direction of the rotatory sensations depends solely upon the axis on which the *head* of the patient is turned, and does not involve any other part of the body. During rotation the sensations

relate to the same direction as the turning. When rotation is stopped the sensations relate to the opposite direction. Fischer and Wodak revealed that the postrotatory sensations show a rhythmic course similar to that of the postrotatory nystagmus (p. 83). For example, after stopping of rotation to the right, the patient has a sensation of being turned to the left (first negative phase). This sensation decreases gradually until it subsides entirely (first intermission). After a short period another turning sensation will occur, but now it is a feeling of motion to the right side (first positive phase). This is followed by another stage of intermission (second intermission) and shortly by another sensation of turning to the left side (second negative phase), etc. Sometimes six such phases can be noticed over a period of fifteen minutes. This rhythmic course can be observed only when the head of the patient is fixed so that no change in position can occur.

*Head-moving test.* It was already observed by the older neurologists that forced head movements can cause intensive vertigo in certain patients. Bárány and Borries were able to produce nystagmus also by forced head movements. The latter author differentiates between kinetic nystagmus—elicited by forced head movements—and static nystagmus, elicited by certain positions of the head. This static nystagmus seems to be identical with positional nystagmus.

Mygind discriminates between nystagmus elicited by the *ampulla* and nystagmus elicited by the *otoliths*. The ampullar type is caused by *sudden changes* in position; the onset is sudden, and the duration is short (a few seconds); there is a dependence on movement and not on position. The otolith type is independent of rapidity of movement; its duration is determined by the position of the head, the onset is slow, there is a dependence on the *position* of the head and not on movement. Bárány found head-moving nystagmus in inflammatory diseases of the labyrinth, while Brunner described this symptom in diseases of the bony inner-ear capsule (para-otitis interna purulenta), in chronic adhesive processes of the middle ear, and in trauma of the inner ear. According to McNally, the vertical canals are diseased when the patient is falling in the direction of the forced head movement, while the utriculus is affected when falling occurs in the opposite direction. However, this theory is not proved as yet.

The technic of the head-moving test is as follows. The head of the patient is bent forward 90° and the eyes are shut (Fig. 26). The examiner stands in front of the patient, holding the head with both hands. He suddenly jerks the head of the patient backward and has him open his eyes. At the same time he watches the eyeballs for nystagmus, which usually is very coarse, of rotatory type, and directed toward the diseased side. The duration is usually very short. Occasionally the head-moving

nystagmus can also be elicited when the head of the patient is forcefully tilted toward the shoulder of the diseased side.

*Head nystagmus.* This reaction is often found in animals, occurs occasionally in sucklings, but is very rare in adults. According to Bárány, it is a "past-pointing reaction of the head" in the direction of the slow component. Alexander turned sucklings in the horizontal plane and described a slow movement of the head in the turning direction.

*Discus-thrower reaction.* The patient is placed in a turning chair with the head fixed and is spun ten times over a sagittal axis. When turning is suddenly stopped, a period of a few seconds must elapse before the

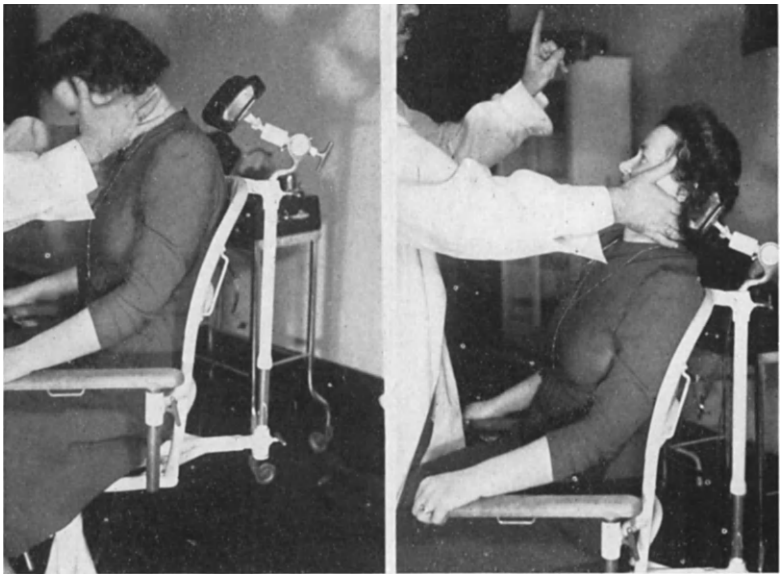

FIG. 26. HEAD-MOVING TEST

patient is ordered to get up from the chair and to hold his arms straight forward. This time is required for the cessation of the postrotatory sensations of the first negative phase. When, for example, the patient is turned to the left, the following reaction can be observed, according to Fischer and Wodak. The trunk of the patient turns slowly to the left and is tilted from the hip joint toward the left side, while the head and arms are markedly turned to the left. At the same time a lowering of the left arm and a raising of the right arm can be noticed. Since this position resembles the posture of a discus thrower, the reaction is called "discus-thrower reaction." If the turning stimulus has been very strong, the patient will fall to the left side. However, when such a falling is prevented,

the discus-thrower posture gradually subsides until the position of the patient becomes normal. Shortly afterward the same reaction will occur, but this time toward the right side. After a while the position of the patient will again be normal. According to Fischer and Wodak, this reaction is caused by vestibular reflexes, while Grahe believes that the reaction is the result of a combination of vestibular reflexes and the body reflexes of Magnus and De Kleyn.

*Falling reaction.* Stimulation of the labyrinth results either in a tendency to fall or in a true falling. The direction of falling is always the same as that of the slow component of the induced nystagmus. For example, a patient with nystagmus toward the right side—i.e., the quick component goes to the right, the slow component to the left—shows a falling or a falling tendency to the left side. When now the head of the patient is turned to the left, the quick component is directed forward, while the slow one is directed backward. Hence the patient now falls backward. In such a way the falling can be directed by the position of the head. It is obvious that the falling reaction can be influenced only as long as the peripheral sense organ (labyrinth and nerve) is still functioning.

The majority of otologists consider the falling reaction as a result of vestibular reflexes. In opposition to them, Fischer and Wodak make a sharp differentiation between "falling reaction" and "vestibular falling." The former can be observed when the patient gets up from the chair immediately after turning is stopped (falling reaction). The latter can be observed only when the patient, after turning stops, remains quiet in the chair for a certain period of time. Only after the first negative phase of postrotatory sensation has subsided does he get up from the chair.

*Past-pointing reaction* (Bárány). The patient is seated in a chair with his arm extended forward and the index finger stretched out. He is ordered to raise his extended arm from a starting point on his knees up to the index finger of the examiner held in the horizontal plane. Then he again goes down to the starting point and up again until he touches the examiner's finger (Fig. 27). He then repeats the same test with his eyes shut. The examiner watches carefully any deviation of the finger. It is important to distract the attention of the patient from the test in order to prevent testing errors. For such purposes there are many modifications of the Bárány test. Almost every author has his own trick. However, all the various methods have the same purpose, namely, the patient must simultaneously perform more or less complicated maneuvers with the other arm. The direction of past-pointing is always that of the slow component of the nystagmus. When, for example, a patient has a nystagmus toward the right, past-pointing to the left side will be noticed, i.e., deviation of the right arm inward and of the left arm outward.

Past-pointing can be examined in relation to the various joints (shoulder, elbow, knee, hip, etc.). For routine examinations, tests on the shoulder joint, as described above, are quite sufficient. Past-pointing can occur either spontaneously or after experimental stimulation of the labyrinth (caloric, turning, galvanic, etc.). Before testing the past-pointing reaction, the spontaneous past-pointing should first be examined.

According to Bárány's theory on past-pointing, there are certain "tonus centers" in the cerebellum that can be stimulated from the vestibular apparatus. The tonic influence of the labyrinth causes an increase of

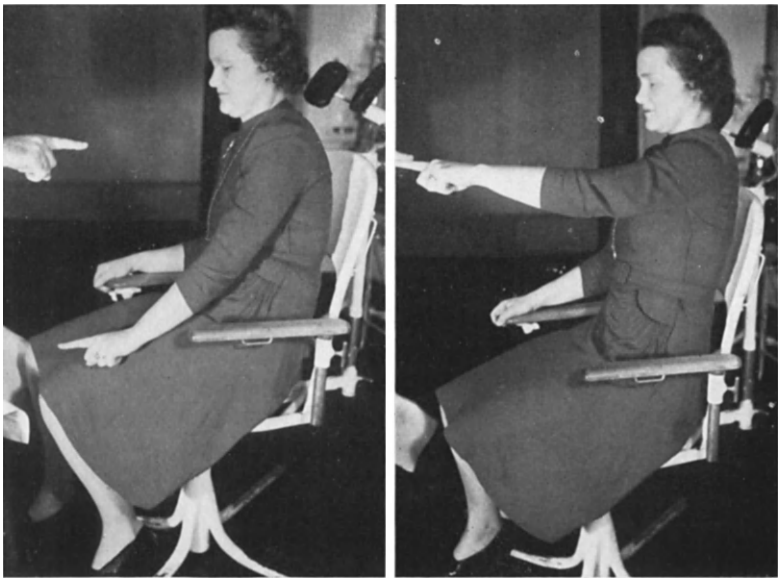

FIG. 27. PAST-POINTING REACTION

tone of the agonists, and a decrease of tone of the antagonists; this in turn leads to a deflection of the arm in the direction of the increased tone, i.e., in the direction of function of the extensors and abductors. Bárány assumes that in the cerebellar cortex there are centers for pointing to the right and the left and upward and downward. Furthermore, he believes that for the various joints (shoulder, elbow, knee, etc.) separate centers in the cerebellum exist. When a person raises or lowers his extended arm, it moves as if governed by two reins, one tending to pull the arm to the left, the other to the right side. When both reins pull with the same power, a certain balance is maintained and no past-pointing can occur. However, if one rein is severed, for example, the one that pulls the arm inward, then the other predominates, with the result of pulling the arm

outward. That is Bárány's explanation of the spontaneous past-pointing. In regard to the experimental past-pointing, he considers the labyrinthine stimulation on one side (caloric, turning, galvanic, etc.) as increasing the tension of one rein with the result that it predominates over the other. Hence it also leads to deviation of the arm to the same side.

These theoretic assumptions and conclusions of Bárány are almost generally accepted as facts. However, there is no proof as yet that such centers in the cerebellum do really exist. According to Goldstein, the cerebellum has a supporting influence upon the flexors and adductors innervated from the forebrain, and an inhibitory influence upon the extensors and abductors. Hence in diseases of the cerebellum the past-pointing is always in the outward direction.

There is scarcely any reaction that has aroused as much general interest as the past-pointing test. The number of experimental and clinical papers published on this subject by otologists, neurologists, and physiologists is tremendous. However, the results and conclusions of the various authors differ to a great extent. The reason for all the divergence of opinion lies in the fact that the past-pointing test represents a very complicated reaction. Not one factor but many are responsible for the reaction. The labyrinth as well as the cerebellum, also visual, tactile, psychic, and other factors determine the results. For example, past-pointing can be elicited by the use of glass prisms, by extreme sideward looking, by head turning, by certain muscular actions, by various skin stimuli, even by hypnosis and suggestion. Furthermore, past-pointing can also occur in connection with vertigo, psychic strain, fatigue, even too tight clothing. It is obvious that all these extravestibular factors influencing the past-pointing reaction must diminish its clinical value as a diagnostic labyrinthine test. It is beyond our scope to discuss all the details of this subject.

From a practical point of view, two questions arise for the otologist: Has the past-pointing reaction clinical significance in diseases of the labyrinth? Has the reaction diagnostic value in diseases of the cerebellum? The answer to these questions could be found in the conclusions to which Bárány came as a result of his tests, providing they are justified. He made the following statements. In unilateral diseases of the *labyrinth* or the *vestibular nerve* there occurs a spontaneous past-pointing with both arms in the direction of the diseased side. The experimental past-pointing reaction is absent on both sides. In unilateral diseases of the *cerebellum* there occurs a spontaneous past-pointing of the homolateral arm in the direction of the diseased side. The experimental past-pointing reaction is absent only in the homolateral extremity. It is not quite clear why the experimental past-pointing on both sides cannot be elicited in cases of unilateral diseases of the labyrinth.

Bárány not only differentiates between labyrinthine and cerebellar diseases, but he uses his test also for further localization of the disease within the cerebellum. For example, a spontaneous past-pointing of the right arm toward the right side can be due either to an *irritation* of the center for outward pointing in the cerebellum, or to a *destruction* of the center for inward pointing. When experimental stimulation of the labyrinth leads to a cessation of the spontaneous past-pointing, a destructive process in the tonus center must be assumed. When, however, experimental stimulation of the labyrinth does not eliminate the spontaneous past-pointing, an *irritative* lesion of the tonus center is to be assumed.

Bárány described exact localization of the disease with the aid of his past-pointing tests. For example, a loss of the inward motion in the right wrist points to a lesion in the antero-inferior part of the biventer lobe of the cerebellum. Loss of the downward past-pointing reaction in the arm speaks for a lesion in the medial part of the superior semilunar lobe. A loss of the past-pointing reaction in the arm toward the outer side makes probable an affection in the lateral part of the inferior semilunar lobe in the cerebellum, etc. All these conclusions sound very encouraging for the clinician.

Many years ago I made large-scale examinations on this subject from a practical point of view. Main attention was given to the question of the clinical significance of the past-pointing reaction. My studies were made on 130 cases of brain lesions associated with an increased endocranial pressure and otogenic complications. In the overwhelming majority of cases the diagnosis was proved either by the operation or by the post-mortem examination.

In summarizing my studies I came to the following conclusions. In cases in which the results of the past-pointing test accord with the other otologic and neurologic symptoms, it can be helpfully used. If, however, a divergence occurs between the past-pointing test and other symptoms, care must be taken in using the test for diagnostic purposes. Subsequently I had many opportunities (as consulting physician of a neurologic clinic) to continue my studies. I also had opportunity to observe many cases of otogenic cerebellar abscesses in the otologic department of the Vienna Policlinic. I saw cerebellar abscesses of such an extent that almost an entire hemisphere had been destroyed, but the past-pointing test was still negative. I therefore cannot see any reason for revising my former position with respect to the clinical value of the past-pointing reaction.

Goldstein stresses the fact that the past-pointing reaction can be of diagnostic value if one knows how to eliminate the various disturbing factors. The patient should be examined in a quiet room where there can be no disturbance of his attention. The light should come from a source in front, so that unequal light on the eyes is avoided. The examiner

should carefully avoid testing the past-pointing reaction in maximal abduction or maximal adduction of the arms, since in such a position past-pointing can be observed also in normal persons. It is further important not to let the patient know of his pointing errors; the examiner should rather correct the errors by his own arms, so that the patient is not aware of them.

*Deviation reaction* (Guettich). The patient, holding his head straight, is ordered to stretch both arms forward and to keep them in this position. The examiner stands behind him, fixing with his hands the chin of the

FIG. 28. DEVIATION REACTION

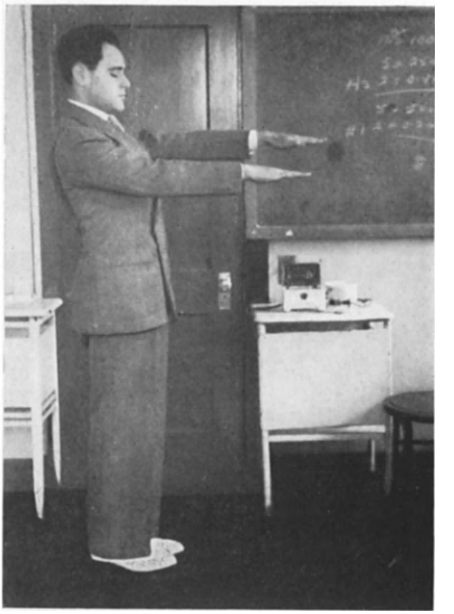

FIG. 29. ARM TONUS REACTION

patient. When he pulls slightly on the chin, the cervical spine of the patient becomes slightly stretched. A normal person will keep his extended arms parallel, while under pathologic conditions a deviation (in the horizontal plane) or a deflection (in the vertical plane) may occur (FIG. 28).

The difference between the past-pointing reaction (Bárány) and the deviation reaction (Guettich) lies therefore in the fact that the former is the result of active motions (lowering and raising of the arms) while the latter tests the extremities at rest. According to Guettich, the deviation reaction has the same diagnostic value as the Bárány test but does not show so many testing errors, since it does not require equally complicated

movements. Guettich explains the reaction as a transfer of the center of gravity.

*Arm tonus reaction* (Wodak). After stimulation of the labyrinth (turning, caloric, galvanic, etc.) the patient is ordered to stretch his arms forward with his eyes shut and to keep the arms in this position. The following reaction can then be observed. The arm on the side of the stimulated labyrinth sinks down, while the other arm moves upward (FIG. 29). Wodak explains this phenomenon as a change in the gravity sensation. The patient has the impression that one half of his body becomes heavier, the other half lighter. The former sensation corresponds to an increase in tone, the latter to a decrease. I cannot see much difference between this reaction and the tests of Guettich or of Bárány.

*Reactions to linear movement.* Reflexes in response to changes of position along a line in space (progressive movement) are common in animals, can occasionally be observed in sucklings, but are very rare in adults. They therefore play no role in the functional tests. There are two reflexes occasionally observed that resemble the reflexes found by Magnus and De Kleyn in animals (p. 96).

In testing *springing-poise reaction*, a suckling is lifted up, held by the examiner's hands around the body. When the child is suddenly lowered, the arms are extremely extended and the fingers stretched out.

When, in order to test *reaction to lifting*, a suckling in squatting position is lifted up, the arms become flexed and the head is lowered. Lowering of the child has an opposite effect.

### REACTIONS AND REFLEXES UPON INADEQUATE STIMULI

These reactions and reflexes have the same character as those elicited by adequate stimuli. There is only a quantitative difference, since they are less intensive and less marked. Otherwise they follow the same rules; for example, the falling and the past-pointing reaction occur in the direction of the slow component of the nystagmus.

*Caloric stimulation.* The *subjective* manifestations are usually reported as turning sensations (J. Fischer and I. Sommer). In order to prove that these turning sensations are elicited by the semicircular canals, we stimulated the various canals in a great number of patients. Our subjects included blind persons, deaf-mutes with and without labyrinthine function, and patients with various diseases of the ear organ. The patients were first examined in a dark room and then in an illuminated one. We found turning sensations after irrigation with cold water in 70 per cent of cases with functioning labyrinths. The direction of the sham movements was not characteristic. However, in those cases in which a certain direction of turning in the dark room had occurred, a change in the illumination

had no influence at all. We came to the conclusion that the turning sensations after calorization depend upon the function of the semicircular canals, but are independent of the function of the eyes.

According to Fischer and Wodak, after caloric stimulation of the labyrinth the following *objective* reflexes can be observed: head-turning reflex, body-turning and tilting reflex, past-pointing reaction, and arm tonus reaction.

C. Hirsch has presented recently a new labyrinthine test which he calls the waltzing test. The patient's ear is irrigated with 5 cc. of water at 15 C. He stands with his head raised 60°, with both arms stretched out straight and with his eyes shut. He is now asked to flex and raise first one knee and then the other. After twenty or thirty seconds he begins to spin in the direction of the slow component of the caloric nystagmus, occasionally turning around 180° or 360°. Hirsch considers the waltzing reaction as a stimulation of the ipsilateral vestibular spinal tract. This test shows a striking resemblance to the treading test (*Tretversuch*) of Unterberger (p. 107).

On bilateral calorization Fischer and Wodak observed in certain cases a phenomenon of retropulsion: "When a patient with his head upright was irrigated with 100 cc. of water at 20 C., he had the impression of being pulled forward (propulsion), and after a short while of being pulled backward (retropulsion). Irrigation with warm water had the opposite effect." The authors considered this symptom as indicative of a lesion of the nucleus ruber.

*Galvanic stimulation.* The *subjective* manifestations depend upon the intensity of the electric stimulus. When only a light current is applied, the patients complain of a vague feeling of dizziness (tactile vertigo). When, however, the galvanic current is strong enough, typical turning sensations occur. The patients usually report sham movements of the surrounding objects, the direction of which depends on the galvanic stimulus—whether the anode or the cathode is applied.

The *objective* reactions are the same as with the caloric test. Stimulation with the anode corresponds to cold irrigation, while stimulation with the cathode has the same effect as warm irrigation. As far as the falling reaction is concerned, there are divergent opinions. Bárány believed that the falling reaction depends upon the position of the head, while others cannot find any relation. In diseases of the frontal lobe the falling occurs, according to French authors, always in the direction of the diseased side, no matter whether the anode or the cathode is used as a stimulus.

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## EVALUATION OF ABNORMAL REACTIONS

The labyrinthine excitability can be changed either quantitatively or qualitatively. The quantitative phenomena include hyper-, hypo-, and nonexcitability, while the qualitative group comprises changes in rhythm, form, direction, etc. These abnormal reactions may further represent all the various stimulations of the labyrinth or may relate to only one or the other stimulus. Thus, for example, the caloric excitability may be normal while the turning reaction is absent, or vice versa. Another combination may be seen in the fact that one and the same stimulus has a different effect on the various semicircular canals. For example, irrigation with cold water reveals a normal reaction for the horizontal canals while the vertical canals do not respond at all, or vice versa, etc. Hence the following seven conditions can be observed with regard to the abnormal excitability of the labyrinth.

### 1. Changes in Relation to Certain Stimuli

It is obvious that many combinations can be observed under the numerous kinds of stimulation. The following types of combination are essential in our discussion.

#### LOSS OF TURNING REACTION, NORMAL CALORIC REACTION

This form is often found in cases of lues; hence it is considered as pathognomonic of inner-ear syphilis. In many cases the loss of the labyrinthine function in response to turning stimuli has come gradually and has not been accompanied by the usual violent manifestations, such as intensive vertigo with turning sensations, nausea, vomiting, disturbance of balance, etc. Therefore the diagnosis of an absence of function often comes as a surprise. This symptomless destruction is also characteristic for inner-ear lues. Neumann assumed for such cases (loss of turning reaction with normal caloric reaction), pathologic changes in the nerve endings of the peripheral sense organ (crista ampullaris). Other authors find in the disproportion between the turning and the caloric reaction proof that the two stimuli do not act upon the same place in the *central* vestibular apparatus.

#### LOSS OF CALORIC REACTION, NORMAL TURNING REACTION

This form is found in cases of inner-ear lues as well as in certain cases of serous labyrinthitis, trauma of the skull, etc. Neumann explained the underlying pathology on the basis of changes in the chemical composition of the endo- and perilymphatic fluid (lymphokinetic apparatus).

#### LOSS OF CALORIC AND TURNING REACTION, NORMAL GALVANIC REACTION

This form indicates a disease of the retrolabyrinthine part of the vestibular apparatus. Such an assumption is based upon the clinical observations of Neumann, who succeeded in eliciting a galvanic reaction in cases of total extirpation of the labyrinth. It is generally accepted that the galvanic current acts upon the vestibular nerve, so that a differentiation between labyrinthine and retrolabyrinthine affection can be made.

#### LOSS OF GALVANIC REACTION, NORMAL CALORIC REACTION

Such results have been observed only occasionally. The reason may be that this form is really rare or that in the majority of cases the galvanic reaction was not tested. According to Rosenfeld the phenomenon indicates a severe lesion of the brain centers.

#### REACTIVE DISSOCIATION

This term is applied by Grahe to cases in which one labyrinth reacts much more easily to turning stimuli, while the other labyrinth responds much better to caloric stimuli. It is held to be a sign of a central lesion.

### 2. Changes in Certain Semicircular Canals

Jones and Fisher described in cases of cerebellopontile angle tumors a loss of caloric reaction in the vertical canals on the affected side, while the horizontal canals were normal. They explained this phenomenon on the basis of anatomic conditions. There is a central differentiation of the fibers from the horizontal canal and the fibers from the vertical canals. The latter lie more exposed than the former. According to Eagleton this symptom indicates an increased intracranial pressure in the posterior fossa. He differentiates between cases with complete loss of caloric function in the vertical canals and cases with only a reduction of the caloric excitability. The former are significant of *infra*tentorial tumors, the latter of supratentorial tumors.

This Jones-Fisher-Eagleton theory is strongly opposed by French authors (Barré). In recent times Maybaum also could not substantiate these findings.

Another symptom described by Eagleton is reduction of the duration of nystagmus elicited by stimulation of the horizontal canals on turning. Such a result occurs in lesions associated with a blockage of the cerebrospinal fluid pathways, such as protective meningitis in the posterior fossa and skull fracture.

### 3. Vestibular Disharmony

Vestibular disharmony occurs when, for example, the primary reaction (nystagmus) is absent, while the secondary reactions (vertigo, falling, past-pointing, etc.) are normal or vice versa. Further combinations can be seen when not all but one or the other secondary reaction is gone or, on the contrary, is markedly increased. Finally, there may be a positive reaction but in an atypical way. The following types of vestibular disharmony are to be noted.

#### ABSENCE OF PRIMARY REACTION (NYSTAGMUS)

On stimulation of the labyrinth no nystagmus occurs. Such a result can easily be mistaken for a loss of excitability of the peripheral labyrinth, if the physician does not examine for the secondary reactions. However, those cases are rather rare and indicate a central lesion somewhere between the vestibular nuclei and the eye nuclei.

#### ABSENT OR ATYPICAL FALLING REACTION

Under normal conditions the falling occurs in the direction of the slow component of the induced nystagmus. When, for example, a nystagmus toward the right side is produced (caloric or turning) the patient falls to the left side. If, however, no falling occurs or there is a falling to the right side, the phenomenon is spoken of as a vestibular disharmony.

According to Jones, the absence of the falling reaction indicates a lesion either in the vestibulo-cerebello-cerebral tract or in the cerebro-cerebello-spinal tract.

Atypical falling reaction has been observed in cases of inner-ear lues and of brain tumors. French authors found that in patients with temporal lobe tumors the direction of falling—after turning—is always toward the side of the affected ear, independently of the induced nystagmus.

#### ABSENT OR ATYPICAL PAST-POINTING REACTION

Under normal conditions past-pointing occurs in the direction of the slow component of the induced nystagmus. If after stimulation of the labyrinth a nystagmus is elicited—for example, toward the right—past-pointing to the left will occur, i.e., deviation of the left arm outward and of the right arm inward (p. 150).

Absence of the past-pointing reaction on both sides, associated with spontaneous past-pointing with both arms in the direction of the diseased side, indicates according to Bárány a unilateral disease of the labyrinth or the vestibular nerve. Absence of the past-pointing reaction only in the homolateral arm, associated with spontaneous past-pointing of the

homolateral arm in the direction of the diseased side, indicates a unilateral disease of the cerebellum.

Absence of the past-pointing reaction is significant of retrolabyrinthine diseases (Guettich). Absence of this reaction is often found in cases of lues and is considered to be the first symptom of a neurolabyrinthitis.

Anyone who undertakes the task of studying the tremendous literature concerned with past-pointing will soon find himself completely confused. Bárány, who inaugurated this test, assumed four centers in the cerebellum, one for each direction. Furthermore, he believed that separate centers for the various joints exist. The past-pointing can be the result of an irritation of one center or of destruction of another center. The past-pointing further depends upon the nystagmus, which may be horizontal, rotatory, vertical, etc. In addition to all this, many extravestibular factors influence the past-pointing test (p. 152). Taking all these points into consideration, one gets a faint idea of the numerous combinations that may theoretically exist.

I agree with Klestadt, who states that anomalies of the past-pointing or falling reaction should be used only for diagnostic purposes, if they occur constantly and if they fit in easily with the other labyrinthine and neurologic symptoms. I came to the same conclusions in my studies of brain tumors in 1920 (p. 153).

#### ABSENCE OF GIRDLE REFLEX

When a patient who has lost his girdle reaction is slightly pushed he will fall over (Shuster). Grant and Fisher observed this phenomenon in lesion of the cerebellum.

### 4. Hyperexcitability

The diagnosis of pathologically increased excitability of the labyrinth often meets with difficulties, since there is a wide physiologic range and since there are no exact methods for determining the normal excitability of the labyrinth in response to the various stimuli (p. 129). Therefore, clinical experiences must substitute for physiologic exactness. If there is doubt as to whether or not hyperirritability is present, the results of the various stimulations (caloric, turning, galvanic, etc.) should be compared, and differences between the right and the left side should also be looked for. Furthermore, attention must be given to spontaneous manifestations such as vertigo, spontaneous nystagmus, spontaneous falling, etc. Many years ago I made the suggestion that the diagnosis of hyperirritability be applied in only those cases in which, besides the usual symptoms, spontaneous nystagmus is present, even though only for a short time. Neumann opposed this suggestion. As far as vertigo is concerned, Alex-

ander and Brunner demand for the diagnosis of increased excitability either the presence of vertigo or at least a previous occurrence of it.

Hyperexcitability may be the result of an irritation of the vestibular nuclei or of the central pathways, or may also occasionally be caused by pathologic changes in the peripheral sense organ. The former type includes lesions in the posterior cranial fossa, such as vascular processes, skull trauma, encephalitis, disseminate sclerosis, and particularly processes with increased intracranial pressure. Neumann described in cases of cerebellar tumors a phenomenon which he called "nystagmoclonus." On stimulation of the labyrinth he observed in these cases an intensive coarse nystagmus that lasted several minutes.

Hyperexcitability may be caused either directly by lesions in the central vestibular organ or indirectly by a distant process in the anterior or middle fossa that exerts a certain pressure upon the central pathways (remote symptom). In the latter case the hyperexcitability occurs usually bilaterally, while in the former only one side is involved.

In cases associated with increased intracranial pressure, I have emphasized the occurrence of sudden changes in the ear symptoms, in the spontaneous manifestations as well as in the labyrinthine reactions. These observations led me to the conclusion that in all such cases repeated examinations have to be performed in order to prevent erroneous results.

Hyperexcitability of a peripheral origin is not as often found as is usually believed. It can be observed in cases of serous labyrinthitis, circumscribed purulent labyrinthitis, inner-ear lues, and in some cases of neuritis of the eighth nerve. Occasionally hyperirritability may be observed only in the very beginning of the disease and may shortly be followed by a hypo-irritability. Finally it can be stated that such an increased excitability may be noted either in the beginning or at the end of a disease, indicating restoration of the labyrinthine functions.

### 5. Hypo-excitability

*Hypo*-excitability always indicates anatomic changes, while *hyper*-excitability, as shown above, may well be of either organic or functional nature. Further, *hypo*-excitability more often originates in the peripheral sense organ, while the opposite is true for *hyper*excitability. If simultaneously there is a deafness in the same ear, the indication of a peripheral site of the lesion is almost certain.

Hypo-excitability is found in cases of labyrinthine suppurations, chronic adhesive processes with secondary degeneration of the inner ear, lues, and retrolabyrinthine lesions such as neuritis of the eighth nerve, neoplasm, trauma, etc.

As far as the question of central origin is concerned, hypo-excitability

often develops from a hyperirritability when the cause, for example increased pressure, persists over a long period of time.

Since the underlying pathology consists of more or less severe irreparable degenerative changes, a restitution of the labyrinthine functions cannot be expected.

### 6. Loss of Excitability

This represents only a higher degree of hypo-excitability. Therefore, everything said above is also true for this condition. Loss of excitability can be found in cases of acquired deaf-mutism, inflammations of the labyrinth, lues, neoplasm, head trauma, etc.

A central origin can be assumed in lesions of the posterior fossa, particularly of either large size or long duration. The prognosis with regard to restoration of the labyrinthine function is usually not good. However, there are certain cases, as of serous labyrinthitis or disseminated sclerosis, in which the function may become normal.

### 7. Qualitative Changes

These changes reflect disturbances in the rhythmic course of the nystagmus, in the relation between slow and fast component, in form and direction, etc. The following six groups are to be noted.

#### DISTURBANCE OF RHYTHM

In certain cases stimulation of the labyrinth produces an irregular nystagmus. Klestadt applied the term "dissected nystagmus" (*Nystagmuszergliederung*). De Kleyn observed a nystagmus of two to three jerks after turning the patient; then came an intermission followed by two to three more jerks. It is obvious that such irregularities can never originate in the peripheral sense organ but are of a central nature.

#### DECREASE OR LOSS OF THE QUICK COMPONENT

*Decrease* of the quick phase leads to a predominance of the slow component. Such a nystagmus appears very slow. *Loss* of the fast component leads to a conjugate deviation of the eyeballs toward the opposite side (p. 64).

This phenomenon was first found experimentally in animals but could later also be observed clinically in man (p. 119). Eagleton described cases of temporosphenoidal abscess in which irrigation of the labyrinth produced a conjugate deviation of the eyeballs in the direction opposite to that of the expected nystagmus. Grant and Fisher found a conjugate deviation instead of a nystagmus in 30 per cent of cases with supratentorial tumors after calorization of the labyrinth.

Brunner states that lesions in the posterior longitudinal bundles may lead to a loss of the fast component if they damage the pathways for the voluntary eye movements. A loss of *both components* of the nystagmus can be observed, according to Brunner, under the following conditions: destruction of the eye nuclei, or destruction of the posterior longitudinal bundle on both sides, or complete destruction of one longitudinal bundle with its arcuate fibers, plus partial affection of the other longitudinal bundle.

### CONJUGATE DEVIATION PLUS NYSTAGMUS

In certain cases stimulation of the labyrinth with cold may lead first to a conjugate deviation of the eyeballs toward the side of the stimulated ear, followed by a nystagmus to the opposite side. The eyeballs remain deviated during the entire reaction (Leidler). This indicates a central lesion.

### NYSTAGMUS PLUS CONJUGATE DEVIATION

On stimulation of the labyrinth with cold, a normal nystagmus toward the side of the nonstimulated ear is first elicited. After a short time a conjugate deviation of the eyeballs toward the side of the stimulated ear can be observed. The eyes remain deviated until the end of the reaction (Leidler). This phenomenon is significant of central lesions.

### INVERTED NYSTAGMUS

When after stimulation of the labyrinth a nystagmus occurs in the direction opposite to that expected, it is spoken of as an inverted reaction. For example, irrigation of the right ear with cold water produces normally a nystagmus toward the left side. If, however, a nystagmus toward the right side is elicited, it is an inverted reaction. The same effect can sometimes be obtained with warm stimuli, while inversion after turning stimulation is rather rare.

This phenomenon has been particularly studied by American otologists (Jones, Fisher, Grant, Shuster, etc.). According to these authors it is significant of brain stem lesions.

De Kleyn and Versteegh were able to produce inverted nystagmus experimentally in rabbits, using prolonged irrigations. In the beginning the nystagmus was typical but on continuation of irrigation the nystagmus became less and less. Before it subsided entirely the direction changed to the opposite of the typical direction (inversion). The authors also succeeded in eliciting this phenomenon in man. However, such prolonged irrigations are very uncomfortable for the patient. In 2 cases of inverted nystagmus—produced by the usual caloric test—the autopsy revealed a tumor of the fourth ventricle.

#### PERVERTED NYSTAGMUS

Under normal conditions stimulation of certain semicircular canals produces certain forms of nystagmus. If, however, the canals respond in an atypical way, the reaction is spoken of as a perverted nystagmus. For example, on stimulation of the horizontal canals a vertical nystagmus occurs. This phenomenon is also significant of lesions of the brain stem (Jones, Fisher, Grant, etc.). According to Aubry and Caussé, absence of induced rotatory nystagmus does not mean paralysis of the vertical canals, but is significant rather of a central lesion within the medulla oblongata.

## DIFFERENTIATION OF PERIPHERAL AND CENTRAL LESIONS

A fundamental mistake made by many otologists originates in their tendency to establish a neurologic local diagnosis based upon the results of the labyrinthine examination. This is because many articles and textbooks list a series of signs and symptoms for differentiation between peripheral and central lesions. Actually the matter is too complicated to allow of any schematization. The otologist can seldom establish a local diagnosis on the basis of single symptoms. The results of his examination should be evaluated only in connection with the findings of the neurologist, the ophthalmologist, the roentgenologist, etc. Thus he is able to render valuable aid to the neurologist without being forced to arrive at a local neurologic diagnosis. For this reason I shall neglect the usual list of symptoms, giving instead the clinical significance of all symptoms and reactions in the order followed in chapter iv.

### 1. Significance of Spontaneous Manifestations

#### VERTIGO

Symptoms pointing to *peripheral* origin are: attacks of true turning vertigo with sham movements of the subject or objects, associated with nausea and vomiting, depending upon the position of the head; spontaneous nystagmus, with falling in the direction of the slow component of the nystagmus; positive otoscopic findings such as acute or chronic otitis media or chronic adhesive processes, etc.; and, finally, complaint of tinnitus and impaired hearing.

Symptoms pointing *against* peripheral origin are: persistent dizziness over a long period of time (months or years), of the same or increasing intensity; vertigo with loss of consciousness; vague dizziness; blurring or double vision, etc.

Symptoms pointing to *central* origin are: vertigo with lateropulsion independent of the position of the head; disproportion between dizziness and nausea and vomiting; falling in the direction of the fast component of

the nystagmus; long duration and marked intensity of vertigo, associated with localized headaches; presence of spontaneous vertigo combined with absence of induced vertigo; negative ear findings and positive neurologic finding.

### SPONTANEOUS DISTURBANCES OF EQUILIBRIUM AND COORDINATION

Symptoms pointing to *peripheral* origin are: spontaneous disturbance of balance, dependent upon the position of the head, with falling in the direction of the slow component of the nystagmus; positive ear finding such as acute or chronic otitis media; spontaneous nystagmus; fistula symptoms; pathologically changed excitability of the labyrinth and impaired hearing.

Symptoms pointing to *central* origin are: spontaneous falling regardless of the position of the head, the falling being in the direction of the fast component of the nystagmus; negative ear finding; positive neurologic finding.

Symptoms pointing to *cerebellar lesions* are: disturbance of kinetic equilibrium, examined by the various walking tests, side-stepping test (Alexander), etc.; disturbance of coordination, such as *asynergie cérébelleuse* (Babinski), dysmetria, adiadokokinesis, disturbances of tone, etc. (chap. iv).

### SPONTANEOUS NYSTAGMUS

Spontaneous nystagmus indicates a lesion in the labyrinthine reflex arc. For further determination as to whether the nystagmus is caused by a disease of the peripheral labyrinth, of the central pathways, or of the peripheral eye organ, attention should be given to the following points: association or dissociation; form; direction; degree; amplitude; frequency; duration. *Dissociation* of nystagmus excludes a peripheral labyrinthine origin.

The *form* of the labyrinthine nystagmus is usually of a mixed type (⇄⇄); the simple forms (→↷ ↑ ) therefore point rather to a central or ocular lesion. Oblique nystagmus (↗) or undulating nystagmus can be only of ocular origin. Vertical nystagmus (⇅) usually indicates a central lesion (region between pons and quadrigeminal bodies). In cases of temporal lobe abscess, vertical nystagmus indicates a very unfavorable prognosis, since it is caused by a rupture of the abscess into the ventricle.

The *direction* of the nystagmus (in inflammatory diseases of the inner ear) is at first toward the side of the diseased ear (irritation effect), later toward the opposite side (destruction effect). When in the further course of the disease the nystagmus changes back toward the affected side (after destruction of the labyrinthine function), it is pathognomonic of an ex-

tension of the process into the posterior cranial fossa. Diseases of the central labyrinthine pathways usually produce a nystagmus toward the affected side. The *degree* of the nystagmus may be helpful in a diagnostic sense, when it is of high intensity (second or third degree). If such a nystagmus is of a peripheral labyrinthine origin, it is always accompanied by severe labyrinthine symptoms, such as vertigo with sham movements, nausea, vomiting, disturbance of balance, etc. Absence of these symptoms in the presence of a nystagmus of second or third degree points against a peripheral origin, but favors the assumption of a central cause, for instance syringobulbia.

*Amplitude* and *frequency* are usually medium in diseases of the labyrinth, while in central lesions the nystagmus is rather coarse and slow. Since, however, too many unknown factors control amplitude and frequency, no further conclusion can be drawn.

The *duration* of a nystagmus may also be utilized for diagnostic purposes. In cases of inflammation of the labyrinth the nystagmus is self-limited. It usually regresses gradually and subsides after days or weeks. Nystagmus persistent in intensity over a long period of time (months or years) points against labyrinthine origin. Central nystagmus is usually persistent and may increase in degree. Ocular nystagmus persists for the duration of life and may not show any change in form or intensity.

### POSITIONAL NYSTAGMUS

Positional nystagmus, i.e., occurrence of nystagmus only with certain positions of the head, has been found in association with (*a*) diseases of the *labyrinth*—the direction of the nystagmus being always the same, regardless of the position of the patient; (*b*) *tumors* and *trauma*—the nystagmus being dependent upon the position of the patient and changing with the change of position; (*c*) *central lesions*—the nystagmus changing in direction even though the patient maintains the same position.

### ASSOCIATED EYE MOVEMENTS (CONJUGATE DEVIATION, CONJUGATE PARESIS)

Disturbances of the associated eye movements are due to lesions in the cortical centers and in the central pathways (diseases of cerebrum or pons). The labyrinthine reflexes exert a certain influence upon these eye movements. Simultaneous involvement of the labyrinthine reflex arc may lead to disturbances of induced nystagmus. Hence the otologist may render diagnostic aid to the neurologist. For example, cold irrigation of the left labyrinth should normally produce nystagmus with the fast component toward the right and the slow component toward the left. If, however, the slow phase can be observed only in the left eye, a nuclear lesion (left

abducens) can be assumed. But if the stimulation produces a conjugate deviation toward the left (which means that the slow labyrinthine component is elicitable), it may be concluded that the lesion is not in the labyrinthine pathways but in the *supranuclear* region.

Differentiation between *cortical* and *pontine* lesions is made by Bielschowsky as follows:

Symptoms pointing to *cortical* origin: Conjugate deviation of the eyes and rotation of the head toward the affected side; transient conjugate paresis with decrease of eye movements toward the contralateral side; ipsilateral paralysis of the facial nerve and of the extremities; normal responses on stimulation of the labyrinth.

Symptoms pointing to *pontine* origin: Conjugate deviation of the eyes and rotation of the head toward the unaffected side; conjugate paresis with loss of eye movements toward the affected side; ipsilateral paralysis of the facial nerve; contralateral paralysis of extremities. Stimulation of the labyrinth elicits a nystagmus with decrease or loss of the quick component and a deviation of the eyeballs toward the opposite side.

## 2. Significance of Induced Reactions

### OPTOKINETIC NYSTAGMUS

The test is used to differentiate a labyrinthine and a nonlabyrinthine spontaneous nystagmus. On turning of the optical wheel, reaction may occur in three possible ways: (*a*) optokinetic nystagmus in the direction opposite to that of the turning; (*b*) optokinetic nystagmus in the same direction; (*c*) no nystagmus at all.

The first result represents the normal response, while the two others are atypical reactions (inversion). The phenomenon of inversion indicates a nonlabyrinthine origin of the spontaneous nystagmus, i.e., a central or ocular site. Atypical optokinetic reaction combined with conjugate paresis of the eyeballs is significant of a beginning increased endocranial pressure (Brunner).

### CALORIC NYSTAGMUS

On caloric stimulation of the labyrinth, the following reactions should be studied:

Changes of *duration* of nystagmus (hyper- or hyposensitivity);
Changes of *latent* period (hyper- or hypo-irritability);
Changes of *duration* plus *latent* period (hyper- or hypo-excitability);
Complete *lack* of excitability;
Lack of excitability in certain canals;
*Qualitative* changes.

The *duration* of the caloric nystagmus expresses the condition of the central vestibular organ (vestibular nuclei and pathways). An increase of duration is usually associated with a decrease of the latent period. There may, however, be cases in which only the duration of the caloric nystagmus is markedly increased (hypersensitivity).

The *latent period* is an expression of the *peripheral* irritability of the labyrinth and has two components: one component relates to the cooling of the tissue (physical factor), the other to the irritability of the labyrinth and to the conductivity of the vestibular nerve (physiologic factor). Separation of the two components is discussed in connection with functional tests (p. 128).

*Hyperexcitability* is more often of *central* than of peripheral origin. The former causation applies to lesions in the posterior cranial fossa, such as vascular processes, skull trauma, encephalitis, disseminate sclerosis, and particularly processes with increased intracranial pressure. Such increased pressure leads usually to a bilateral hyperexcitability, while the other central lesions affect mainly only one side. Peripheral hyperexcitability does not occur as often as is generally believed. It can be found in cases of early labyrinthitis, inner-ear lues, or neuritis of the eighth nerve.

*Hypo-excitability* more often originates in the *peripheral* sense organ. It always indicates anatomic changes, while hyperexcitability may well be of either organic or functional nature. *Peripheral* hypo-excitability is found in connection with labyrinthine suppurations, chronic adhesive processes with secondary degeneration of the inner ear, lues, and retrolabyrinthine lesions such as neuritis of the eighth nerve, neoplasm, trauma, etc. A positive otoscopic finding and impaired hearing in the same ear makes peripheral origin almost certain. *Central* hypo-excitability may develop from a hyperexcitability when the cause, for example increased endocranial pressure, persists over a long period of time.

*Complete lack of caloric excitability* represents a higher degree of hypoexcitability. It can be found in cases of acquired deaf-mutism, suppuration of the labyrinth, inner-ear lues, neoplasm, head trauma, etc.

*Loss of caloric reaction in the vertical canals* on the affected side, associated with a normal response in the horizontal canals, is significant of cerebellopontile angle tumors (symptoms of Eagleton, Jones, Fisher).

*Qualitative changes* include disturbances in the rhythmic course of the caloric nystagmus, loss of the fast component, the inverted and perverted nystagmus reaction, etc. It is obvious that such irregularities can never originate in the peripheral sense organ but have a central site, particularly in lesions of the brain stem.

When the *warm-cold contrast reaction,* i.e., alternating irrigations with cold and warm water, reveals that nystagmus toward one side can much

more easily be elicited than movement toward the other side, a central lesion may be assumed (p. 128).

### BILATERAL CALORIZATION

This test may render diagnostic aid to the neurologist. It is indicated in cases in which a differentiation has to be made between lesions of the posterior cranial fossa and lesions in the middle and anterior fossa. The method applies particularly to processes of increased endocranial pressure (supra- and infratentorial tumors). The test is called positive when after bilateral calorization a nystagmus of second or third degree is elicited, and negative when either no nystagmus or nystagmus of the first degree or vertical nystagmus is produced.

A positive reaction points to a lesion in the posterior cranial fossa. The phenomenon of "retropulsion" (Fischer and Wodak) is significant of a lesion of the nucleus ruber. On bilateral calorization, such patients get the impression of being pulled forward (propulsion) and shortly thereafter of being pulled backward (retropulsion).

### TURNING NYSTAGMUS

As in the case of the caloric nystagmus, efforts have been made to utilize the *turning* stimulation for various diagnostic purposes. Some reactions are still in the experimental stage and have not as yet been introduced into the routine clinical examination. Attention should be given to:

Differentiation between peripheral irritability and central sensitivity;
Central tone difference;
Hyperexcitability;
Hypo-excitability or loss of excitability;
The phenomenon of compensation;
Lack of excitability of certain canals.

Differentiation between *peripheral irritability* and *central sensitivity* can be made by study of the various phases of the postrotatory nystagmus (p. 83). The first phase expresses peripheral as well as central excitability, while the second, third or following phases relate only to the sensitivity of the central vestibular organ.

*Central tone difference* can be diagnosed when, for instance, the second phase of one side and the first phase of the other side are markedly increased. This test corresponds to the warm-cold contrast reaction of the caloric examination.

*Hyperexcitability* is more often of central than of peripheral origin. Everything said in relation to caloric hyperexcitability can be applied here.

*Hypo-excitability* or loss of excitability usually originates in the peripheral sense organ, as described in our discussion of the caloric reaction (p. 163).

The *phenomenon of compensation* (Ruttin) is usually observed in cases where a unilateral *complete* destruction of the peripheral sense organ has taken place, but does not occur if only a part of the labyrinth has been destroyed. The average figures of compensation are: for the horizontal canals, ten to fifteen seconds; for the frontal canals, six to eight seconds. For the vertical canals no compensation exists (p. 139).

*Lack of excitability* of certain canals may occasionally be found in cases of inner-ear lues. According to Eagleton, a reduction of the turning nystagmus elicited by the horizontal canals is significant of lesion associated with a blockage of the cerebrospinal fluid pathways. Such lesions are protective meningitis in the posterior fossa and skull fractures.

### FISTULA SYMPTOMS

Differentiation must be made between the true and the pseudo fistula symptom.

The *true fistula* symptom is found in cases in which a circumscribed defect exists in the bony labyrinthine capsule. There are, however, cases in which the reaction is negative in spite of an existing fistula, namely, when the defect is covered by polypi or cholesteatomatous masses (FIG. 37). There is great controversy over the direction of the induced nystagmus. It must, however, be borne in mind that the direction does not play any major role. The only important factor for the diagnosis of a fistula is whether or not eye movements can be elicited upon pressure or aspiration.

The *pseudo fistula* symptom can be produced in certain cases of inner-ear lues (particularly congenital syphilis) with a normal drum membrane finding. The symptom may be due to the luetic inflammation of the eighth nerve, which leads to increased conductivity for mechanical stimuli. Other forms of pseudo fistula symptom are of a vascular origin, such as pressure on the carotid artery, pressure on the veins of the neck, etc.

### GALVANIC REACTION

The purposes of the galvanic test are: (*a*) differentiation between labyrinthine and retrolabyrinthine diseases; (*b*) differentiation between peripheral and central diseases; (*c*) determination of slight differences in the excitability of each side.

*a*) Differentiation between *labyrinthine* and *retrolabyrinthine* diseases can be made by comparison of the galvanic examination with the other labyrinthine reactions. Loss of function in response to caloric, turning, and mechanical stimuli, as well as to the galvanic current, points to a

labyrinthine lesion. Loss of function in response to caloric, turning, and mechanical stimuli, with normal response to the electric current, is significant of a retrolabyrinthine disease (vestibular nerve affection).

*b*) Differentiation between *peripheral* and *central* diseases has recently been made by determination of the vestibular chronaxia (p. 143). In central lesions, chronaxia yields normal results, while in peripheral diseases changes of the vestibular chronaxia occur. However, this test has not as yet been introduced into the routine clinical examination.

*c*) Determination of *slight differences* in the *excitability* of each side can be revealed by use of the double galvanic test (p. 143). When the stimulating electrode is used as the cathode, the nystagmus is directed toward the side of the greater excitability, while when the stimulating electrode serves as the anode the nystagmus is directed toward the side of the labyrinth with the lesser excitability.

### POSTURAL REFLEXES AND REACTIONS

In this group belong the tests for postural sensation, for head-righting reflexes, for position of the head, for compensatory eye movements, and for positional nystagmus. None of these various tests has found its way into the routine clinical examination. The reasons are that the role played by the otolithic organ in man has not been proved satisfactorily, that many of these tests are still in the laboratory stage, and that many of them require more or less complicated apparatus.

### HEAD-MOVING TEST

This test may render valuable help in diagnosing inflammatory diseases of the bony labyrinthine capsule (para-otitis interna purulenta). The test may further be positive in cases of chronic adhesive processes in the middle ear and in cases of trauma of the inner ear.

### FALLING REACTION

*Typical* falling occurs in the direction of the slow component of the induced nystagmus.

*Atypical* falling is observed in some cases of inner-ear lues and of brain tumors.

*Absence* of falling is, according to Jones, significant of lesions either in the vestibular cerebello-cerebral tract or in the cerebro-cerebello-spinal tract.

### PAST-POINTING REACTION

Bárány introduced this test for (*a*) differentiation between labyrinthine and cerebellar diseases, and (*b*) local diagnosis in cases of cerebellar lesion.

*a*) Differentiation between *labyrinthine* and *cerebellar* diseases can be made on the basis of the following results:

In unilateral diseases of the *labyrinth* (or vestibular nerve) there occurs a spontaneous past-pointing with both arms in the direction of the diseased side. The experimental past-pointing reaction is absent on both sides.

In unilateral diseases of the *cerebellum* there occurs a spontaneous past-pointing of the homolateral arm in the direction of the diseased side. The experimental past-pointing reaction is absent only in the homolateral extremity.

*b*) *Local diagnosis* in cases of *cerebellar lesions* can be made on the basis of these facts:

Loss of the inward motion in the right wrist points to a lesion in the antero-inferior part of the biventer lobe of the cerebellum.

Loss of the downward past-pointing reaction in the arm indicates a lesion in the medial part of the superior semilunar lobe.

Loss of the outward past-pointing reaction in the arm makes probable an affection in the lateral part of the inferior semilunar lobe, etc.

However, I do not believe that one is justified in drawing such precise conclusions from the past-pointing test. There are too many extravestibular factors that influence the outcome of the reaction, thus diminishing its clinical significance. There is, furthermore, no anatomic proof that such pointing centers in the cerebellum really exist. It can therefore be said that in cases in which the results of the past-pointing test accord with the other otologic and neurologic symptoms, it can be helpfully used; if, however, there is a divergence between the past-pointing reaction and the other symptoms, care must be taken in using the test for diagnostic purposes.

The same holds true for the deviation reaction (Guettich) (p. 154) and the arm tone reaction (Wodak) (p. 155).

### CLINICAL SIGNIFICANCE OF COMPARISON OF TESTS

Comparative studies of the various reactions may render some additional diagnostic help. We shall here discuss (*a*) the differences in response to the various stimuli, (*b*) the "vestibular disharmony."

*a*) *The differences in response* to the various stimuli comprise the following results:

*Loss* of *turning* reaction, with *normal caloric* reaction. This disproportion is significant of inner-ear lues.

*Loss* of *caloric* reaction, with *normal turning* reaction. This form is found in cases of inner-ear lues as well as in certain cases of serous labyrinthitis, trauma of the skull, etc.

*Loss* of *caloric* and *turning* reaction, with *normal galvanic* reaction. This is indicative of a disease of the retrolabyrinthine part of the vestibular apparatus.

*Loss* of *galvanic* reaction, with *normal caloric* reaction. This is rather rare, but has occasionally been observed in cases with severe lesions of the brain centers.

*Reactive dissociation.* This term is applied to cases in which one labyrinth reacts much more easily to turning stimuli, while the other responds much better to caloric stimuli. This disproportion points to a central lesion.

*b*) *Vestibular disharmony* occurs when, for instance, the primary reaction (nystagmus) is absent, while the secondary reaction (vertigo, falling, past-pointing, etc.) is present or atypical, or vice versa. Here are some examples:

*Absence of primary reaction* means that on stimulation of the labyrinth no nystagmus will occur, while the other reactions seem normal. It indicates a central lesion somewhere between the vestibular nuclei and the eye muscle nuclei.

*Absence of the past-pointing reaction* is significant of retrolabyrinthine disease (Guettich). It is also often found in cases of lues and is considered to be the first symptom of a neurolabyrinthitis.

*Absence of the falling reaction* indicates a lesion in the vestibulo-cerebello-cerebral tract (Jones).

*Atypical falling reaction* is found in cases of inner-ear lues and brain tumors.

*Absence of the girdle reflex* has been described by Grant and Fisher in cases of lesion of the cerebellum.

# V

# Primary Diseases of the Labyrinthine Capsule: Otosclerosis

By *Louis E. Wolfson*

## 1. Historical Viewpoints

Otosclerosis, the enigma of otology, after many years of world-wide research work, still presents the problem of unknown etiology. The great diversity of opinion is shown by the confusion in nomenclature seen in such terms as otosclerosis, osteospongiosis progressiva, ankylosis of the stapes, ostitis metaplastica chronica, ostitis vasculosa, circumscribed ostitis fibrosa, capsulitis labyrinthica, osteofibromatosis, etc. Even though we know that the term otosclerosis is wrong, since sclerosis does not exist at all, we still use the old name applied by Politzer fifty years ago.

The number of theories dealing with otosclerosis is legion. Not only do the various otologists in all countries have their own ideas, but even the same author may change his viewpoint. Thus, O. Mayer, considered one of the best research authorities on otosclerosis, has changed his theory four times.

Politzer was the first to recognize otosclerosis as a primary disease of the labyrinthine capsule. He described the otosclerotic foci as proliferating bone growths occurring in certain areas of the capsule, i.e., "sites of predilection." As long as the foci lie within the bony labyrinthine capsule, no clinical symptoms can be observed in the patients. When, however, the endostal or periostal layers of the capsule are overstepped, partial or complete ankylosis of the stapes joint will occur. This in turn leads to a lesion of the sound-conducting apparatus associated with a normal drum and a normally functioning eustachian tube. Here the genius of the pioneer of otology becomes evident, in that he made such precise and plain statements fifty years ago.

Later on confusion began to arise, when some authors described cases in which otosclerotic patches were found not only in the sites of predilection but also in other places, such as the round window (Fig. 30). Other observers found the labyrinthine capsule completely normal in Politzer's site of predilection, but were able to demonstrate otosclerotic foci in the depth of the cochlear capsule, or in the fundus of the internal auditory meatus (Fig. 31). While some otologists based their diagnoses upon the clinical signs and symptoms as outlined by Politzer (typical history, otoscopic

findings, and functional test), other authors demanded the microscopic proof in postmortem cases. Which point of view is justified? It all depends on the problem presented. That is, if one is dealing with a patient whose impaired hearing must be diagnosed in order to be treated, one must follow the suggestion of Politzer; on the other hand, if one intends to make

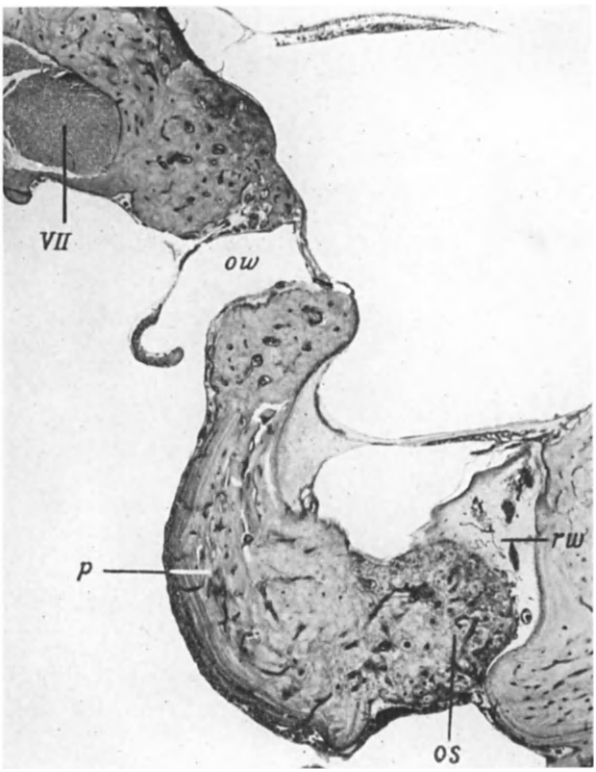


FIG. 30. PHOTOMICROGRAPH OF CASE OF OTOSCLEROSIS: VERTICAL SECTION THROUGH MIDDLE AND INNER EAR, SHOWING LARGE OTOSCLEROTIC TUMOR (*os*) ENTIRELY BLOCKING LUMEN OF ROUND WINDOW

*p* = promontory; *VII* = nervus facialis; *ow* = oval window; *rw* = round window.

scientific contribution to the problem of otosclerosis, one should base one's diagnosis upon the histologic examination of the petrous bones.

## 2. PATHOLOGIC ANATOMY

Otosclerosis represents a disease of the bony labyrinthine capsule. It is therefore advisable to discuss first the histologic structure of normal bone and the osteogenesis of the petrous bone. This is the only way of understanding why otosclerosis occurs only in the bone of the ear organ

and has never been observed in other bones of the skeleton, either in the long medullated bones or in the short membranous bones. It also explains the failure to produce otosclerosis experimentally.

The bony labyrinthine capsule comprises three layers: (*a*) the innermost layer, or endostal capsule, consisting of a few lamellae of membranous bone; (*b*) the external layer, or periostal capsule, consisting, in the newborn, of reticulate fibrillar bone that later on becomes compact lamellar bone, the lamellae being arranged parallel to the surface; (*c*) the medial layer, or enchondral capsule, which is very thick and consists of compact lamellar

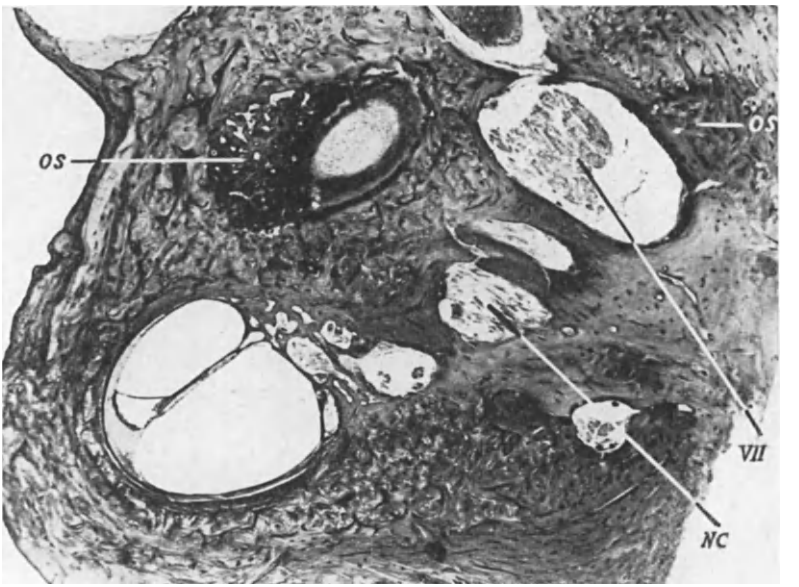


FIG. 31. PHOTOMICROGRAPH OF CASE OF OTOSCLEROSIS: VERTICAL SECTION THROUGH COCHLEA AND INTERNAL AUDITORY MEATUS, SHOWING OTOSCLEROTIC FOCI (*os*) IN COCHLEAR CAPSULA

*NC* = nervus cochlearis; *VII* = nervus facialis.

bone that incloses the remnants of the original cartilaginous capsule—these cartilaginous islands being called interglobular bodies (Manasse).

The *osteogenesis* of the petrous bone shows essential differences in comparison with the ossification of other bones of the skeleton. It was already known to Siebenmann that the bony labyrinthine capsule reaches its ultimate size and shape in the newborn. The physiologic growth of bone is the result of resorption and apposition processes. Whereas in the long medullated bones such processes take place through a period of two decades, the growth in the labyrinthine capsule soon comes to a standstill be-

cause of lack of resorption in the enchondral capsule, as O. Mayer showed. This explains the difference in the histologic structure. The labyrinthine capsule always contains remnants of cartilage and embryonic tissue. The coincidence of portions of fetal bones and fully developed bones forms the basis for the genesis of the otosclerotic foci. For further details of structure and osteogenesis of the otitic capsule, the reader is referred to the

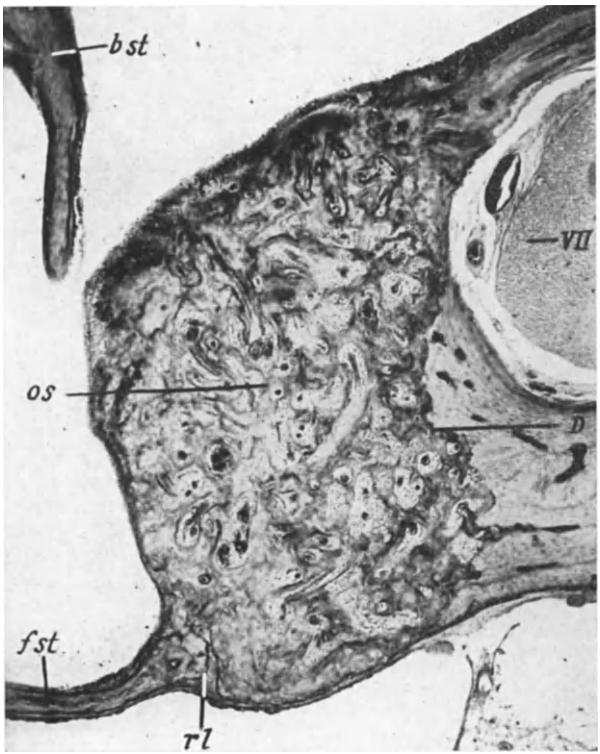


FIG. 32. PHOTOMICROGRAPH OF CASE OF OTOSCLEROSIS SHOWING LARGE OTOSCLEROTIC FOCUS (*os*) WITH LARGE MEDULLARY SPACES AND NEWLY FORMED BLOOD VESSELS

*f st* = footplate of stapes; *b st* = branch of stapes; *VII* = facial nerve; *rl* = ring ligament, partly ankylosed; *D* = demarcation line between otosclerotic focus and normal bone.

studies of Wittmaack, Nager, Kosakobe, T. H. Bast, Anson and Wilson, Guggenheim, Guild, Anson and Martin, etc.

Manasse described an embryonic focus in front of the oval window that he considered as a local malformation; it forms the basis for the development of otosclerosis. O. Mayer, corroborating the existence of such an island of fetal cartilage, differs, however, in his interpretation of it. His

studies led him to the belief that this focus represents a part of a synchondrosis that extends from the oval window in a forward and upward direction to the cochlear capsule. In contrast to Manasse, he cannot see any pathology in this cartilage rest. T. H. Bast made large-scale examinations of residual cartilages and defective ossification and their relation to otosclerosis. He observed that there are six such regions of the petrous bone: (1) fissula ante fenestrum; (2) fissula post fenestrum; (3) the infracochlear region; (4) the base of the styloid bone; (5) the petrosquamous suture; (6) the semicircular canals.

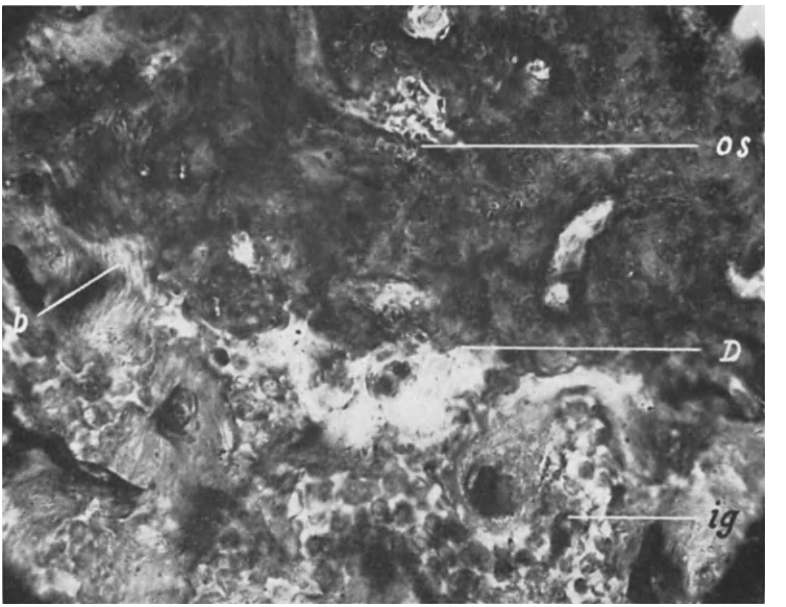


FIG. 33. PHOTOMICROGRAPH OF CASE OF OTOSCLEROSIS: SECTION THROUGH COCHLEAR CAPSULE UNDER HIGH MAGNIFICATION

*D* = demarcation line between otosclerotic focus (*os*) and normal bone (*b*) showing large islands of cartilage (*ig*).

In the light of our discussion of the normal histology and the osteogenesis of the labyrinthine capsule, the pathologic anatomy of otosclerosis becomes more understandable. Summarizing, the following can be stated:

1. Otosclerosis represents a disease of the bony otitic capsule occurring in circumscribed patches.

2. There is usually a sharp line of demarcation between the normal and the diseased bone (FIG. 32).

3. There is a marked difference in the tingibility of the bone with hematoxylin-eosin.

4. Changes of the structure of the otosclerotic bone occur. The diseased part shows the structure of a nonmature reticulated bone. Whereas the cells of the normal bone have a certain size and shape, and a certain arrangement at regular intervals, with their longitudinal axes parallel to the lamellae, no such regularity can be found in the diseased bone. There the cells are coarse, irregular in shape, usually increased in number, and irregular in arrangement.

5. Interglobular bodies are absent in the diseased area. FIGURE 33 illustrates an interglobular body that is sharply cut off at the line of demarcation.

6. Very large medullary spaces are seen in the otosclerotic focus. The medullary substance may show in certain places the character of lymphoid tissue; in other places it may be poor in cells but rich in fibers.

7. Engorged, newly formed blood vessels can be observed in the medullary spaces in the diseased bone.

8. Resorption of bone takes place by action of osteoclasts in the Howship lacunae; occasionally perforating Volkmann canals can be found.

9. There are no changes of the bone surrounding the otosclerotic focus.

## 3. ETIOLOGY

A study of the literature concerned with the etiology of otosclerosis reveals the amazing fact that only a few diseases exist that are not brought into relation with otosclerosis in the effort to explain its genesis. It is therefore impossible to deal with all the numberless hypotheses. In order to discuss the most important theories, an attempt is made here to classify them into certain groups.

### THEORIES OF INFLAMMATION

According to Manasse, otosclerosis originates in the blood vessels of the compact bone, with formation of granulation tissue and with a new formation of bone. He does not believe in resorption of the old bone by action of osteoclasts, but assumes rather a substitution of the old bone by a simple displacement, due to the ingrowing new bone. He therefore calls the process ostitis chronica metaplastica. He furthermore observed that the blood vessels growing from the focus into the surrounding capsule are surrounded by blue-staining cementum, which he calls "blue mantles." According to Brunner, these blue mantles consist of a precollagenous mass characteristic for beginning otosclerosis. O. Mayer cannot see any pathology in the presence of blue mantles. Gunther thinks that the blue-mantled vessels draw calcium from the normal capsule and deliver it to the growing focus (see below).

Alexander gives to otosclerosis the name ostitis vasculosa. The large

engorged newly formed blood vessels would justify the term vasculosa, but the absence of any inflammatory changes does not accord with the term ostitis.

### THEORIES OF DISTURBANCES OF CIRCULATION

Wittmaack considers otosclerosis as the result of localized venous stasis in the petrous bones. There is often a tendency to develop collateral circulation in order to overcome the disturbance. The local stasis leads to a loss of calcium in the bone, called halisteresis, which in turn causes later a resorption of the bone in the surroundings of the dilated vessels. Wittmaack tried to prove his theory by experimental research work on chickens. He produced local stasis, disturbing the circulation in the sinuses of the semicircular canals. He was able to produce rebuilding processes in the surrounding bone that, according to his statement, showed a striking resemblance to the changes in otosclerosis. O. Mayer categorically rejects the results of Wittmaack, which he says are absolutely untrue. He also opposes the conclusion Wittmaack draws from his experiments. He does not believe in halisteretic resorption of the bone. On the contrary, he claims that this bone is newly formed bone (osteoid).

It is interesting to note that Mayer in his first theory of otosclerosis also believed in disturbances of circulation. He made the statement that the otosclerotic foci usually occur in areas that correspond to the distribution of the arteriae nutriciae of the petrous bone.

Sparer considers otosclerosis as a local bone disease due to local alterations in circulation as a result of vasomotor imbalance of the nerves controlling the blood vessels. These nerves in the tympanic plexus are connected with the sphenopalatine ganglia. Gray believes that otosclerosis is the result of a defective vasomotor response in the small vessels of the ear. Defect of the vasomotor mechanism leads to disturbance of the nutrition of certain areas in the petrous bone.

### THEORY OF TUMOR

According to O. Mayer the otosclerotic patches represent local malformations that he classified in the group of hamartoma. The growing tendency is expansive, such as benign tumors usually show. The pressure of the growing tumor causes resorption of the bone. A similar point of view is maintained by Bruehl, Lange, J. Fischer, etc. A glance at FIGURES 30 and 31 supports the impression of a tumor. The otosclerotic new growth appears inlaid in the labyrinthine capsule.

### THEORY OF CONGENITAL ORIGIN

Alexander stated first that the otosclerotic foci already exist in early childhood, even in the fetal period of life. The clinical manifestations occur in the period of puberty or sometimes still later. The same viewpoint is taken by Bast, Guild, and Guggenheim. Hammerschlag, who considers otosclerosis as a heredodegenerative lesion, claims that deterioration of the germinal substance is responsible.

### THEORY OF CONSTITUTION

Bauer and Stein, who undertook large-scale clinical examinations, found a great number of degenerative signs in patients with otosclerosis. J. Fischer describes in otosclerosis many morphologic anomalies in the inner ear, which he considers as the anatomic basis of an inferiority of the ear organ in cases of otosclerosis. He believes in a disposition to otosclerosis when there are disturbances of the enchondral ossification and degenerative changes in the inner ear.

### THEORY OF TRAUMA

Bruehl explains ankylosis of the stapes as a result of mechanical irritations. The steady vibrations of the stapes and the tension of the annular ligament in its anterior part represent a continual stimulation. This in turn leads to rebuilding processes in the bone in the region of the oval window. As an analogy, he refers to the ankylosis seen in the vertebral column, which results from rebuilding processes due to continual mechanical irritation.

The latest theory of Mayer is based upon the assumption of a traumatic origin of otosclerosis. He considers the bony changes as callus formations resulting from healed spontaneous fractures in the otitic capsule. He explains the way he arrived at such conclusions. Studying a great number of petrous bones, he discovered many small fissures that often occurred in the same sites in the various specimens. The fissures contained loose fibrous tissue resembling the fibrous osteoid found in healed skull fractures. Similar fissures have been described by Alexander in cases of congenital deafness and by J. Fischer in otosclerosis. Mayer draws the following conclusions from his systematic examinations. The histologic structure of the fissures makes it obvious that they represent healed fractures filled with callus tissue. Because of the typical site of occurrence, they cannot be the result of an external trauma, but must rather be considered as spontaneous fractures due to pressure exerted in a certain direction upon the labyrinthine capsule. These bony formations tend to increase the firmness of the labyrinthine capsule. Since the traumatic theory cannot ex-

plain all the problems involved, Mayer adds to his mechanical hypothesis the factor of constitution, i.e., the disposition to hyperplastic bone formation in cases of otosclerosis.

### THEORY OF SYSTEMIC DISEASES OF THE SKELETON

The efforts to explain the etiology of otosclerosis led the otologists to the belief that there must be some relation to diseases of the skeleton, such

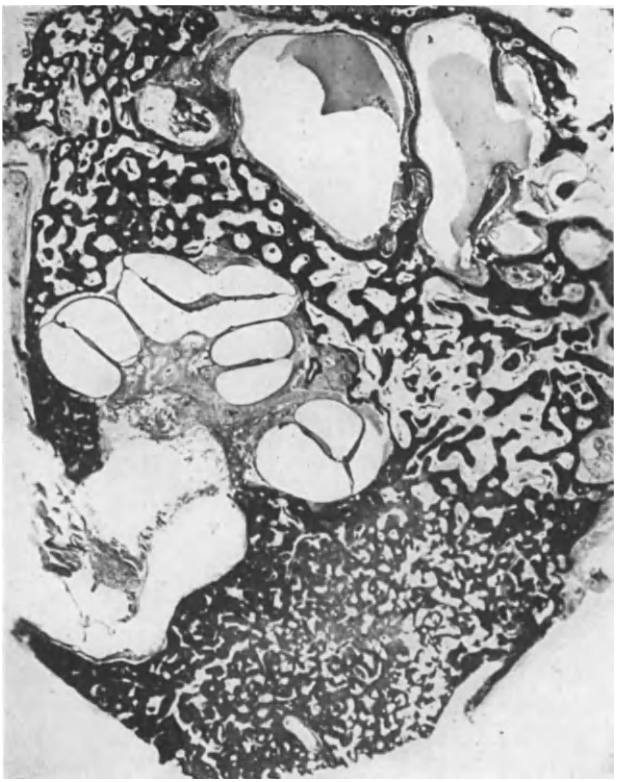

FIG. 34. PHOTOMICROGRAPH OF CASE OF PAGET'S DISEASE

Axial section through cochlea, showing spongious bone of entire cochlear capsule with large medullary spaces in between.

as ostitis fibrosa (Recklinghausen), ostitis deformans (Paget), osteogenesis imperfecta, osteopsathyrosis, etc. Mayer, who considers otosclerosis as a circumscribed form of ostitis fibrosa, suggests the term osteofibromatosis instead of otosclerosis. The changes in the fibrous medullary substance, with the rebuilding processes in the bone, made him think of a connection between the ostitis fibrosa and the otosclerotic foci. FIGURE 34 presents

a photomicrograph of a case of ostitis deformans (Paget) published by J. Fischer. A glance at the picture shows the difference between a circumscribed otosclerotic focus and the widespread changes in Paget's disease, where the entire inner-ear capsule is involved in rebuilding processes. FIGURE 66 reproduces a photomicrograph of a case of osteogenesis imperfecta.

The first case in which a histologic examination of the ear organ was made was published in 1920 by J. Fischer. He found disturbance in the enchondral and in the periostal ossification of the otitic capsule, further traumatic changes in the form of spontaneous fractures with intense callus formation, and finally a number of defective changes.

Osteopsathyrosis associated with blue sclerosis and otosclerosis has been described by many authors (Bigler, Ruttin, van der Hoove, De Kleyn, etc.).

### THEORY OF DISTURBED ENDOCRINE GLANDS

The well known fact that the clinical symptoms of otosclerosis depend upon certain conditions, such as puberty, pregnancy, lactation, menopause, makes it understandable that otologists looked for disturbances in the function of the endocrine glands. Brunner considers otosclerosis as a dystrophic disease due to dysfunction of the glands. Frey believes in a hypofunction of the parathyroid with latent tetany. Leicher found in 75 per cent of otosclerosis cases a reduction of the calcium content of the blood serum, while the rest of the cases showed a marked lability in the calcium metabolism. Other authors describe in cases of otosclerosis a positive pituitary reaction of Abderhalden. Mayer could not find any pathology in investigating the pituitary gland.

There have further been claims that otosclerosis is somewhat related to osteomalacia and rickets. Erdheim was able to produce changes in the bone similar to rickets by extirpation of the parathyroids in rats. Klose obtained the same results after destroying the thymus. J. Fischer, in an article entitled "Studies of Pathological Anatomy of the Petrous Bone," reports on his experimental and pathologic investigations. He made extirpations of the various glands (thyroid, parathyroid, ovary, testicle) in a great number of rats and examined all the bones. He could not find any changes resembling rickets, osteomalacia, or otosclerosis. He further examined the petrous bones of patients with osteomalacia and with rickets but did not reveal any otosclerotic foci.

Bernstein and Gillis think that otosclerosis may have some relation to the sex hormones, because of the dependency upon certain age factors and because of the predominance of incidence in females.

### THEORY OF VITAMIN DEFICIENCY

The importance of vitamin D for the bone structure of the labyrinthine capsule is stressed by many otologists (J. H. Jones, L. Gunther, Guggenheim, Baer, etc.). This vitamin is essential for calcium absorption and utilization. Guggenheim states that the capsular bone around the otosclerotic foci degenerates, losing its calcium and phosphorus and finally becoming resorbed. The enormous demand for calcium and vitamins during adolescence is often not adequately supplied, so that some withdrawal of calcium from the capsule occurs. The author comes to the conclusion that resorption around the focus plus endocrine stimulation of the reticular bone is responsible for the spread of the dystrophic area.

## 4. Clinical Diagnosis

The clinical symptoms and signs largely depend upon the size and place of the otosclerotic foci. As long as the bone proliferation has not reached the surface of the labyrinthine capsule, no clinical symptom will occur. The same holds true for small otosclerotic patches somewhere in the depth of the cochlear capsule or in the vicinity of the internal auditory meatus. Such foci are revealed only in the course of a microscopic examination post mortem. Clinical importance attaches only to the typical otosclerosis cases that usually do not present much diagnostic difficulty.

The clinical diagnosis of a typical otosclerosis is based upon, first, case history; second, otorhinoscopic examination; and, third, functional test.

In taking the history, attention should be paid to five points: (1) positive family history; (2) dependence upon certain age factors, such as puberty, pregnancy, lactation, menopause; (3) tinnitus, which often is described as low tones, or as noise rhythmic with the pulse—since these subjective noises are usually very intense and continuous, the patients are much more annoyed than in other catarrhal diseases of the middle ear; (4) impairment of hearing, gradually increasing; (5) apparent ability to hear better in a noisy place (paracusis Willisii).

On otorhinoscopic examination, the drum membrane either appears normal or shows a pinkish glow in the region of the promontory.

The functional test reveals a loss of the lower tone limit, while high sound pitches are heard almost normally. There is, further, an increased duration of hearing by bone conduction, a negative Rinne and a negative Gellé. After inflation of air by Politzer's method, no improvement of hearing can be found. This is in contrast to the condition in other catarrhal diseases of the middle ear or in chronic adhesive processes.

All these typical signs and symptoms may lose more and more of their characteristics when the otosclerotic disease steadily advances. Owing

to secondary degeneration of the nerve ending places, signs of a lesion of the sound-perceptive apparatus soon will interfere. The prolonged bone conduction is more and more reduced and may finally be replaced by a shortened bone conduction. Hearing of high tones also becomes impaired, and this may result finally in a complete loss of the higher tone limits. The Gellé test cannot be performed any more because of the poor hearing of the patient. If the degenerative processes also involve the labyrinth, vertigo, nausea, and disturbances of equilibrium may be found. Besides these otologic symptoms, otosclerotic patients often show psychic changes; they are shy, suspicious, worrying, sensitive, and usually talk with a very low voice.

### 5. Treatment

The basis for any rational treatment of a disease lies in the proper knowledge of its etiology. The complete lack of such knowledge in otosclerosis explains the failure of all the therapeutic measures applied hitherto. It would fill volumes to discuss all the numberless methods of treatment suggested by the various otologists in all the countries. It can safely be stated that almost all the treatments used in general medicine have been applied to cases of otosclerosis. The methods can be classified into two main groups, conservative and surgical.

#### CONSERVATIVE METHODS

A study of the literature shows that the treatment of otosclerosis was always in accordance with the theory of its origin in fashion at the given time.

In the early times, otosclerosis was treated with politzerization, with use of catheters or bougies, with pneumomassage, and with injection of pilocarpine (Politzer). For the tinnitus the patients were given sedatives.

When the theory of disturbances of circulation was in force, drugs were administered to control the blood circulation. The details will not be discussed here. Sparer advocated injection of alcohol into the sphenopalatine ganglion to cope with the vasomotor imbalance of the nerves controlling the blood vessels.

When the theory of diseases of the bone skeleton was in fashion, the patients were given phosphorus, cod liver oil, calcium phytin, nucleogen, protylin tablets, etc.

When the theory of disturbances of endocrine glands was in vogue, the treatment consisted of administration of thyroid extract, thyroidectin, thyroxin, pituitary extract, parathyroid extract, fluorine, estrogens, etc. A. Lewy made experimental studies of the influence of fluorine on the bony

labyrinth and advocated its application in otosclerosis. Gray and Goldstein recommended intratympanic injections of thyroxin at weekly intervals.

Frey and Krieser considered otosclerosis as a result of dysfunction of the parathyroid. They therefore advocated X-ray treatment of the parathyroid in small doses, combined with X-ray treatment of the petrous bone in larger doses. For technical details the reader is referred to the original article. The authors stated that the X rays check the pathologic bone growth in the labyrinthine capsule.

Mortimer, Wright, Thomson, and Collip used estrogens which they insufflated daily, in a dose of 1 cc. of oil containing 1,000 international units of an estrogen, for periods of three to six months. Bernstein and Gillis state that administration of sex hormones causes dilatation of the peripheral vessels and increased circulation of the blood, which might cause absorption of the embryonic bone in otosclerosis. They had particularly good results when using the heterogenic hormone. G. E. Shambaugh, however, could not find any improvement after use of the estrogenic treatment.

Guggenheim, who makes halisteretic processes responsible for the spread of otosclerotic foci, advocates the following treatment to produce a reversal of halisteresis:

1. Analysis of diet;
2. Correction of diet to normal requirements for age, type of work, etc.;
3. Vitamin C, 1,000 to 3,000 international units daily;
4. Calcium, 3 to 6 Gm., and phosphorus, 6 to 12 Gm. daily;
5. Vitamin D, 3,000 to 6,000 U.S.P. units daily;
6. Vitamin B, 1,000 to 2,000 Sherman units (in case of tinnitus).

### SURGICAL METHODS

Of the many treatments advocated for otosclerosis, the most intriguing, and that appearing to offer some solution to the problem, is surgery. The various surgical treatments have for their main purpose the establishment in another part of the labyrinth of a substitute window to take the place of the oval window with its fixation of the footplate of the stapes.

Kessel in 1876 was the first to report an operation for otosclerosis. He removed the footplate of the stapes. He reported that the operation was a failure. Passow in 1897 made a fistula on the promontory and got improvement in hearing of a very short duration. Bárány in 1910 was the first to suggest a fistula in the semicircular canal. His operation produced improvement for a short time. In 1914 Jenkins made a fistula in the horizontal canal and covered the fistula with a Thiersch graft and then with a flap from the external auditory canal. These improvements were also of

only short duration. Holmgren in 1917 and until 1935 made a large number of these fistulas of the labyrinth, using various parts of the labyrinth and various substances to cover the fistula. Most of these cases eventually had a closure of the fistula by bone formation.

Sourdille in 1924 devised his so-called tympanolabyrinthopexy. His procedure was designed to create a thin epithelialized membrane of scar tissue to provide a covering for the fistula in the semicircular canals. The operation consisted of three or more stages. In the first stage the skin and periosteum of the posterosuperior wall of the auditory canal were removed and the denuded bony area was allowed to heal with a thin epithelial membrane that was later used to cover the fistula. The second stage, four or five months later, consisted of a radical mastoid operation; the head of the malleus was also resected. The membranous flap was placed in such a way as to seal off the tympanic cavity and cover the site of the fistula of the horizontal canal. The third stage followed four to five months later. The portion of the flap over the horizontal canal was elevated and a fistula made in this area, which was immediately covered with this flap. If the fistula became filled with bone, this operation was repeated at intervals of three to four months, until a permanent opening was formed.

Sourdille reported improvement of long standing in many of his cases, although audiometric tests were lacking and very little information was given as to X-ray findings. When Sourdille's optimistic reports were published, Holmgren decided to repeat his operations, and used tissue that is non-bone-forming, as Thiersch graft or fat, and various prostheses such as rubber, stent, and paraffin. Holmgren did much experimental work on monkeys, in whom the labyrinthine capsule is similar to that of man, and tried pressure, erosion, electrolytic action of metals, irradiation, radium, peritoneum, fascia, muscle, and platinum wire. Nager examined the auditory capsule in Holmgren's cases histologically and found bony formation in all of them, but noted that the specimens with squamous cell epithelial tissue produced less bone and that radium reduced bone regeneration. When I visited Holmgren in Stockholm in 1937, he had had almost 200 cases in human subjects, with a small percentage of good results—that is, results lasting more than six months. Several of his cases had retained their hearing for six years.

In 1926 Lempert devised a one-stage operation with some similarity to Sourdille's method. It consisted of (1) creation of a troughlike fenestra in the bony capsule of the external semicircular canal, with the aid of a polishing and burnishing burr; (2) "incorporation of a newly created fenestra within the confines of a newly constructed, air-filled, and hermetically sealed tympanic cavity"; (3) "reconstruction of the osseous external canal

to permit access of sound waves to a newly made fenestra in the semicircular canal."

Lempert first reported 120 cases and claimed practical and physiologic hearing in 69 cases. Ten cases showed audiometric improvement not sufficient for practical hearing. Twenty-seven cases were unimproved. Fourteen cases showed further impairment in hearing. In 79 cases in which there was improvement, tinnitus disappeared. In 100 cases the fenestra remained open.

Lempert gave detailed operative findings and audiometric studies of these cases. He considered operation indicated when: (1) the hearing loss is bilateral and progressive; (2) the stapes is fixed within the fenestra ovalis but the membrane of the round window is normal; (3) hearing by air conduction in 512, 1024, and 2048 double vibrations has declined to a level that makes conversational hearing impossible, while bone conduction is normal or declines to a level of not less than 30 decibels; (4) the tympanic membrane is normal; (5) no infection of the ear exists; (6) the bony walls of the external ear are normal; (7) the eustachian tube is patent; (8) the patient is in normal health.

In November, 1941, Lempert reported 375 cases, and at this time reported also a revision of technic in the last 75 cases. He called this procedure the *fenestra novo-ovalis operation.* He attributed the fact that the fistula remained open in a very large percentage of his cases to (1) the fact that burrs delayed osteogenesis; (2) careful removal of the endostium and the shaving of the inner and osteal cambium layer beneath the inner margins of the bony walls of the fenestra; (3) epithelial lined surface, such as Shrapnell's membrane; (4) the small amount of postoperative trauma, due to endaural and antauricular approach. The reasons he gave to explain those cases in which osteogenesis recurred were: (1) nonremoval of the fibrous endostium membrane; (2) formation of fibrous connective tissue from the perilymphatic space to between the bony walls of the fenestra; (3) blood remaining in the perilymphatic space; (4) bone dust; (5) fistula formed posterior to the ampulla.

Lempert reasoned that the fenestra should be created in that part of the vestibular labyrinth that has the largest circumference of perilymphatic space and is in a position anterior to the ampulla of the semicircular canal and not posterior to it. Thus the fenestra will be nearest to Shrapnell's membrane. The dome of the vestibule was chosen as the most suitable and advantageous place for the creation of a new labyrinthine fenestra. This area occupies a position immediately adjacent and superior to the impeded fenestra ovalis and communicates directly with the scala vestibuli.

The operation is as follows. The temporal bone is exposed, and the

mastoid antrum is opened. The bony mastoid cell structure is partially exenterated and the perilabyrinthine cell structure is removed, exposing to view the mastoid aspect of the basal labyrinthine portion of the petrous pyramid and exposing the semicircular canal. The mastoid aspect of the posterior, superoposterior, and superior bony walls of the external auditory canal is skeletonized by removing all the bony cellular mastoid structure adjacent to these walls, thus exposing the entire epitympanic recess, the incudomalleolar joint, and the anterior malleolar ligament. These bony walls of the external auditory meatus are removed from the cutaneous membrane that lines these walls, including the corresponding portion of the sulcus tympanicus. Care must be taken not to disturb the outer dermal layer that binds and holds together the cutaneous lining of the external auditory canal and the tympanic membrane. The pyramidal eminence is then removed, exposing to view the chorda tympani branch of the facial nerve and the long process of the incus with the incudostapedial joint. The capsule of the incudomalleolar joint is divided. The body of the incus is separated from the head of the malleus, and after the long process of the incus is disconnected from the stapes, the incus is removed and discarded; in removing the incus, care must be taken not to injure the chorda tympani nerve.

This exposes to view the ampullated ends of the external and superior semicircular canals, the posterior half of the roof of the vestibule, the oval window with the stapes *in situ* within the tympanic wall of the vestibule, and the tympanic transverse portion of the facial canal lying external and superior to the oval window and separating it from the dome of the vestibule. Then the head and neck of the malleus are amputated up to the attachment of the anteromalleolar ligament. By means of a polishing burr, size 3, a fenestra measuring about 5 mm. in length and 2 mm. in width is created through the dome of the bony capsule of the vestibule down to the endostial membrane lining the lumen. The fenestration is begun anterior to the ampullated ends of the superior and the external semicircular canal and is carried forward above and external to both the tympanic and the epitympanic course of the facial nerve. With the aid of a Zeiss loop, the bony margins of the fenestral walls are shaved smoothly with dental spoon excavators until the fenestra assumes the desired shape and size. The inner cambium layer underneath the inner margins of the bony walls of the fenestra are also shaved. Finally, the bony walls are polished and burnished with a 24-carat gold burnishing burr. This completes the exposure of the perilymphatic membranous labyrinth.

With the aid of Zeiss magnifying spectacles, the endostium that forms the outer protective wall of the membranous perilymphatic labyrinth is irrigated carefully by means of a rubber-bulb syringe and washed with

warm physiologic solution of sodium chloride, in order to remove the particles of bone dust that may have settled along the walls of the bony fenestra and on the outer surface of the endostial membrane. The presented surface of the endostium is carefully shredded with a special needle and removed from the underlying perilymph by the aid of gentle irrigation with warm physiologic sodium chloride. This results in a fenestra in the membranous perilymphatic labyrinth, exposing to view the membranous endolymphatic labyrinth. The area exposed to view is the utricle. The fenestra novo-ovalis vestibule is now complete. The drum membrane is freed from the entire superior margin and the posterior half of the inferior margin of the sulcus tympanicus and thus converted into a movable membrane. The tympanomeatal membrane is so placed that the tympanic portion seals the entire tympanic air space, with Shrapnell's membrane covering the fenestra novo-ovalis.

Hughson approaches the surgical problem from a different point of view. In his experimental study, in collaboration with Crowe, on the function of the round window, during the time when the Weaver and Bray experimental work was begun, Hughson found that under the stress of extreme acoustic stimulation, or even under ordinary stimulation with the round window blocked, auditory response was greatly enhanced. In 38 cases, over a four-year period of observation, he operated to block the round window, and felt that he had obtained appreciable improvement in the hearing of these deafened patients.

Hughson makes an incision in the posterior half of the drum, and with a long cotton applicator gently rubs the membrane as near as possible to the area of the round window. A small amount of fascia is inserted in this area and a sterile piece of cotton is inserted into the ear. He found that hearing gradually improved after this operation, for from 15 to 35 decibels. He has very thoroughly studied the hearing both before and after operation, making as many as ten audiograms to get a proper idea of the hearing before and after. Eighty-five per cent of his cases showed a definite improvement.

*In summary, the goal of all the various surgical technics* described above is the formation of a permanent fistula for the establishment of a new path for sound waves, in place of the foramen ovale with its fixation of the stapes. Despite the marked improvement in surgical technic for making a new fistula, the essential problem in otosclerosis seems to have been overlooked. Otosclerosis is not a simple disease of the sound-conducting apparatus, but a primary disease of the bony labyrinth capsule; the progress of this disease and the reasons for this progress remain unknown.

The disease occurs in circumscribed patches of otosclerotic foci of bone in the various parts of the otic capsule, often involving the neurosensory organ. Even though the surgical problem of a permanent fistula may be

solved, the underlying disease is not cured, nor is the progress of the disease stopped. On the other hand, although surgical treatment does not cure otosclerosis, the fact that in a number of cases improvement up to approximately five years' duration has been shown, justifies the operation at the hands of a skilful surgeon.

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# VI

# Inflammatory Diseases of the Inner Ear

By *Joseph Fischer*

## LESIONS OF THE LABYRINTH

THE FOUNDATION for clinical work in diseases of the labyrinth was laid by Jansen fifty years ago. He was also the first to institute major surgical procedures for the treatment of suppurations of the labyrinth. In the ensuing years more and more work was done on this subject by various otologists such as Hinsberg, Gradenigo, Neumann, Ruttin, Fraser, Dench, Canfield, Richards, Lund, Uffenorde, Bourguet, Botey, Hautant, Eagleton, Friesner and Braun, Jones, etc.

Great importance must be given to the studies of the pathologic anatomy of the labyrinthine diseases made by Politzer, Alexander, Goerke, Manasse, Zange, Nager, etc. Further progress was finally due to the improved methods of labyrinthine testing devised by Bárány, Ruttin, Kobrak, etc.

In spite of all this clinical and research work, there is still great dissension not only with regard to diagnosis and surgical treatment but even with respect to nomenclature and classification. For example, distinction is made between acute and chronic labyrinthitis, circumscribed and diffuse, manifest and latent, primary and secondary forms, etc. There is another differentiation between serous, fibrinous, serofibrinous, seropurulent, and purulent labyrinthitis. A further differentiation is made as between exudative, plastic, and necrotic types. From the etiologic point of view there is a scarlet labyrinthitis, a tuberculosis or a cholesteatoma labyrinthitis, etc. With regard to the development of the infection, the disease is spoken of as a tympanogenic, a meningogenic, or a metastatic labyrinthitis. Another classification is made on the basis of the site of the disease. Jansen differentiates, according to the three parts of the inner ear (cochlea, semicircular canals, and otolith organ), three forms of labyrinthitis: otitis interna cochlearis, otitis interna labyrinthica, and otitis interna macularis.

Some other common classifications may be mentioned. Ruttin differentiates five groups of inflammatory diseases of the labyrinth, as follows: (1) chronic circumscribed labyrinthitis; (2) diffuse manifest serous labyrinthitis; (3) diffuse manifest purulent labyrinthitis; (4) diffuse serous induced labyrinthitis; (5) latent purulent labyrinthitis. Hinsberg separates the traumatic forms from the labyrinthitis following otitis media.

The latter can be subdivided into four groups: (1) labyrinthitis following a common otitis media; (2) acute labyrinthitis following a scarlet otitis media; (3) labyrinthitis following a protracted otitis media (mucosus infection); (4) labyrinthitis following a chronic otitis media. Lund differentiates four groups: (1) labyrinthitis chronica diffusa destructiva; (2) labyrinthitis circumscripta; (3) labyrinthitis serosa; (4) labyrinthitis acuta diffusa destructiva.

In 1926 I wrote an article, "Indications for Surgical Procedures in the Inner Ear," wherein I presented the standpoint that obtained in the clinic of Alexander in Vienna. I used a new classification outlined by my chief and myself. We classified the inflammatory diseases of the labyrinth into the following groups:

1. Otitis interna serosa;
2. Para-otitis interna purulenta;
3. Peri-otitis interna purulenta;
4. Otitis interna purulenta;
5. Otitis interna purulenta complicata.

For purposes of instruction, another group is here added:

6. Otitis interna traumatica.

It is obvious that this classification also has its deficiencies. All such efforts must be arbitrary, since nature does not always follow strict rules. There always will be cases that represent a transition from one classification to another, or where the patient has in one part of the inner ear certain changes characteristic for one type, while at the same time another part may show the pathology of another type. Finally there are cases with metastatic infection of the inner ear arising from remote foci. However, we found that for practical purposes our classification can be applied to the overwhelming majority of cases.

## 1. Otitis Interna Serosa

### ETIOLOGY

We must differentiate between a genuine otitis serosa (Alexander) and serous forms of otitis that are usually forerunners of purulent internal otitis. The latter does not represent a disease *sui generis*.

The genuine serous otitis may follow an acute otitis media as well as a chronic one. Occasionally it may also occur immediately after a radical mastoid operation (serous induced labyrinthitis). Trauma plays an important role in the development of the disease.

Common pathways for spread of the infection into the inner ear are the windows in the medial wall of the middle ear. It is not a rupture through

the windows, but a slow migration of toxins, for instance through the secondary tympanic membrane, that causes serous otitis in the inner ear.

### PATHOLOGIC ANATOMY

The inner-ear spaces are filled with a homogeneous fluid that contains almost no cells. The accumulation of fluid not only may extend over the entire peri- and endolymphatic spaces of the cochlea and the labyrinth

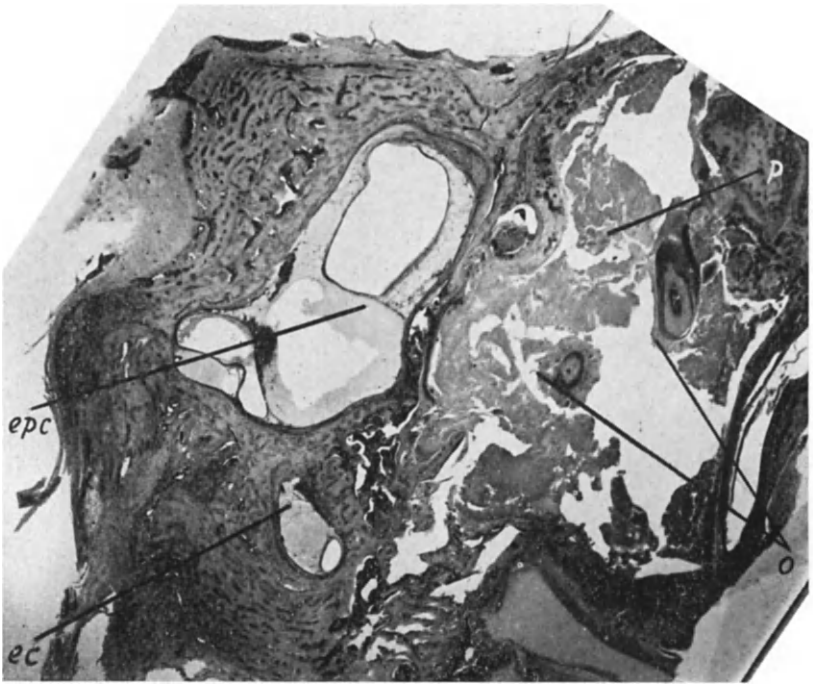


FIG. 35. PHOTOMICROGRAPH OF CASE OF OTITIS INTERNA SEROSA

Vertical section through middle and inner ear, showing middle ear filled with pus (*p*) imbedding ossicles (*o*), while only diffuse serous exudation appears in inner-ear spaces. Exudation is to be noted in perilymphatic cistern (*epc*) and in perilymphatic spaces of semicircular canals (*ec*), while endolymphatic spaces are intact.

(FIG. 35), but there may also be a serous infiltration of the tissue of the membranous inner ear. Such an infiltration may occur in the spiral ligament, in the stria vascularis, or in the nerve ending places.

In a differential-diagnostic respect, there are two diseases that may be mistaken for genuine serous otitis. One is hydrops labyrinthi (Manasse); the other is the initial stage of purulent internal otitis. The former is characterized by venous stasis, which is not found in genuine serous otitis.

The latter is characterized by the presence of numerous mono- and polynuclear leukocytes in the exudate, which also cannot be found in genuine serous otitis.

### CLINICAL COURSE

In the beginning of the disease there are symptoms and signs usually called "manifestations of irritation," while later on these are replaced by "manifestations of destruction."*

Among the manifestations of irritation are sudden onset of vertigo in the form of numerous short dizzy spells, nausea, vomiting, malaise, spontaneous nystagmus of second to third degree, usually toward the diseased side, disturbance of equilibrium, and occasionally slight rises in temperature. The climax of this stage is reached on the second or third day.

The manifestations of destruction include: rapid impairment of hearing until complete deafness results; loss of labyrinthine excitability; spontaneous nystagmus in the direction of the sound ear. The loss of function of cochlea and labyrinth is of only short duration, lasting usually from one to two days, followed by gradual improvement. Approximately a week after onset of the disease, hearing and labyrinthine excitability are restored.

### DIFFERENTIAL DIAGNOSIS

While the differentiation of genuine serous otitis from a pathologic point of view is relatively easy, the clinical separation from the initial stage of purulent otitis may be very difficult. Ruttin believes that the earlier a labyrinthitis occurs—following an acute otitis media—the greater is the likelihood of its being the genuine form and the less is the danger. On the other hand, the later the symptoms occur, the greater is the probability of a purulent form and the greater the danger. Another differentiation may sometimes be made on the basis of the general aspect of the patient. The general state of the patient is more severe in the purulent otitis than in the serous forms.

Postoperative labyrinthitis is discussed below (p. 220).

### TREATMENT

The treatment is conservative. Absolute rest in bed is required, with darkening of the room in order to keep away any irritation. The patient himself usually finds a position in bed that gives him the least vertigo. He lies mostly on the side of his sound ear. He should have a light diet and should be kept under sedatives (bromides or morphine hypodermic). Recent results of chemotherapy are very encouraging.

* Braun and Friesner apply the terms "stimulation disharmony" and "destruction disharmony."

## 2. Para-otitis Interna Purulenta

### ETIOLOGY

Para-otitis may occur in the course of either acute or chronic otitis media. The former is more often found in children or in cases where a retention in the antrum or mastoid exists. The latter is usually associated with cholesteatoma or tuberculosis. The bony changes may occur in any part of the medial wall of the middle ear, but the prominence of the horizontal canal is the most common site. The fact that this region is a favored site for the establishment of bone diseases can be explained by anatomic reasons: the bony wall of the horizontal canal lies freely exposed in the depth of the narrow antrum, where it is subject to increased pressure caused by the poor conditions of drainage.

### PATHOLOGIC ANATOMY

The bony labyrinthine capsule becomes diseased either by direct continuation of the inflammation in the middle ear or by way of the blood channels, particularly those in the haversian canals. The bony destruction is caused by the Volkmann "penetrating canals" and by lacunar resorption due to the action of osteoclasts. In this way the haversian canals become more and more enlarged. Figure 36 shows such large haversian resorption spaces in the bony labyrinthine capsule, between the horizontal and the superior vertical canal.

As long as the bony erosion is only superficial, there are usually no changes in the inner ear itself. The deeper the bony disease goes and the nearer it comes to the inner-ear spaces, the more changes may be expected. The membranous inner ear usually shows changes of a low degree—for instance, circumscribed exudation in the horizontal canal or in the perilymphatic cistern, or an increase of perilymphatic tissue, while the endolymphatic parts are mostly intact.

### CLINICAL COURSE

In the beginning of this disease there may not be any characteristic symptoms as regards the inner ear. In the further course, however—when the bony destruction continues—the manifestations of irritation can be observed, although in a much lesser degree than as described in the previous chapter.

Vertigo is present but not very marked. Spontaneous nystagmus is of the lowest degree, often toward both sides, occasionally toward the affected side. Sometimes there is no nystagmus at all, probably because at the time of examination the labyrinth is not in a stage of irritation. In such a

case the diagnosis can be made only when repeated examinations over a certain period of time have been performed.

The manifestations of irritation become suddenly more marked when in chronic cases an acute exacerbation of the middle-ear process occurs, or when in acute cases a retention of pus in the antrum or mastoid exists. The labyrinthine excitability may be either normal or pathologically in-


FIG. 36. PHOTOMICROGRAPH OF CASE OF OTITIS INTERNA PURLENTA

Section through medial wall of middle ear, between superior vertical canal (*sc*) and horizontal canal (*hc*). Suppuration in middle ear (*sm*) has led to circumscribed disease of bony wall. *iH* = infection in haversian canals; *d* = dura of middle fossa.

creased. A very valuable test for the diagnosis of para-otitis is the positive head-moving test (Brunner). The head of the patient is bent forward 90° and the eyes are shut. The examiner stands in front of the patient, holding the head with both hands. He suddenly jerks the head backward and has the patient open his eyes. If now a nystagmus occurs (usually rotatory, coarse, toward the diseased side), the test is positive.

#### TREATMENT

Elimination of the source of infection by surgical procedures in the middle ear is indicated. If para-otitis has occurred in the course of an acute otitis media, a simple mastoidectomy, with exposure of the diseased bony portion, should be performed. In chronic cases, however, a typical radical mastoidectomy with exposure of the diseased bony region in the horizontal canal is an absolute necessity. It must be borne in mind that curetting of the diseased bone, or removal of granulations in the region of the semicircular canals, must be carefully avoided. Such procedures are very dangerous and absolutely not necessary. A large exposure of the affected bony parts is sufficient to bring about complete healing.

Great attention must also be given to the after-treatment. Any accumulation of pus in this region must be avoided, and signs of a traumatic internal otitis (p. 220) must be watched for carefully.

### 3. Peri-otitis Interna Purulenta

#### ETIOLOGY

Peri-otitis is more often associated with chronic than with acute otitis media. Cholesteatoma and tuberculosis play the main roles in the etiology. As far as the pathways of infection and the site of the bone disease are concerned, the facts are the same as those presented in the preceding section in relation to para-otitis.

#### PATHOLOGIC ANATOMY

The ostitic changes in the labyrinthine capsule are not only found on the surface or in the lateral parts of the wall—as in cases of para-otitis—but involve the entire thickness of the bony capsule in a certain region. If such a process leads to a free communication between the middle and the inner ear, it is spoken of as a penetrating fistula (Fig. 37). However, we often observe that the bony wall, for instance that of the horizontal semicircular canal, is completely destroyed, but a thin endostal layer remains intact (Fig. 38). It appears strange that such a thin layer should offer more resistance than the hard thick bony wall.

The changes in the peri- and endolymphatic parts of the inner ear depend not as much upon the extent of the bony involvement as upon the length of time in which the bone becomes diseased. The more time elapses before the entire wall of the labyrinthine capsule is involved, the better are the chances for an establishment of organization and restoration processes in the inner ear. A comparison of Figure 37 with Figure 38 clearly illustrates these facts.

Figure 37 is a microphotograph of a case of chronic otitis media with a

very slowly growing cholesteatoma. The perilymphatic space of the horizontal canal is mostly filled with connective tissue (scar), partly with granulated tissue in the stage of beginning organization. The endo-

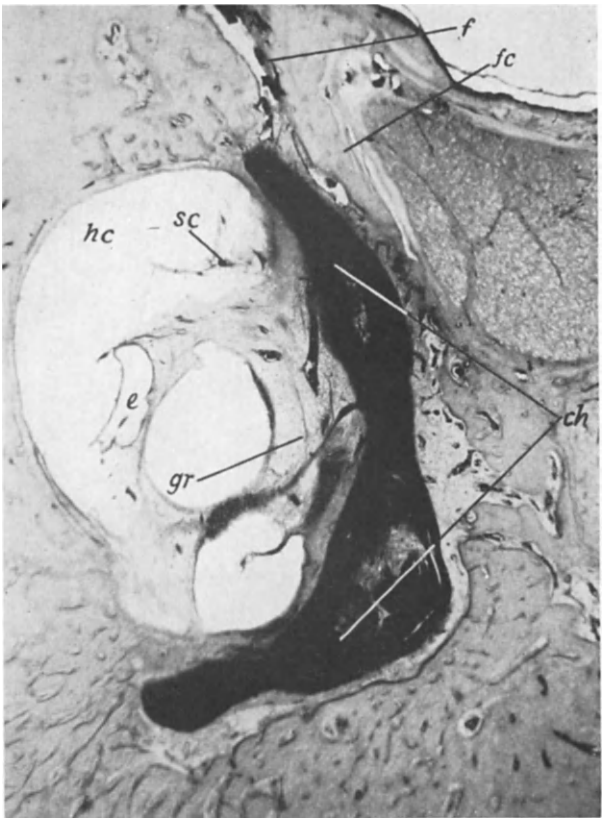


FIG. 37. PHOTOMICROGRAPH OF CASE OF PERI-OTITIS INTERNA PURLENTA IN CHRONIC CHOLESTEATOMA OTITIS MEDIA

Section through medial wall of middle ear, showing penetrating fistula (*f*) located between facial canal (*fc*) and horizontal canal (*hc*); cholesteatoma pearl (*ch*), slowly growing into depth, that has led to complete destruction of bony lateral wall of horizontal canal; perilymphatic space of latter mostly filled with connective tissue scar (*sc*), partly with granulation tissue (*gr*); endolymphatic space (*e*) empty but contracted from without by scar formations.

lymphatic space is normal but the lumen is narrowed by compression of the scar tissue.

FIGURE 38 shows a case of tuberculosis otitis media with a rapid course and with a complete destruction of the bony wall. The membranous horizontal canal shows the same necrotic masses as the bony canal. The

other parts of the labyrinth were more or less intact. The cholesteatoma otitis extended over a long period of time, led to a circumscribed, almost cured labyrinthitis, while the tuberculosis case with its progressive course resulted in complete destruction.

Although the prominence of the horizontal canal is the most common site of the disease, there are sometimes other parts of the bony labyrinthine capsule (promontorium, facial canal) involved. FIGURE 39 shows a case of chronic otitis media with destruction of the fallopian canal.

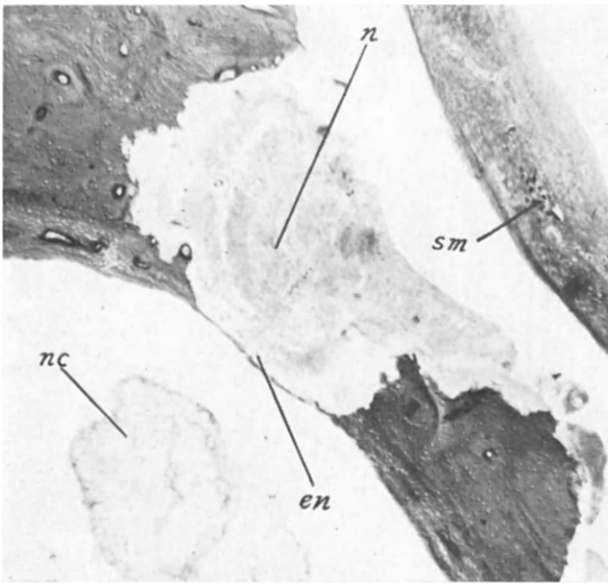


FIG. 38. PHOTOMICROGRAPH OF CASE OF PERI-OTITIS INTERNA PURULENTA IN TUBERCULOSIS OTITIS MEDIA

Section in region of prominence of horizontal semicircular canal: suppuration process in middle ear (*sm*) has led to complete destruction of bony labyrinthine capsule in this place, only a thin endostal layer (*en*) remaining intact. *n* = necrotic masses replacing bone; *nc* = necrosis in horizontal canal.

The destruction of bone takes place either by action of osteoclasts or somewhat differently in cases of tuberculosis and cholesteatoma. In tuberculosis cases the central caseation processes of the myriads of tubercles lead to numberless small sequestra that make the bone look "moth-eaten" (FIG. 38). In cholesteatoma cases the bone is destroyed by three different factors: first, by the active growing tendency of the cholesteatoma destroying any tissue that is in the way (like a neoplasm); second, by pressure atrophy of the bone; and, third, by the common chronic suppura-

tion processes that are always associated with the cholesteatoma. However, the bony defect reveals a smooth surface. FIGURE 37 clearly illustrates these facts. A cholesteatoma pearl, growing deep into the bony labyrinthine capsule between the facial and the horizontal canal, has led to a complete destruction of the lateral wall. Between the matrix of the pearl and the bone, a vascular granulation tissue has helped also to erode

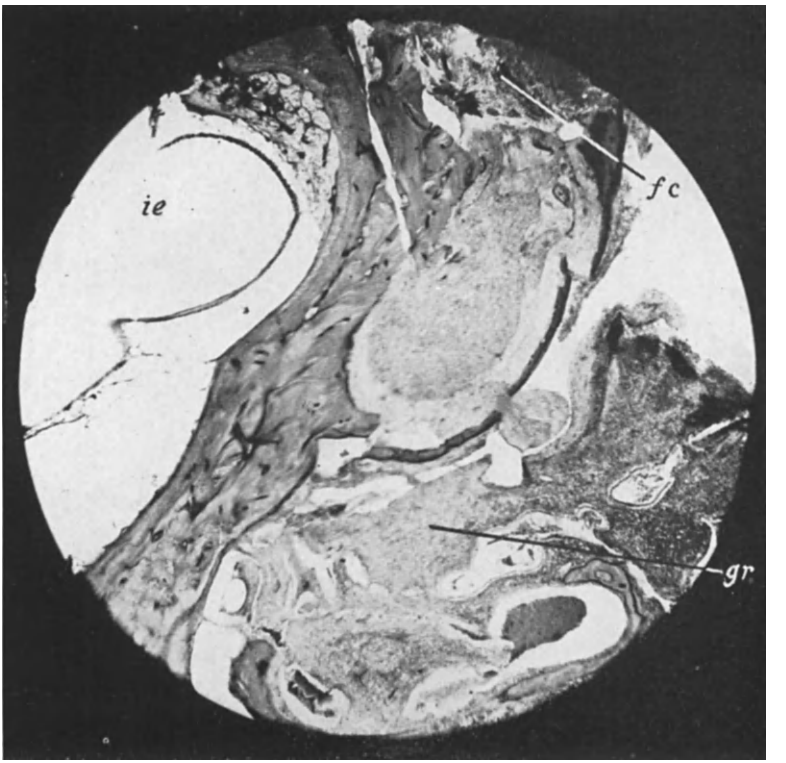


FIG. 39. PHOTOMICROGRAPH OF CASE OF PERI-OTITIS INTERNA PURULENTA IN CHRONIC OTITIS MEDIA

Vertical section, showing destruction of wall of bony facial canal (*fc*); tympanum cavity filled with pus and granulation tissue (*gr*); inner ear (*ie*) intact.

the bone. However, the entire defect of the bony canal wall is covered by the cholesteatoma pearl. This explains the clinical fact that on functional examination the fistula test was negative in this case.

### CLINICAL COURSE

The manifestations of irritation (vertigo, disturbance of equilibrium, spontaneous nystagmus toward one or both sides, etc.) occur in the form

of repeated attacks. The development of a penetrating fistula can usually be diagnosed on the basis of the case history. Such a patient complains of dizziness over a long period of time. But among all these attacks there occurs one that he will never forget. The patient knows the exact date; he can describe the character of the vertigo, the direction of the sham movements, the duration, etc. This attack marks the time at which the communication between middle and inner ear (penetrating fistula) became evident. Never before or after is such an attack experienced by the patient. On functional examination the labyrinthine excitability appears pathologically changed (hyper- or hypo-), the hearing is impaired, and the fistula test is positive. Occasionally the fistula reaction can be produced by pressure on the tragus (p. 140). Such a patient complains of dizziness and nausea in the morning when he washes his face and touches the ear. On the other hand, a negative fistula test does not necessarily mean absence of a fistula, since any mass (cholesteatoma, polyp, crusts, etc.) may block the lumen of the fistula (FIG. 37).

The further clinical course of the disease depends upon the underlying pathology. For clinical reasons two different types of peri-otitis must be denoted: one is the stationary, the other the progressive form.

*Stationary type.* The clinical signs and symptoms remain over a long period of time more or less unchanged, or even become less and less in intensity. The pathologic basis is represented by a circumscribed lesion in the perilymphatic part of the inner ear (occasionally in the endolymphatic part) with a tendency to scar formation (FIG. 37).

*Progressive type.* From the very beginning of the disease, there is a tendency to spreading and becoming diffuse. The manifestations of irritation very soon are replaced by the manifestations of destruction. The hearing acuity is more and more reduced, and instead of hyperirritability a hypofunction of the labyrinth will be found. The fistula reaction, previously positive, becomes more and more difficult to elicit and is finally completely negative. In this stage peri-otitis is often difficult to differentiate from otitis interna purulenta (cf. next section) and can be regarded as a transitional form between the two types. The pathologic picture also shows a remarkable resemblance to that found in otitis interna purulenta.

### TREATMENT

In view of the great difference in the clinical course of the two forms of peri-otitis, it seems logical that the treatment of the two types also should be quite different.

*Stationary form.* The treatment is the same as in para-otitis. Its chief aim is the elimination of the source of infection in the middle ear, with an

extensive exposure of the diseased parts of the labyrinthine capsule. However, it must be borne in mind that radical mastoidectomy should never be performed during an acute flare-up. It is very important to wait until all signs of the acute exacerbation have subsided.

Another point to be stressed concerns the technic of operation. Greatest care must be taken to avoid any concussion when working with chisel and hammer (a new set of chisels should be used). In working with sharp curets, scraping of diseased bone of the labyrinthine capsule or removal of granulations should be carefully avoided. In examining the fistula, no probe should be used, and any unnecessary manipulations should be avoided, since even rough sponging of these areas may be dangerous. Excessively tight packing with gauze should also be avoided.

The same care must be given to the after-treatment. Repeated changes of bandage to prevent retention are advisable. There should be no forced irrigations of the wound cavity. I cannot see any necessity for irrigating at all. In cases with a profuse discharge, we make the bandage in such a way that only the retro-auricular wound is covered, while the pinna is exposed. This enables either the nurse or the patient himself to change the strip of gauze in the ear canal as often as necessary.

Patients who have undergone mastoidectomy in cases of peri-otitis interna purulenta should be kept in the hospital much longer than patients with normal labyrinth. An increase of vertigo and spontaneous nystagmus, associated with a decrease of cochlear and labyrinthine function, may be significant of a traumatic internal otitis (serous induced labyrinthitis) or of an otitis interna purulenta (p. 221).

*Progressive form.* The treatment is the same as in otitis interna purulenta. Here we may only briefly stress these points: The first part of the treatment is conservative. There should be strict bed rest until all labyrinthine symptoms have subsided. It may take six to ten weeks to achieve this aim. The second part of the treatment is surgical (radical mastoidectomy). If one starts immediately with a mastoidectomy, one may easily be surprised by an intracranial complication.

### 4. Otitis Interna Purulenta

#### ETIOLOGY

Otitis interna purulenta follows either an acute or a chronic otitis media. In the former, the middle-ear process is usually severe, being caused by very virulent germs and being associated with retention of pus in the attic or the antrum. In the latter there is either an acute exacerbation of a cholesteatoma otitis or a tuberculosis otitis. Pathways of infection are the oval or the round window, the blood or lymph channels, or fistulas in the

labyrinthine capsule. However, the inner ear may also be infected from the opposite direction, namely, from the cranial fossae by way of the internal auditory meatus or the aqueducts. FIGURE 40 shows the course of meningitis descending along the eighth nerve into the inner ear.

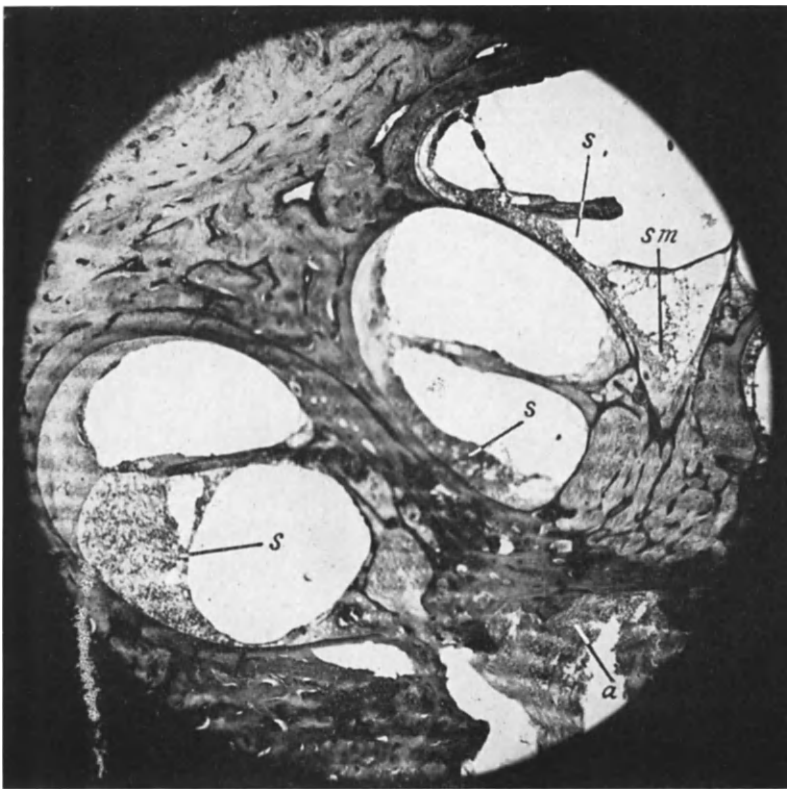


FIG. 40. PHOTOMICROGRAPH OF CASE OF OTITIS INTERNA PURULENTA IN MENINGITIS
Axial section through cochlea, showing abscess in fundus of internal auditory meatus (*a*) and suppuration (*s*) in scala tympani of turns of cochlea and in modiolus (*sm*).

## PATHOLOGIC ANATOMY

In *acute* cases, four stages of the labyrinthine infection can be distinguished:

1. *Stage of acute exudation.* This must be regarded as the initial stage or as a forerunner of the purulent inflammation. There is an accumulation of fluid, rich in cells and fibrin (exudate) in the inner-ear spaces, and also a hyperemia with extravasations into the vicinity. For differentiation of this condition from genuine serous otitis, see page 198.

2. *Stage of cell infiltration.* There is a widespread infiltration not only in the inner-ear spaces but also in the membranous inner ear itself.

3. *Stage of suppuration.* There is an accumulation of pus and granular tissue, leading to destruction not only of the membranous inner ear (Corti organ, nerve, ganglion cells) but also of the bony inner ear. FIGURE 41 shows complete destruction of the organ of Corti, the spiral nerve, and the spiral ganglion. The large-scale bony destruction may lead to fistular

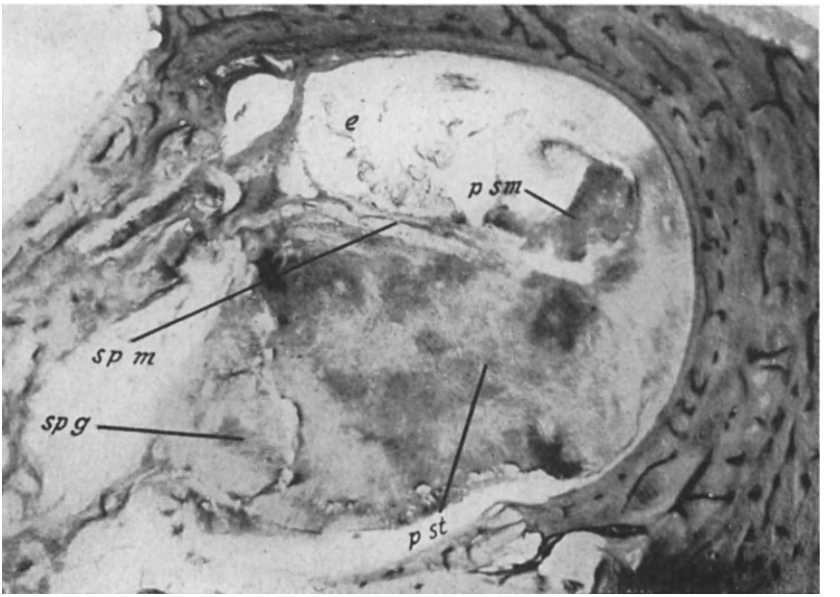


FIG. 41. PHOTOMICROGRAPH OF CASE OF OTITIS INTERNA PURULENTA IN ACUTE OTITIS MEDIA

Section through middle turn of cochlea, showing complete destruction of organ of Corti, of nerve fibers between lips of spiral membrane (*sp m*), and of spiral ganglion (*sp g*). *p st* = pus in scala tympani; *p sm* = pus in scala media; *e* = exudate in scala vestibuli.

rupture of the labyrinthine walls outward into the middle-ear spaces or inward into the cranial fossae.

4. *Stage of regression and healing.* In certain parts, organization processes (resorption of exudate, formation of connective tissue, etc.) take place. This leads to a confinement of the suppuration and destructive processes, which gradually are more and more replaced by scar formation. The final result is a complete healing in spite of the large-scale destruction. Later on the connective tissue scar may be transformed by action of osteoblasts into a bony scar. FIGURE 42 shows a stage in which connective

tissue and bone formation are seen in the cochlea, while FIGURE 43 shows a stage in which large exostoses in the vestibule and in the cochlea have entirely replaced the connective tissue.

However, in certain cases there is no tendency to heal at all. The stage of regression does not occur, and the destruction finally results in an intracranial complication (p. 217).

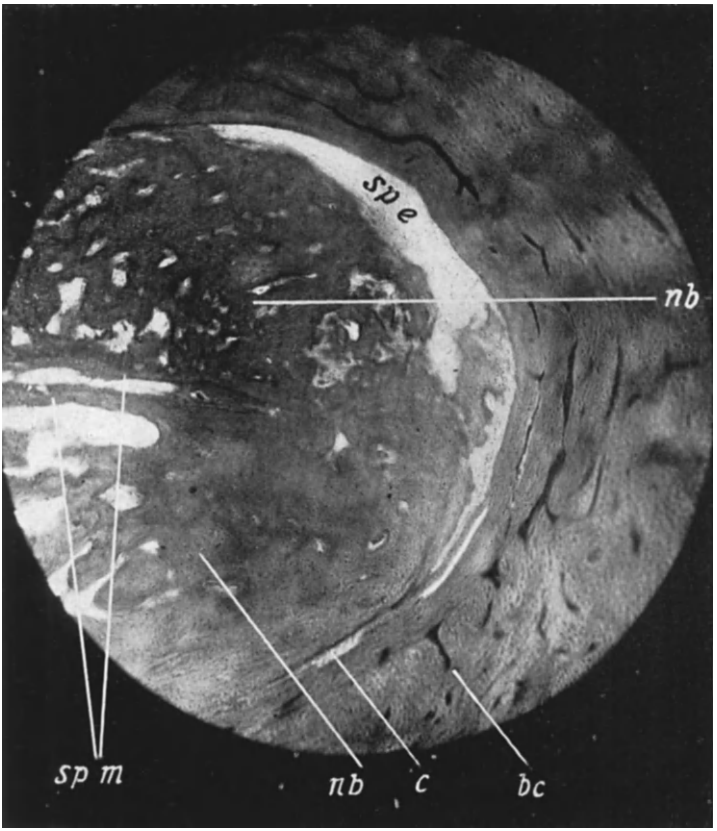


FIG. 42. PHOTOMICROGRAPH OF HEALED CASE OF OTITIS INTERNA PURULENTA: SECTION THROUGH FIRST TURN OF COCHLEA, SHOWING ENTIRE LUMEN FILLED WITH BONE

*c* = contour line between new-formed bone (*nb*) and bony cochlea (*bc*); *sp e* = remnants of spiral ligament; *sp m* = bony spiral membrane.

In *chronic* cases, the four stages are usually very difficult to distinguish, since the repeated acute attacks disturb the pathologic picture. Occurrence of different stages at the same time may often be noticed. FIGURE 44 shows granulation tissue and also beginning organization in the

first turn of the cochlea, while in the second turn suppuration and in the third turn exudation are present.

In chronic cholesteatoma cases, the slow course of the disease leads often to a circumscribed lesion in the inner ear, as described in the preceding section. This benign course, however, may suddenly be changed when an acute exacerbation of the cholesteatoma otitis occurs. Within a sur-

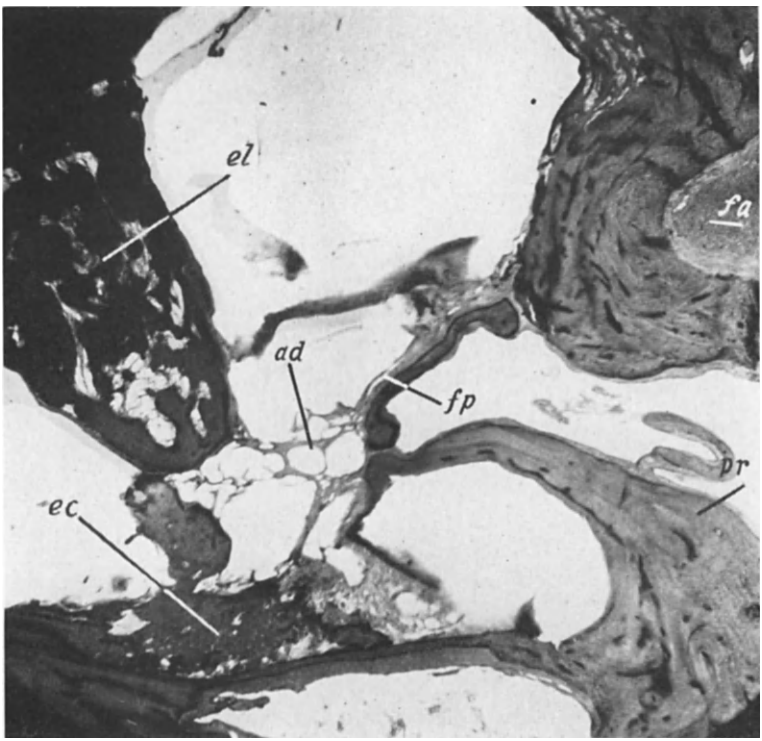


FIG. 43. PHOTOMICROGRAPH OF HEALED CASE OF OTITIS INTERNA PURULENTA: VERTICAL SECTION THROUGH MIDDLE AND INNER EAR, SHOWING MEMBRANOUS INNER EAR DESTROYED AND REPLACED BY CONNECTIVE TISSUE AND BONE

*pr* = promontory; *fa* = facial nerve; *fp* = footplate of stapes; *el* = large exostosis coming from medial wall of labyrinth; *ec* = exostosis in scala tympani of cochlea; *ad* = adhesions with inner surface of stapes plate.

prisingly short time the entire inner ear can be completely destroyed. The danger of a cholesteatoma, therefore, lies in such acute exacerbation.

In severe cases of chronic otitis media, a destruction of the supplying blood vessels may lead to sequestration of large parts of the inner ear. FIGURE 45 illustrates a process of this kind in which the entire cochlea, the bony as well as the membranous structure, has become sequestrated.

## CLINICAL COURSE

The clinical picture of the otitis interna purulenta may be manifold, depending upon various factors, such as the stage in which the patient is seen, the type of the internal otitis (foudroyant or protracted form), whether the infection of the inner ear has followed an acute otitis media or a chronic one, etc.

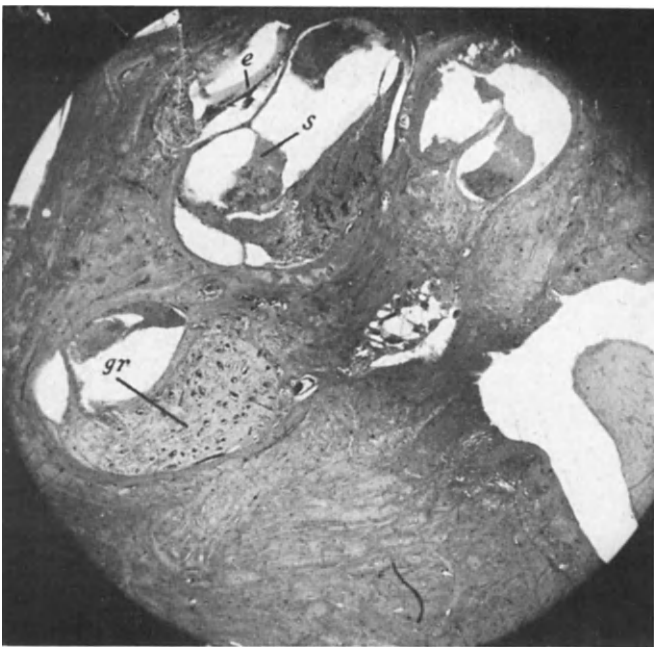


FIG. 44. PHOTOMICROGRAPH OF CASE OF OTITIS INTERNA PURULENTA IN CHRONIC OTITIS MEDIA: AXIAL SECTION THROUGH COCHLEA

*gr* = granulation tissue in first turn of cochlea; *s* = suppuration in second turn; *e* = exudation in third turn.

For clinical purposes it is advisable to use here the same four-stage characterization as for the pathology:

*First stage.* The clinical symptoms are not very marked. There is a vague dizziness, malaise—slight, not localized—headache, tinnitus of a high character (noise like ringing bell, high-pitched sound). Furthermore, there is often a spontaneous nystagmus of lowest degree, usually toward both sides, but occasionally only toward the side of the affected ear.

*Second stage.* There is gradual increase of dizziness, which soon assumes the character of a typical turning vertigo, and which occurs in the form of spells. There are sham movements of either surrounding objects or of the

patient himself. The former type is more common than the latter. The objects usually move in the direction of the quick component of the spontaneous nystagmus, so that the vertigo is dependent on the position of the head. Other symptoms are a spontaneous nystagmus of first to second degree, of a rotatory-horizontal form, toward the side of the diseased ear, further spontaneous disturbances of the equilibrium, and spontaneous past-pointing in the direction of the slow component of the

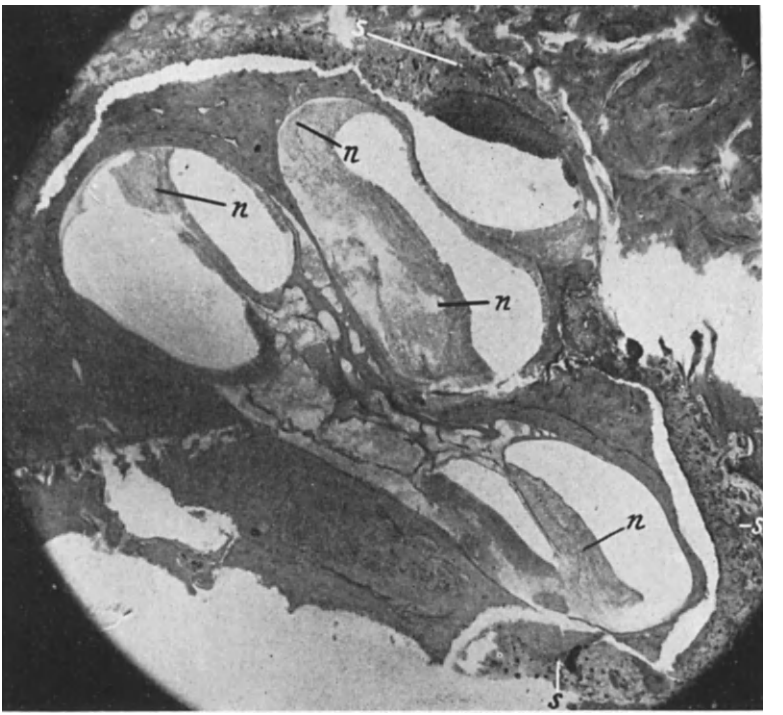


FIG. 45. PHOTOMICROGRAPH OF CASE OF OTITIS INTERNA PURULENTA IN CHRONIC OTITIS MEDIA

Axial section through cochlea, showing sequestration of entire bony cochlea caused by circumvallate suppuration (*s*) due to interruption of blood supply. Necrotic masses (*n*) are seen in membranous cochlea.

nystagmus. This past-pointing occurs first only with the arm of the affected side, but later on with both arms. The patients usually complain of nausea, vomiting, and malaise. The functional tests reveal a decrease of hearing and an increase of labyrinthine excitability. The fistula symptom is either positive or negative. All these symptoms and signs are regarded as manifestations of irritation.

*Third stage.* There is a gradual decrease of all signs of irritation. Vertigo, nausea, and vomiting become less and less intensive, subsiding completely within a period of ten to fourteen days. The spontaneous nystagmus switches toward the other side (that of the sound ear), the labyrinthine excitability decreases more and more until no irritability at all exists. The same happens to the cochlear function, which shows less and less hearing until complete deafness is reached. All these symptoms and signs are regarded as manifestations of destruction.

*Fourth stage.* After the destruction of cochlear and labyrinthine function, there are two possibilities with respect to the further course of the disease. One is a localization of the infection, with regressive processes, such as organization, scar formations (FIGS. 42, 43). This in turn leads to healing of the inner-ear disease, although with complete deafness and loss of labyrinthine excitability in relation to caloric, turning, and mechanical stimuli. However, when a certain period of time has elapsed, it looks as though the function for turning stimulation has been restored (phenomenon of compensation of Ruttin, p. 139). A glance at FIGURE 41 shows clearly that such a course is impossible, since a completely destroyed sense organ can never regain its function. It is not the peripheral sense organ that is responsible for the phenomenon of compensation, but rather a shift in the condition of the central vestibular excitability.

The second possibility with respect to the further course of the internal otitis in the fourth stage is a further spread of the infection beyond the anatomic boundaries of the inner ear, i.e., into the cranial fossae. It is then spoken of as an otitis interna purulenta complicata (see next section).

The clinical picture just outlined applies to the majority of cases of otitis interna purulenta. However, there are some forms that run a different clinical course and that therefore will be discussed separately:

*Foudroyant (fulminant) form.* This type of internal otitis follows a severe acute otitis media caused by very virulent germs. A sudden rupture through the windows leads to a diffuse invasion of all spaces of the inner ear with the virulent bacteria. The symptoms are extremely severe. A differentiation of four stages is impossible, since the infection rapidly runs through all stages almost simultaneously. The patients are caught by surprise in the sudden onset of terrific vertigo, nausea, and vomiting. The attacks are so severe that the patients may collapse. There is a continuous vomiting for a certain period of time. The spontaneous nystagmus shows the highest degree and its direction is toward the side of the sound ear. The patients become deaf and have no labyrinthine excitability. After a short time an intracranial complication becomes evident.

Many years ago I observed this foudroyant form in the course of severe influenza. Following is the history of such a case.

A 26-year-old white male acquired an acute otitis media in the left ear, during the course of severe influenza. On the next day he woke up with severe labyrinthine symptoms, such as terrific vertigo, nausea, vomiting, nystagmus of third degree in the direction of the sound ear. Hearing in the left ear was gone. On the third day vertigo and nausea were less intense, there was no vomiting at all; the nystagmus was still present. On the fourth and fifth days the patient felt much better, the nystagmus was only of first to second degree, the neurologic findings were normal, the lumbar puncture finding normal, the eyeground normal. In the night of the sixth day there was a sudden onset of severe vertigo, nausea, vomiting, rise in temperature (101 F.) rigidity of the neck, Kernig's sign, and loss of consciousness. A few hours later the patient died. Autopsy revealed a diffuse purulent meningitis and bronchopneumonia. From the beginning of the otitis media to the death from meningitis, a period of less than a week elapsed. A similar case was described by Jansen in which the infection ran through all these stages in five days. Although he operated on the fourth day (radical mastoidectomy plus resection of the labyrinth), Jansen did not succeed in saving the patient's life.

*Protracted form.* This type, usually following chronic otitis media, is characterized by its almost symptomless course. The manifestations of irritation may not be noticed either by the physician or by the patient himself. The same holds true for the manifestations of destruction: the deafness is not diagnosed, since the patients have usually been hard of hearing for many years; the loss of labyrinthine excitability is not found because it is not looked for. The condition is spoken of as a latent form of internal otitis; it appears latent to the patient as well as to the physician. But even if an experienced otologist does examine for the inner-ear functions and finds them absent, he still will have difficulties in diagnosing a purulent internal otitis. The loss of function is only significant of a destructive process, but we must distinguish whether the infection is still present or whether the process has healed with scar formation. In cases in which the middle ear is dry we cannot even say whether or not an inflammatory lesion of the inner ear exists, since the loss of function may be the result of secondary degenerative atrophic processes following chronic catarrhal conditions or chronic adhesive processes in the middle ear. A latent purulent otitis interna may be overlooked for a long time, until an acute exacerbation of the otitis media (often associated with cholesteatoma), produces manifest symptoms and signs relating to the inner ear.

### TREATMENT

The treatment of otitis interna purulenta must be conservative, with watchful waiting for eventualities. There must be strict rest in bed with a minimum of movements and darkening of the room in order to prevent

any irritation. The patient is kept under sedation (bromides, morphine hypodermically) and given a light diet. A tremendous amount of patience is necessary, not only on the part of the patient but also on the part of the physician and of the nursing personnel, since it may take six to ten weeks until all labyrinthine symptoms have subsided and the patient can be allowed to leave his bed. Any relaxation of the strict rules may have a fatal effect upon the patient. It is obvious that such a treatment can be undertaken only if the physician has a chance to observe the patient through a certain period of time and to watch carefully for any symptoms that do not belong to the clinical picture of an internal otitis but are significant rather of a beginning spread into the cranial fossae. Such early symptoms and signs consist of changes in the general aspect of the patient, as restlessness, insomnia or drowsiness, marked malaise, localized headaches (very important), rise in temperature, sudden changes in pulse rate, pleocytosis in the lumbar puncture fluid (more than 10 cells in 1 cu. mm.). In the very beginning of an intracranial complication, the other findings in the cerebrospinal fluid, such as pressure, color, chemical composition, etc., are usually still normal. Finally the eyeground must be examined for engorged blood vessels or beginning papillary edema (see below).

I am fully aware that many otosurgeons will emphatically object to such a conservative treatment, with the argument that it is too dangerous and involves too many risks. However, histologic findings have shown that there is often a tendency to heal in spite of large-scale destruction in the inner ear. Clinical observations on deaf-mutes prove these facts. On the other hand, the labyrinthine resection must be regarded as a serious operation even when performed by a skilful otosurgeon and therefore involves a number of risks (p. 230). Considering the unquestionable fact that a great number of cases of purulent internal otitis end in a complete healing when left alone, the conservative treatment appears justified, provided that a careful observation of the patient is possible. If any symptom or sign of an impending intracranial complication occurs, the labyrinthine resection must be performed immediately.

When the conservative treatment leads to a complete healing of the internal otitis, a radical mastoid operation should be performed at a later date in order to eliminate the source of infection in the middle ear.

### 5. Otitis Interna Purulenta Complicata

#### ETIOLOGY

If the infection has overstepped the normal boundaries of the inner ear, it is spoken of as otitis interna complicata. The infection may either use the anatomic pathways, such as the oval or the round window, the cochlear

or the vestibular aqueduct, and the internal auditory meatus, or may force its way by destruction of the bony walls that separate the inner ear from the cranial fossae. Occasionally a metastatic invasion may also occur. Complicated internal otitis follows either an acute or a chronic otitis media. In acute cases the middle-ear process is usually very severe, being caused by extremely virulent germs. There is often a rupture through the windows with sudden invasion of the entire inner ear. In the chronic cases the complication is mostly the result of an acute flare-up of the middle-ear otitis. The cholesteatoma plays the main role in such cases. It has been shown previously that the cholesteatoma, which grows rather slowly, usually leads to circumscribed lesions in the inner ear. Since it takes a relatively long time before the cholesteatoma has affected the entire thickness of the bony wall, there is plenty of time for protecting processes in the labyrinth, so that only circumscribed, walled lesions in the labyrinth are found, which usually show a great tendency to heal. The course is quite different, however, if an acute exacerbation of the cholesteatoma occurs. After a short time the infection spreads over the entire inner-ear spaces and from there to the cranial fossae. The danger of a cholesteatoma, therefore, lies in an acute flare-up, particularly when associated with a mastoiditis. Statistics show that among the intracranial complications that occur in the course of an acute exacerbation of the middle ear, a cholesteatoma is found in more than 90 per cent of cases.

### PATHOLOGIC ANATOMY

Spread of the infection from the inner ear into the cranial fossae is favored by the increased pressure in the inner ear and by the extensive destruction of the bony walls. FIGURE 46 illustrates a case of acute otitis media with rupture through the oval window. There is an invasion of all inner-ear spaces with pus. In the common cross between the superior and the inferior vertical canal, the suppuration has broken through, leading to a destruction of bone between the middle and the posterior cranial fossa. This in turn has caused an intracranial complication. FIGURE 47 illustrates a case of chronic otitis media that has led to a fistula on the medial wall of the inner ear. The membranous inner ear has been completely destroyed by the suppuration. In this case the intracranial complication is caused by destructions of the bone in the region of the superior petrosal sinus.

### CLINICAL COURSE

The clinical picture depends largely upon the site of the complication. For clinical purposes we must differentiate between extradural and intradural complications. To the former belong pachymeningitis externa

(extradural abscess), to the latter leptomeningitis, empyema of the endolymphatic sac, and cerebellar abscess. While leptomeningitis represents the most common complication of acute internal otitis, the other complications are more frequent in a course of chronic internal otitis. Intracranial complications following internal otitis are separately discussed in chapter vii.

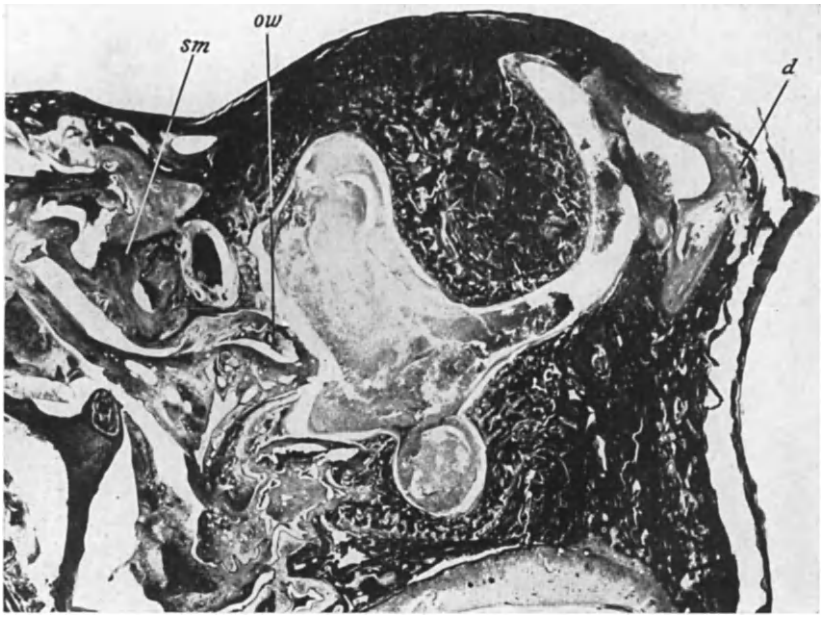


FIG. 46. PHOTOMICROGRAPH OF CASE OF OTITIS INTERNA PURULENTA COMPLICATA IN ACUTE OTITIS MEDIA: VERTICAL SECTION THROUGH MIDDLE AND INNER EAR

Suppuration of middle ear (*sm*) has broken through oval window (*ow*), spreading all over inner ear. Destruction of bone (*d*) between middle and posterior fossa is site of intracranial complication.

### 6. OTITIS INTERNA TRAUMATICA

Traumatic inner-ear lesions differ from the genuine forms in course, treatment, and prognosis and must, therefore, be discussed separately. Clinical observations have proved that traumatic infections are mostly severe and afford bad prognoses. There are two main reasons for this: one is the absence of any walling or protecting processes when the infection suddenly invades the inner ear; the other, that extensive lacerations with loss of continuity of structure in the inner ear have been caused by the trauma, so that a secondary infection can rapidly spread all over.

The clinical course depends upon the kind of trauma, the site of the

injury, and the virulence of the bacteria. With regard to the kind of trauma, four groups may be differentiated: skull fractures, gunshot lesions, foreign-body injuries, and operative damages.

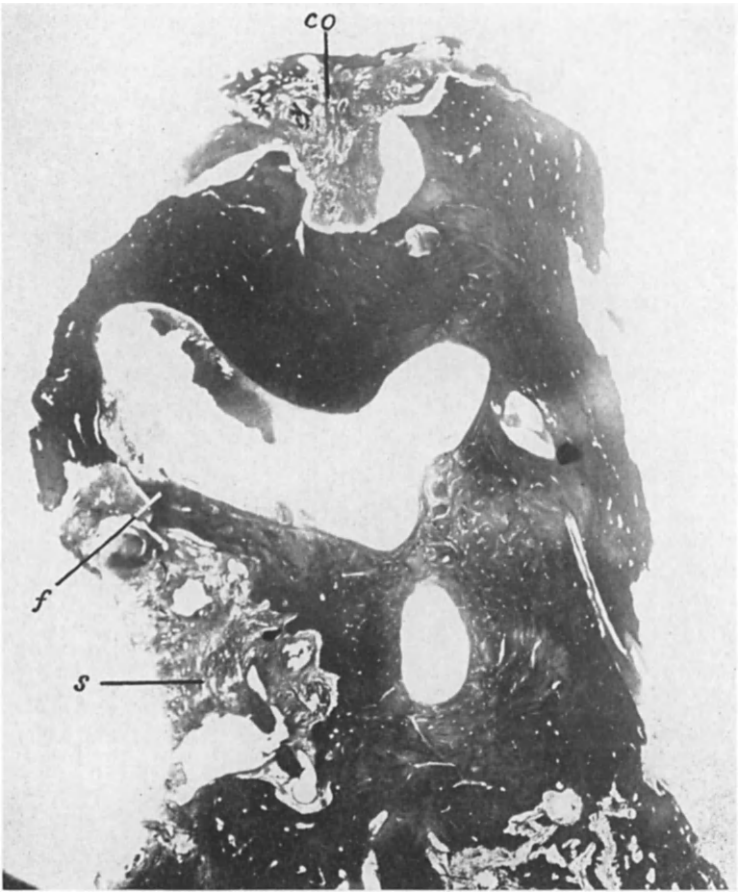


FIG. 47. PHOTOMICROGRAPH OF CASE OF OTITIS INTERNA PURULENTA COMPLICATA IN CHRONIC OTITIS MEDIA: VERTICAL SECTION THROUGH MIDDLE AND INNER EAR, SHOWING MEMBRANOUS INNER EAR DESTROYED

*s* = suppuration and granulation in middle ear; *f* = fistula in labyrinthine capsule; *co* = site of complication, in region of superior petrosal sinus.

## SKULL FRACTURES

Infections of the inner ear mostly follow the transverse skull fracture. The fracture line runs along the posterior surfaces of the pyramid in the region of the internal auditory meatus, leading to laceration in the vestibule. Usually the lateral wall of the labyrinth is involved, so that a communication between inner and middle ear results. In cases in which there is a

combination of transverse and longitudinal skull fracture, an additional rupture of the drum membrane and external auditory meatus may be noticed. This in turn leads to a communication between inner, middle, and external ear (p. 345).

The clinical picture depends upon whether or not the brain is involved by the trauma. If there is a severe damage of the brain, the labyrinthine symptoms may be entirely masked by the brain symptoms. If the patient later on regains consciousness, the stage of irritation of his labyrinthitis may be over and he may feel very comfortable. On examination, a complete loss of cochlear and labyrinthine function may be revealed.

If the brain is not affected, or only slightly, the manifestations of irritation can easily be noticed.

*Treatment.* Early operation of the middle and inner ear is necessary. The operation must be performed as soon as the diagnosis of infection of the inner ear has been made. Here, however, comes the difficulty. How can we say that the inner ear has become secondarily infected? It is impossible to distinguish between an infected traumatic lesion of the labyrinth and a noninfected one, since the clinical symptoms are the same (Hinsberg, Klestadt). For this reason we must direct our attention to the *middle* ear. If in a case of traumatic injury of the inner ear a suppurative process exists simultaneously in the middle ear, we must assume also an infection of the inner ear and must operate immediately.

### GUNSHOT WOUNDS

We must differentiate direct and indirect injuries (p. 366). The former are caused by the projectile, the latter by fractured and splintered bone and by change in atmospheric pressure during detonation. *Direct* injuries of the inner ear are so grave in effect that the victims usually die immediately. If they survive, the danger of meningitis is great. *Indirect* lesions show more or less the same conditions as those in skull fractures but have a better prognosis. Preysing believes that a hemorrhage on the brain surface may lead to circumscribed walling processes that help to check a further spread of infection. According to Zange, the brain is pressed into the defect of the bony capsule, thus closing the opening and checking a further infection. If the lateral wall of the labyrinth is injured, a communication with the middle ear is opened.

*Treatment.* Early operation of the labyrinth is required. In contrast to the procedure indicated in relation to the genuine forms of internal otitis, we should not wait for signs of a beginning intracranial complication.

### FOREIGN-BODY INJURIES

Objects introduced into the ear (chiefly because of itching) are usually long and thin, such as knitting needles, lead pencils, etc. The common

site of injury is the oval window. Besides these accidental trauma, there are cases of mutilation, particularly among soldiers. I had plenty of opportunity, during four years of the first world war, to collect a great deal of material. The methods and objects used in order to escape the battle-front were almost unbelievable. In order to place irritating drugs as deep as possible in the ear canal, men introduced them by means of goosequills, ramrods, pipescrapers, barbed wire, etc. The inner-ear lesion was the result either of a mechanical damage caused by the instrument, or of a chemical injury caused by the irritating substance. Most of the substances had a cauterizing effect (hydrochloric acid, essence of acetic acid, sodium hydroxide, gunpowder, dynamite, organic drugs). This led to profuse suppuration and ulceration—processes like those in severe diphtheritic infections. I saw cases in which the bony walls of the middle ear were entirely exposed, devoid of periosteum. The infection went through either the oval or the round window into the inner ear, and in one case through the facial canal.

The clinical picture in cases of window rupture is similar to that in the genuine purulent otitis of the foudroyant type. The treatment required is early labyrinth operation.

### OPERATIVE DAMAGES

The most dangerous accident that can occur during a mastoid operation is *luxation of the stapes*. Secondary infection of the inner ear occurs in almost every case, since the middle ear always shows a suppuration. The luxation of the stapes causes a sudden invasion of the large perilymphatic cistern by infected material. According to Jansen, the secondary infection takes place within twelve hours after the operation. The clinical symptoms are very violent. The manifestations of irritation become more and more intensive within hours, and may be followed quickly by meningitis symptoms (rise in temperature, changes in lumbar puncture fluid, hypersensitivity to light, tachycardia, etc.). On the second or third day the meningitis may be fully developed. Labyrinth operation at the earliest possible date is imperative. In the majority of cases the operation is performed too late. Although the mortality rate is very high, Jansen has described some cases that survived.

Injuries to the *horizontal semicircular canal* show a relatively benign course. They do not tend to become infected. The clinical symptoms occur immediately after operation, in contrast to the effect of luxation of the stapes, where the symptoms appear six to twelve hours after the operation. Another differentiation from the latter situation can be made on the basis of the further course. In cases of semcircular canal injuries, the symptoms gradually subside. There may also sometimes be a palsy of the facial

nerve, which lies just beneath the injured horizontal canal. For the overwhelming majority of cases, the treatment should be conservative.

Another operative accident may happen during *extraction of polypi*. The clinical course depends upon where the polyp comes from. If its pedicle is inserted on the stapes, a luxation of this ossicle may be caused (see above). Many years ago I had an accident I shall never forget. Just after I had removed a polyp that seemed to come from the antrum, the patient turned suddenly pale and his face was covered with sweat; this was followed by convulsions and collapse. Although the patient recovered soon and had no sequelae, I shall always remember the few anxious hours I experienced.

Great care should also be given to the procedure of removing polyps or remnants of polyps by means of cauterization. There are two types of caustics in use, one with relatively superficial effect, the other with deep action. However, in neither case are we able to determine the exact limit of effect, nor can we know in advance the irritation reaction of the labyrinth. I do not advise the use of any cauterization for ear polyps.

Accidents during paracentesis of the drum are usually not serious. There is one exception, namely, luxation of the stapes. Such an accident can happen only if the head of the patient is overtilted toward the shoulder, so that the horizontally introduced instrument may hit the incudostapedial joint.

There are, finally, traumatic lesions of the inner ear occurring *without any accident* during operation. These are usually in the form of a low degree of inflammation (serous induced labyrinthitis). The symptoms often occur between the second and the fourth day after operation.

For practical purposes, therefore, we can differentiate as follows. In operative injury of the *horizontal canal*, the labyrinthine symptoms occur *immediately* after the operation; in injury of the *stapes*, the symptoms follow *six* to *twelve hours* after the operation; in *induced labyrinthitis*, the symptoms usually appear at some time from the *second* to the *fourth day*.

### 7. Indications for Surgery

A study of the treatment of internal otitis reveals that great diversity of opinion exists with respect to the indications for surgery. Some otologists advocate surgical measures even for circumscribed lesions in the labyrinth in order to check a further spread, while others wait for symptoms of beginning complication before they operate. Although judgments based on individual skill and experience are very valuable, the indications for surgical measures should be more uniformly agreed on. Why is there no diversity of opinion in respect to other inflammatory lesions, such as extradural abscess, sinus thrombosis, or cerebellar abscess? The answer

is simple. These lesions represent surgical diseases, since spontaneous healing almost never occurs. Quite different, however, are the conditions in inflammatory lesions of the inner ear. These lesions often show a tendency to heal spontaneously in spite of widespread destruction of the inner ear (cf. histologic findings, p. 208).

Clinical studies made by Alexander and the writer on the inmates of various deaf-mute institutes in Vienna revealed that more than half of them had had diffuse suppuration processes in the inner ear. Although none had any surgical interference at the time, all these cases healed completely, although with loss of the cochlear and labyrinthine functions. On the other hand, it must be borne in mind that a labyrinth operation, even though performed by an experienced and skilful otosurgeon, is always a major surgical procedure, thus involving a number of risks.

But even when a complete healing has been obtained, the extensive bone defects due to operative removal will always represent a locus minoris resistentiae. The exposed dura of the middle and the posterior fossa, covered by scar tissue and scalp, decreases the resistance to mechanical injury. All these facts lead us to the conclusion that surgery in the inner ear should be undertaken only if absolute necessity arises. A relative indication represents no justification at all.

It is beyond the scope of this book to discuss the many viewpoints obtaining among the otologists of the various countries. The Vienna school was split on this subject into two parties: one that can be called the radical group was represented by Neumann and his followers; the other, the conservative group, by Alexander and his pupils.

Neumann classified the suppurations of the labyrinth with respect to treatment into the following categories:

1. Function of cochlea and labyrinth intact. Mastoid operation reveals fistula. Labyrinth operation is contra-indicated.

2. Function of cochlea absent, of labyrinth present. Mastoid operation reveals fistula. Labyrinth operation is indicated only when fever and spontaneous nystagmus occur.

3. Function of cochlea present, of labyrinth absent. Mastoid operation reveals fistula. Labyrinth operation is indicated only when other symptoms, particularly fever, exist.

4. Function of cochlea present, of labyrinth absent. Mastoid operation reveals no fistula. Labyrinth operation is indicated when other symptoms, particularly fever, exist.

5. Function of cochlea and labyrinth absent. Mastoid operation reveals fistula. Labyrinth operation is indicated.

6. Function of cochlea and labyrinth absent. Mastoid operation reveals no fistula. Labyrinth operation is indicated when spontaneous

nystagmus exists. If there is no nystagmus, the operation should be performed only when fever or meningitis symptoms are present.

7. Function of cochlea absent, of labyrinth present. Mastoid operation reveals fistula. Labyrinth operation is not indicated.

According to Ruttin, labyrinth operation is indicated when the labyrinth does not react to any stimuli (caloric, turning, fistula) and the hearing acuity is lost.

At a suggestion of my chief, Alexander, I described fifteen years ago the standpoint maintained in our clinic in Vienna, here briefly summarized. The inflammatory diseases of the inner ear can be classified with respect to treatment according as they require:

1. *Operative* measures in the *middle ear:* These are aimed to eliminate the infection source in the middle ear. According to the underlying process, either a simple mastoidectomy or a radical mastoid operation must be performed. This treatment applies to para-otitis interna purulenta (group 2) and peri-otitis interna purulenta (group 3 or stationary form).

2. *Operative* measures in the *middle and inner ear* as a one-stage procedure: This is aimed to eliminate the source of infection in the middle ear and to check a further spread of infection into the cranial fossae. This treatment applies to otitis interna purulenta complicata (group 5) and otitis interna traumatica (group 6).

3. *Conservative* treatment, strictly avoiding any therapeutic trauma: This is aimed not to disturb the spontaneous healing tendency. This treatment applies to otitis interna serosa (group 1) and to otitis interna purulenta (group 4).

There is no doubt that a number of otosurgeons uphold a more radical standpoint. However, the great danger for the patient's life is not at the hands of the conservative nor of the radical group, but of those who try to make concessions in one or the other direction, thus taking a middle course.

When, for instance, in a case of otitis interna purulenta (group 4), an otologist, instead of being very conservative or very radical (middle- and inner-ear operation), first performs a mastoidectomy to see whether the lesion will then heal, he may shortly be surprised by the appearance of intracranial symptoms. There are two reasons why the complication occurs: one is the operative trauma, which causes a propagation of the infection, the other the insufficiency of a mastoid operation for draining the inner ear.

### 8. Operative Technic

The main difficulties in the technic of labyrinthine surgery must be attributed to the anatomic course of the facial nerve, which runs through

the entire field of operation (Fig. 48). All the methods—and all modifications of them—used by the otosurgeons of the various countries have the same aim, namely, to avoid injury to the facial nerve. For that reason some otologists operate above the nerve (suprafacial methods), others behind the nerve (retrofacial methods) or beneath the nerve (infrafacial methods). Uffenorde operates directly over the nerve. He deliberately

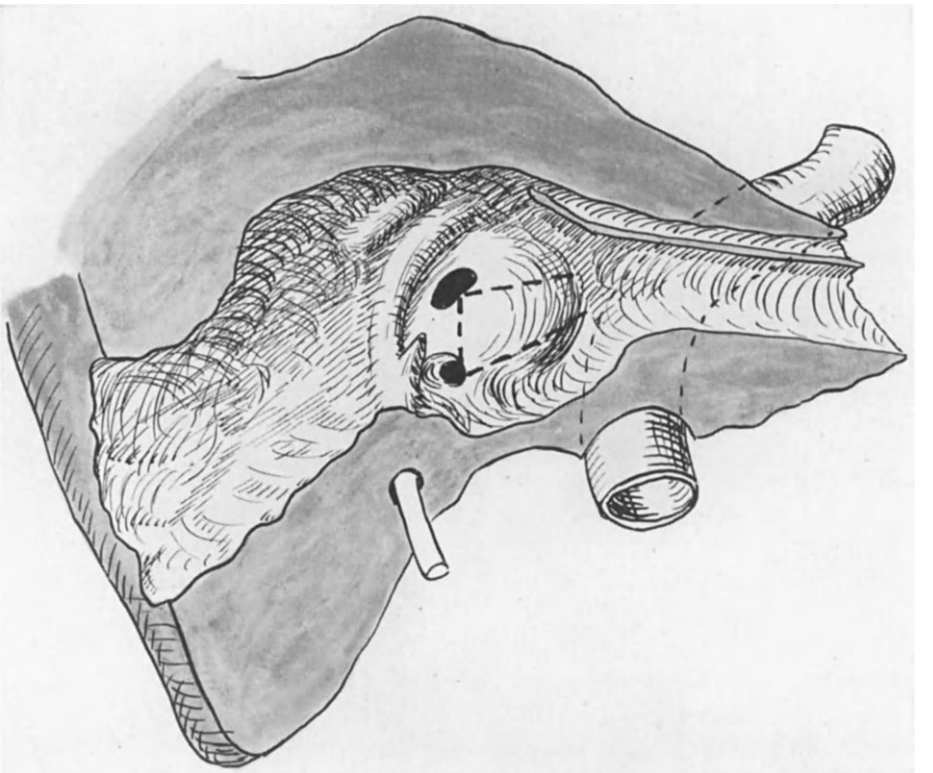

Fig. 48. Diagrammatic Section Showing Medial, Posterior, and Anterior Wall of Middle Ear

Dotted lines illustrate technic for removal of promontory in labyrinth operations.

exposes the facial nerve in an area from the pyramidal eminence to the anterior knee.

Other differences in technic arise in accordance with the various approaches to the labyrinth. There are procedures in which the cochlea and tympanic portion of the vestibule are primarily opened (Hinsberg, first method, Hautant-Rendu, Bourguet, first method); others in which the anterior limb of the horizontal canal (Botey and Richards) or the posterior

limb (Jansen, first method, Frey and Hammerschlag, Bourguet, second method, etc.) is first opened; while other surgeons approach the labyrinth from Troutman's triangle in front of the lateral sinus (Jansen, Neumann, Ruttin, Blumenthal, etc.).

Some operate only on certain parts of the labyrinth, while others perform a complete extirpation of the inner ear (Jansen, Neumann, Ruttin). There are operators who carefully prevent any exposure of the dura, while others start surgery of the labyrinth with a large exposure of the lateral sinus and the dura of the middle and the posterior fossa (Jansen, second method, Neumann, second method, Alexander, Ruttin). Finally, it should be mentioned that some methods differ only in respect to the order of their various steps. For example, Hinsberg opens first the cochlea and vestibule and then the horizontal canal, while Botey follows the opposite order.

It is beyond the scope of this book to describe in detail all the numerous methods and modifications. I shall discuss in detail only one example of the tympanic methods and one of the retrotympanic. Before going into the technical details of the operations, something must be said about the instruments. In our clinic we used the labyrinth set of Alexander. It contained series of gouges in sizes from no.'s 5 to 14, these being longer than the usual Alexander gouges. There were, further, rongeurs with long and stout branches, tissue forceps (also longer than usual), flexible silver probes, a set of curettes, sharp double spoons (one end round, the other oval), blunt-edged spatulae, a flat sinus protector, and a flat chisel for removal of the promontory. Some operators use, instead of gouges and chisels, electrically driven burrs. The question as to which method is the better one can be answered simply in this way: The best method is that with which the operator is most familiar. As I have not much experience with the electric burr, I can only quote the claims of the literature. The fine bone dust caused by the burr may disturb the anatomic picture. For example, it may close the small openings of the cross-sected semicircular canals, which are badly needed for orientation. Other disadvantages of the burr are the operative injuries. If the facial nerve is caught by the burr it is badly torn, while if the nerve is struck by the gouge it will often heal with restoration of function.

As a preliminary step to any labyrinth operation, a thorough radical mastoidectomy must be performed. Some surgeons operate in two stages, while others prefer a one-stage procedure. In our clinic we used to operate upon middle ear and labyrinth in one sitting.

We shall now describe the respective technics of operation.

### HINSBERG-BOURGUET METHOD

This procedure starts with the removal of the promontory by three strokes of the chisel (FIG. 48). The first stroke is executed parallel to and

beneath the fallopian canal. Care must be taken not to injure the superior rim of the oval window, as this would endanger the facial nerve. The second stroke is made at the base of the promontory, while the third stroke is carried out in front of the facial ridge between the oval and the round window. After removal of the bone chip, the lower whorl of the cochlea is exposed. The opening in the cochlea is then enlarged by means of a sharp curette or a small chisel until the second whorl and the modiolus

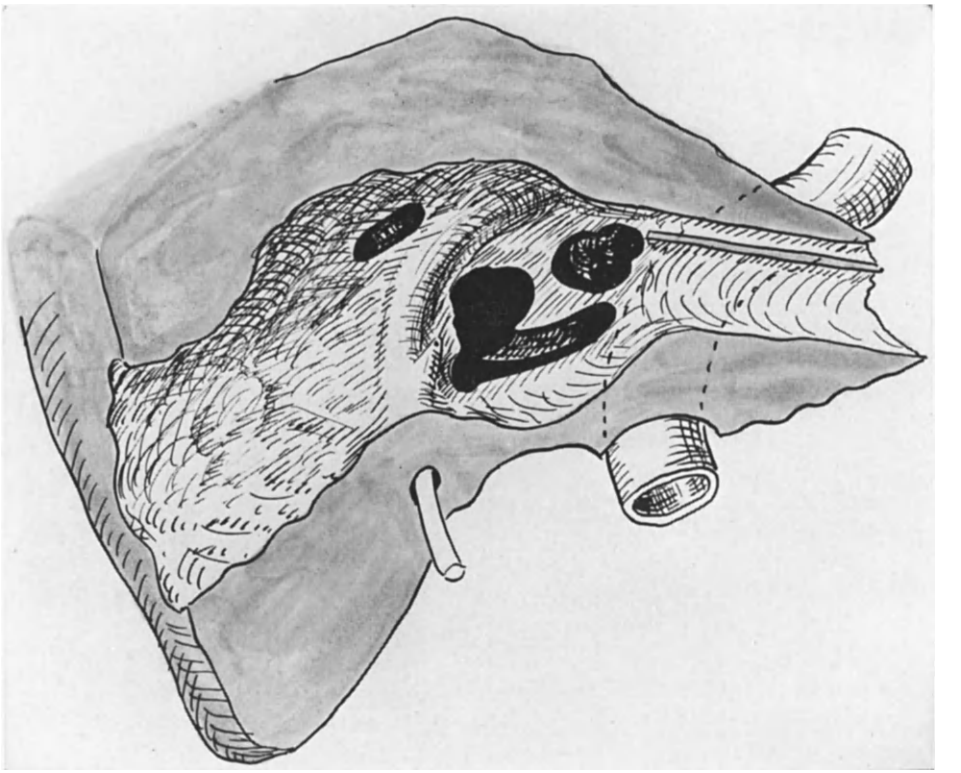

FIG. 49. DIAGRAMMATIC SECTION SHOWING COMPLETED HINSBERG-BOURGUET LABYRINTH OPERATION

can be seen. In order to expose the top of the cochlea, the bone must be removed anteriorly as far as the canal for the tensor tympani muscle, which lies just above the tympanic mouth of the eustachian tube. Special care must be taken not to injure the carotic artery because of the close relation of this region to the vessel. With the entire cochlea exposed, a hooked probe is introduced into the opened vestibule in an upward and outward direction, leading into the horizontal semicircular canal. The lateral

wall of the horizontal canal is then removed by chiseling in an oblique direction from above (from the tegmen tympani). In this manner operative damage of the facial nerve can usually be avoided. Upon the exposure of the ampullae of the horizontal and the superior vertical canal, the operation is completed (FIG. 49).

### JANSEN-NEUMANN METHOD

The first step consists of an exposure of the lateral sinus and removal of the bone in Troutman's triangle. According to the original method, exposure of the dura of the posterior fossa is not essential. However, in

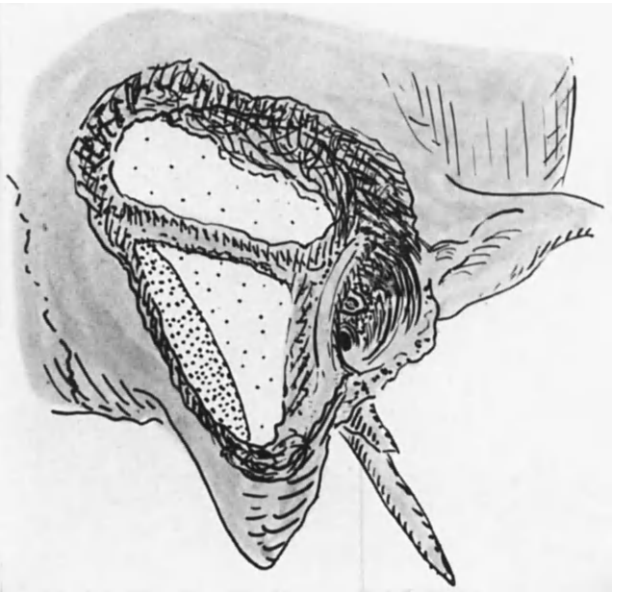

FIG. 50. DIAGRAM SHOWING JANSEN-NEUMANN OPERATION WITH DURA OF MIDDLE AND POSTERIOR CRANIAL FOSSA AND LATERAL SINUS UNCOVERED

our clinic we principally uncovered the dura both of the posterior fossa and of the middle fossa at the beginning of the labyrinth operation. The reasons were: the exposure makes a thorough inspection of both cranial fossae possible; it further simplifies some technical difficulties of the labyrinth operation; it improves the conditions for drainage; and, should the postoperative course indicate more surgery, for instance upon the brain, no major chisel work is then necessary. This may be of great importance when the general condition of the patient is very poor. Having exposed both cranial fossae, the bone bridge between them is resected, thus exposing the superior petrosal sinus (FIG. 50).

The next step is to remove the bone portion between the anterior border of the lateral sinus and the facial ridge. This can be achieved by cutting with the gouge, thus separating thin layers parallel to the posterior pyramidal wall. The concave surface of the gouge faces the dura of the posterior fossa, while the convex surface is directed toward the posterior pyramidal wall. With continuing removal of the bone in shavings or thin chips, two openings in the pyramid soon become visible (FIG. 51). The upper hole represents a cross section of the common crus (superior and inferior vertical canals), while the lower opening is formed by the cross-sected inferior vertical canal near its ampullar end. These openings

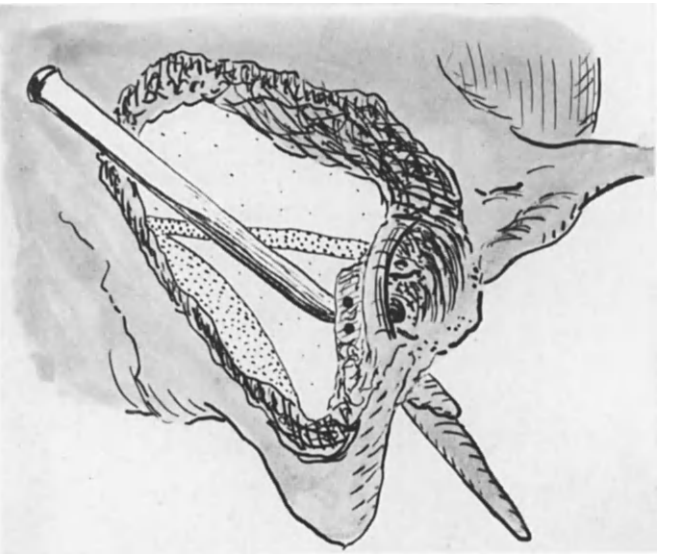

FIG. 51. DIAGRAM SHOWING JANSEN-NEUMANN OPERATION: EXPOSURE OF SUPERIOR PETROUS SINUS AFTER REMOVAL OF BONY BRIDGE BETWEEN CRANIAL FOSSAE, AND CORRECT POSITION OF CHISEL BETWEEN OPENINGS OF VERTICAL CANALS

should be the guides for the further work. Continuing to chisel between the two holes will soon produce a third opening, representing a cross section of the horizontal semicircular canal. This opening is oval-shaped and lies approximately midway between the two previously made holes, but somewhat more exteriorly. On further chiseling in this direction, the opening becomes larger as it leads directly into the vestibule. By removing the prominent parts of bone on the posterior pyramidal wall by parallel undermining shavings, the lateral border of the internal auditory meatus will finally be reached and should be removed (FIG. 52). This completes the retrotympanic work. A flat chisel is then placed vertically upon the

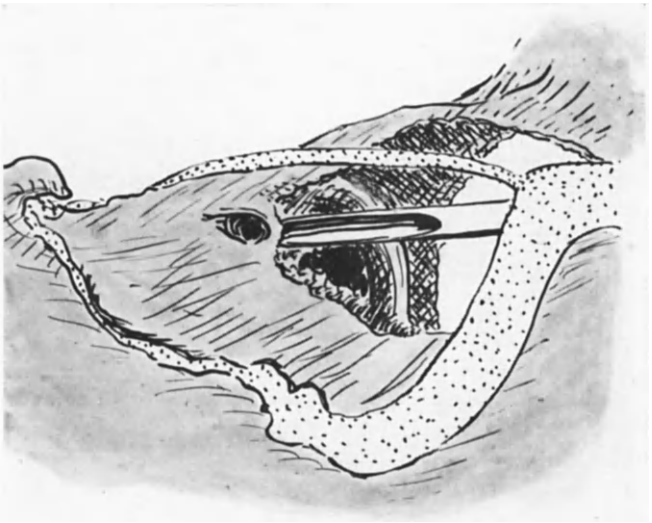

FIG. 52. DIAGRAM SHOWING JANSEN-NEUMANN OPERATION: MEDIAL VIEW OF PYRAMID, AND CORRECT POSITION OF GAUGE FOR REMOVAL OF LATERAL MARGIN OF INTERNAL AUDITORY MEATUS

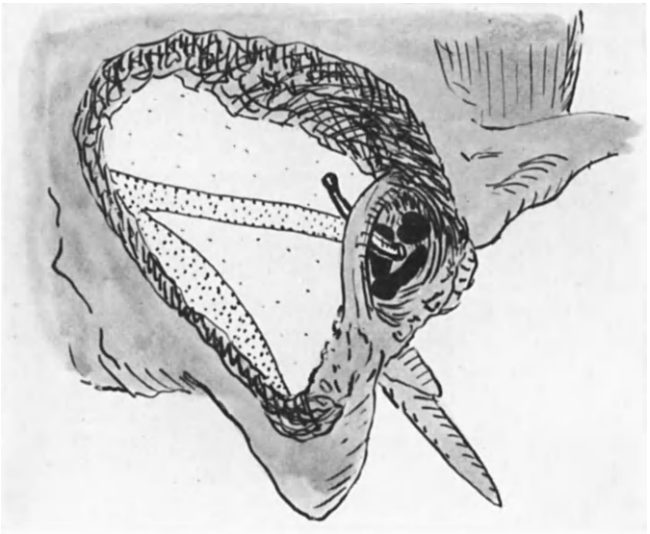

FIG. 53. DIAGRAM SHOWING JANSEN-NEUMANN OPERATION, COMPLETED: PROBE BENT AT RIGHT ANGLE, INTRODUCED IN TYMPANIC PART OF VESTIBULE, EMERGES FROM RETROTYMPANIC PART

promontory just in front of the facial ridge and the bony plate between the oval and the round window is removed. After enlargement of the exposure of the tympanic portion of the vestibule, a hooked probe is introduced in front. The head of the probe now appears in the retrotympanic portion of the vestibule (FIG. 53).

The retrolabyrinthine cavity is loosely packed with iodoform gauze. The wound cavity remains open until completely closed by granulation. The after-treatment is similar to that in radical mastoidectomy.

### OPERATIVE INJURIES: TECHNICAL PRECAUTIONS

The Jansen-Neumann method represents a serious major operation requiring an exact knowledge of the topographic anatomy and a certain amount of personal skill. Some difficulties may arise when the preceding radical mastoidectomy has not been performed thoroughly enough. A high facial ridge, a prominent facial spur, an overhanging lateral attic wall, etc., may interfere.

Another technical difficulty may be due to an improper position of the gouge on removal of the posterior pyramidal wall. In order to cut thin bone chips, the chisel should be held parallel to the posterior wall, i.e., almost horizontally. If the operator has not exposed enough of the cranial fossae, the remaining bone in the petrosal angle or behind and above the sinus may force him to hold the gouge too vertically, so that he is unable to slice off the bone. To prevent waste of time in unsuccessful efforts, he should simply take off the overhanging edges in order to have plenty of room for his chisel work on the pyramid. An operator unfamiliar with the labyrinth operation may further have some difficulties in removing the hard solid bone of the pyramid, if he shaves only the bone on the surface. By failing to cut the deep parts, he leaves stairs in the bone, which become thicker after each chisel stroke. By this means he will never reach the margin of the internal auditory meatus. The incomplete cut with the gouge has also another disadvantage: it makes it very difficult to remove the fractured but not entirely cut bone chip. Forcible efforts to get the piece out with tissue forceps or stout rongeurs may lead to lacerations of the dura of the posterior fossa. In order to avoid injury to the dura, it is advisable to separate the dura from the underlying bone before chiseling, and to protect it by blunt-edged spatulae or by a sinus protector. The instrument should be held by the assisting physician. Special care must be taken when working in the area of the endolymphatic sac, because the dura is particularly adherent in this region. In order not to tear it, a circular incision should be made into the dura with a sharp knife.

Operative accidents may occur in chiseling on the pyramid. Any deviation from the direction (outlined by the two openings) may lead to

severe damage. A deflection of the gouge in an upward direction may cause an injury to the superior petrosal sinus. A deviation downward may endanger the jugular bulb, while a departure outward from the posterior pyramidal wall may cut the facial nerve. The facial nerve may further be injured by chiseling off of the lateral margin of the internal auditory meatus or by removal of the promontory (see Hinsberg method).

Which of the numerous labyrinthine methods should really be employed depends entirely upon the indication for surgical intervention. It is obvious that with strict indications as outlined in the preceding section, the Jansen-Neumann operation is the only method of choice.

## INFLAMMATION OF THE PETROUS PYRAMID

### 1. Pathways of Infection: Pathology

Inflammatory processes of the petrous pyramid may occur in the course of acute or subacute otitis media, or of an acute exacerbation of a chronic otitis. The lesions often follow surgery of the mastoid (simple mastoidectomy).

A study of the literature, particularly, of the years 1930–36, creates the impression that a new form of disease has been detected. As a matter of fact, Troeltsch as much as half a century ago directed attention to the perilabyrinthine spaces as pathways of infection of the petrous pyramid. At the same time Gruber and Koerner stressed the importance of the carotic canal as a route of infection.

In order to understand the pathology, symptomatology, and treatment of all the inflammatory lesions of the pyramid, a thorough knowledge of the anatomy and pneumatization processes of the petrous bone is necessary. The reader is therefore referred to chapter i (cf. anatomy of perilabyrinthine spaces). We agree with Guild, who stresses the extremely wide range of "normal variations" of the adult human petrous pyramid. Although the pneumatization processes are very complicated, and there is hardly a petrous bone that exactly resembles any other in structure, certain tracts can nevertheless be observed where pneumatization takes place. Schlander assumes three different tracts of pneumatization between the middle ear and the tip of the pyramid: one running along the tegmen and above the internal auditory meatus to the tip; another extending from the medial wall of the antrum to the tip; and another running above the cells of the eustachian tube and below the cochlea to the tip. Tobeck describes four pathways: (1) a *posterior* pathway originating in the antrum; (2) a *superior* pathway coming from the epitympanic recess; (3) an *inferior* pathway originating in the hypotympanic recess; and (4) an *anterior*

pathway that pneumatizes first the lower wall of the bony tube and finally may reach into the petrosal part behind the carotic canal. Kopetzky distinguishes between a coalescent osteitis and an osteomyelitis of the petrous bone. The former occurs, according to Kopetzky and Almour, in fully pneumatized temporal bones—it cannot occur in any other type (diploetic or sclerotic); the latter occurs in diploetic bones (Eagleton).

The course of inflammation of the petrous pyramid depends upon the underlying pathology. In mild cases there are acute inflammatory swelling of the mucous linings of the various cells and serous exudation, as found in nonsurgical mastoiditis (see p. 239). All these changes resolve spontaneously. If we had the opportunity to examine microscopically the perilabyrinthine cells in every case of acute otitis media or mastoiditis, we should find these changes much more frequent than is generally believed. Fortunately all these slight changes usually subside in the further course of the disease. In severe cases, however, abscess formation, necrosis of the bone, and fistulation into the adjacent parts may take place. The fistula may lead into the posterior or the middle fossa, into the base of the pyramid, or into the labyrinth or carotic canal.

### 2. Clinical Diagnosis

The clinical picture of infections of the petrous pyramid depends largely upon the pathways of infection, the sites of the lesions, and the extensions —i.e., whether the infection remains intrapyramidal or whether a fistula develops. In the latter cases, symptoms and signs may arise either from the middle or posterior fossa, from the labyrinth or cochlea, or from the venous plexus surrounding the carotid artery. In noncomplicated cases, however, the diagnosis usually does not present much difficulty, and can be made on the basis of the following factors:

1. Symptoms originating from the fifth nerve, such as orbital pain, particularly in the depth of the orbit—occurring mostly at night—and occasionally toothache.
2. Symptoms originating from the sixth nerve, such as double vision due to palsy of the abducens. This symptom may, however, often be absent.
3. Slight septic temperature.
4. Otorrhea extended over a long period of time, occasionally subsiding and recurring.
5. X-ray examination should be made early to study the structure of the petrous bone, and should be repeated to determine the progress of the disease. However, it must be borne in mind that one cannot rely too much on roentgen findings.

Besides this characteristic form, there may be cases of infection of the petrous bone that show no signs or symptoms.

Before discussing the treatment of these lesions, attention should be directed to a complex of symptoms known as the "Gradenigo syndrome." It consists of diplopia, due to homolateral abducens paralysis, and pain in the temporoparietal region of the affected side, occurring in the course of an acute otitis media. It must be emphasized that infection of the petrous pyramid and Gradenigo's syndrome are not always identical. The latter cannot be considered as a clinical entity, since it comprises a number of lesions, such as osteitis of the tip of the petrous bone, extradural abscess or circumscribed leptomeningitis about the tip of the petrous bone, toxic neuritis of the sixth nerve, cerebral edema, etc.

### 3. Treatment

The otologic practitioner is amazed when he studies the numerous major surgical procedures advocated in recent years by the various authors. His experience has taught him that almost all cases of infection of the petrous bone recover when he employs the usual surgery and when he takes care to effect a thorough drainage. Although a great deal more than 90 per cent of cases can be treated this way, it must still be admitted that a few other cases showing severe pathologic changes do require special procedures. For the experienced otologist who knows his anatomy, and who has carefully watched the clinical symptoms and the course of the disease, the surgery presents no problem. He will use his own judgment as to the method of approach, trying to follow the pathways taken by the infection. We shall here briefly mention three of the various methods of approach: the extrapetrosal, the intrapetrosal, and Lempert's approach. The first includes various subdural approaches to the apex, while the second is limited to drainage of the apex through the temporal bone, and the third is an endaural approach.

In the extrapetrosal or subdural approach, the dura of the middle cranial fossa is separated from the superior surface of the petrous bone through (1) the simple mastoidectomy exposure, which is inadequate because it does not allow for a search of the inner tympanic wall for fistulas into the anterior perilabyrinthine space; (2) Eagleton's technic, in which the dural elevation takes place in front of the subarcuate fossa and of the emergence of the petrosal nerves, injury to the dura being thus minimized. Cerebral displacement is allowed for by removal of squama.

In the intrapetrosal approach, the drainage of the apex is through the temporal bone. The technics generally followed are those of Ramadier and Almour. They are the operations of choice in acute apicitis, because they afford a thorough exposure of all avenues of infection to various points of the perilabyrinth. They afford drainage at the most dependent point of the apex. Surgical approach should be conducted in an orderly manner.

Exploration should be made of the cells around the labyrinth and toward the zygoma, and extension of the search, should the disease not be located, can proceed by means of a radical exposure of the field and by directing attention to the peritubal and carotid cells.

By the Ramadier technic, a tympanomastoidectomy is first performed; then the external aspect of the perilabyrinth is searched for a possible fistula. If none is found, the carotid canal is next opened, or the anterior tympanic wall directly below the orifice of the eustachian tube. In conditions in which there is a collection of peripetrosal pus, the Lempert operation is even more thorough. After a mastoidotympanectomy has been performed, and an exposure and inspection of the basal labyrinthine portion of the petrous pyramid has been accomplished, Lempert creates a surgical intrapetrosal approach to the apical carotid portion of the petrous pyramid, and exenterates the entire bony cellular structure of this area by exploration of the apical course of the internal carotid artery.

It is generally agreed that there is no one surgical approach in petrositis that will adequately meet all surgical requirements. The optimal time for surgical intervention is often difficult to determine. Individual surgical judgment will always be the determining factor in this disease.

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# VII

# Intracranial Labyrinthogenic Complications

By *Joseph Fischer* and *Louis E. Wolfson*

## 1. Viewpoints and Classification

The otogenic intracranial complications have at all time represented a problem of greatest interest to clinicians. It would take volumes to mention all the numerous authors of the various countries who have made contributions toward clarifying this problem. In the United States the following authors will be noted: Blake, Cushing, Dandy, Dean, Dench, Eagleton, Frazier, Fisher, Friesner, Grant, King, Kopetzky, Macewen, Mosher, Lillie, Richards, Tobey, etc. Details of their work are dealt with in relevant chapters.

Intracranial complications may follow acute as well as chronic otitis media. It is not known why in some cases complications develop early, while in others, after many years of suppuration, they do not occur at all. It is natural for the layman to believe that complications are due to faulty treatment, whereas the physician tends to stress the patient's failure to follow instructions properly or to come for early treatment.

There are a great number of cases in which the patient was seen on the first day of the otitis media and in which a proper treatment was given, and still the complication has occurred. However, it must be admitted that neglecting the disease may play an important role, as can be seen from the relatively high percentage of intracranial complications among the cases of the Alexander clinic in Vienna. These patients usually came from remote small places in the country, e.g., from Poland. When such a patient finally decided to undertake the long trip to Vienna, an intracranial complication was either in the making or already well developed.

As far as the physician's fault is concerned, it is generally believed that surgical procedures instituted either too late or too soon were responsible for the development of complications. Some physicians do only a paracentesis of the ear drum even in the fourth or fifth week of an otitis media, when symptoms of a mastoiditis are already present. On the other hand, some otologists advocate a mastoid operation in the first week of an otitis, even in the first days if the course does not run satisfactorily. Neither standpoint seems justified. There is an ardent controversy over the indication for such "premature operation." Alexander has again

and again warned of premature surgical intervention, which, in his belief, favors the development of intracranial complications. Upon his suggestion J. Fischer delivered an address before the otologic meeting in Hamburg in 1926 on the subject of early operation in cases of "mastoidism." This term was applied to cases of acute otitis media in which mastoid symptoms (except drooping of the canal wall) occurred in association with fever, particularly high in children, in the first week of the otitis. The underlying pathology consists of an acute diffuse hyperemia of the mucous lining of the mastoid cells with a serohemorrhagic content. The development of small circumscribed inflammatory infiltrations in the very beginning of the otitis gives rise to brief exacerbations of the clinical symptoms, i.e., fever, edema of the tip of the mastoid, tenderness or pressure, etc. All these symptoms gradually subside in the second week of the otitis when treated conservatively. If, however, such a mastoidism is operated upon in the first week of the disease, the operative trauma may cause an increase in intensity of the short flare-ups, thus eliciting a number of threatening clinical symptoms. This in turn causes the operator to open the mastoid again and to perform more surgery either on the sinus or the dura. In the further course, real intracranial symptoms will develop. This finally leads to the death of the patient. In recent times Goodyear stressed the same points and warned of too early operation of the mastoid.

The etiology of the otitic intracranial complications is still unknown. Some authors attribute the main role to the virulence of the bacteria. However, thorough studies of the bacteriology of the otitic complications have not proved this theory satisfactorily. Other authors explain the development of complications on the basis of anatomic factors, such as various types of pneumatization, presence of dehiscences in the bony walls, or abnormal course of blood vessels between middle ear and cranial fossae, etc. There are others who make the trauma, especially the operative trauma, responsible. Although none of the various theories is proved as yet, the experienced otologist knows that the retention of pus due to an insufficient drainage plays a certain role in the development of intracranial complications. Hence, all our treatment, the conservative as well as the operative, must be aimed to avoid such retention.

With respect to the classification of the complications, Alexander differentiates three forms:

1. Complications of the first order: These follow an ordinary acute or chronic otitis media. There is neither an acute exacerbation nor a mastoiditis (extradural abscess of middle fossa, meningism, temporal lobe abscess).
2. Complications of the second order: These follow the surgical type of otitis media (extradural abscess of posterior fossa, sinus thrombosis, meningitis).

3. Complications of the third order: These follow either an internal otitis or an extra- or intradural suppuration (empyema of sac, internal pachymeningitis, secondary meningitis, cerebellar abscess).

From a practical point of view the best classification is the following:

1. Extradural lesions (pachymeningitis externa, extradural abscess);
2. Intradural lesions (empyema of endolymphatic sac, sinus thrombosis);
3. Subdural lesions (pachymeningitis interna, leptomeningitis, brain abscess).

The pathways of infection will be discussed in connection with the various forms of complications.

Friesner and Braun state that middle-ear infections, traveling along the natural openings, lead to a diffuse suppurative process (meningitis), while those that do not follow preformed paths usually cause circumscribed accumulation of pus (brain abscess, subdural abscess).

The local diagnosis of an intracranial complication may sometimes be very difficult, occasionally not possible at all. However, the experienced clinician will recognize, in such cases, at least the probable existence of an otitic complication. This, in turn, is a direct indication for a surgical measure in the mastoid. In a number of cases the operation may reveal the exact localization of the complication. The inexperienced clinician tends to find different, nonotogenic explanations for the various symptoms. Alexander used to begin his lectures on intracranial complications with the statement that it is the duty of the otologist to acknowledge every intracranial symptom that occurs in the course of an otitis as one of otitic origin. The great danger for the patient's life arises when the otologist allows these symptoms to be "taken out of his hands" by other clinicians. For illustration of his statement he used the following example.

A child with an otitis media and intracranial symptoms such as fever, headaches, malaise, loss of appetite, insomnia, etc., was examined by an otologist who proposed surgical intervention. The parents, however, in their anxiety, insisted upon further consultations. A number of physicians (pediatrician, internist, neurologist, etc.) upon examination of the child offered varied diagnoses, e.g., central pneumonia, influenza, incubation of childhood disease, etc. As time passed and the intracranial symptoms became more marked, the consulting clinicians withdrew. The otologist stood alone. The local diagnosis was now obvious but the operative risk was tremendously increased.

## 2. Extradural Lesions (Pachymeningitis Externa)

### Etiology and Pathologic Anatomy

External pachymeningitis may follow acute as well as chronic otitis media. If there is an extensive collection of pus between dura and bone,

it is spoken of as an extradural abscess. With respect to the localization of the lesion, four groups must be distinguished: the lateral (superficial) abscesses of the middle cranial fossa, the medial (deep) abscesses of the middle fossa, the lateral (superficial) lesions of the posterior fossa, and the medial (deep) abscesses of the posterior fossa.

This book deals chiefly with the lesions in the *posterior* fossa. The superficial abscesses are in the region of the sigmoid sinus, while the deep ones lie medial to it. The former usually follow acute otitis media, while the latter are more often seen with chronic otitis media. The pathways of infection from the middle ear to the posterior fossa are the medial wall of the mastoid, the lateral sinus, the inner ear, the perilabyrinthine cell system, and the blood channels. According to the route of infection, the clinical course will vary greatly. In cases in which there is a fistula through the medial wall of the mastoid, the extradural abscess may be uncomplicated. Abscesses developed on the route of the infection through the sinus are associated with symptoms of a sinus thrombosis. If the infection spreads to the inner ear, the labyrinthine symptoms govern the clinical picture. An infection along the pneumatic cells around the inner ear may show no symptoms until after a period of time, when the labyrinth becomes secondarily involved. The hematogenous way of infection is rather rare. Kopetzky found it in cases of hemorrhagic mastoiditis.

From a pathologic-anatomic point of view, three types of pachymeningitis externa may be distinguished:

1. The *granulating form* (fungus), characterized by production of granulation tissue on the outer surface of the dura (FIG. 55). This is considered as a process of protection against further spread of the infection (Jansen, Eagleton). In some cases the formation of granulation tissue may assume enormous proportions. One of us (Fischer) has published a case in which an excessively large granulation tumor developed from the dura.
2. The *exudative form*, in which extensive collection of pus may lead to a separation of the dura from the underlying bone. According to Goerke, the extradural abscesses are mostly found in the region of the lateral sinus, because of the loose contact between dura and bone in this area. More medially, toward the tip of the pyramid, the dura becomes more adherent.
3. The *necrotic form*, characterized by the green-yellow color of the diseased dura, can usually be noticed macroscopically. There is a sharp demarcation line around the diseased parts, and there is often a fistula from the bone to the dura (FIG. 54).

The further course of an extradural abscess can vary. There may be either a resorption of the pus, with the result of a spontaneous healing (rather rare), or a rupture outward into the spaces of the petrous bone.

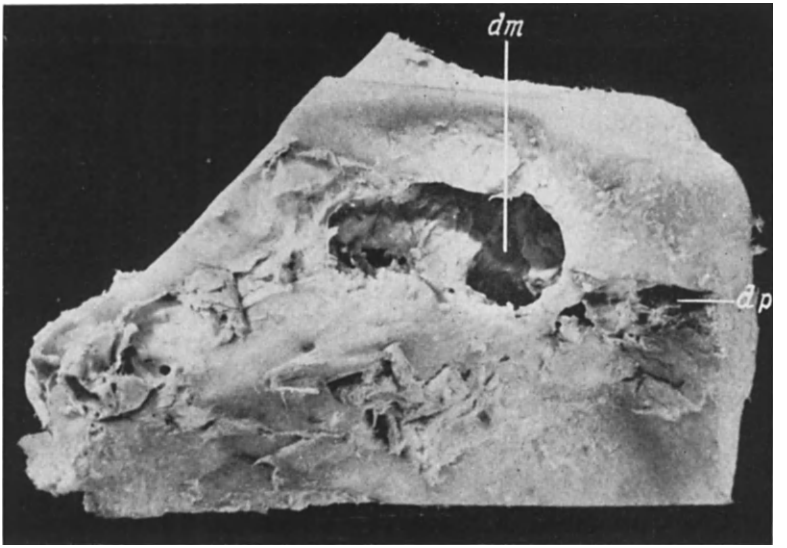


FIG. 54. PHOTOGRAPH OF PETROUS BONE, SHOWING LARGE DESTRUCTION IN MIDDLE CRANIAL FOSSA (*dm*) AND IN POSTERIOR FOSSA (*dp*)

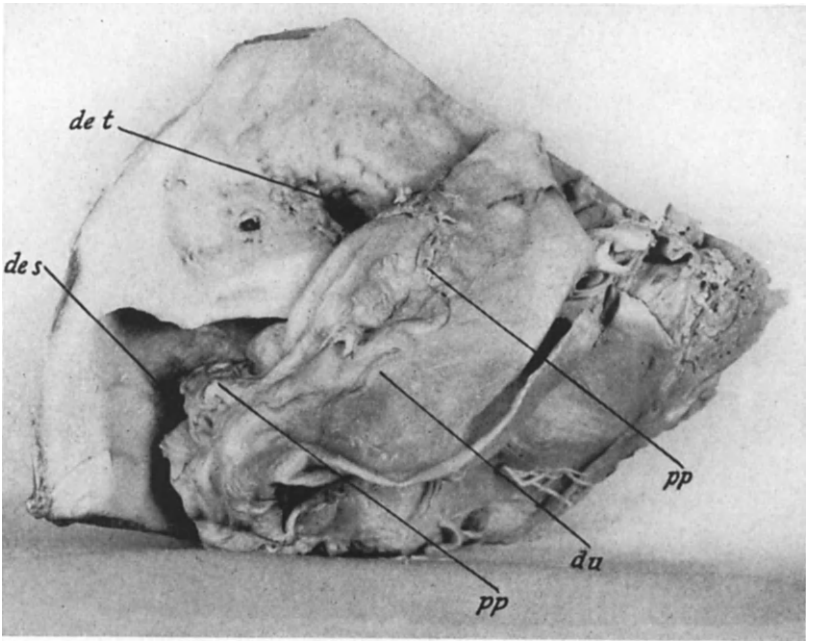


FIG. 55. PHOTOGRAPH OF PETROUS BONE OF CASE OF EXTERNAL PACHYMENINGITIS OF MIDDLE AND POSTERIOR CRANIAL FOSSA

Dura (*du*) separated from underlying bone and reflected. Corresponding with defects in bone of tegmen tympani (*de t*) and of sinus region (*de s*), dura shows pachymeningitic patches (*pp*).

A further rupture to the outside of the mastoid may lead to abscesses in the soft tissue surrounding the ear or the neck (retropharyngeal, parapharyngeal abscess) (FIG. 56). Another possibility is the rupture of an extradural abscess inward toward the leptomeninges and the brain.

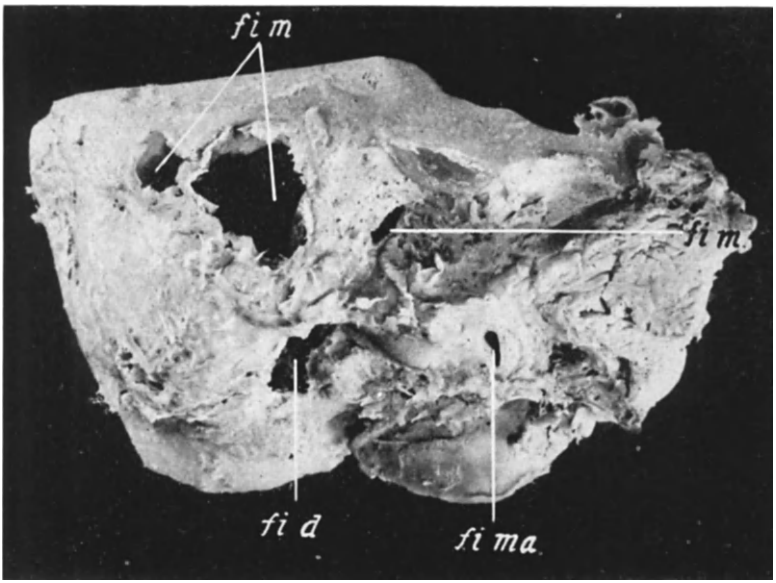


FIG. 56. PHOTOGRAPH OF PETROUS BONE, SHOWING MULTIPLE FISTULAE

*fi m* = fistula in mastoid; *fi d* = fistula in digastric groove; *fi ma* = fistula in mandibular fossa.

### SYMPTOMS AND DIAGNOSIS

It is obvious that the clinical picture will depend largely upon the pathways of infection, upon the location and size of the extradural abscess, and upon the simultaneous presence or absence of other intracranial complications. In cases of medial or deep abscesses of the posterior fossa, symptoms of an internal otitis or of local brain pressure may prevail, while the lateral or superficial abscesses may show the symptoms of a sinus thrombosis. The diagnosis of an extradural abscess is difficult, because an uncomplicated lesion may run a symptomless course, while in the other forms the symptoms of the complication predominate. However, the experienced otologist will often find some signs and symptoms that do not fit into the picture of a common mastoiditis but suggest rather an extradural complication in the posterior fossa. Such signs and symptoms are as follows:

*Secretion.* An abundant discharge from the ear, particularly in at-

tacks, suggests an extradural abscess. In such cases the secretion may first be very profuse, then gradually subside or stop entirely for several hours, and suddenly again become very abundant. Goerke considers the diagnosis of an extradural abscess as very probable when, with the increase of the discharge, a decrease of the cerebral symptoms occurs.

*Pulsation.* In some cases of acute otitis media there is a pulsation of pus. If after the operation the pulsation subsides, but recurs after a period of time, this is evidence of an extradural abscess (Alexander, Marx). A number of authors do not agree with these conclusions. Although the question is still in dispute, one thing is definite, namely, a pulsation in the *early stage* of an acute otitis media is *not significant* of an extradural abscess.

*Headaches.* Unilateral headaches, particularly intense during the night, are—according to Goerke—characteristic of extradural abscesses. Brunner gives an exact localization of the headaches for the various forms of pachymeningitis externa: in cases of superficial (lateral) abscess of the posterior fossa, the patients refer to headaches in the occipital or frontal region; in cases of deep (medial) abscess of the posterior fossa, the headaches occur either in the regions just named or in the neck; in cases of superficial (lateral) lesion of the middle fossa, there are complaints of headaches in the temporal area; and in cases of deep (medial) abscess of the middle fossa, the patients refer to pain in the depth of the orbits. Although for a number of cases this may be correct, it should be borne in mind that the clinical significance does not lie in the various locations of the headaches, but in the fact that a *localized* headache exists. However, this symptom plays an important part in all intracranial complications.

*Pulsating pain.* Some patients complain of throbbing pain synchronous with the rhythm of the pulse. The sudden interruption of such a spontaneous pain indicates a rupture of an extradural abscess into the cranial fossa (Scheibe).

*Tenderness on pressure.* In cases of extradural abscess of the posterior fossa there may be a tenderness not on the mastoid but approximately one inch behind the mastoid.

*Impediment to motion of the head.* An extensive collection of pus in the posterior cranial fossa may interfere with the active and passive movements of the head. As a result the head is usually tilted toward the nonaffected side.

*Cerebral symptoms.* The deep (medial) abscesses of the posterior fossa may show brain symptoms such as fatigue, drowsiness or restlessness, bradycardia, vomiting, nystagmus, elevated temperature, change in the eyeground, etc. However, it must be borne in mind that all these symptoms do not belong to the clinical picture of an uncomplicated extradural

abscess. They point rather to an involvement of the leptomeninges or the brain. Koerner found these symptoms more frequent in children than in adults.

*X-ray findings.* X-ray examinations may occasionally render helpful aid, particularly in cases with extensive destruction of the bone.

#### TREATMENT

When the diagnosis of an extradural abscess has been made, the treatment can be only surgical, in spite of some reports of spontaneous healing. The operation must expose the dura to such an extent that the pachymeningitic area is surrounded by sound tissue.

It has been shown above that the diagnosis of an uncomplicated extradural abscess is often very difficult. In such doubtful cases the experienced otologist will at least recognize the surgical character of the otitis media and will therefore perform an operation on the mastoid. In a great number of cases he will then arrive at the right diagnosis, guided by the diseased bone, by granulations, or by a fistula.

The further course of the disease is usually satisfactory. A complete healing is obtained when the primary infection in the mastoid has been eliminated, when the extradural abscess has been emptied, and conditions for a good drainage to the outside have been established. If, however, after the operation the cerebral symptoms persist or become more marked, it can be assumed that the extradural abscess was not the only intracranial complication. It appears certain that either the leptomeninges or the brain is also involved.

The application of chemotherapy is discussed in chapter viii.

### 3. Intradural Lesions

#### EMPYEMA OF THE ENDOLYMPHATIC SAC

The endolymphatic duct ends in the form of a blind sac on the posterior surface of the pyramid, halfway between the internal auditory meatus and the lateral sinus. The sac is imbedded in a duplication of the dura. A suppuration between the two layers of the dura is called an empyema of the endolymphatic sac. The infection usually spreads from the middle ear to the inner ear and via the vestibular aqueduct into the endolymphatic sac. Some authors have described an opposite way of infection—for instance, a cerebellar abscess or a sinus thrombosis that caused secondarily an empyema of the sac.

The disease has more scientific interest than practical importance. On the one hand, an exact diagnosis cannot be made on the basis of the clinical examination; on the other hand, the empyema of the endolymphatic sac

is almost never the only intracranial complication. It is usually combined with symptoms of an extradural abscess, a cerebellar abscess, or a sinus thrombosis. It has been suggested by Goerke that in cases with vague intracranial symptoms of the posterior fossa, an empyema of the endolymphatic sac may be present.

### SINUS THROMBOSIS

*Etiology and pathways.* The inner ear plays a minor role in the etiology of sinus thrombosis. The condition usually develops directly from diseases of the mastoid. We therefore shall not discuss it in extenso.

Thrombosis of the lateral sinus follows acute otitis media as well as chronic otitis approximately in the same incidence. The infection is usually monobacteriogenic and is caused particularly by hemolytic streptococcus, while pneumococcus is found rather seldom.

The pathways of infection are the following:

Direct continuity from the mastoid (75 to 82 per cent). The suppuration in the mastoid usually causes a destruction of the bony sinus wall, with perisinuous lesions that in turn lead to affection of the inner wall of the vessel (sinus phlebitis) with consecutive formation of thrombi (mural or obturating); finally the thrombi become infected. The most common site of thrombosis of the lateral sinus is the upper knee (FIG. 57). Other places are the lower knee or the jugular bulb FIG. 58).

Primary thrombosis of the jugular bulb, in which the infection spreads through the floor of the hypotympanum.

Labyrinthogenous thrombosis, which is a rather rare occurrence. The infection may spread through the saccus endolymphaticus or through fistulae in the labyrinth.

Perilabyrinthogenous thrombosis by way of the numerous cell spaces around the labyrinth.

Metastatic thrombosis by way of the veins of the mastoid (osteothrombotic phlebitis, Koerner).

Kopetzky distinguishes between two types of infection: (*a*) infection by contiguity of tissue, which he calls extravenous extension; (*b*) infection by extension of small thrombi into the larger vein, which he calls intravenous extension. While the former kind is usually observed with the coalescent type of mastoiditis, the latter is found chiefly with the hemorrhagic type of mastoiditis. In regard to this latter type, Kopetzky states that the pathology is situated within the blood vessels *at the time* of onset of the primary mastoidal disease. This explains why the septic symptoms are observed early in cases of hemorrhagic mastoiditis.

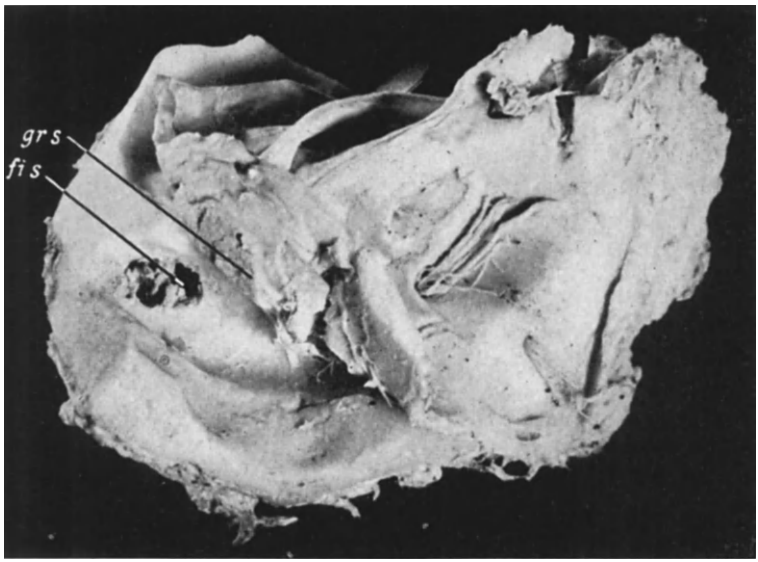


FIG. 57. PHOTOGRAPH OF PETROUS BONE SHOWING FISTULA IN UPPER KNEE OF GROOVE OF LATERAL SINUS (*fi s*). DURA, REFLECTED MEDIALLY, IS COVERED WITH GRANULATION (*gr s*)

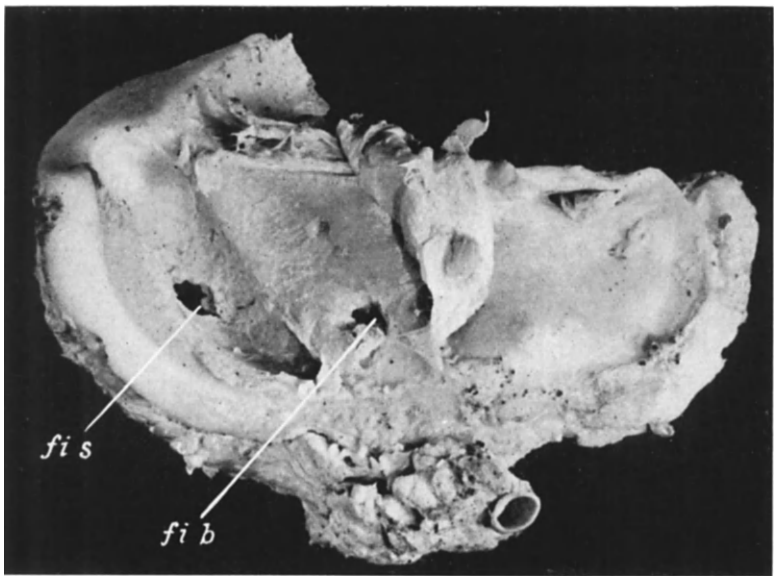


FIG. 58. PHOTOGRAPH OF PETROUS BONE SHOWING TWO FISTULAE
*fi s* = fistula in sinus groove; *fi b* = fistula in sinus bulb.

With regard to the various routes of extension of the infected thrombi along the blood channels, Alexander differentiates four groups:

The cranial group (brain abscess, encephalitis, meningitis);
The thoracal group (lung abscess, mediastinitis);
The abdominal group (infection of the liver, spleen, kidney, bladder);
Metastases of skin, joint, muscles, and bones.

*Symptomatology.* The symptoms and signs of sinus thrombosis are usually characteristic. The temperature is intermittent, with either sudden or slight remissions. In the former cases, the temperature of 104 F. drops suddenly down to normal or subnormal; in the latter there are remissions of from 1 to 2 degrees within a day. A severe chill often initiates the sudden rise of temperature. The number of attacks of chills, however, varies greatly. There are cases where one occurrence of chills is soon followed by another, while in some patients only one severe attack is observed. If in such a case the chills happen to occur during the night, the occurrence easily can be overlooked. In cases where chills fail to be noticed, a profuse sweat may be observed. The authors saw cases in which neither chills nor fever was present, but this is fortunately a rare incident. Headaches are often associated with sinus thrombosis, while complaints of vertigo and nausea are rather rare.

A very significant sign is a sudden change in secretion, particularly in acute cases. Marked decrease or complete cessation of secretion in the ear, associated with a rise in temperature, makes a sinus thrombosis probable. Another symptom is tenderness to pressure, not on the mastoid but about 2 inches behind it.

Changes in the eyeground may occur if the intracranial pressure is increased (Blau, Friesner, Lillie). Lillie observed papilledema following ligature of the jugular vein, which he attributes to interference with the normal circulation of the inferior petrosal sinus. Dean makes a serous meningitis responsible for the eye changes.

Blood examinations present a very valuable diagnostic help. According to Kopetzky, decreasing hemoglobin percentage—by successive readings at regular intervals—indicates an advance of the septic process. A complete surgical eradication of the foci within the mastoid produces a halt of the hemoglobin reduction. Bacteriologic studies of the blood have been made by Brieger, Leutert, Libman, Goldman, and many others. In spite of the great value of positive cultures, one cannot rule out the possibility of a sinus thrombosis if the cultures are negative.

In order to determine which side is involved in a sinus thrombosis, various manometer tests are used:

Tobey-Ayer test: The initial spinal fluid pressure in the lumbar region is first noted. Then compression of the internal jugular vein of the

affected side is instituted and the spinal pressure is again noted. A patient with patent sinus will now show a marked rise in pressure, while a patient with an obstructing thrombus does not show any such rise. However, in one case of double mastoiditis, etc., with sinus thrombosis, Wolfson found the Tobey-Ayer test of no value. The patient's illness began with an acute suppurative otitis media of the right ear, with a similar involvement of the left ear five days later. Both mastoids on X-ray examination showed bilateral destruction of the bone ten days after the onset. A double mastoidectomy was performed. Five days after operation, symptoms of a sinus thrombosis with high temperature were observed. A lumbar puncture with compression of the jugular vein showed only a slight increase (150 mm. to 180 mm.) in the intraspinal pressure, while compression of the left jugular vein showed a marked increase (150 mm. to 250 mm.). The right lateral sinus was widely exposed, and the appearance found to be normal; then the left lateral sinus also was exposed, and a large clot was found and removed.

Crowe-Beck test: The arrangement is the same as in the Tobey test, but the conjunctival vessels are observed. After compression of the patent jugular vein, a filling or engorgement of the vessels can be noticed. In the presence of an occluding thrombus, no changes in the eye vessels occur.

Freedman test: A needle is introduced into the jugular vein and the pressure is estimated. If there is an obstructing thrombus, a drop in the intravenous pressure can be noticed.

Besides the sinus thrombosis described above, there is a *postoperative sinus thrombosis* which represents an entity in itself. As the name implies, the disease follows surgical procedures on the mastoid. Brunner reports 154 cases of sinus thrombosis operated on in the Policlinic in Vienna. In 86.1 per cent of these cases, the correct diagnosis was made before operation, while in 13.9 per cent of the cases the thrombosis was diagnosed after the operation. Brunner stresses the fact that two types of sinus thrombosis following mastoid operation must be sharply distinguished. One type consists of cases in which sinus thrombosis has previously been present but in a latent form, the other type comprises the true postoperative sinus thromboses. He gives the following differential-diagnostic points:

Latent sinus thrombosis is more often found in association with acute otitis media, while postoperative sinus thrombosis follows more usually after chronic otitis media.

In latent sinus thrombosis, the sudden rise in temperature (indicating the complication) occurs immediately after the operation on the mastoid, while in postoperative sinus thrombosis it appears after an interval (usually two weeks).

In latent sinus thrombosis one finds most extensive local thrombi, while in postoperative sinus thrombosis (if operated right after the first rise in temperature) mostly mural thrombi are found. Brunner finds the prognosis in latent sinus thrombosis very good, while the prognosis in postoperative sinus thrombosis is less favorable.

*Treatment.* All otologists agree that the treatment of sinus thrombosis must be surgical, with the aim of eliminating the primary source of infection. There is, however, great diversity of opinion with respect to the other procedures, such as exposure of the lateral sinus, puncture or incision of the sinus, ligation of the jugular vein, etc. There is furthermore a dispute not only about the indication for ligation or resection of the internal jugular vein, but also about the time when this should be done. One group of otologists demands ligation of the jugular vein as soon as the diagnosis of septic thrombosis of the sinus is made, usually before the lateral sinus is opened. The early surgical procedure is supposed to prevent the dissemination of the infection to other parts of the body. This point of view is not shared by a group of authors who strongly oppose the ligation of the vein. A great number of otologists maintain a standpoint in between. They reserve the ligation for certain selected cases. Thus, Haymann gives the indications for ligation as follows: (*a*) in case of a thrombosis of the jugular vein; (*b*) in case of thrombosis of the jugular bulb; (*c*) in case of severe changes of the wall of the sinus, provided that the central part of the thrombus could not be removed during operation; (*d*) in cases of mural thrombosis with failure to eliminate the disease in the local area, or when the signs of severe general infection persist.

Ballenger recommends ligation of the internal jugular vein when the sigmoid sinus is completely blocked by an infected thrombus. He refers to statistics evidencing more favorable results if ligation is done where there is a complete blockage of the sigmoid sinus and worse results when the sinus has a current of blood passing through it.

In the clinic of Alexander, the surgical treatment of sinus thrombosis had a threefold aim: ligation of the jugular vein, elimination of the primary source, and exposure of the lateral sinus. The order of the surgical procedures was dependent upon the diagnosis made either before or during operation. In cases where diagnosis was made before operation, surgery began with ligation and resection of the internal jugular vein, followed by operation on the mastoid (simple antrotomy or radical mastoidectomy). The last step consisted of an extensive exposure of the sigmoid sinus, even though the bony plate did not show any pathology. When ligation of the jugular vein yielded current blood, and when the sinus wall was normal in color and tender on palpation, no further surgery was done. When, however, the sinus showed changes in the current of blood, indicating a thrombosis, the wall was incised and the thrombus removed.

When the diagnosis of sinus thrombosis could not be made before operation, surgery began on the mastoid. As soon as the existence of a sinus thrombosis became evident, the operation on the ear was interrupted and ligation of the jugular vein was performed. Then the operation on the mastoid was resumed and all the necessary surgery was done. The mortality rate was approximately 20 per cent.

Review of the literature shows that although more than sixty years have elapsed since Zaufal recommended ligation of the jugular vein, the problem is still in dispute. The arguments against ligation are based on reports about undesirable complications following ligation and failure to prevent further dissemination of the infection.

Eagleton, Rohrback, and others have described circulatory disturbances following ligation of the jugular vein, such as swelling and edema of the face, headaches, hyperemia, edema of the brain, etc. Meltzer, studying the material on sinus thrombosis of the Massachusetts Eye and Ear Infirmary in Boston, states that the majority of the undesirable complications occur after ligation of the jugular vein. He finds in 40 per cent of cases a secondary infection of the wound in the neck. Metastasis occurs about four times as often after ligation as before it. We have never in our cases seen any serious complication due to the ligation. The argument that ligation does not prevent spread of the infection, particularly in cases of retrograde infection, is partly true. However, one has to bear in mind that ligation blocks the main pathways of circulation, thus at least diminishing a further spread into the body. A preoperative ligation of the jugular vein makes it possible to accomplish all surgical procedures in one stage. It is certainly no advantage for the patient when he has to be operated on a second or a third time, particularly when his general condition is getting worse with the advance of the sepsis. When Harkness in a recent article writes that, with the advance of chemotherapy, the day of primary ligation of the jugular vein prior to any surgery has passed, the statement seems somewhat overenthusiastic.

A number of nonsurgical measures have been recommended as useful in supporting the operative treatment. Kopetzky particularly advocates blood transfusions when the hemoglobin is low (below 55 per cent) or when it shows a gradual reduction in successive counts. Transfusion with whole blood supplies additional antibacterial elements and also restores the red blood cells and the hemoglobin. Chemotherapy (chap. viii) may be of great help. In a recent article Bancroft recommends the drug heparin combined with the sulfa drugs. It should be given intravenously in physiologic solution of sodium chloride over a period of three to five days. Other measures are increased fluid intake, urotropin, Pregl's solution mercurochrome, metaphen, etc.

## 4. Subdural Lesions

### pachymeningitis interna (subdural abscess)

A review of the literature shows great diversity of opinion concerning nomenclature and classification of these lesions. The cause for the controversy lies in the varying underlying pathology. Figure 59 illustrates the normal anatomy of the meninges. It shows the fissure-shaped subdural space confined by two walls. The external wall is formed by the inner layers of the dura, the internal wall by the outer layers of the arachnoidea. Both walls are lined by endothelial cells.

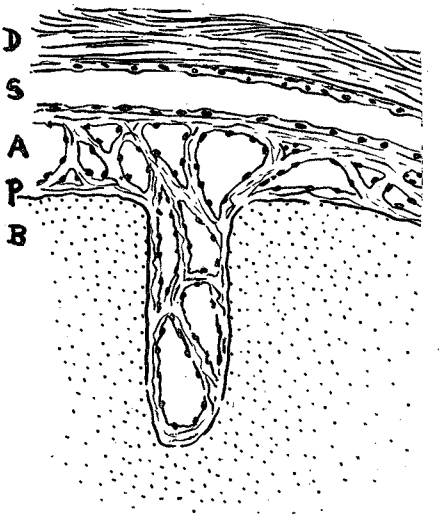


Fig. 59. Diagram Showing Anatomy of Meninges
D = dura mater; S = subdural space; A = arachnoidea; P = pia mater; B = brain.

If the pathology is limited to the inner layer of the dura, the condition is spoken of as a pachymeningitis interna. A suppuration within the space is called a subdural suppuration or a subdural abscess. However, if the arachnoidal wall is greatly involved, the term pachyleptomeningitis should be applied.

The infection usually spreads from the mastoid to the external layer of the dura, then to the internal layer, the subdural space, the leptomeninges, and finally to the brain. Kopetzky considers such an abscess as a form of suppurative meningitis that has been localized by the protective processes of the meninges. It is the result of infection from without inward. However, we have seen cases in which the pathway of infection was the opposite—for instance, a cerebellar abscess or a leptomeningitis may

secondarily lead to an infection of the subdural space. If at the beginning of the inflammation adhesive processes develop, the suppuration may well be walled off, leading to a circumscribed subdural abscess (Macewen, Koerner). However, if such adhesions are lacking, a sudden invasion of pus may extend over large areas in the subdural spaces (Heine).

The disease more often follows a chronic otitis media than an acute one. The most common cause is the acute exacerbation of a cholesteatomatous otitis.

The *diagnosis* of a pachymeningitis interna cannot be made *before operation*, for the reasons that (*a*) circumscribed inflammatory patches on the inner layer of the dura do not cause any clinical symptoms at all; (*b*) subdural suppurations in the posterior cranial fossa are usually accompanied by symptoms of increased endocranial pressure; (*c*) in cases in which the arachnoidea is involved, the signs and symptoms of a leptomeningitis (rigidity of neck, vomiting, convulsions, etc.) will dominate the clinical picture.

Examination of the spinal fluid may occasionally be of diagnostic value. Goerke suggests a subdural lesion when, in the presence of meningitis symptoms, the cerebrospinal fluid either is normal or shows only a slight increase of cells. The same can be assumed in cases in which the fluid first is cloudy but becomes clear, although the clinical signs of meningitis grow more intense. Difficulties of *correct diagnosis* may still exist *during* the mastoid *operation*, when upon exposure of the dura no fistula is revealed and the outer surface of the dura does not show any pathology macroscopically.

The *treatment* consists first of an elimination of the primary source of infection. According to the nature of the underlying otitis media, either a simple antrotomy (acute cases) or a radical mastoidectomy (chronic cases) should be performed. There must further be an extensive exposure of the dura by removal of large portions of bone. In cases with circumscribed lesion on the inner surface of the dura, these surgical measures may be quite sufficient to check further spread of infection and to bring about complete healing. If there is a walled-off subdural abscess with a dural fistula, large incisions in the form of a cross should be made into the dura. Proper drainage to the outside should be established. Alexander advocated excision of the edges of the fistula. These cases do not permit as good a prognosis as the first-mentioned. In those cases in which extensive invasion of pus has taken place within the subdural spaces, with severe changes of the arachnoidea, the prognosis is absolutely grave. All our surgical procedures usually cannot prevent a fatal outcome.

The administration of chemotherapy, which has produced encouraging results in the treatment of meningitis, is discussed in chapter viii.

## LEPTOMENINGITIS

*Classification.* The general picture of meningitis is manifold and confusing. There are forms of meningitis with the most alarming clinical symptoms but nevertheless meager postmortem disclosures. On the other hand, cases with a very mild clinical picture may on autopsy show widespread suppuration on the base or convexity of the brain, with involvement of brain tissue. The same can be said with respect to the clinical course. Some cases of acute otitis media may lead within a few days under fulminating symptoms to a rapid death, while other cases show a protracted course extended over a period of weeks or months. Besides this, there may be forms of meningitis with several flare-ups and remissions, called intermittent meningitis (Brieger). There is finally a great difference in the clinical picture as between cases of meningitis following otitis media and cases in which other intracranial complications occur simultaneously.

This great variety of manifestations can be explained by many factors, such as age of the patient, disposition, anatomic conditions, pathways of infection, virulence of the germs, underlying pathology, trauma, etc. This in turn may explain all the contradictions of opinion among the various otologists with regard to diagnosis, treatment, and prognosis of meningitis.

Many efforts have been made to bring some order into the confusion. In order to break down the complex of symptoms, the otologists have tried hard to separate forms that have the same clinical symptoms and course, and to classify them in certain groups as special entities.

The differentiation between circumscribed and diffuse meningitis is not justified. A case of meningitis may be clinically diagnosed as circumscribed, while the postmortem findings reveal a diffuse lesion, or vice versa. Some authors deny the existence of a circumscribed meningitis and consider all the cured cases as pachyleptomeningitis or as suppuration in the subdural space.

Another classification is based upon the kind of exudation present (serous, hemorrhagic, purulent). Difficulties with this classification arise when differentiation has to be made between a serous meningitis as a forerunner of purulent meningitis or as a disease per se (p. 225).

Alexander classified the various forms of leptomeningitis according to etiologic factors. He differentiates between primary noncomplicated forms and secondary forms. The former are the cases of meningitis in the course of noncomplicated otitis media, the latter the cases arising in the course of otogenic complications. His classification is as follows:

Meningitis with acute otitis media (noncomplicated);
Meningitis with chronic otitis media (noncomplicated);
Meningitis with otitic brain abscess;
Meningitis with sinus thrombosis and extradural abscess;
Meningitis with labyrinthitis.

Similar views were taken by Link, Goercke, Brunner, etc. Kopetzky classifies otitic meningitis into two main types: protective meningitis and dangerous meningitis. The former includes serous meningitis and meningitis sympathica, the latter comprises the suppurative processes within the meninges. Eagleton believes that the leptomeninges act as a barrier to further extension of infection—that they contain a protective mechanism "so that any infection which may be brought to the brain by the circulating blood will be instantly counteracted, digested, and removed."

Finally, there are classifications of meningitis based upon the lumbar puncture test. Fleischman differentiates four groups:

Simple meningism: fluid normal, pressure over 200 mm. of water;
Acute inflammatory hydrocephalus: fluid normal, pressure markedly increased;
Collateral meningitis: increased cells and albumin, decreased sugar and chlorides;
Disseminated purulent meningitis: markedly increased pressure, leucocytes, albumen; markedly decreased sugar and chlorides; presence of bacteria not necessary.

Fleischman himself admits that this classification is not sufficient for clinical purposes.

Dwyer makes the diagnosis of otitic purulent meningitis only if the organism is demonstrated in the spinal fluid by smear or culture or both. He considers a persistent cloudy fluid without organisms as a sign that the infection is still extradural. In his opinion, the pathways of infection are: (*a*) anatomically preformed routes; (*b*) pathologic pathways; (*c*) blood and lymph channels. According to Kopetzky, the infections usually follow the blood vessel route rather than the anatomically preformed channels.

### SEROUS MENINGITIS

Anatomic proof establishing the actual existence of serous meningitis per se has not been given as yet, since the patients usually recover and postmortem corroboration is lacking. It is, however, generally assumed that an edema of the meninges and of the superficial parts of the brain does occur. Efforts to separate serous meningitis from the other forms of leptomeningitis are largely for clinical reasons. It is, however, important to set certain limits for regarding serous meningitis as a clinical entity.

Goercke is right when he states that the clinicians often label varying diseases as one, just because of one common symptom, such as the fluid findings. Alexander excluded all cases of serous meningitis as a forerunner of purulent meningitis, and of concomitant meningitis in cases of brain abscess. He found in serous meningitis per se a normal spinal fluid, though with slightly increased pressure. Severe cerebral symptoms following lumbar puncture point to an early stage of purulent meningitis.

The same holds true for cases showing increase of cells and albumen, which many authors still regard as serous meningitis. High initial temperature is an indication in favor of a diagnosis of early purulent meningitis, even though the spinal fluid at that time does not show much pathology. Changes in the eyeground do not belong to the clinical picture of serous meningitis and must be regarded as indicating an early stage of purulent meningitis. Among the clinical symptoms of serous meningitis, Alexander observed rigidity of the neck, bradycardia, lagophthalmus, loss of consciousness even through several days. Marked changes in the clinical picture are in favor of a diagnosis of serous meningitis, provided that presence of a temporosphenoidal abscess can be ruled out.

Alexander used to say that there is no sense in diagnosing a case as serous meningitis just because of the lumbar puncture findings, and assuming a good prognosis, only perhaps to be forced to change one's mind twenty-four hours later when the cerebrospinal fluid becomes purulent.

The treatment of serous meningitis consists of a quick elimination of retention. Thorough antrotomy with large exposure of the dura should be performed, but the dura should not be incised. As a support to the surgical treatment, chemotherapy should be used (chap. viii).

### PURULENT MENINGITIS

In the discussion concerning the symptomatology of meningitis, relatively more attention is given to meningitis of the *posterior* cranial fossa. The diagnosis of a fully developed meningitis is rather simple, but for the sake of treatment and prognosis the disease should be recognized as early as possible. Following are the various symptoms and signs pointing to meningitis:

General conditions: There is an intense malaise; the patients look severely sick and overtired; there are, further, mental excitement, insomnia, and restlessness. The temperature is elevated, the pulse is often very slow, occasionally rather fast; it is not the pulse rate, however, but its variability (sudden changes) that is significant of the impending complication.

Localized headaches: The patients complain of headaches in the frontal, parietal, or occipital region, or in the depth of the orbita. It is, however, not the site of the headache that is pathognomonic, but the fact that the headache is localized in a certain area. This must be considered as an important early symptom.

Disturbances in sensitivity and sensibility: There are often signs of hyperesthesia and hyperalgesia. Tenderness of the eyeballs represents an early symptom of meningitis. The patient is ordered to shut his eyes; a slight pressure upon the eyeballs that would not

have any effect upon a normal person causes severe pain particularly over the eye of the affected side. There may also be a hypersensitivity to optic (photophobia) and acoustic stimuli.

Vasomotor changes: Dermographism, erythema, flushes, etc., are noted.

Vomiting: Vomiting of labyrinthine origin is always associated with nausea and belongs to the clinical picture of internal otitis during the stage of irritation. Vomiting of meningeal origin is unaccompanied by nausea (projectile vomiting). If, in the course of a purulent internal otitis, vomiting subsides concomitantly with the other manifestations of irritation, and if later on this symptom again occurs, it is suggestive of a complication.

Vertigo: This symptom is found in purulent internal otitis during the stage of irritation and usually subsides with the destruction of the inner-ear function. A recurrence of vertigo in this stage points to a spread of the infection into the posterior fossa.

Spontaneous nystagmus: Great attention must be given to this symptom. In noncomplicated cases of internal otitis, nystagmus of second to third degree is in the direction of the diseased ear during the stage of irritation, and changes its direction toward the sound ear in the stage of destruction of the inner-ear function. If then in the further course of the disease the nystagmus switches back again in the direction of the affected ear, it is obvious that this nystagmus cannot be elicited by the destroyed labyrinth but must be taken to point rather to a lesion in the posterior cranial fossa (Neumann).

Lumbar puncture: Examination of the spinal fluid plays an important role in the diagnosis of early meningitis. It must include physical characteristics and the cytologic, chemical, serologic, and bacteriologic findings. An early stage of meningitis may be revealed by the increase of cells (pleocytosis). There is, however, great dissension with respect to the number of cells necessary to warrant speaking of a pleocytosis. Mygind and Lund consider a finding of more than 2 cells in 1 cu. mm. as a pathologic increase. Knick, Pappenheim, etc., insist on more than 5 cells, while other authors claim that more than 10 or even 20 cells are required to justify the diagnosis of pathologic changes. We consider 8 to 10 cells as the upper physiologic limit. Attention must be given not only to the quantitative but also to the qualitative determination—that is, whether monocytes or polynuclears or erythrocytes are present. According to Dwyer, an increase in lymphocytes points to tuberculosis or syphilis, while a large number of polymorphonuclears indicates either an extradural infection or a purulent meningitis. An increase of erythrocytes may occasionally be seen if a small blood vessel was hit in tapping. In such a case the spinal puncture has to be repeated.

For the bacteriologic examination Kopetzky insists that cultures must be made immediately after tapping and that a large amount of fluid should be used for centrifugation. Eggston reports about the difficulties that may occasionally arise in typing the bacteria. It may sometimes take twelve to twenty-four hours, occasionally a few days. The diagnosis is easier from a Gram stain or from the "hanging drop," but it is less exact. Dwyer observed great variations in staining reaction, particularly with pneumococci. The same organism may at times be gram-positive or gram-negative, depending upon age and the degree of destruction by the infection.

Kopetzky states that in cases with positive bacteriologic findings the meningitis can usually be recognized from the clinical picture. He therefore recommends for the early diagnosis of meningitis a thorough examination of the chemistry of the fluid. His conclusions are:

*Early signs of meningitic reaction* can be seen in the increase of pressure of the spinal fluid associated with a decrease of carbohydrates; the chlorides, carbonates, and the *p*H are normal, while the lactic acid is slightly increased.

*Signs of progressing meningitis* can be seen in lowering of the *p*H, the chlorides, and the carbonates. The bicarbonate content of the spinal fluid is often as much as 33 per cent below that of the blood plasma. There is further an increase of albumin and globulin.

*Changes in the eyeground* may or may not occur. There are sometimes engorged vessels, particularly on the affected side, papilledema, or optic neuritis. These changes are particularly marked in cases with increased endocranial pressure.

*Treatment.* A review of the literature reveals great fluctuation in the methods of treatment of otitic meningitis. Periods of hopelessness have been followed by periods of overenthusiasm. Thus, thirty to thirty-five years ago it was generally accepted that otitic meningitis was not amenable to any surgery. Later on Jansen, Koerner, etc., categorically demanded operative elimination of the primary source of infection. Brieger and Goercke saw only one contra-indication to surgery, namely, a moribund condition of the patient. Alexander went still farther, stating that there is no contra-indication at all, and that he operated even in the terminal stage of a case in an attempt to save the patient's life. In recent times the miraculous results of chemotherapy, as reported in the literature, have aroused such enthusiasm that a number of otologists find no reason for resort to surgery any longer. Such an attitude obviously reflects a high degree of enthusiasm. We agree with Goercke, who expects success in treating otitic meningitis only when the following four aims are fulfilled:

1. *Prevention* of further spread of *infection*;
2. *Destruction* of the invading *bacteria* and their toxins;
3. *Drainage of the products* of inflammation from the subarachnoidal spaces;
4. *Decompression of the brain* to prevent damage to vital parts.

1. *Prevention* of further spread of *infection*: The elimination of the primary source of infection consists of an operation on the mastoid. In acute cases a simple antrotomy should be performed, while in chronic cases a radical mastoidectomy or, if necessary, a labyrinth resection should be done. In any case, the dura must be exposed to a large extent. If there is a deep-seated focus in the pyramid (apicitis petrosa), elimination can be accomplished by various approaches (Voss, Kopetzky and Almour, Eagleton, Lempert, Friesner, etc.) (see p. 233).

In cases of secondary meningitis in the course of other intracranial complications, it is obvious that, besides the operation on the mastoid, surgery on the respective sites is required. In case of thrombosis of the sigmoid sinus, the medial sinus wall should be incised after removal of the occluding thrombus.

2. *Destruction* of the invading *bacteria* and their toxins: There have always been efforts to invent remedies that when introduced into the body would kill the germs. This aim, however, has not been realized as yet. The sulfa drugs, although very effective in the treatment of infections, are not capable of destroying the bacteria. They only decrease the invasiveness of the latter. This in turn is of great help in the battle between bacteria and antibodies. The conclusion that can be drawn, therefore, is that the sulfa treatment has the greatest effect when combined with two other therapeutic factors: one is the elimination of the primary source of infection by surgery, the other consists of a strengthening of the antibodies (immunotherapy). A discussion of chemotherapy will be found in chapter viii, together with all the data concerning technic, dosage, etc.

Immunotherapy has been in use for a long time, but has yielded relatively encouraging results only in recent times. For therapeutic purposes, vaccines as well as sera have been used. The former consisted either of a polyvalent vaccine or of autovaccine. Unfortunately the time was too short in cases of meningitis to prepare the vaccine. The sera used were either antistreptococci or antipneumococci. Immunotherapy is now of great value in supporting chemotherapy. Steele, Gottlieb, and Brann designate the indications for addition of antipneumococcus serum to chemotherapy as follows: (*a*) presence of bacteremia; (*b*) failure of spinal fluid cultures to become sterile twenty-four hours after the start of chemo-

therapy; (*c*) failure of the patient to improve clinically with chemotherapy alone; (*d*) when the patient is under 2 years of age; (*e*) when the patient is over 40 years of age.

3. *Drainage of the products* of inflammation from the subarachnoidal spaces: The method of incision into the dura, with introduction of a drain, has almost as many followers as opponents. Manasse recommends incision only if the dura shows great tension and signs of a disease, or when on puncture of the subdural space pus occurs. Goercke rejects such a therapy. Alexander finds it effective particularly for meningitis of the posterior fossa. Cunning splits the dura with three or four parallel incisions to permit a free flow of spinal fluid. Kopetzky advocates exposure of the visceral inner surface in order to break the contiguity of the skeletal venous blood vessels; he stresses the importance of keeping the cerebrospinal fluid circulating. Cerebrospinal lavage with Ringer-Locke solution is recommended by Knick, Eagleton, Borries, etc. Continuous spinal drainage by laminectomy has been performed by Paget, Friedrich, Wickart, etc. A number of authors advocate drainage of the various cisterns. Holmgren suggests drainage of the cisterna pontis. Goercke reports about a number of cases with good results in which he drained the cisterna pontis lateralis. Haynes and Kopetzky have opened the skull with a trephine at a point 1 inch behind the posterior margin of the foramen magnum, draining the cisterna magna. Similar steps have been taken by Day, Dandy, etc. Eagleton finds the greatest amount of inflammatory exudate collected at the base of the brain in the cisterna and recommends drainage of this area.

4. *Decompression of the brain* to prevent damage to vital parts: The inflammatory increase in volume of the structures inclosed in the capsule of the skull may be due to various causes, such as active hyperemia, disturbances of circulation of the fluid, increased permeability of the capillaries, increase of intracellular content, etc. To prevent damage to vital parts of the brain, the increased pressure must be relieved. There are numerous methods, as follows:

*Lumbar puncture* affords only a temporary relief of the intraspinal pressure. Some otologists repeat the procedure every other day, while others apply it at eight-hour intervals. It is safer to puncture frequently, withdrawing small amounts of fluid at a time. Brunner withdraws 30 cc. of spinal fluid and compensates with an injection of 20 to 25 cc. of air, in order to prevent adhesions within the leptomeninges. Other authors prefer the ventricle puncture. In regard to puncture of the various cisterns, see the discussion of drainage above. For reduction of the intracranial pressure, Eagleton recommends ligation of the carotid artery on the affected side. Another method of relieving the pressure consists

of forced drainage of the spinal fluid. Kubie combines continuous drainage of the spinal fluid with an intravenous administration of hypotonic solution of sodium chloride (2,000 cc. intravenously every day). Neal, Jackson, and Appelbaum do not recommend establishment of permanent drainage. The radical methods, such as laminectomy, trephining of the cisterna magna, or other forms of forced drainage, have no advantage over repeated lumbar punctures.

*Blood transfusions* not only should be given to increase the action of the antibodies but are, according to Kopetzky, of great value for keeping the spinal fluid circulating. By influencing the chemistry of the fluid, the tendency to blocking of the fluid is much decreased.

Among the nonsurgical measures for relieving the increased pressure are dehydration, low salt intake, hypertonic solutions, venesection, etc.

*Prognosis.* Otitic purulent leptomeningitis has always had the highest mortality rate among all intracranial complications. In recent years, since the introduction of the sulfa drugs, more and more cases with complete healing are described in the literature. There is no doubt that strikingly good results must be attributed to the sulfa drugs. One must, however, bear in mind that progress has also been made in the diagnosis of early stages of meningitis and, further, in surgical elimination of the primary source of infection. This is particularly true as regards cases in which there is a deep-seated focus in the pyramid. While earlier statistics show a mortality rate of more than 95 per cent, recent figures range between 20 and 60 per cent. The reasons for such marked differences among recent reports in the literature lie in the manifold picture of meningitis. It has been stated above that the course of an otitic leptomeningitis is dependent upon numerous factors, such as etiology, pathways of infection, anatomic conditions, underlying pathology, virulence of the bacteria, age and constitution of the patient, time of surgical intervention, etc. In a recent paper, Brunner directs attention to this fact. He shows that meningitis in young people affords a much better prognosis than in older persons; he has further found that meningitis en route to the posterior fossa (mostly chronic otitis media) has a much milder course than meningitis en route to the middle fossa or with multiple routes (mostly acute otitis media). Another factor responsible for the clinical course concerns the degree of pathologic change in the cerebral cortex: the more intense the encephalitic changes, the graver the prognosis. The important role of the virulence is obvious.

Taking all these facts into consideration, we come to the conclusion that a uniform treatment of otitic purulent meningitis cannot be expected. It is necessary instead to handle each case individually. Here, however, arise the difficulties for the clinician; to overcome them, the otologist

must know well his anatomy, pathology, and bacteriology, and must be familiar with all the surgical procedures. It is obvious that many years of clinical experience and a certain amount of diagnostic intuition are necessary for such difficult tasks.

## 5. Cerebellar Abscess

### GENERAL CONSIDERATIONS

A study of cerebellar abscesses of otitic origin reveals the fact that there is an amazing diversity of opinion concerning not only symtomatology and diagnosis but also treatment and prognosis. One difficulty arises from the mistake, usually made in the various textbooks and handbooks, of discussing the cerebellar abscess together with all other brain abscesses. Alexander has repeatedly stressed this point. In his classification of intracranial complications, he distinguishes between the temporal lobe abscess as a complication of the first order, and the cerebellar abscess as a complication of the third order (p. 239). It is, therefore, wrong to discuss the cerebellar abscess first within the category of all brain abscesses and to differentiate it only later when symptomatology and diagnosis are discussed.

A thorough study of the underlying pathology is absolutely necessary. It is obvious that a cerebellar abscess developed along the pathways through the labyrinth will show symptoms that are entirely different from those of an abscess following a sinus thrombosis or of an extradural abscess. Pathologic anatomy helps in understanding the clinical course, the success or failure of our treatment, etc. It is further important to know the location of the abscess—whether it is a superficial (cortical) or a deep abscess, whether it has a dorsal or a ventral site. Finally, the clinical course and prognosis depend upon whether the abscess is acute or chronic and whether destructive processes (progressive encephalitis) or fibrovascular processes (capsule formation) prevail.

### ETIOLOGY

The cerebellar abscess follows usually a chronic otitis media or an acute exacerbation, but less frequently an acute otitis media. The statistics of the literature cannot be used as a proof, since they cover all the brain abscesses. As far as the relation between cerebellar abscess and temporal lobe abscess is concerned, the ratio seems to be 1 to 3. With regard to the age of the patients, statistics show that cerebellar abscesses are most frequent in the age period between the second and third decade of life, which is usually true of all intracranial complications. Furthermore, there is a prevalence of incidence in male over female; figures dealing with the location of the abscess (right or left side) are so contradictory that no conclusion should be drawn.

It can be said in general that in chronic cases the cerebellar abscess follows the route through the labyrinth, while in acute cases the abscess develops from a sinus thrombosis or from an extradural abscess of the posterior fossa.

A different view is taken by Turner and Fraser, who found cerebellar abscesses as a complication of suppurative labyrinthitis resulting from chronic otitis media in only 3 cases out of 26. Atkinson also described infection spreading via the labyrinth in only 3 cases out of 14. In chronic cases the cholesteatoma plays the main role (Neumann).

### PATHS OF INFECTION

In order to understand the pathways of infection, we should distinguish between the routes from the middle ear to the dura and the routes from the dura to the brain. In addition there is a metastatic infection occurring usually as part of a general pyemia. Table 2 analyzes the various routes of infection. FIGURE 60 illustrates a large destruction of the pyramid by an abscess with routes of infection through the labyrinth and the lateral sinus. FIGURE 61 shows a cholesteatoma in which the infection spread via the labyrinth, cochlea, and internal auditory meatus into the cerebellum.

### PATHOLOGIC ANATOMY

Otogenic brain abscesses usually develop in the medullary substance just beneath the cortex by way of retrograde thrombosis of pial vessels. Friedmann studied their development experimentally. After injection of cocci into the vessels he noticed small infarcts and accumulation of polynuclear leucocytes leading to extensive edema and malacia of brain tissue (circumscribed purulent encephalitis). By confluence of the numerous disseminated spots the brain abscess is formed.

Fremel, who studied the morphology and development of cerebellar abscesses, found in the very beginning a diffuse inflammation in the superficial parts of the medullary substance, but no tendency to involve the deep portions of the white matter. This in turn results in formation of very flat but extensive abscesses with fissure-shaped lumens. Since the extension of the abscesses is along the surface, large parts of the hemisphere are usually involved.

The fact that the white matter is first to become diseased can best be explained by the difference in blood supply of the cortex and the medullary substance. That of the former originates in the pial vessels, while the latter is supplied by end arteries (Fremel, Hoffmann). The zone underneath the cortex is particularly poor in blood supply.

TABLE 2.—*Pathways of Infection*

| A. Middle Ear ⟶ Dura | B. Dura ⟶ Brain | C. Metastatic |
|---|---|---|
| 1. Translabyrinthine<br>tympanic cavity ⟶ oval window ⟶ internal meatus<br>tympanic cavity ⟶ round window ⟶ endolymphatic duct*<br>tympanic cavity ⟶ promontory ⟶ bony fistula<br>tympanic cavity ⟶ horizontal canal ⟶ bony fistula | 1. Direct continuation<br>dura ⟶ leptomeninges ⟶ cortex<br><br>2. Indirect route<br>dura ⟶ pial vessels ⟶ white matter of brain | Hematogenic spread without any topographic relation between source of infection and brain abscess: usually part of a general pyemia |
| 2. Perilabyrinthine<br>attic ⟶ posterosuperior cell tract ⟶ tip of pyramid<br>mastoid ⟶ infralabyrinthine cell tract ⟶ tip of pyramid<br>antrum ⟶ subarcuate tract ⟶ posterior cranial fossa | | |
| 3. Transfacial<br>fallopian canal ⟶ internal meatus ⟶ posterior cranial fossa | | |
| 4. Through sinus or mastoid<br>mastoid ⟶ lateral sinus ⟶ posterior fossa<br>mastoid ⟶ Troutman triangle ⟶ posterior fossa | | |

* Infection via the cochlear aqueduct leads usually to diffuse purulent meningitis.

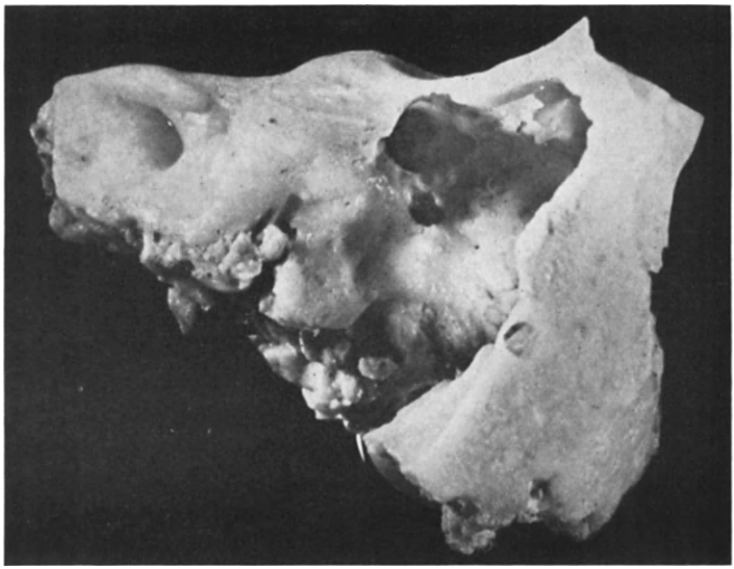

FIG. 60. PHOTOGRAPH OF PETROUS BONE OF CASE OF CHOLESTEATOMA
Destruction of lateral half of petrous pyramid and fistula in labyrinth.

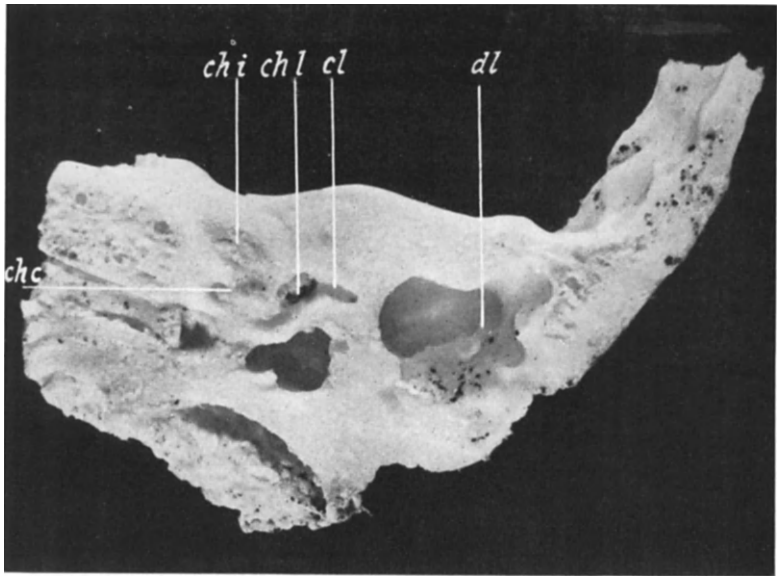


FIG. 61. PHOTOGRAPH OF PETROUS BONE OF CASE OF CEREBELLAR ABSCESS
*dl* = destruction in lateral part of petrous bone; *ch l* = cholesteatoma in labyrinth; *cl* = lateral semicircular canal; *ch c* = cholesteatoma in cochlea; *ch i* = cholesteatoma in internal auditory meatus.

In a pathologic-anatomic respect, the cerebellar abscesses may be differentiated, according to Brunner, into three groups:

1. *Flat fissure-shaped abscess* (Fremel). This is the most common abscess. The inflammatory process takes place in the superficial layers of the medullary portion of the brain, just below the stratum granulosum of the cortex. The main portion of the white matter is displaced, but not destroyed, and the nuclei are not involved. The inflammation has a tendency to spread and to penetrate the cerebellar sulci, thus forming

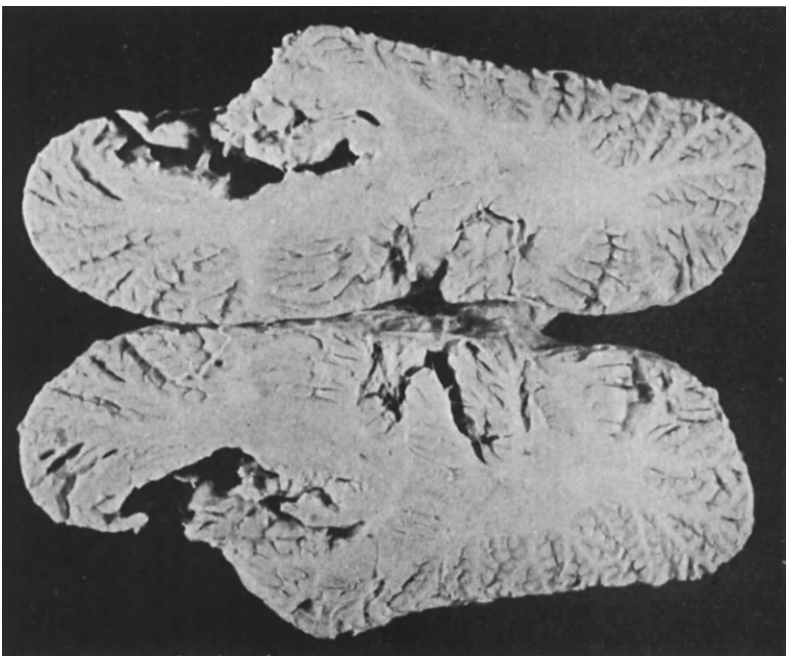

FIG. 62. FISSURE-SHAPED FLAT CEREBELLAR ABSCESS WITH TENDENCY TO EXTEND MEDIALLY

pockets and recesses of the abscess. These flat fissure-shaped abscesses may become very large, extending from the anterior surface of the hemisphere to the posterior pole of the cerebellum (sagittal direction), and may spread over the vermis to the other side of the hemisphere (frontal direction) (FIG. 62).

2. *Cortical abscess.* This mainly follows a thrombosis of the lateral sinus or a pachymeningitis of the posterior fossa. The infection spreads from the inner wall of the sinus to the attached cortex of the cerebellum; Lillie, however, believes in a metastatic way of spread.

3. *Deep cerebellar abscess.* This is rather rare. The abscess develops by means of thrombosis of the vessels supplying the white matter, thus destroying large portions of brain tissue, including the nuclei (FIG. 63). Microscopic examination of the abscess shows that the pathologic changes involve much more brain tissue than appears macroscopically. Homén and Hoffmann describe four zones of changes in the brain: the innermost or exudative zone shows necrotic brain particles and many leucocytes;

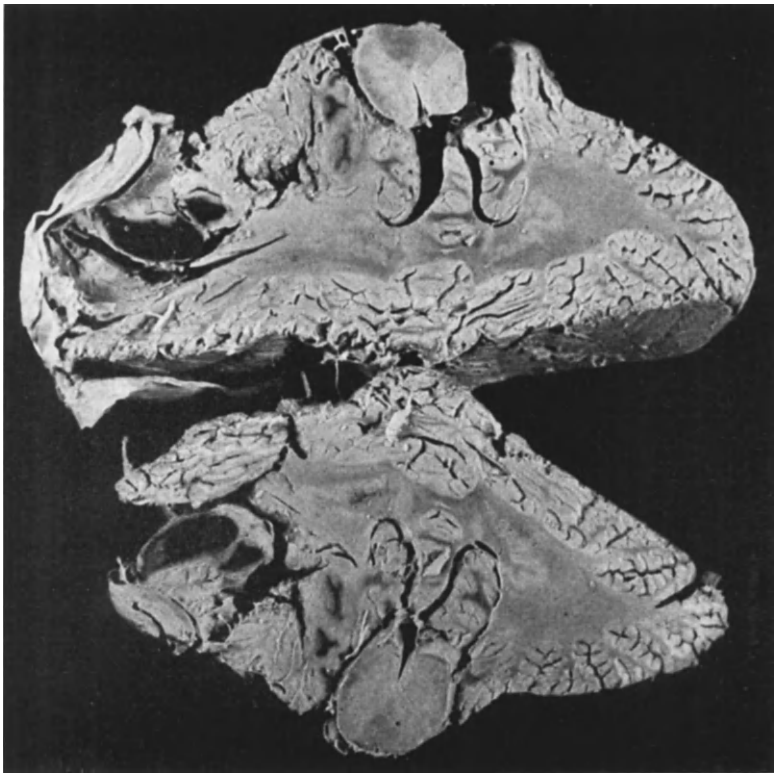

FIG. 63. CEREBELLAR ABSCESS IN VENTRAL PARTS WITH TENDENCY TO EXTEND DORSALLY

then comes the zone of infiltration, then the fibrovascular zone, and finally the zone of edematous infiltration and rarefaction of tissue. Eagleton and Miodowski distinguish three layers, namely, the granulation, the demarcation, and the irritation zones.

Particular stress must be laid upon the inflammatory edema in the adjacent parts of the abscess and upon the hydrocephalus, which often leads to a dilatation of the ventricles. These explain the variety of symptoms, the clinical course, and also the prognosis

The greatest interest, however, has always been given to the pathologic changes connected with the encapsulation of the abscess; and yet these factors are mainly unknown. We cannot explain satisfactorily why in certain cases a pyogenic membrane is sooner or later formed, while in other cases encapsulation does not take place at all. Some authors assume a connection between encapsulation and the bacteriology of the infection. Neumann claims that the diplococci are capsule-forming bacteria; others attribute this potentiality to certain types of fungi. It is generally believed that the presence of anaerobes prevents encapsulation. There is no proof as yet of all these theories. It is further assumed that the processes leading to formation of a capsule originate in the vessels and in the connective tissue. It is a general belief that encapsulation is concomitant with ripening of the abscess, or with a standstill of the infection, or even with healing processes. However, certain observations in the literature do not corroborate such an assumption. Macewen, Brunner, and Schnierer describe cases in which a spread of the infection through an intact capsule took place. There have also been cases in which rupture and fistulation of a relatively thick capsule were observed. It is further believed that the capsule becomes thickened as time elapses. Pathologic studies on the part of Aschoff, Passow, Koerner, etc., did not suffice to prove such an assumption. These authors came to the conclusion that the thickness of a capsule cannot be used as a measurement of the duration of the brain infection. The discussion, therefore, shows that the various factors pertaining to encapsulation are still unknown.

### BACTERIOLOGY

Most of the statistics deal with the bacteriologic findings in brain abscesses in general, while in relation to the cerebellar abscesses only casual examinations are reported. In spite of numerous large-scale examinations, there is no uniformity at all with respect to the bacteriology. Hasslauer found the streptococcus to be the most frequent agent, with the staphylococcus in second place, and the diplococcus in third place. Other authors consider the Staphylococcus aureus as the most frequent agent. Some investigators describe the colibacillus as very common, while others claim that it is particularly rare. A study of the literature shows that almost every type of aerobe as well as anaerobe has been found. Streptococcus haemolyticus seems to be most common.

Neumann attributed to the diplococcus type the tendency to form a pyogenic membrane, while the anaerobe germ prevents such a formation. Ghon opposes this theory, showing that many cases of brain abscess caused by anaerobe bacteria had a definitely pyogenic membrane. According to Lund, the presence of the colibacillus must be considered a bad sign.

Linck and Lund believe that staphylococci and streptococci favor the encapsulation of the abscess, while the presence of gram-negative rods does not allow capsule formation.

### SYMPTOMATOLOGY

The clinical picture is manifold, depending upon a number of factors, such as the underlying pathology, the size and location of the abscess, the type of the accompanying complication, and the stage in which the patient is seen by the examiner.

For clinical reasons, the differentiation of four stages (as used in all other groups of intracranial complications) is advisable:

The *initial* stage represents the time necessary for the infection to pass from the ear to the brain. Its duration can approximately be determined as from one week to two months.

The *latent* stage is very poor in symptoms, as the name implies, and has approximately the same duration as the initial stage.

The *manifest* stage, which shows more clinical symptoms, lasts only a short time, i.e., from a few days to two weeks.

The *terminal* stage, characterized by symptoms of ventricular rupture and meningitis, is very short, usually of a few days' duration.

It must, however, be admitted that such a differentiation, made for demonstration purposes, is arbitrary and often is not actually observable. In certain cases the initial stage may be followed by the manifest stage, which rapidly leads to the terminal stage, or the manifest stage may be followed by a second period of latency.

In order to study the clinical course of the cerebellar abscess we follow the classification of Bergman, who distinguishes these groups of symptoms: (*a*) general symptoms, (*b*) general brain symptoms, and (*c*) local brain symptoms. The latter group can be subdivided into focal and remote symptoms.

*General symptoms.* There is an intense malaise; the patient looks severely sick, the face is pale, the expression tired, the tongue coated; there may be gastric disorders and emaciation. The temperature in the initial stage is often high, occasionally combined with chills; in the latent stage, it is normal or subnormal; while in the terminal stage it rises again, owing to the meningitis.

*General brain symptoms.* The notable changes of this group are as follows:

*Headache* is very significant of cerebellar abscess. It is often the first symptom in the initial stage, becomes less intense during the latent stage, and is very marked in the manifest stage. Since this symptom is caused

by increased brain pressure, it will be found particularly in cases with progressive encephalitis and consecutive edema and hydrocephalus. The location of the headache need not necessarily correspond to the location of the abscess, but is often in the forehead (Neumann).

*Psychic* changes are reported by various authors, suddenly subsiding when the abscess is found and drained.

*Vomiting* is very common and more intense than in connection with other brain abscesses. It is of the intracranial type, i.e., projectile, without nausea or any muscular activity and independent of meals.

The *pulse* is often very slow and resistant (pressure pulse). In the terminal stage it may become more rapid, owing to paresis of the vagus nerve.

The *respiration* is rapid, irregular. In the terminal stage there may be a Cheyne-Stokes type.

*Vertigo* is reported as being very intense and enduring. If the infection spreads through the inner ear, there is dizziness of the character associated with a labyrinthine lesion.

*Rigidity of the neck* is more often observed in connection with cerebellar abscess than with other brain abscesses or with cerebellar tumor.

*Local brain symptoms.* The two subdivisions of this group include:

Focal symptoms: These are found particularly in cases in which the abscess is located in the deep portion of the medullary part of the brain, thus involving the nuclei of the cerebellum. Since the cerebellum is known to be the regulating apparatus for the motions of the body and the tone of the musculature, a disease in this organ will cause disturbances of coordination and tone. Lack of cooperation of the various muscles and groups of muscles on the diseased side will lead to a unilateral ataxia (hemi-ataxia). Such symptoms are: homolateral hemi-ataxia of the trunk and upper and lower extremities, ataxic gait, disturbances in side-stepping (Alexander); past-pointing (Bárány); *asynergie cérébelleuse* (Babinski); adiadokokinesis, adynamia, dysmetria, disturbances of tone, etc. (p. 105).

Remote symptoms: One of the most important symptoms is spontaneous nystagmus. Since the cerebellar substance cannot produce any nystagmus, the presence of the latter must be regarded as a remote brain sign, due to pressure upon the brain stem and the vestibular nuclear region. The nystagmus found in cerebellar abscesses is usually very slow, coarse, horizontal, and in the direction of the diseased side. Occasionally a nystagmus to both sides may be observed, but more markedly in the direction of the diseased side. There is often a sudden change in direction and intensity. In cases where the cerebellar abscess was developed by the route through the labyrinth, the differentiation between a peripheral labyrinthine and a central nystagmus may be very difficult (p. 275).

Another remote symptom is represented by *conjugate deviation* of the eyeballs. The deviation is almost always toward the sound side, and may be associated with a conjugate paresis of the eyeballs in the direction of the affected side. This symptom, which can also be observed in other intracranial complications, is elicited by lesions either of the cortex or of the pons (p. 64). The theory of a cerebellar elicitation as assumed by some authors has not been proved as yet. It appears more probable, however, that these disturbances of the associated eye movements are due to increased local pressure upon the central vestibular area.

*Disturbances of speech* (cerebellar dysarthria) are occasionally observed. Some authors (Stenvers) assume a center of coordination of speech in the cerebellum. Gordyschewski distinguished cerebellar dysarthria from the bulbar and mixed type. The former points to a lesion of both semilunar lobes or of the medial parts of these lobes.

*Compulsory posture of the head*, i.e., flexion of the head and neck in a forward or sideward position, with or without rigidity of the neck, is often observed. There are many explanations for this symptom. According to Alexander, this position of the head is maintained by the patient in order to have the least possible degree of vertigo. Other authors (Oppenheim) believe in a damage of the posterior longitudinal bundle or in a compression of the posterior roots of the cervical nerves.

*Lesions of the basal cranial nerves* may involve the third, fifth, sixth, and seventh nerves. The oculomotor nerve is not as often affected as occurs in connection with the temporal lobe abscess, and is usually only partly diseased. Affection of the trigeminus nerve causes a homolateral hyporeflexia or areflexia of the cornea and neuralgic pain. Abducens nerve palsy with diplopia, strabismus, etc., is very characteristic for cerebellar abscess according to Alexander, while Neumann regards this symptom as very rare. A palsy of the facial nerve is explained by Neumann as caused by increased pressure upon the nerve stem at its entrance into the internal auditory meatus. In such cases the velum palatinum is also involved in contrast to a palsy of the facial nerve at the site peripheral to the geniculate ganglion. Palsy of the contralateral facial nerve must be explained by an accompanying meningitis.

Finally it should be emphasized that, owing to the hydrocephalus associated with cerebellar abscess, symptoms from the cerebrum may be observed.

## DIAGNOSIS

The diagnosis of a cerebellar abscess is often very difficult. This can be seen in the various statistics, which show that the cases of nondiagnosed cerebellar abscesses outnumber those of all other brain abscesses. The

reason for the difficulties lies in the fact that some cases run an almost symptomless course. On the other hand, the cerebellar symptoms may be obscured by the various complications accompanying the abscess. It can best be understood by means of the classification of Alexander (p. 239).

The *temporal lobe* abscess represents a complication of the first order. This in turn means that the brain abscess follows directly a disease of the middle ear, thus showing the cerebral symptoms very clearly.

The *cerebellar abscess* represents a complication of the third order. This means that the brain abscess has developed on the basis of other complications (internal otitis, sinus thrombosis, extradural abscess, etc.) and therefore the symptoms are obscured. However, there will be a number of cases in which the diagnosis can be made at the proper time. In order to achieve this aim, an exact case history and thorough examination from an otologic-ophthalmologic and neurologic standpoint are absolutely necessary.

The *case history* must clear up the following questions. Is there an inflammatory disease of the ear or has such a condition recently been present? Is the ear process on the same side as the brain lesion? Is the otitis media an acute or chronic infection, or an exacerbation of a chronic infection? If a flare-up of a chronic otitis is not found, has any been previously observed? Is there any coincidence between the first symptoms of the cerebellar abscess and the beginning of the acute exacerbation of the chronic otitis media?

The *otologic examination* reveals, in the majority of cases, a chronic otitis (cholesteatoma) with fetid discharge and with a marginal perforation in the upper posterior quadrant, indicating a destructive lesion in the bone (antrum, mastoid), while a perforation in the Shrapnell membrane (attic suppuration) points rather to a temporosphenoidal abscess. In cases of cerebellar abscess following acute otitis media, the symptoms of sinus thrombosis or pachymeningitis of the posterior fossa are dominant.

In chronic processes, however, the infections usually follow the pathways through the labyrinth, leading first to diffuse internal otitis. In such cases the otologic examination must include functional tests of the cochlea and labyrinth.

Eagleton, Fisher, and Jones observed in cerebellar abscesses a reduction of caloric excitability first in the vertical semicircular canals. Irrigation of the various canals may reveal hypo-irritability or complete loss of function of the vertical canals, while stimulation of the horizontal canals yields a normal response.

The *ophthalmologic* examination is chiefly concerned with changes in the eyeground, which may occur as manifestations from slightest engorgement of the blood vessels up to the most marked choked disk. Statistics

on such changes vary to a great extent; the average figures give a frequency of approximately 50 per cent of eyeground changes in cases of cerebellar abscess (Neumann, Lund). Coleman observed choked disk in the same frequency as in connection with brain tumors. Lillie stresses the great clinical importance of papilledema, which in his cases at no time exceeded 2 diopters in degree. He insists on repeated examinations of the eyeground in order to determine the proper moment for surgical interference, and recommends drainage of the brain abscess when choking has attained its maximum and the disks have become stationary. Coleman does not agree with Lillie, stating that there is no definite relation between the size of the abscess and the degree of choked disk. He found the highest degree of papilledema in some of his smallest abscesses, while the largest abscess often had a normal eyeground. J. Fischer made the same observations as Coleman in his studies of brain tumors in 1921.

In the *neurologic* examination we have to look for focal symptoms, such as cerebellar ataxia, asynergia, adiadokokinesis, etc. The tests of these various disturbances of coordination and tone are described in chapter iv.

The lumbar puncture represents a very important clinical examination. It can be helpful not only for diagnosing the brain abscess but also for the control of the clinical course of the infection and for the prognosis. This in turn requires repeated tappings at certain intervals. There is great diversity of opinion concerning the danger of lumbar puncture. A number of otologists and neurologists consider this procedure as dangerous in cases of brain abscess, basing their assumption on reports of sudden death following a spinal tap, particularly if too much fluid escapes. Cains and Donald describe such accidents after withdrawal of 10 cc. of liquor. Therefore they recommend withdrawal of not more than 1 cc. Other authors are opposed to the traditional fear of such a manipulation. Generally, it can be said that this danger is overrated. We used to drain spinal fluid from patients in recumbent position, tapping very slowly and withdrawing not more than 4 to 5 cc. of fluid, and we never experienced any accident.

It must be borne in mind that there are cases of cerebellar abscess in which the fluid is found to be normal even up to the death of the patient. Grabscheid published 4 cases of the Alexander and Brunner clinic, none showing any changes in the spinal fluid. According to Alexander, a slightly cloudy but sterile fluid with an increase of leucocytes denotes a nonperforated brain abscess. Knick considers marked changes in the cell count of the fluid, after punctures repeated at short intervals, as characteristic for brain abscess. If the fluid findings show improvement, but the clinical symptoms become worse, the existence of a cerebellar abscess is most probable. Alexander stated that if the fluid within a period of eight to ten days (after repeated tappings) yields normal findings, the presence of

meningitis can be ruled out; if in such a case symptoms of an intracranial complication occur, the diagnosis of brain abscess, sinus thrombosis, or extradural abscess can be made. According to Haymann, the presence of eosinophiles with the increase of lymphocytes must be regarded as a favorable prognostic sign. Woltmann believes that a decrease in the total number of cells, associated with a predominance of lymphocytes, indicates a better encapsulation of the abscess and a greater resistance.

For *roentgenologic* studies of the ventricles and subarachnoidal spaces, inflation of air or oxygen has been used. There are two main methods. One consists of inflation into the ventricle by ventricular puncture (ventriculography, Dandy): the air spreads from the third and fourth ventricles via the cisternae into the subarachnoidal spaces. The other method uses the spinal puncture (encephalography, Bingel): the inflated air spreads retrogressively from the spinal subarachnoidal space into the cisterna magna and from there into the cerebral subarachnoidal spaces and into the ventricles. X rays taken with the patient in occipital position produce in normal cases the typical picture resembling a butterfly. On the antero-posterior film the lateral ventricles will show moderate symmetric dilatation, and the third ventricle will appear dilated and in the midline. In the postero-anterior view the posterior horn on the involved side will be smaller than that on the opposite side.

### DIFFERENTIAL DIAGNOSIS

The differentiation of a cerebellar abscess from other complications depends largely upon the pathways of infection. It has been mentioned above that an abscess developed in a course of chronic otitis media usually follows the pathways through the inner ear, while an abscess in acute otitis media follows more often an infection of the posterior fossa (sinus thrombosis, extradural abscess). Another point that must be taken into consideration concerns the inflammatory edema and the hydrocephalus that accompany the cerebellar abscess. The more intense these pathologic changes are, the more marked are the remote symptoms due to the increased general brain pressure. This in turn may give rise to cerebral symptoms of the middle or anterior fossa. We shall here discuss only the most important differentiations.

*Internal otitis and cerebellar abscess.* The problem presented here does not relate to the question whether an internal otitis or a cerebellar abscess exists, but rather to the question whether only an internal otitis or an internal otitis plus cerebellar abscess is present. If we have opportunity to observe the patient through a period of time, particularly in the beginning of the labyrinthine infection, the differentiation may be easy. The symptoms will then present the manifestations of irritation, such as in-

tense turning vertigo, nausea, vomiting, nystagmus of second to third degree, chiefly in the direction of the diseased ear, and a functioning labyrinth. Later on the irritation symptoms gradually subside and are replaced by manifestations of destruction, such as nystagmus of second to third degree in the direction of the nonaffected ear, and loss of cochlear and labyrinthine function. When in the further course of the disease the nystagmus changes again in the direction of the affected ear, in combination with recurrence of vertigo and disturbances of balance and coordination, the presence of a cerebellar abscess seems probable (Neumann). It is obvious that this nystagmus toward the side of the diseased ear could not be elicited by the labyrinth that has previously been destroyed. Neumann formulates the following differential-diagnostic observations:

Nystagmus in the direction of the diseased ear may be due to either a circumscribed labyrinthitis or a cerebellar abscess. In the case of the former, the caloric reaction will be intact, the fistula symptom positive, and the hearing acuity still present.

Nystagmus in the direction of the diseased ear associated with complete loss of the inner-ear functions points to a disease in the posterior cranial fossa.

Nystagmus in the direction of the diseased ear in which the function of the inner ear cannot be determined requires a labyrinth operation. If after the operation the nystagmus in the direction of the diseased ear persists, the diagnosis of a cerebellar abscess becomes certain.

Nystagmus in the direction of the nondiseased ear associated with loss of the inner-ear function may be due either to a diffuse internal otitis (destructive stage) or to a cerebellar abscess. The differentiation can be made by means of a labyrinth operation. A nystagmus caused by internal otitis must subside within two to three days, while a nystagmus caused by a cerebellar abscess will persist or become more intense or change to the direction of the diseased side.

*Temporal lobe abscess and cerebellar abscess.* The differentiation is easy if the focal symptoms are well marked, but becomes more difficult as the remote symptoms prevail. The following are some signs and symptoms in favor of a diagnosis of temporal lobe abscess: aphasia, homonymous hemianopsia, pain over the temporal or parietal lobe, tenderness on percussion over these areas, acute otitis media, particularly of the epitympanic type.

Symptoms in favor of a diagnosis of cerebellar abscess are: hemi-ataxia, vertigo, nystagmus, pain over the occipital or frontal lobes, tenderness on percussion over these areas, rigidity of the neck, compulsory posture of the head, involvement of certain brain nerves, chronic otitis media (choles-

teatoma) or acute exacerbation, mastoiditis, internal otitis, sinus thrombosis, or extradural abscess of the posterior cranial fossa.

However, there will always be cases in which a differential diagnosis cannot be made. One of us (Fischer) observed a case of acute exacerbation of a chronic otitis media in which a radical mastoidectomy was performed. The operation revealed a pachymeningitis of the middle fossa. Soon after the operation the temperature rose to 105.5 F. and the patient had attacks of chills. There was no nystagmus nor symptoms of meningitis. The eyeground and spinal fluid were normal and the labyrinth function intact. The operator ligated the jugular vein and punctured the sinus, which proved to contain free blood. After this operation the patient felt much better but still had a very high temperature. Five days later amnesic aphasia developed. This symptom, in conjunction with the pachymeningitis of the middle fossa (revealed at the first operation), as well as the normal labyrinthine function and the absence of any spontaneous nystagmus, pointed to the probability of an abscess in the temporal lobe. Puncture of the brain in this region had no successful result. Two days later the patient died. The autopsy revealed a cerebellar abscess in the ventral region.

A case of just the opposite kind is described by Hinsberg. In a course of radical mastoidectomy a perisinuous abscess and a sinus phlebitis were found. There was also spontaneous nystagmus, which made the diagnosis of a cerebellar abscess very probable. However, repeated punctures of the cerebellum yielded a negative result, and the patient died shortly after. The autopsy revealed a large abscess of the right temporal lobe. There is another very interesting case which one of us (Wolfson) was able to observe from the very beginning to the end, over a period of many years:

A girl 15 years of age was first operated on for acute mastoiditis on the right side. A simple antrotomy led to complete recovery. Approximately three years later, there was a recurrent mastoiditis following a head cold. The mastoid was operated on again and the diseased cells thoroughly removed. Two weeks later the temperature suddenly rose to 104 F.; the patient had vertigo, nausea, vomiting, and terrific headaches. Lumbar puncture revealed clear fluid; the lymphocyte count was 122, red cells 586. The mastoid was operated on again; exposure of the lateral sinus and of the dura of the middle and posterior fossae gave no evidence of pathology; there was also no lead to any petrositis. On the following day the patient developed a facial palsy on the affected side and complained of vertigo and headaches. On consultation, the neurologist made a diagnosis of brain abscess, probably cerebellar abscess. The parents, however, demanded conservative treatment with watchful waiting. The patient was given the routine sulfanilamide treatment. In the further course the patient improved more and more. After seven weeks of stay in the hospital, she was discharged in very good condition, except for the facial palsy, which remained unchanged. Eight days later the patient was again admitted to the hospital, complaining of headaches, vertigo, nausea, and vomiting. The eyeground first showed slightly engorged vessels on the right side, but a few days later papillary

edema could be observed. The consulting neurosurgeon (Horrax) made the diagnosis of a cerebral abscess. The parents agreed to transfer the patient to the Lahey Clinic for brain surgery. Pneumoventriculography, on the left side, made the existence of a large right temporal lobe abscess almost certain. The operation consisted of right temporal decompression with multiple taps to try to locate the abscess, and subtemporal decompression with uncapping of the herniated, edematous cortex. No abscess was found and the patient had an uneventful recovery.

This case illustrates clearly the difficulties that may occasionally occur in the diagnosis of the site of a brain abscess. While one consultant assumed a cerebellar abscess, the other located the abscess in the temporal lobe. Operation on the brain failed to disclose any abscess. The patient recovered and is now, four years after the operation, in normal health except for her facial palsy, which still persists.

*Purulent meningitis and cerebellar abscess.* A differentiation between these two diseases can usually be made. The diagnostic difficulties, however, occur when the question arises whether a purulent meningitis or a cerebellar abscess plus meningitis exists. There are some differential-diagnostic points:

The general brain symptoms in meningitis are more acute, more violent, more enduring: remissions do not usually occur as they do in the case of cerebellar abscess, where the focal symptoms are more marked.

The pulse in meningitis is a combined septic and pressure pulse and is absolutely faster but relatively slower (as compared with the temperature). The pulse in cerebellar abscess is a typical pressure pulse, i.e., slow and resistant.

The temperature in meningitis is very high and continuous. The temperature in the case of cerebellar abscess is usually slightly elevated or normal.

The fluid findings in meningitis show marked changes in pressure, color, transparency, chemical composition, cytology, bacteriology, etc. The fluid in the case of noncomplicated cerebellar abscess shows relatively slight changes or no changes at all, particularly in regard to bacteriology.

Rigidity of the neck combined with positive Kernig sign and infected fluid indicates a meningitis. Rigidity of the neck with negative Kernig sign and sterile fluid, combined with compulsory head posture in a forward or sideward inclination, points to the presence of a cerebellar abscess (Nuehsmann).

Convulsions, tonic or clonic, are common in meningitis, while rare in cerebellar abscess.

Spinal symptoms are common in meningitis but rare in cerebellar abscess.

It is obvious that there always will be cases in which a local diagnosis of abscess cannot be made. If, however, an experienced clinician would only

consider the existence of an inflammatory process within the posterior cranial fossa, a great step forward would be made. For just this gives the indication for a surgical intervention that often reveals the routes of infection and makes the local diagnosis possible.

### OPERATIVE TREATMENT

In the beginning of this chapter, it has been stated that there is an amazing diversity of opinion with respect to the various problems concerning cerebellar abscess. This particularly applies to the question of surgical treatment. There is hardly one point on which an agreement can actually be noted. This dissension concerns, for instance, the approach to the abscess, the exploration, the proper moment for surgical intervention, the phases of the operation (one- or two-stage procedures), and numerous technical problems, such as puncture through an intact dura or incision of the dura, use of knives or needles, drainage or tamponades, etc.

So far as the approach to the abscess is concerned, there is a basic difference in the technic of the neurosurgeons and that of the otosurgeons. The former approach the cerebellum from the outside of the skull, far from the diseased petrous bone, while the latter use the routes from the inside, i.e., through the mastoid. The determinant for the neurosurgeons is the prevention of a secondary infection of the brain, since the operation is performed in a sterile field. The otosurgeons on the other hand claim for their method the following advantages: they eliminate the source of infection; they follow the same pathway through which the infection traveled from the middle ear to the brain, and which therefore is the best guide to the abscess. The otitic brain abscesses usually lie in the vicinity of the ear and should therefore be traced from the ear organ (Koerner). Macewen advocates the approach from both directions. Ramadier starts with the Cushing operation for decompression and then performs the mastoidectomy, carefully avoiding any contact between the two fields of operation. He then waits a certain period of time to see the results of these surgical procedures. If all the symptoms gradually subside, no further operation seems necessary. If, however, the symptoms persist or become more intense, he approaches the cerebellum for exploration and drainage of the abscess en route through the sterile field.

The proper moment for surgical interference is another point of debate. Some authors, particularly the neurosurgeons, maintain the standpoint that operation of a cerebellar abscess should be postponed until the abscess becomes ripe, i.e., until encapsulation has taken place. Frazier believes that an abscess in the making is not a surgical lesion and he attributes many of the failures of the past to ill-timed operations. Grant states that drainage of a brain abscess before encapsulation has occurred has been

uniformly disastrous. According to Eagleton and to Westphal, the capsule formation begins seventeen days from the onset of the brain infection. Most of the authors agree that for complete encapsulation a period of from four to six weeks seems to be necessary. Efforts have been made to determine the moment of ripening of the abscess. Some authors recommend for such reasons repeated eyeground examinations (Dandy, Lillie). The latter advocates drainage of the abscess until the choking has attained its maximum and the disk has become stationary. Woltmann determines the encapsulation processes by repeated spinal fluid examinations. He believes that a decrease in the total number of cells, in association with a predominance of lymphocytes, indicates a better encapsulation and a greater resistance.

It must be admitted that the prognosis of an encapsulated abscess is much more favorable than that of an abscess that is not walled off at all. The suggestion of the neurosurgeons for postponing the operation until the abscess becomes ripe would be ideal if we were able to determine the moment of ripening. This, however, is frequently a difficult task in spite of the various methods described in the literature. First of all, the factors in the formation of a capsule are still unknown (p. 268); some cases do not show any encapsulation at all. Shuster found, out of 11 cases of cerebellar abscess, 8 cases with no sign of encapsulation. Among 5 cases with recovery, none showed a capsula around the abscess. Second, encapsulation is not necessarily identical with ripening of the abscess (p. 268). Third, developing of a capsula is said to take from one to six weeks, a period that is not very definite for practical purposes. Fourth, it is often difficult to determine definitely the time of onset. Fifth, there is often no relation between size of abscess and degree of choked disk.

Some authors (Dench, Krause, etc.) operate in two stages. The first phase consists of a mastoid operation, with exposure of the posterior fossa. The uncovered dura is swabbed with iodine in order to produce adhesions within the meninges and to seal off the subdural spaces. In such a way the danger of a secondary infection of the brain spaces is said to be avoided. Some otologists use phenol (Macewen) or other chemical irritants, or electricity in the form of electrocoagulation of the dura (Kaplan). The second stage of the operation is performed as soon as the adhesions seem to have been formed (one to six days after the first operation).

Neumann believes that the general fear of a secondary infection is much overrated. Every experienced otosurgeon has had cases in which an operative injury of the dura mater within an infected field—as the mastoid region is supposed to be—has taken place without development of a secondary meningitis. Furthermore, we know that after an incision through the dura a prolapse of the brain occurs that seals off the subdural

spaces, thus preventing spread of the infection. We assume more danger for the patient in waiting for such adhesions than in a one-stage operation.

In regard to exploration of the brain abscess, the same diversity can be observed as in respect to the approach. Some neurosurgeons advocate puncture of the brain through the closed skull, as used by Neisser and Pollack. On certain parts of the skull, openings are made by means of a burr; then a cannula is introduced, piercing through all the layers directly into the brain. Other neurosurgeons first perform an osteoplasty with exposure of the dura and then puncture the brain (Adson). The otosurgeons follow the pathways of infection, i.e., from the mastoid to the brain, exposing the dura to a large extent. If there is a fistula in the dura, the puncture is likely to be successful. If the dura is intact but shows granulation or pachymeningitic changes, we may use these as guides. Finally, should the uncovered dura look normal macroscopically, we may still be guided by the bony destructions and granulations and pus in the mastoid cavity. Some authors puncture through the intact dura after swabbing it with iodine, while others reject such a procedure as being dangerous, but advocate an incision of the dura first, followed by puncture of the brain.

An old point of controversy is as to whether a puncture cannula, an aspiration needle, or a knife should be used for the exploration of the brain abscess. Each operator claims advantage for his method and rejects the others as being dangerous. Alexander used special calibrated curved scalpels. If one uses a needle, he stated, he can easily miss the abscess, or the lumen of the needle may be blocked up by thick pus or by brain particles, thus yielding a negative result. Others are afraid to use knives because of the danger of a hemorrhage or of damaging brain tissue. There is no doubt that each method carries a certain danger in itself.

With respect to the operative technic, the two most extreme standpoints will here be discussed. One group of authors (Dandy, Dean, Lemaître, etc.) recommend surgical procedures limited to a minimum, such as a small incision of the dura, repeated tappings with a needle, filiform drains, etc. Another group (Eagleton, Bruenings, King, Alexander, Brunner, etc.) call for extensive operations on the bone as well as on the brain. Eagleton exposes the dura of the whole cerebellar fossa of the affected side, while the bone over the unaffected hemisphere is freely removed. He further ligates the descending portion of the lateral sinus (cf. also Friesner and Braun).

The commonest place for opening a cerebellar abscess lies in front of the sinus within Troutman's triangle. According to Eagleton the intracerebellar pressure is less there than in the lateral parts.

All the numerous surgical procedures described in the literature can be classified into the following four types (Alexander):

1. Methods of evacuation of the brain abscess by incision and drainage, according to the surgical principles applied to abscess in any part of the body;
2. Methods of evacuation of the brain abscess by puncture or aspiration and drainage with filiform drains;
3. Methods of evacuation of the brain abscess by removal of brain substance and drainless after-treatment;
4. Methods of evacuation of the brain abscess and after-treatment with permanent tampons.

*Type 1.* These methods are very common. As far as exploration and evacuation of the brain abscess are concerned, the technic is satisfactory. However, with regard to after-care and final cure of the patient, the results mostly do not satisfy. After a short temporary improvement there is often a relapse with recurrence of all symptoms. In spite of a large incision and an apparently good drainage, the encephalitis in the depth advances, leading to formation of pockets and recesses in the cavity and of new abscess.

The main efforts of all operators have always been concentrated on establishing a good drainage. Every surgeon has his own ideas about the kind of drains to use and the way of introducing and changing them. Among the kinds used are glass drains (Nuehsmann), perforated glass drains (Koerner), rubber drains (Ruttin), rubber drains wrapped in iodoform gauze (Neumann), copper wire drains (Mosher), resorbing drains or decalcified chicken bones (Macewen), iodoform wicks (Alexander), oiled silk drains (Moskovitz), gutta-percha drains (Bárány), the tracheal cannula (Koerner), the Grant cannula, etc. Instruments used for introduction of the drain under control of the eye are the encephaloscope (Eagleton), the Killian speculum, the bronchoscope (Henke), etc.

This variety in the treatment shows clearly the lack of satisfaction with the results obtained. However, there are certain cases of cerebellar abscess, such as cortical abscesses or small, well localized medullary abscesses, which have yielded a good result with this method.

*Type 2.* This method was first described by Lemaître. After puncture of the brain abscess and aspiration by means of a needle, a filiform drain is introduced. As often as the dressing is changed in the course of the after-treatment, the filiform drain is replaced with one of a larger size, until a drain with a lumen of about 2 cm. diameter is used. The method of Dandy is similar. He advocates repeated tappings of the abscess by means of a thin needle and slow drainage in the after-care. For small

abscesses with high intracranial pressure, Coleman recommends Dandy's method combined with surgical decompression.

*Type 3.* Efforts to convert the closed abscess cavity into an open excavation by removal of brain substance were made by Boenninghaus and Bruenings in Europe and very successfully by King in America. Because of the great importance of King's method, as it was first used in the operation of temporal lobe abscesses, his technic will here be described in detail. The bone over the site of the abscess is removed over an area about 3 to 4 cm. in diameter. The dura is incised in the lines of a cross, creating six pennant-shaped flaps that are reflected over the bony margin and sutured to the underlying surface of the scalp flaps. The subdural and subarachnoid spaces are sealed off by packing the circular defect with three strips of iodoform gauze. Then follows exploration (needle or cannula) and incision of the brain at the point where it overlies the abscess cavity. The incision in the cortex of the presenting brain is carried to a point on the margin of the cavity and then around in a circular manner so that the entire roof of the cavity is removed ("unroofing"). When normal brain tissue is cut, there may be a considerable amount of bleeding; only slight oozing occurs when the brain tissue is diseased. After unroofing the cavity, the floor of the abscess cavity becomes less concave, flattens out, and tends to rise, thus diminishing the depth of the cavity. After packing with iodoform gauze, a fenestrated rubber dam is placed over the cavity. No drainage at all is used.

This method is very effective, representing the open type of abscess treatment. Unroofing allows the remaining portion of the cavity to herniate outward, thus preventing any secondary pocketing. The rubber dam protects the surface of the postoperative prolapse. Another very important improvement is the drainless after-treatment.

*Type 4.* The method of permanent tamponade was discovered accidentally and was formerly applied in the treatment of traumatic brain abscesses due to gunshot injuries. During the first world war, Albert (Vienna) made the following observation. When in certain periods (after an offensive battle) a tremendous number of traumatic brain abscess cases were admitted, so that a regular change of dressings was impossible, the patients showed a great improvement. He tried now deliberately to leave the bandage on as long as possible (three to seven weeks), and got amazingly good results. Similar experiences were described by Bárány, Feuchtinger, Leidler, Mayer, Marschik.

There is a great difference between traumatic and otogenous brain abscesses and one cannot apply the experiences pertaining to the one type in the treatment of the other. If a patient in the course of the after-care suddenly shows a high temperature, chills, pressure pulse, etc., much more courage is necessary for leaving the tampon in than for taking it out.

However, the number of otogenous brain abscesses treated in this way is too small to allow of any decisive judgment of the method.

We have presented the various points of view and have critically discussed all the pros and cons. What the reader really wants to know is the best technic for operating a cerebellar abscess. Should he follow the advice of the neurosurgeons, or should he operate via the mastoid? The method will largely depend upon a proper diagnosis. If the abscess originates from the ear and the preoperative diagnosis was certain, he should proceed by way of the mastoid. If, however, the focus seems questionable, the neurosurgical approach should be made. The surgical treatment of an otogenic cerebellar abscess must include the following three aims:

1. *Elimination of the primary source of infection in the mastoid.* This consists of a simple mastoidectomy in acute cases, a radical mastoidectomy in chronic cases, a labyrinthectomy in cases with routes of infection through the labyrinth, and a sinus operation in cases in which the abscess developed by the route through the sinus.

2. *Decompression.* In order to combat encephalitis effectively, the squamous portion of the occipital bone of the affected side should be largely removed.

3. *Operation on the brain.* In cases of cerebellar abscess developed by the route of the sinus, an incision into the medial wall should be made after removal of the thrombus or after double ligation of the sinus. From this point the cerebellum should be explored and the abscess emptied and drained. In the case of cerebellar abscess by the route through Troutman's triangle, a large exposure of the sinus and posterior fossa should first be made. The dura is then incised in front of the lateral sinus and the cerebellum explored. If the abscess is found, a large counterincision should be made into the cerebellum in the retrosinusal region and drains introduced. In the event that the further clinical course does not run satisfactorily, the part of the cerebellum between the two incisions (anterior and posterior to the sinus) should be removed.

In cases of cerebellar abscess by the route through the labyrinth, the method of King (p. 232) should be used. The dura is incised in the lines of a cross, thus yielding four flaps that are carefully separated from the surface of the brain. The diseased part of the hemisphere is then circumcised and capped. Since there is no danger for the ventricle, drainage tubes can be introduced, in contrast to the methods required in the case of temporosphenoidal abscesses.

### 6. Temporosphenoidal Abscess

The temporal lobe abscess will be briefly discussed, since the overwhelming majority of cases develop by the route through the middle ear,

thus leaving the inner ear intact. The infection usually spreads through the epitympanum to the roof of the middle ear. Koerner found in 37 out of 40 cases of temporal lobe abscess a circumscribed necrosis of the tegmen with changes in the dura. Eagleton, however, believes that pathologic changes in the bone are very often lacking. FIGURE 64 illustrates such destruction of the roof of the middle ear. The route of infection through the labyrinth is rather rare. In such cases the infection spreads through the superior vertical canal, causing bony necrosis on the arcuate eminence. Such abscesses have a much more medial location than the common abscesses. The typical site of a temporal abscess is the basal portion of

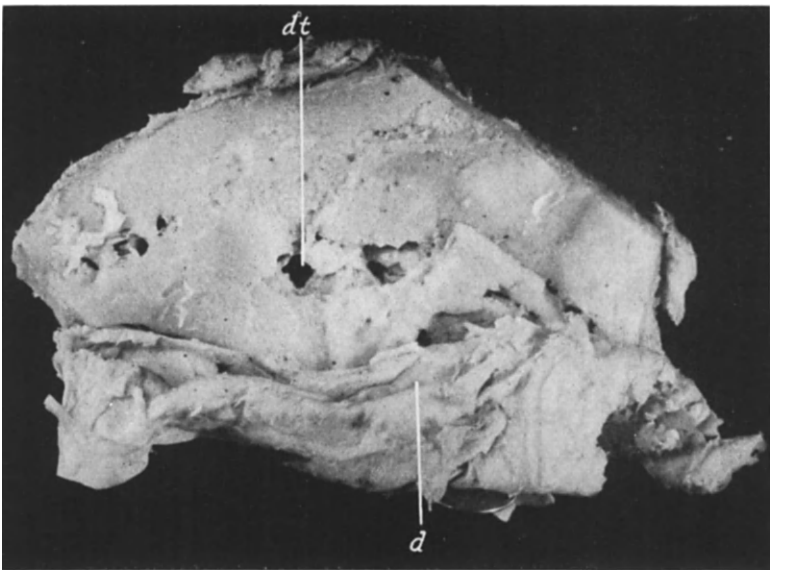


FIG. 64. PHOTOGRAPH OF PETROUS BONE OF CASE OF TEMPOROSPHENOIDAL ABSCESS
*d t* = destruction of tegmen tympani and tegmen antri; *d* = dura of middle cranial fossa reflected downward.

the temporal lobe (gyrus fusiformis, lingula, gyrus temporalis inferior). While the infection often spreads *per continuitatem* from the middle ear to the dura, the further extension into the brain is the same as in the case of cerebellar abscess (p. 263), i.e., by way of retrograde thrombosis of pial vessels. As far as pathologic anatomy, capsula formation, and bacteriology are concerned, the same considerations hold as those outlined in the discussion of cerebellar abscess. With regard to growing tendency, it can generally be stated that temporosphenoidal abscesses may reach a far greater size than cerebellar abscesses. This is particularly true in respect to the extension in the medullary substance (Macewen,

Preysing). Another difference can be seen in the fact that otitic temporal lobe abscesses usually do not show as great a tendency to form pouches and pockets (Brunner).

### SYMPTOMATOLOGY

For discussion of the symptomatology we shall use the classification of Bergmann:

*General symptoms.* The patients usually look very tired and sick, with flabby skin. Loss of appetite, constipation, and emaciation occur. The temperature is slightly elevated in the initial stage, but very high in the manifest stage.

*General brain symptoms.* The patients usually complain of severe headaches either in the temporal region or in the forehead, occasionally in the occipital region; these are not relieved by medication. The symptom becomes very significant if it is persistent over a considerable period of time and if it is localized, regardless of the region. On movements of the head an increase in the pain is recorded. Change in personality can often be observed. In the further advance of the disease, drowsiness and somnolence occur. Other symptoms that are very common in association with cerebellar abscesses, such as vomiting, vertigo, nystagmus, rigidity of the neck, etc., are rather rare in the case of temporosphenoidal abscess. Pressure pulse and disturbances in respiration are also less frequent than in the case of cerebellar abscess. Changes in the eyeground are often found. Comparative statistics show these changes to be more frequent in connection with temporal lobe abscesses but more severe (choked disk) in cases of cerebellar abscess. Changes in the cerebrospinal fluid have been described in the discussion of cerebellar abscess (p. 273).

*Local brain symptoms.* These may be subdivided as in relation to cerebellar abscess:

Focal symptoms: In order to understand the focal symptoms, it is necessary to know the centers and paths located in the temporosphenoidal lobe. There are the cortical speech center (on the left side), the acoustic center for the contralateral ear, and the optic radiation, the latter situated in the depths of the temporal lobe and running to the calcarine fissure. Hence the focal symptoms should be aphasia, contralateral deafness, and hemianopsia.

There is usually an amnesic aphasia, which is a certain feature of the various types of sensory aphasia. Isolated motor aphasia does not belong, according to Brunner, to the typical picture of an otitic temporal lobe abscess. It either points against the probability of otitic origin of the abscess or is in favor of the assumption of an otogenic but diffuse brain complication (meningitis, encephalitis).

A patient with amnesic aphasia is unable to name objects shown him, although he recognizes the identity of them. In cases with only a slight degree, it is often very difficult to detect aphasia, since the patient describes the objects correctly. If, for instance, he is shown a knife, his answer may be, "It is for cutting," or, if shown a key, "It is for locking or opening." When directly asked to name the object, he answers, "I know but I cannot say." With the further advance of aphasia, he uses the wrong expression—e.g., he calls a lead pencil a chair. If now the examiner asks him, "Is it a chair?" he answers with "No"; if asked whether it is a fork, he again says, "No"; but when asked whether it is a lead pencil, he smiles and says, "Yes." Some patients have special difficulty in recalling names of persons whom they know very well, even of persons of their own family. One of us (Wolfson) observed such a case with a marked loss of memory for personal names. A patient 48 years of age with a chronic otitis media had two main complaints, i.e., persistent terrific headaches localized over the left temporal region and inability to recall the names of his best friends. The latter fact upset him very much. A radical mastoidectomy was performed, with large exposure of the middle cranial fossa. On exploration of the brain a temporosphenoidal lobe abscess was found, emptied, and drained with a Mosher wire basket; the patient recovered. It was interesting to observe how his amnesic aphasia gradually subsided. While he soon gained ability to name objects, it took him very long to recall names of persons.

In spite of the great clinical significance of amnesic aphasia in cases of temporosphenoidal abscess, it must be admitted that this symptom has also been found in connection with various other intracranial lesions. Brunner considers the presence of amnesic aphasia in the course of an otitis media as an absolute indication for exposing the middle cranial fossa, but not indication enough for exploring the temporal lobe.

The symptom of *contralateral loss of hearing* has not much practical value since the auditory function is controlled by both hemispheres. Many authors do not believe in a central origin of the diminished hearing, but explain it rather by the assumption of a basilar meningitis accompanying the temporal abscess.

The symptom of *hemianopsia* is very important for the diagnosis of a temporosphenoidal abscess. There is either a homonymous or a quadrant hemianopsia.

Remote symptoms: These symptoms are not only not characteristic at all, but may instead confuse the clinical picture, thus making the diagnosis more difficult. They depend largely upon the size and growing tendency of the abscess, upon the intensity of the encephalitis, the collateral edema, and the hydrocephalus. Pressure upon the various parts

of the brain such as the motor area, the internal capsula, the sensory centers, etc., may cause a variety of symptoms. Pressure upon the posterior cranial fossa, for instance, can elicit symptoms of a cerebellar or a pontine lesion.

Ventriculography may play an important role in diagnosing temporal lobe abscesses by showing displacement and distortion of the lateral ventricle (p. 274).

### TREATMENT

The surgical treatment of temporosphenoidal abscesses must follow the same lines as in that of cerebellar abscesses, i.e., elimination of the source of infection, decompression, and operation upon the brain.

*Elimination of the primary source of infection.* This is accomplished by a simple antrotomy in cases of acute otitis media, and by a radical mastoidectomy in the case of chronic otitis media.

*Decompression.* In order to combat encephalitis, the overlying bone must be removed, the bony opening measuring 3 to 4 cm. in diameter.

*Operation on the brain.* The most effective surgical procedure consists of the method of King (p. 282).

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# VIII

# Chemotherapy

By *Louis E. Wolfson*

## Classification and General Use of the Sulfa Drugs

The desire to find a substance that will destroy micro-organisms in the body, without detrimental effect on the host, is as old as our knowledge of micro-organisms, being the ideologic factor in most diseases. Koch first attempted this form of treatment in cases of septicemia by using mercury bichloride, but found it ineffective because it is inactivated by the blood proteins. Later, Ehrlich explained as his aim a "therapia sterilisans magna" (to be achieved by a chemical substance that kills the disease organisms in the body without harm to the host).

Unquestionably we have begun to achieve results in this quest. Apart from the drugs used in protozoa diseases, in lues, and possibly in leprosy, the sulfonamide preparations are by far the most important. In 1935 Domagk found a substance called prontosil effective in pneumococcic infections of white mice. This prontosil had been built up some years previously by Klarer and Mietsch who, with Domagk, were employed by the same laboratories. Domagk first successfully applied the drug experimentally in infection without any harmful effect on normal tissue. Further investigation with prontosil by Trefouels, Nitti, and Bonet showed that the prontosil was split up in the body and that the really effective chemical was sulfanilamide, which itself had been built up many years before by Gelmo, but with no suggestion as to a therapeutic use. Butler and other Englishmen discovered the great effectiveness of these drugs in streptococcus infections in man.

An enormous amount of work has been done in the past several years on these and similar drugs relative to their effect on an increasing number of diseases. There are at present four drugs of the sulfonamide group that are practically important: sulfanilamide, sulfapyridine, sulfathiazole, and sulfadiazine. The last named is poorly soluble and is used mostly as a local disinfectant in dysentery and similar conditions, and is not of great importance for our further discussion here.

The mechanism of action is not completely understood, but it seems certain that these drugs interfere with the normal growth and propagation of the micro-organisms. As far as we know, their effect on the defensive forces of the body is only indirect. The production of antibodies is un-

changed. The effect of the drugs impairs the production of the microorganisms and slows up their invasive power so that the defense forces of the body are able to overcome the infection. The unaffected tissue is more resistant, but it is important to know that pus or destroyed tissue remains uninfluenced.

### SULFANILAMIDE

Sulfanilamide is the oldest in the list of sulfonamide drugs. Its effect in hemolytic streptococcic infections is marked, and in all these infections, for example in erysipelas, it is dramatically effective and very much used. It has a very low effect in staphylococcic infections, but is useful in pneumococcic, meningococcic, and gonococcic infections and in infections with Clostridium welchii, Brucella melitensis, and Escherichia coli.

The effect depends on the level of sulfanilamide in the blood. The aim of any therapy is a constant sufficiently high level for a long time. In general it is necessary to maintain this level for some time after a normal temperature is reached. The level of sulfanilamide for a strong effect on streptococci is in general 10 mg. per 100 cc. In very severe infections it is possible for a short time to increase the dose up to a level of 15 mg. per 100 cc., which nearly always causes toxic symptoms. In some less dangerous conditions, and in pneumococcic infections, a level of 6 to 8 mg. per 100 cc. is usually sufficient. The spinal fluid shows a level 25 per cent lower than that in the blood.

The disadvantage of this therapy lies in the toxicity of the drug. The most serious effects of sulfanilamide relate to the skin, bone marrow, kidney, and blood; the nervous system is also affected. Dizziness, tinnitus, malaise, and headache are frequent. Alcohol, which has similar effects, accentuates these symptoms. The patient, if he gets the drug while ambulatory, has to be warned not to drive a car or plane after the administration of sulfonamide preparations. Toxic symptoms of the gastro-intestinal tract are also frequent, such as anorexia, nausea, vomiting, or diarrhea.

The most marked and most common toxic sequela of sulfanilamide administration is cyanosis. It was shown that sulfanilamide produces methemoglobin. Wendel therefore introduced the therapy of methylene blue in sulfanilamide poisoning. Usually the effect is striking. Severe cyanosis is also relieved within thirty minutes by slow intravenous injection of methylene blue (methylthionine chloride) in a dose of 1 to 1½ mg. per kilogram of body weight, given in a 1 per cent aqueous solution.

Marshall and Walzl do not agree that methemoglobin is the only reason for cyanosis in sulfanilamide therapy and have shown that some dark oxidation products of sulfanilamide, which themselves are harmless, have the power to increase cyanosis. It seems certain that methemoglobin is

produced and that in this lies the point of danger that is announced by cyanosis, while relatively harmless substances increase cyanosis.

Sometimes high fever and chills appear after the administration of sulfanilamide; this should put an immediate stop to continuing use of the drug. Further, dermatitis and allergic reactions may occur and indicate cessation of sulfanilamide therapy. It has been claimed that irradiation by sun or ultraviolet light may be concerned in such reactions; irradiation therefore has to be omitted.

TABLE 3

| Body Weight (Kg.) | Initial Dose by Mouth (Gm.) | Maintenance Dose Every 4 Hr., Day and Night (Gm.) | Dose in First 24 Hr. | |
|---|---|---|---|---|
| | | | Gm. per Kg. of Body Weight | Total |
| 70 | 4.8 | 1.2 | 0.15 | 10.5 |
| 60 | 4.2 | 0.9 | 0.15 | 9.0 |
| 45 | 3.6 | 0.9 | 0.18 | 8.1 |
| 35 | 3.6 | 0.9 | 0.23 | 8.0 |
| 23 | 3.0 | 0.6 | 0.26 | 6.0 |
| 11 | 1.8 | 0.3 | 0.3 | 3.3 |

TABLE 4

| Body Weight (Kg.) | Calculated Daily Dose (Gm.) | Gm. per Kg. of Body Weight | Dose by Mouth Every 4 Hr., Day and Night (Gm.) |
|---|---|---|---|
| 70 | 5.4 | 0.08 | 0.9 |
| 60 | 5.4 | 0.08 | 0.9 |
| 45 | 5.4 | 0.12 | 0.9 |
| 35 | 4.2 | 0.12 | 1 of 1.2<br>5 of 0.6* |
| 23 | 3.6 | 0.16 | 0.6 |
| 11 | 1.8 | 0.16 | 0.3 |

* 1 Dose of 1.2 Gm. followed by 5 doses of 0.6 Gm. each.

The question of the production of acidosis has caused an intensive discussion. The method of combining sulfanilamide with large amounts of sodium bicarbonate is now in general abandoned and at least superfluous in patients with normal digestion.

A dangerous toxic effect is due to the destructive influence of the drug on red blood cells, leading to severe anemia of the hemolytic type. Even more dangerous is the destructive effect on white blood cells and the tendency to agranulocytosis. Such occurrences are reason enough for watching carefully red and white blood cells in any course of sulfanilamide therapy.

Damage to liver or kidney is very rare and may occur only in specifically sensitive patients.

The therapy is aimed to reach as quickly as possible the necessary blood level and to maintain it for some days longer than the disease lasts. The general method is to administer a relatively large dose and to continue every four hours with a somewhat smaller dose. For adults the initial dose is usually 2 Gm., the maintenance dose 1 Gm. every four or six hours.

The amounts of sulfanilamide necessary to establish effective blood levels (10 to 15 mg. per 100 cc.) quickly in patients with severe hemolytic streptococcal, meningococcal, gonococcal, pneumococcal, or Welch bacillary infections are given by the table of Long and Bliss (Table 3). The amounts of sulfanilamide necessary to establish effective blood levels (4 to 8 mg. per 100 cc.) in patients with mild or moderately severe tissue infections in which sulfanilamide therapy is indicated are shown in Table 4.

### SULFAPYRIDINE

This drug was prepared by Ewins and Phillips and introduced as a therapeutic drug by Whitby. It has been proved more effective than sulfanilamide in streptococcic infections and in infections with the Friedlaender bacilli in some pneumococcic infections, less effective in typhoid infections. and very effective in strepococcic, meningococcic, and gonococcic infections. This superiority in relation to staphylococcic or Friedlaender infections constitutes its importance. In animal experiments it is much less toxic than sulfanilamide. An important difference between it and sulfanilamide is its much lower solubility. This is the cause of much lower absorption from the intestines and also decreases the toxicity. The low and quite irregular adsorption is the reason for difficulties in reaching a regular and lasting blood level. In sulfapyridine therapy the control of the blood level is therefore not so important as in sulfanilamide therapy.

The mechanism of the action of sulfapyridine seems to be similar to the effect of sulfanilamide. It also inhibits growth and invasiveness of the micro-organisms to such an extent that the humoral and cellular defenses of the body can cope with the infection.

The toxic symptoms differ to some extent from the sequelae of sulfanilamide. Cyanosis is very rare and by far not so important. Fever, rashes, hemolytic anemia, and agranulocytosis are seen and important to the same extent as in sulfanilamide therapy. Nausea and vomiting are much more common, but without effect on the acid-base ratio. The most common complication, however, is damage to the kidney and hematuria; this does not occur in sulfanilamide therapy. It is caused by the low solubility of sulfapyridine itself and its acetylization products, so that the drug precipitates in crystals in the urinary tract. High amounts of fluid are therefore

necessary to increase the solution and to eliminate the drug from the urinary tract.

Sulfapyridine reaches a relatively higher level than sulfanilamide in the cerebrospinal fluid, corresponding to from 60 to 80 mg. per 100 cc. of the blood level.

The dosage resembles in general the amounts given in sulfanilamide therapy. We begin with 2 Gm., followed every four hours by 1 Gm., later prolonging the interval to six hours and some days later decreasing the dose to ½ Gm. Sulfapyridine and especially the more readily soluble sodium sulfapyridine are often given intramuscularly or intravenously, especially if severe vomiting prevents oral administration or if a very quick effect is desired. Because of the strong alkaline reaction of the solutions, the intravenous injection has to be performed extremely slowly, in a 0.5 per cent solution, 0.05 Gm. per kilogram of body weight, 200 to 300 cc. per hour, and never more than 5 cc. per minute. An excellent and very practicable way of administration, especially often used for children, is rectal administration in 1 per cent sodium bicarbonate.

#### SULFATHIAZOLE

This form was described by Forbinder and Walter and by Lott and Bergeim. It is much more soluble than sulfapyridine and therefore better adsorbed. Its toxicity is very low; a blood level of 20 mg. per 100 cc. is in general adequate. It is very effective in the same infections as those in which sulfapyridine is used, and also in some in which the latter is ineffective, as in infections with Streptococcus faecalis.

Toxic complications are rare, and cyanosis, acidosis, and vomiting very rare. But hematuria and injury to the kidney are very common and represent the great disadvantage of this drug. It has to be given therefore with large amounts of fluid. The intravenous administration of a 5 per cent solution of sodium sulfathiazole is possible but rarely necessary for the quick absorption and quick effect of orally administered sulfathiazole. The dosage is in general the same as for sulfapyridine.

#### SULFADIAZINE

This was first described as sulfaparamidine by Robbin, Williams, Winnek, and English. To avoid confusion with sulfapyridine, the name was later changed to sulfadiazine. It resembles in its effect the other sulfonamide preparations, and has been proved effective in all the infections in which sulfathiazole is effective. Its toxicity is very low, though not negligible. Use and dosage are similar to those described above for sulfathiazole.

### 2. Use of Sulfa Drugs in Otology

Because chemotherapy has produced such miraculous results, not only in general practice but also in our branch of medicine, almost every patient

has been given sulfa drugs. The most striking results, however, have been achieved in cases of otogenic purulent meningitis, which represented a mortal disease prior to the adoption of this treatment. Reports from all over the country from that time on agreed that the advent of chemotherapy marked a turning point in otologic history. To mention all the individual papers dealing with chemotherapy would fill volumes and would lead only to more confusion (we therefore shall refer only to some articles that show the development of sulfa therapy up to the present time).

Here are some reports of 1938 and 1939. Horan and French treated 607 patients in the ordinary way and observed 138 cases of mastoiditis, while among 155 patients treated with sulfanilamide there were only 7 cases with mastoiditis, and these revealed no further complications at all. These authors also compared the average time required for healing and found that the time for the first group amounted to 65 days, for the second group to 23 days. Other reports tended to show that the sulfa drugs not only shorten the healing time and prevent occurrence of mastoiditis, but are also effective in curing a fully developed mastoiditis without surgical intervention. Baker and Bradford observed 4 such cases. There are further reports that diffuse otogenic meningitis has been cured by the use of chemotherapy without surgery in many instances. Such results led Wyllie to the statement that "in the otitic condition myringotomy may be necessary but further operation is usually not required."

Reports coming out subsequently showed less optimistic figures, gave warnings in regard to the masking effect of the drug, and showed the efforts made by the various authors to explain the failure of the sulfa drugs in certain conditions. The great diversity of opinion was illustrated in a panel discussion on sulfonamides at a gathering of the American Otological Society in Rye, N. Y., in 1940. There was no agreement, no common platform.

A very good paper concerned with objective explanations for the failure of sulfa drugs in certain cases was published in 1940 by Lyons and Ganz. They believe that the clinical aim in the chemotherapy of hemolytic streptococcus infections should be to secure dramatic improvement within twelve hours and a normal temperature and an arrested infection within forty-eight hours. Failure to achieve such a response may be caused by the following factors: (1) inadequate blood concentration of the chemotherapeutic agent; (2) deficiency of antibacterial antibodies; (3) coexistent erythrogenic toxemia of scarlet fever; (4) a focus of inflammatory fixation (undrained pus, necrotic tissue); (5) intravascular or perivascular sepsis (thrombophlebitis, endocarditis); (6) drug toxemia.

The ill effects of sulfanilamide are well known in general medicine. The first fatal case in otology was reported in 1940 by Kasnetz. The patient died of agranulocytosis, which developed six days after the administration

of the drug. In cooperation with Damashek, I had the opportunity of treating 2 cases of agranulocytosis. Death in such cases is probably due to the overwhelming of the body, stripped of its granulocytic defenses, by the invading infection. We treated the patients with transfusions, pentose nucleotides, liver extract, and large doses of sulfathiazole, and succeeded in saving their lives. The recoveries may be credited, at least in part, to the effect of the sulfonamide on the sepsis, thus allowing spontaneous leucocytic regeneration in the bone marrow to take place.

As to the question of the time for the administration of the drug, it is believed that the earlier the drug is employed, the better are the results. There is more effective contact of the drug with the body fluids in the early stage. On the other hand, a great number of otologists are opposed to this treatment of acute otitis media in the beginning when there are no signs or symptoms to indicate that the disease will run an atypical course. Fowler, Jr., advises postponement of chemotherapy for a week or ten days after the onset of the otitic infection. If there is no improvement by that time, the sulfonamide should be given in maximal doses. Giving insufficient doses of the drug to ambulatory patients is unjustifiable. The drug should be continued for at least a week after acute symptoms subside.

While there is a certain agreement as to the great value of chemotherapy in relation to otogenic complications, great dissension exists with respect to sulfa treatment of noncomplicated otitis media. Lindsay and his coworkers recommend sulfa therapy in all cases of otitis media. Ganz, Lyons, and Ferguson advise early treatment of acute otitis with myringotomy and sulfonamides, with supplemental immune serum for those patients not clinically convalescent within forty-eight hours. Lupton suggests the employment of chemotherapy as an adjunct to surgical treatment and not as a substitute for it. This means that after surgical elimination of the focal infection, the sulfa drugs should be administered. Fuerstenberg applied sulfanilamide locally in the mastoid cavity after the mastoid operation. He used 10 to 15 Gm. of sulfanilamide powder and found that the wounds healed admirably and with very little secretion.

A number of otologists oppose the use of the sulfa drugs in cases of acute otitis media for two reasons. In the presence of an inclosed focus or of necrotic bone, the sulfonamides fail to fulfil the aims of chemotherapy, i.e., sterilizing of the focus. On the other hand, an undesirable masking of the clinical symptoms and signs may often occur. They further direct attention to the fact that even in case of recovery the prolonged conservative chemotherapy results in a permanent damage of the hearing acuity. All these various arguments have led many otologists to the conclusion that chemotherapy should be limited to the treatment of otogenic intracranial complications. It is obvious that chemotherapy in such cases must be

combined with surgical elimination of the primary focus and with supplementary immunotherapy. Following is a schedule for the treatment of meningitis as outlined by Steele, Gottlieb, and Brann:

1. Initial lumbar puncture with cell count, Gram staining, and culture.
2. Sulfa drug administration. Sulfanilamide dosage: initial dose of 40 grains; repeated doses of 10 to 20 grains every four hours until a blood level of 10 mg. per 100 cc. or more and an optimal spinal fluid level have been reached; thereafter, twenty-four-hour maintenance dose of 15 to 30 grains every four hours as necessary to maintain the blood and spinal fluid levels, continued for several days after the spinal fluid has become negative. In the event of relapse, the drug should be resumed at once.
3. Repetition of lumbar puncture every twelve hours until cultures are sterile. Control of cell counts and fluid concentration of sulfa drugs.
4. Repetition of blood examination until the blood stream is sterile. Control of blood concentration of the sulfa drug.
5. Indications for addition of antipneumococcus serum to chemotherapy:
   *a*) Presence of bacteremia;
   *b*) Failure of spinal fluid cultures to become sterile twenty-four hours after the start of chemotherapy;
   *c*) Failure of the patient to improve clinically with chemotherapy alone;
   *d*) When the patient is under 2 years of age;
   *e*) When the patient is over 40 years of age.

In recent years progress has been made through the introduction of new sulfa drugs into the chemotherapy and through advance in clinical experience. The best information on present-day views can be found in an article by Osgood. To determine whether chemotherapy is indicated or not it is important to weigh the risk of use of the drugs against the risk of the disease and to know that the organism producing the disease is susceptible to the action of the drug. At present the chemotherapeutic agent of choice in the majority of infections is sulfadiazine. Against the most severe staphylococcus or Streptococcus viridans infection, the drug of choice is neoarsphenamine, either alone or in combination with sulfadiazine. The latter should be administered every four hours day and night, preferably by mouth, in quantities sufficient to maintain a blood level of 5 to 8 mg. per 100 cc. and sometimes of 10 mg. per 100 cc. Local collections of pus should be drained, and crystalline sulfathiazole in powdered form should be introduced either as a dry pack for the wounds or in heavy suspension in saline solution or in some viscid water-soluble suspending medium that is nonirritating and nontoxic. Unless there are toxic reactions,

or danger of occurrence of these, administration of the drug should not be discontinued until about a week after the temperature has become normal and the cultures negative.

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# IX

# Facial Palsy

By *Louis E. Wolfson*

## 1. Anatomic and Physiologic Basis

For a thorough understanding of the diagnosis and surgical treatment of facial palsy one must have a knowledge of anatomy and physiology. Figure 65 illustrates diagrammatically the course of the facial nerve, its branches, and its connection with the other brain nerves. The facial nerve originates in the facial nucleus in the upper and posterior part of the medulla oblongata, near the junction of the medulla and the pons; from there the fiber passes through the fourth ventricle, forming a knee around the nucleus of the sixth cranial nerve. The facial nerve emerges on the brain surface on the posterior margin of the brachium pontis and runs in an anterior and lateral direction to the internal auditory meatus. There the nerve enters the upper compartment and extends into the fallopian canal to the hiatus spurius, where the geniculate ganglion is located. Two-thirds of the nerve fiber enters the ganglion, while one-third passes close by. From there the facial nerve runs at an angle of 100° downward and backward (upper knee), between the horizontal semicircular canal and the oval window. In the antrum there is another sharp turn downward (lower knee) where the nerve passes behind the posterior wall of the external canal to the stylomastoid foramen. Having passed the foramen, the nerve continues to the parotid gland, forming a plexus within the gland. On the anterior border of the parotid gland the nerve fibers disperse in the form of a fan in order to supply the various muscles of the face.

The *branches and connections within the ear* are (Fig. 64): the nervus petrosus superficialis major; the ramus anastomoticus with the plexus tympanicus; the nervus stapedius; the chorda tympani; the ramus anastomoticus with the auricular branch of the vagus.

The *branches and connections outside of the ear* are: the nervus auricularis posterior; the nervus digastricus; the ramus anastomoticus with the nervus glossopharyngeus; the plexus paroticus; the ramus anastomoticus with the nervus auriculotemporalis; the rami temporales; the rami zygomatici; the rami buccales; the rami mandibulares; the rami colli.

The physiologic function of the nerve is still in dispute. Hunt calls the facialis a mixed nerve, claiming that the geniculate ganglion corresponds to a spinal ganglion. Others believe that the facial nerve is a purely motor nerve. Such variance of opinion can be explained on the basis of the nu-

merous connections of the nerve with other brain nerves and of its gross association with fibers of different function. There are, for instance, con-

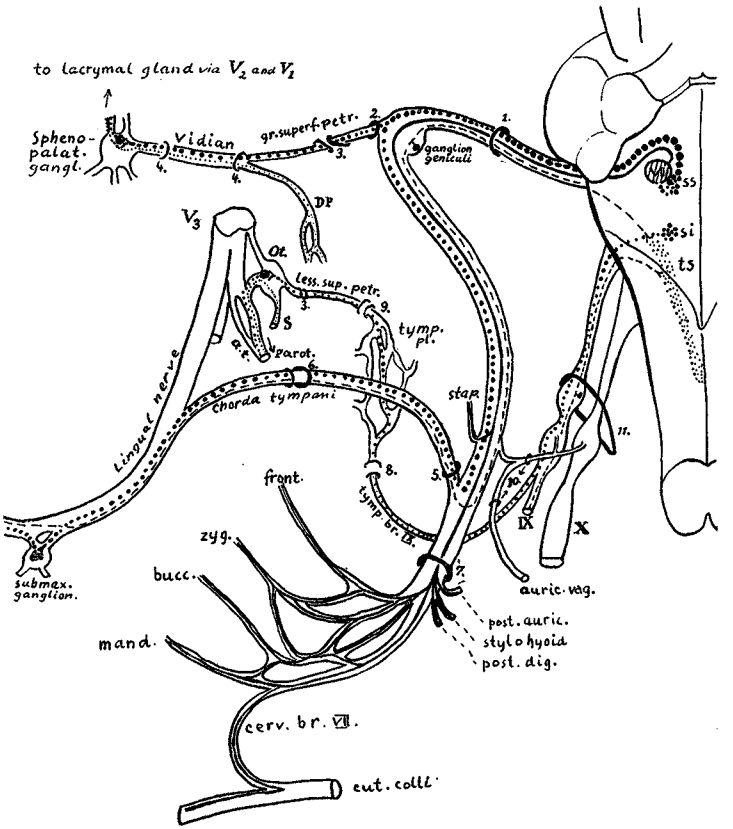


FIG. 65. DIAGRAM SHOWING BRANCHES AND CONNECTIONS OF FACIAL NERVE

*1.* = internal auditory meatus; *2.* = hiatus of facial canal; *3.* = foramen lacerum (represented twice); *4.* = vidian canal; *5.* = entrance of chorda tympani; *6.* = exit of chorda tympani; *7.* = stylomastoid foramen; *8.* = tympanic canaliculus; *9.* = opening in tegmen tympani; *10.* = opening for auricular branch of vagus nerve; *11.* = jugular foramen; *VII.* = motor nucleus of facial nerve in pons; *ss* = superior salivatory nucleus; *si* = inferior salivatory nucleus; *ts* = nucleus of tractus solitarius; *gr. superf. petr.* = greater superficial petrosal nerve; *DP* = deep petrosal nerve; *less. sup. petr.* = lesser superficial petrosal nerve; *tymp. pl.* = tympanic plexus; *parot.* = nerve to parotid gland; *a.t.* = auriculotemporal nerve; *stap.* = nerve to stapedius muscle; *tymp.br.IX.* = tympanic branch of glossopharyngeal nerve; *auric.vag.* = auricular branch of vagus nerve; *post.auric.* = posterior auricular nerve; *stylohyoid* = nerve to stylohyoid muscle; *post.dig.* = nerve to posterior belly of digastric muscle; *front.* = frontal branches of facial nerve; *zyg.* = zygomatic branches of facial; *bucc.* = buccal branches of facial; *mand.* = mandibular branch of facial; *cerv.br.VII.* = cervical branch of facial; *cut.colli* = nervus cutaneus colli of cervical plexus.

voying the facial nerve, fibers with secretory functions, such as the production of saliva, tears, sweat, or fibers with gustatory function.

The motor function of the facial nerve involves the following muscles: the muscles of expression, the rudimentary muscles of the pinna, the buccinator muscles, the stylohyoid, the stapedius, the posterior belly of the digastric, and the muscle fibers of the platysma. There is, however, no innervation of the muscles of the soft palate from the facial nerve, as as-

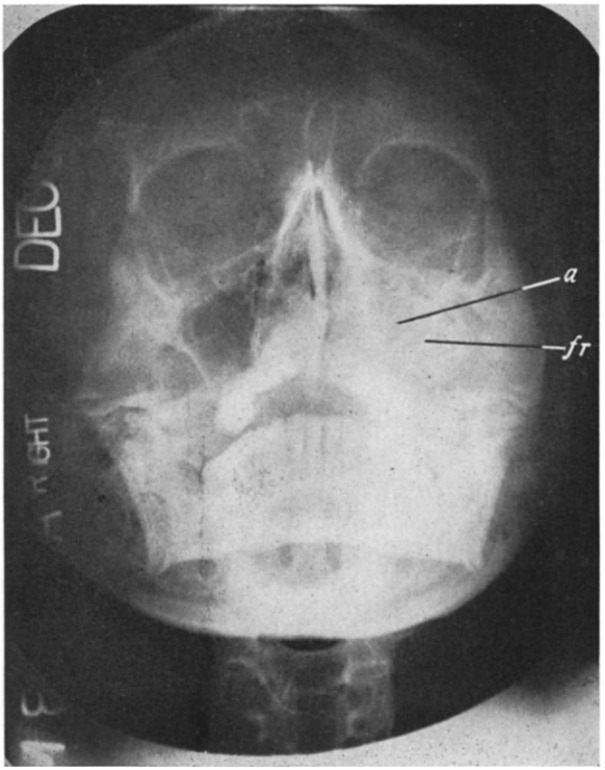


FIG. 65 A. X-RAY PICTURE OF FRACTURE OF LEFT MALAR BONE
*a* = antrum; *fr* = fracture.

sumed by many authors. It can therefore be stated briefly that the facial nerve represents the motor innervation for the mimetic musculature of the face. With respect to function, the facial nerve can be divided into three main branches—the eye, the nose, and the mouth branch. Each part shows a certain functional independence.

### 2. CLASSIFICATION AND ETIOLOGY

According to the site of the lesion, three types of facial palsy can be differentiated, namely, nuclear, cortical, and peripheral palsy. With

respect to the extent of the disease, one must distinguish between total palsy, i.e., affection of all three branches of the nerve, and partial palsy, in which only one or two branches are involved. With regard to the severity of the lesion, there may be either a complete palsy or a slight paresis of the nerve.

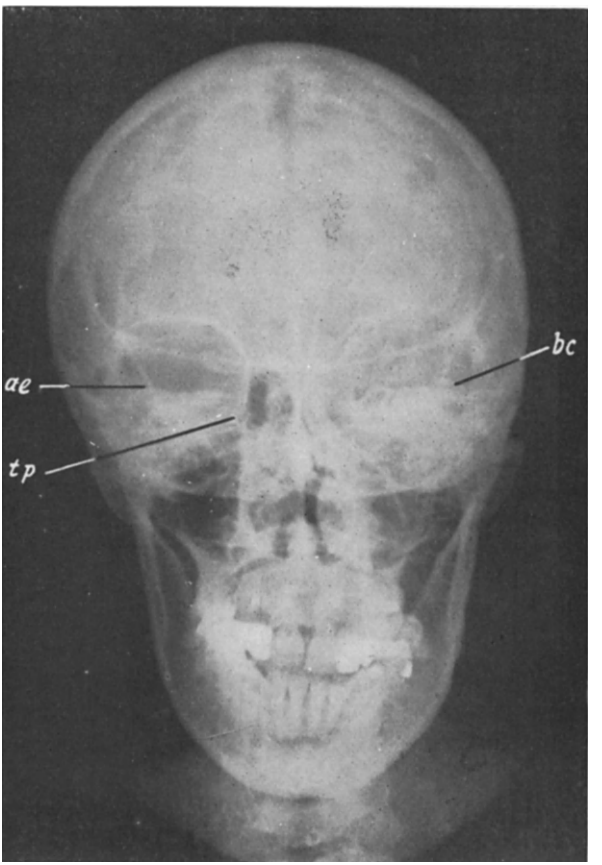


FIG. 65 B. X-RAY PICTURE OF FRACTURE OF APEX OF PYRAMID
*ae* = arcuate eminence; *tp* = tip of pyramid; *bc* = bone chip.

The *nuclear lesion* is found in diseases of the pons or of the medulla oblongata. The palsy usually occurs homolaterally, involves all three branches, and produces pathologic changes in the electric irritability of the nerve (p. 307). There is often a combination with palsy of the sixth nerve.

The *cortical lesion* is found in cases of meningitis or temporal lobe abscess or in extensive affection of the cerebellum with symptoms of pressure upon the fourth ventricle. The palsy usually occurs contralaterally, is only par-

tial (mostly in the mouth branch) and leaves normal electric irritability of the nerve.

The *peripheral lesion* is found in diseases that involve the nerve stem or any site from the internal auditory meatus to its termination. The palsy occurs homolaterally, is usually total, and produces pathologic changes in the electric irritability of the nerve. The peripheral palsy is the most important lesion and will therefore be discussed in detail.

From an *etiologic point of view*, the following groups must be distinguished: (*a*) otogenic inflammatory facial palsy, (*b*) facial palsy in connection with skull trauma, (*c*) facial palsy due to operative injuries, (*d*) facial palsy in connection with tumors, (*e*) facial palsy of unknown origin.

### OTOGENIC (INFLAMMATORY) FACIAL PALSY

Facial palsy may occur in the course of an acute otitis media. There are often signs of retention of pus within the middle-ear spaces or dehiscences in the fallopian canal.

In cases of chronic otitis media, tuberculosis or cholesteatoma is mainly responsible for the palsy, since either leads to extensive destruction of the bone. Facial palsy in the course of a labyrinthitis points to a spread of the infection into the internal auditory meatus (otitis interna purulenta complicata, p. 215).

### FACIAL PALSY IN CONNECTION WITH SKULL TRAUMA

The facial nerve is particularly endangered in cases of transverse fracture of the skull. The fracture line usually goes through the internal auditory meatus or the vestibule. In the latter case the fracture may reach the lateral wall of the vestibule, which is also the medial wall of the middle ear. This in turn may lead to a laceration of the facial nerve in the fallopian canal. If the palsy occurs right after the trauma, a tearing of the nerve seems likely, while a palsy in a later state points rather to a compression of the nerve due to a hematoma or a secondary infection. FIGURES 65 A and 65 B show roentgenographs of a case of traumatic facial palsy as recently observed by the writer; the fracture involved the malar bone and the petrous pyramid.

### FACIAL PALSY DUE TO OPERATIVE INJURIES

Facial palsy following surgical intervention in the mastoid may have different causes, such as a direct blow from a chisel or burr, simple exposure of the nerve at any place within the ear, hemorrhage into the nerve tissue, compression of the nerve by a sharp bony chip or by a sequester, or postoperative neuritis due to secondary infection of the nerve. The diagnosis depends largely upon the moment at which the palsy has occurred. A

sudden palsy during operation or immediately after makes the inference of a direct blow very likely; a palsy occurring a day after operation, with gradual progression, points rather to the probability of a hemorrhage or an exposure of the nerve. When, however, the palsy develops three to six days after the surgical procedure, slowly progressing in intensity, a secondary infection (neuritis) can be assumed. When labyrinthine symptoms occur simultaneously with the palsy, it may indicate that the inflammatory process is continuing into the inner ear and the endocranium.

#### FACIAL PALSY IN CONNECTION WITH TUMORS

To this group belong cases of palsy associated with acoustic tumors, cerebellopontile angle tumors, and the malignant neoplasm of the ear organ (p. 327).

#### FACIAL PALSY OF UNKNOWN ORIGIN

This group includes cases in which an apoplectiform palsy occurs in all three branches of the nerve following a severe cold (rheumatic palsy). It further includes cases of facial palsy associated with herpes zoster (herpes oticus, Koerner), with geniculate ganglionitis (Hunt), with toxic neuritis (Bell's palsy), and finally cases of facial palsy in the course of a polyneuritis cerebralis menieriformis (Frankl-Hochwart). It would go beyond the scope of this book to discuss all these forms in detail.

### 3. Clinical Signs and Symptoms

The clinical picture depends upon the site of the lesion and upon the underlying pathology. Whereas rheumatic palsy occurs suddenly and involves all three nerve branches, the otogenic inflammatory type often shows symptoms as forerunners of the palsy. Thus the case history may bear some significant clues. There may be, for instance, a patient with a chronic otitis media complaining of difficulties in eating, particularly liquid food. As often as he drinks he must wipe his mouth and he cannot understand why he is so clumsy. It is evident that this is not due to any clumsiness but to the slight paresis of the mouth branch of the facial.

The most common symptoms and signs of facial palsy involving the three nerve branches are:

Lesions of the *first branch:* The skin of the forehead of the diseased side is smooth, shows no wrinkles, and cannot be frowned. The eye cannot be closed, owing to palsy of the ring muscles (lagophthalmus). This in turn dries up the cornea, which may lead to a keratitis or even to an iridocyclitis. When the patient is ordered to shut his eyes, the ball moves upward (Bell's phenomenon). Further, no lid reflexes are elicitable on the diseased side. The tear secretion in the very beginning of the palsy usually increases but later decreases.

Lesions of the *second branch:* The nose is deviated toward the sound side,

the nasolabial fold is absent, and the nostril of the diseased side is somewhat narrower.

Lesions of the *third branch:* The mouth is deviated toward the normal side, the angle of the mouth is somewhat lowered on the diseased side, there is a flow of saliva on the affected side. The disturbances of the mimetic musculature can best be observed when the patient is asked to laugh, to whistle, or to say words with many labials.

The examination of the *electric irritability* of the nerve is another important factor for the prognosis and treatment of a facial palsy. In testing the irritability, one must differentiate between direct stimulation of the muscles and indirect stimulation through one of the nerve branches supplying the muscles. The stimulus used is either the continuous electric current (galvanic stimulation) or the interrupted current (faradic or sinusoidal stimulation).

In cases of peripheral palsy of the facial nerve, a change in the normal irritability usually takes place: one or two weeks after the onset of the palsy, a decrease can be noticed of the indirect irritability to galvanic and faradic currents and of the direct irritability to faradic currents. Direct galvanic stimulation, however, reveals an increase of irritability for a certain period of time (one to two months). Later on, when irreparable damage has occurred, the direct galvanic irritability gradually subsides.

Besides these quantitative changes of the electric irritability, there may also be qualitative alterations. The normal muscle responds with a jerking contraction; the cathodic closure contraction prevails over the anodal closure contraction. In cases of facial palsy the muscle responds in a slow, sluggish manner and the law of contraction is reversed, i.e., the anodal closure contraction predominates over the cathodic closure contraction.

If all the changes, quantitative as well as qualitative, are well marked, the phenomenon is spoken of as a "complete degenerative reaction" of the nerve. If, however, only some pathologic changes exist, it is called a partial degenerative reaction. In general, it can be stated, a complete degenerative reaction indicates a grave prognosis for the facial palsy. The same holds true for a partial degenerative reaction that gradually becomes complete. In cases, however, in which a short time after the onset of the palsy (two to three weeks) only quantitative changes occur, the prognosis is very favorable. Fowler found that in cases of severe injury to the facial nerve, associated movements of various parts of the face occur after recovery. He believes that these phenomena are caused by splitting of axons in the neuroma, so that one axon innervates several parts of the face.

The various localizations of lesion are:

Between the auriculotemporal nerve and the chorda tympani: palsy of the mimetic musculature, with reduction of mobility of the head and neck.

In the branch of the chorda tympani: palsy of the mimetic musculature, with disturbances of the sense of taste. In cases of complete destruction of the chorda, the potassium thiocyanate test yields a negative result.

In the branch of the stapedius nerve: palsy of the mimetic musculature, with tinnitus and hyperacusis. The latter symptom does not express an increase of hearing but a pathologic hypersensitivity to acoustic stimuli and should, therefore, be called dysacousis; the hearing acuity is decreased.

Lesion in the geniculate ganglion: palsy of the mimetic musculature, with disturbances of tear and sweat secretion, and gustatory disturbances.

Lesion within the internal auditory meatus: palsy of the mimetic musculature, with disturbances of tear and sweat secretion, but no gustatory disturbances.

### 4. Treatment

The therapeutic measures can be classified into three groups: (*a*) causal treatment, (*b*) conservative treatment, and (*c*) surgical repair.

#### CAUSAL TREATMENT

All efforts are directed to eliminating the cause of the facial palsy as quickly as possible. If the lesion has occurred in the course of an acute otitis media without any mastoid symptoms, a large paracentesis opening may suffice to cure the case. If, however, fever, pain, and drooping of the canal wall occur, a thorough antrotomy has to be performed.

Chronic cases indicate a thorough radical mastoidectomy with large exposure of the diseased bony walls of the middle ear. In the presence of labyrinthine signs and symptoms, a one-stage radical mastoidectomy with labyrinthectomy and exposure of the internal auditory meatus is necessary.

If facial palsy follows any mastoid surgery, an immediate change of dressing has to be made. For such a purpose the patient must be brought back to the operating room. A thorough inspection of the wound cavity will often reveal the cause of the palsy in the form of a sharp bony chip, or a fracture of the wall of the fallopian canal, or a sequester in the facial region, which can easily be taken care of. It may also be possible to find the site of the direct blow to the nerve. If both ends are dislocated, one can try to approximate them, which often brings about healing without any suture of the nerve.

In a case in which I was operating, in which the facial nerve had been cut by an otologist during a radical mastoid operation, there was a defect of just over 23 mm. Not being sure that I would be able to approximate

both ends of the nerve, my assistant cut down on the anterior femoral cutaneous nerve while I was removing more of the posterior external wall. To my surprise I was able to reroute the facial nerve so that there was good approximation, and it was unnecessary to use any other tissue.

Finally, there may be found a simple exposure of the nerve in a small area, with compression from too tight a packing, or with retention of secretion around the exposed nerve.

### CONSERVATIVE TREATMENT

The purpose of all the conservative methods lies in the prevention of degeneration of the muscles of the face. This can be achieved by use of the interrupted (faradic) current. The electric stimulus is applied to the nerve branches (indirect) as well as to the muscles (direct). The patient is given, once or twice a day through five to ten minutes, faradic current in an intensity he can just bear. In cases in which faradic irritability no longer exists, galvanic current is used. Should the galvanic irritability of the nerve also be lost, then direct muscular stimulation must be used.

Other conservative treatments supporting the electrotherapy are massage and exercises of the mimetic musculature. It is advisable to ask the patient to control his muscle exercises before a mirror. Some authors claim good results with diathermy, which is said to increase the blood circulation within the affected muscles.

### SURGICAL REPAIR

In 1830 Sir Charles Bell published his book on the nervous system of the human body, in which he discussed the anatomy and physiology of the seventh nerve. Faure in 1898 performed the first nerve anastomosis. Since then much has been written on facial paralysis. Ballance, Gillies, Cushing, Duel, Blair, Tickle, and Sheehan have made great contributions on this subject.

Much work has been done and many constructive efforts made for the relief or cure of facial paralysis. At the present time we have several appropriate operations dealing with the peripheral type of facial paralysis, which give satisfactory results.

When the facial nerve is intact and there is complete peripheral facial palsy, decompression of the facial nerve in its mastoid and intratympanic course is indicated, providing of course that galvanic stimulability is preserved and faradic stimulability is lost. Bell's palsy is perhaps the most frequent form of facial paralysis in these cases, and if no evidence of return of facial movement is seen by the end of six months, this operation is indicated.

Facial paralysis occurring with a chronic suppurative process in the

mastoid, but with faradic and galvanic stimulability present, requires a classic mastoidectomy and uncovering of the facial nerve in its mastoid and intratympanic course. The facial nerve is first located emerging from the stylomastoid foramen by opening the mastoid cortex and tip. The facial nerve is then exposed in its canal throughout the mastoid and intratympanic course, with removal of such mastoid cells and bone as is necessary. It is when the continuity of the facial nerve has been disrupted, and a restoration of this nerve pathway is sought, that various operations are undertaken.

There is first the end-to-end suturing of the severed nerve, or the insertion of a nerve graft between the distal and proximal ends of the nerve. If the defect in the facial nerve is less than 6 mm., then end-to-end anastomosis can still be achieved by traction on the proximal and distal ends of the nerve. If a defect of more than 6 mm. is encountered, there is still a possibility of end-to-end anastomosis by rerouting the facial nerve wherever possible. This can be done in cases where there is as much as 23 mm. of defect. There are some cases, however, in which end-to-end anastomosis, even by rerouting, is found to be impossible. In these cases a nerve-grafting operation is advised. First a radical mastoidectomy is performed, followed by adequate exposure and decompression of the facial nerve. The damaged part of the nerve is excised and a nerve graft is taken from the anterior femoral cutaneous nerve. The section needed is inserted between the proximal and distal segments of the facial nerve in such a way that the ends are in accurate apposition. The graft area may be covered with gold foil (sutures may or may not be used to hold the graft in place). No antiseptics, hemostatics, or local anesthetics are used. The area should be free of blood when the nerve graft is inserted. Dressings are changed every two to four days. The postauricular incision is left open to facilitate postoperative dressings, and is closed by plastic operation after the graft becomes covered with healthy granulation tissue.

In cases, however, in which the injury to the facial nerve is proximal to the geniculate ganglion and distal to the stylomastoid foramen, or in which the nerve throughout its course has been destroyed, recourse must be had to anastomotic operations to restore the facial movements. Anastomosis of the distal segment with the spinal accessory, the hypoglossal, and other motor branches in the neck is used. It is preferable, however, to use a spinal facial anastomosis whenever possible. The spinal accessory nerve is exposed as it penetrates the sternocleidomastoid muscle, through an oblique incision beginning about 1 cm. behind the ear, well above the mastoid tip, and carried down along the anterior border of the muscle to the level of the cricoid cartilage. The nerve is freed from the underlying soft tissue as it passes upward beneath the posterior belly of the digastric muscle. The

spinal accessory nerve is sectioned close to the muscle and the proximal end carried under the posterior belly of the digastric muscle and sutured to the distal end of the facial nerve trunk with fine silk. The distal end of the spinal accessory nerve is turned downward, and after division of the descending branch of the hypoglossal nerve, it is sutured to the proximal end of the descending branch of this nerve, in order to maintain tone in the muscles supplied by the spinal accessory nerve. In the event that diagnosis does not promise full functional restoration by nerve grafting, another method is available. Facial muscles that have fallen into disuse may be reanimated by contact with other muscles that retain their innervation.

There are many methods of *muscle suspension*. In cases in which there is a degeneration of the peripheral portion of the nerve and atrophy and fibrosis of the facial muscles, operation for reanimation of facial muscles by direct nerve suture and nerve graft or anastomosis with a contiguous motor nerve cannot be done. Therefore, other mechanical procedures have been used, such as fascia lata strips, as popularized by Blair, or muscle transplants from the temporal muscle, which are relocated in strategic positions. Lexer, Gillies, and Sheehan have various methods of using the temporal muscle. Halle's operation, in which he uses the pedicles in such a way that when they are relocated the motor action of the muscle still runs from origin to insertion, is in my opinion the best method of all. The insertions of muscles are cut at the point of attachment and not at the point of origin, as is done by Lexer, Gillies, and Sheehan. A reversed fishhook incision is made with complete cut of skin and subcutaneous tissue as far forward as the nose and mouth, and as far downward as the mandible, care being taken to avoid the parotid duct in front of the ear, and extending below and around the ear.

The operation is done under local anesthesia by injection of the sphenopalatine ganglion. The middle third of the temporal muscle is divided, after the separation of the fascia covering it, from its attachment to the lip of the coronoid process of the mandible. It is brought forward, one segment being placed in the substance of the occipitofrontalis muscle above the eyebrow, the second slip inserted into the orbicularis in the upper lip, and the third slip into the orbicularis in the lower lip, as far to the median line as possible. The anterior half of the masseter muscle is freed from its attachment to the inferior border of the mandible and is brought forward in the substance of the cheek through the incision in the nasolabial fold. One attachment is inserted into the angularis muscle of the nose, the second into the orbicularis of the upper lip, and the third into the orbicularis of the lower lip, as far to the median line as possible. All pedicles are sutured into their new positions with medium-fine silk sutures. At the same time a cosmetic meloplasty is done, with removal of all re-

dundant skin adjacent to the nasolabial fold and along the line of the reversed fishhook incision.

Union of the temporal fascia is made by means of interrupted figure-of-eight sutures of fine chromic catgut; the skin sutures are of interrupted medium-fine silk. All skin sutures are removed within three to four days at the latest and replaced with coaptive devices to maintain incision apposition.

Sheehan's operation appears to give the best results. First, reductions are made in the bony arches beyond which the temporal muscle strips are to pass. There are two reasons for this. One is that the disfiguring protuberance that would be caused by the strips passing over is avoided. Second, the ends of the strips can be carried farther—far enough in one direction to invest the muscles about the nose and mouth, and in the eyelid pair far enough to meet at the inner orbital canthus. The muscles about the mouth and nose receive the largest strip of temporal muscle. It is here turned on its pedicle and brought down through a pouch made by undermining the skin. Reanimation in this area of the mouth and nose operates to correct the imbalance of forces that accounts for the characteristic distortion. Next, two narrow half-thickness strips are turned to the orbital area, with the eyelid skin undermined. These strips are carried close up to the rim margins to the inner canthus, where they are made fast to each other and to the periosteum. According to Sheehan, return of motion to the upper lid is frequently observed before the patient leaves the operating table.

The third phase of the procedure is directed to assuring activity and, as far as possible, coordination of the muscles of the forehead and eyebrow regions. A single strip is carried to that area and its end strands are interwoven with those of the muscle below. Fat and fascia are introduced in the denuded areas.

Brunner aims to correct only the mouth branch of the facial nerve. Using local anesthesia, he makes an incision in the vestibulum oris along the mandible backward and along the anterior border of the masseter muscle upward. After exposure and incision of the masseter muscle, the inferior insertion of the anterior part is cut off from the mandible, and the muscle flap is fixed by one or two bridle sutures. From an arched incision through the mucous membrane near the angle of the mouth, a tunnel to the masseter muscle is made, and the flap is pulled forward and anchored to the angle of the mouth.

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# X

# Congenital Diseases

By *Joseph Fischer*

The old classification of congenital and acquired diseases is based upon etiologic and clinical points of view. Because of its simplicity it is generally used. Deaf-mutism, for example, is classified as congenital in conjunction with a positive family history. The diagnosis of an acquired deaf-mutism is made either when this symptom is absent or when reports show that trauma or infections have been present. From a scientific point of view, such a distinction does not seem justified. Modern research on the subject of constitution has shown that there is a difference between lesions introduced by the germinal substance (constitutional changes) and changes acquired during the intra- or extra-uterine period of life (conditional changes). The term "congenital" includes, therefore, all constitutional and all intra-uterine acquired diseases. On the other hand, the constitutional disturbances are not necessarily always congenital but may occur in a later period of life. In other words, constitutional lesions need not be congenital, while congenital lesions need not be constitutional. In contrast to the old differentiation, this classification is based upon scientific points of view. It must, however, be admitted that difficulties often arise in the diagnosis. There are many constitutional anatomic changes that show a great resemblance to those found as results of inflammation.

The congenital diseases of the inner ear can be differentiated as (*a*) changes of the bony inner ear and (*b*) changes of the nerve and sensory apparatus.

## 1. Changes of the Bony Inner Ear

### Systemic Diseases of the Bones

This group includes rare forms such as osteogenesis imperfecta, chondrodystrophia foetalis, congenital myxedema (endemic cretinism), etc. I made the first investigation of the ear organ in osteogenesis imperfecta and found disturbances of the enchondral and periosteal ossification, with the result of an underdevelopment of the bony labyrinthine capsule; Figure 66 illustrates the abnormal shape and unusually small size of the bony cochlea in the case studied. Later a second case of osteogenesis imperfecta was described by Nager with similar findings. Nager further investigated 4 cases of chondrodystrophia and found changes in the enchondral

inner-ear capsule, with an increase of the intraglobular cartilage. In examining the ear organ of one case I found the cartilage cells unusually small and the zone of proliferation of chondrodystrophia much narrower than normal. Otologic studies of the petrous bones in cases of congenital myxedema (endemic cretinism) revealed an increased periosteal ossification of the inner-ear capsule (Nager). The disturbances of development of the inner ear in the systemic diseases of the bones are as follows: changes concerning the enchondral ossification (chondrodystrophia); changes concerning the periosteal ossification (congenital myxedema); and changes concerning the entire ossification (osteogenesis imperfecta).

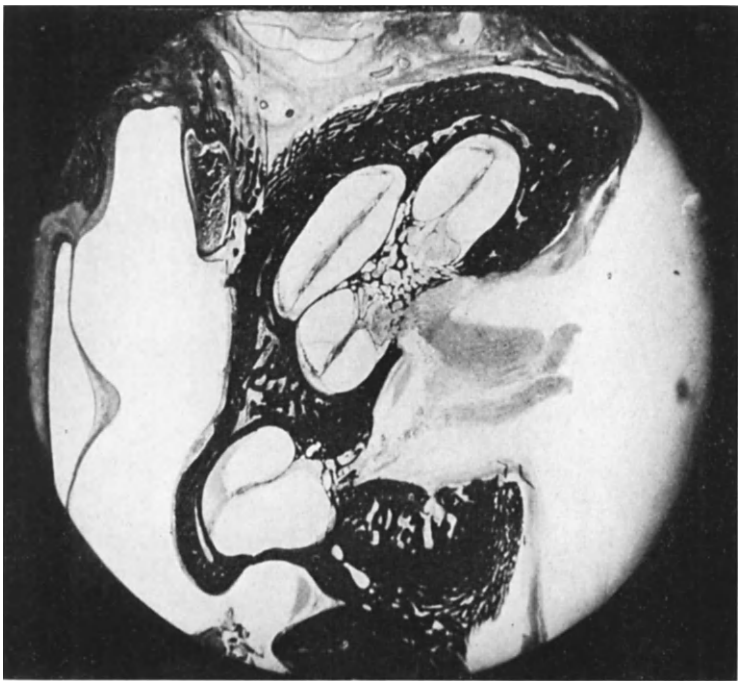

FIG. 66. PHOTOMICROGRAPH OF CASE OF OSTEOGENESIS IMPERFECTA: AXIAL SECTION THROUGH COCHLEA, SHOWING AMAZING FLATNESS AND SMALLNESS OF BONY COCHLEA

### OTOSCLEROSIS

Alexander classified otosclerosis with the congenital diseases (p. 183). Politzer had already emphasized that otosclerosis represents a primary disease of the bony labyrinthine capsule. There are usually circumscribed areas of pathologically changed bone. These bony spots occur in certain

places, such as the oval or round window, the prominence of the horizontal canal, or the region of the internal auditory meatus. Alexander was the first to state that these otosclerotic centers are of congenital origin. They may remain for many years in a latent state. In certain age periods, mainly in the stage of puberty, however, they start to grow and become manifest. Bauer and Stein examined patients with otosclerosis for clinical signs of degeneration. The great number of such signs (status degenerativus) and the frequency with which they occurred in the various patients led the authors to the belief that otosclerosis has a heredodegenerative origin. In 1920, in an article entitled "The Constitutional-congenital Character of Otosclerosis [etc.]," I described a number of morphologic anomalies of the inner ear that I considered as expressions of a constitutional inferiority of the ear organ.

#### ARRESTED MALFORMATIONS

This group includes cases of deaf-mutism showing lack of a cochlear whorl, defects of the modiolus or of the septi of the scales, etc. In the absence of the bony spiral membrane, there is one scala communis instead of a scala tympani and a scala vestibuli. Brunner states that these anomalies are connected with the development of the membranous cochlea, which, however, has no influence upon the ossification of the bony labyrinthine capsule. Guild considers these anomalies as overdifferentiations rather than as arrest of development.

### 2. Changes of the Nerve and Sensory Apparatus

Complete aplasia of the acoustic nerve is extremely rare. There is more often an atrophy of the nerve. The nerve bundle is thinner than usual and contains more connective tissue than true nerve fibers. It may often be difficult to distinguish such a primary atrophy from the secondary degeneration found as a result of intra-uterine inflammatory processes. Atrophy may not only involve the nerve stem but may also be found in the peripheral distributions of the nerve and in the ganglia.

As far as the sensory organ is concerned, a complete aplasia is extremely rare. The hypoplasia includes quantitative as well as qualitative changes. There is either a decrease in the number of hair cells, or a loss of their cilia, or changes in the structure. The cochlear apparatus is far more often involved than the labyrinthine part of the inner ear. This can be explained on the basis of the difference between them in resistance (p. 317). The two most common congenital diseases of the cochlear apparatus are deaf-mutism and chronic progressive inner-ear deafness. Because our discussion deals chiefly with the labyrinth, these conditions will be only briefly described.

### DEAF-MUTISM

The most common form of deaf-mutism is the sacculocochlear type. The degenerative processes include the nerve and the sensory organs of the sacculus and the cochlea, whereas the utriculus and the three semicircular canals are normally developed. This discrimination can be explained on the basis of embryologic and phylogenetic factors. The inner ear develops from two different portions: the pars superior gives origin to the utriculus and the three canals; the pars inferior, to the cochlea and the sacculus. The derivatives from the pars superior can be traced back in phylogenetic descent as far as the lowest types of animals. The cochlea, however, occurs first in amphibians and birds, while the characteristic spiral slope is found only in the higher species of mammals. This great difference in the phylogenetic age of the two parts is expressed in their different resistances (Alexander).

The sacculocochlear type represents 60 to 70 per cent of congenital deaf-mutism. The *clinical* picture is characterized by loss of hearing while the labyrinthine excitability remains intact. The case history contains reports of heredity in the direct ascent or of the occurrence of deaf-mutism in various members of the same family. There are occasionally heredodegenerative signs in other sensory organs, for example, in the eye. In some cases a consanguineous marriage of the parents is revealed. Great care must be applied, however, in taking the history. It is a psychologic fact that the parents usually try to find some external cause for the deaf-mutism, such as trauma or diseases of childhood. In doubtful cases it is better to rely on results of functional tests than on the case history. Deafness with eventual remnants of hearing (tone islands), associated with normal labyrinthine function, points rather to the heredodegenerative type. A condition in which there is complete loss of hearing and of labyrinthine function favors more the diagnosis of acquired deaf-mutism. It seems logical that an inflammation of the inner ear—tympanogenic or meningogenic—will not stop at an ideologic landmark but will rather involve the whole inner ear.

### CHRONIC PROGRESSIVE INNER-EAR DEAFNESS

This disease shows a striking relation to deaf-mutism. Alexander and Manasse found a progressive atrophy of nerve fibers in certain portions of the acoustic nerve. The destroyed nerve elements were replaced by connective tissue. The *clinical* picture is characterized by a progressive decrease of hearing in the presence of a normal drum membrane and a normal eustachian tube. The tuning fork test reveals a shortened bone conduction and a decrease of the upper tone limit. The labyrinthine function is

usually intact. The hereditary origin and the underlying pathology explain the failure of any treatment.

### ANOMALIES OF THE LABYRINTH

Heredodegenerative changes of the labyrinth are not so frequent by far as such changes of the cochlear apparatus. Because of lack of any clinical

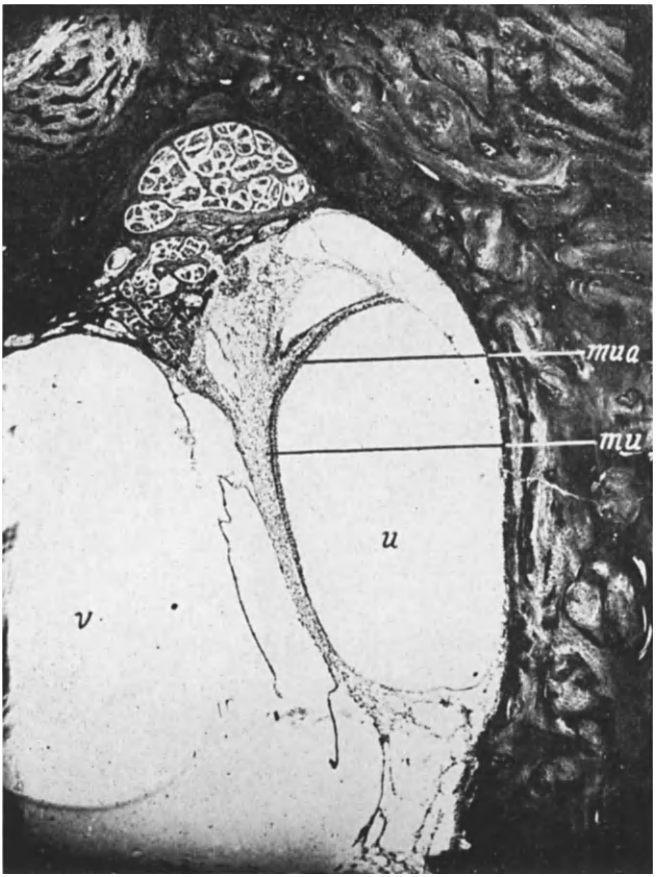


FIG. 67. PHOTOMICROGRAPH OF VERTICAL SECTION THROUGH INNER EAR SHOWING VESTIBULE (*v*) WITH UTRICLE (*u*): NEURO-EPITHELIAL FOLD, CALLED MACULA UTRICULI ACCESSORIA (*mua*), ON MACULA UTRICULI (*mu*)

symptoms they are usually accidentally discovered on autopsy. Hence they have not much clinical importance.

To the minor forms of labyrinthine anomalies belong the various atypical epithelial formations, epithelial segmentations, cysts, crests, and perilymphatic septa. These epithelial formations usually lie in the medial

part of the utricle near its bottleneck and in the lateral wall of the sinus utricularis inferior. In a very rare anomaly on the macula utriculi that I have described, there was a circular fold of neuro-epithelium segmenting the utricle (FIG. 67). In a topographic sense its position was vertical to the macula utriculi and also to the macula sacculi; it therefore was in the third dimension. I applied the term macula utriculi accessoria and compared it, in a physiologic sense, with the lagena of animals.

Complete aplasia of the labyrinth has been described in only one case in the literature (Michel). Aplasia or hypoplasia of some nerve endings or obliteration of endolymphatic spaces has occasionally been observed.

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# XI

# Neoplasms

By *Joseph Fischer*

## 1. Neoplasms of the Inner Ear

Primary tumors of the labyrinth are extremely rare. There is usually an extension of a neoplasm from any part of the ear organ or within its vicinity into the inner ear. These secondary tumors may further represent metastases of a malignant growth of a remote organ. The most common types of secondary neoplasm are (*a*) endothelioma, (*b*) sarcoma, (*c*) carcinoma.

### Endothelioma (Meningeoma)

Tumors of this type occur more often than the literature shows because of the great dissension over nomenclature. Many cases therefore appear as carcinoma or sarcoma. Borst distinguishes endothelioma of lymphatics, of blood vessels, of serous surfaces, and of the dura, on the basis of origin. Dura endothelioma, however, has to be considered as a type by itself. It may derive from the endothelium or the stroma of the dura or from the arachnoidal processes in it. The latter form has particularly been stressed by Cushing (meningeoma). There are only a few histologic investigations of the ear organ. I published 2 cases which I observed clinically and examined histopathologically. One of them was that of a 5-year-old girl with a marked swelling of the orbitotemporal region. On postnasal examination a tumor of the pharyngeal vault was visible. Autopsy revealed a new growth extending from the base of the skull to the middle cranial fossa, penetrating the orbit and also the eustachian tube. Histologically all cavities of the middle ear were filled with tumorous masses. Remarkably, the neoplasm was confined to the anatomic landmarks of the middle ear. There was, however, a slight penetration in the region of the horizontal canal and at the top of the bony cochlea. The facial nerve and geniculate ganglion were both infiltrated by tumor cells.

The other case was that of a 47-year-old woman who died of a heart attack. The otoscopic and functional examinations gave normal findings. On autopsy, a large meningeoma of the posterior cranial fossa was found. The size of the tumor made it difficult to determine its origin. The histologic examination, however, pointed to an origin in the area of the knee of the lateral sinus; the tumor had grown medially up to the vestibular aqueduct along the posterior pyramidal surface. There was also a marked dilatation of the cerebral ostium of the cochlear aqueduct. Striking enough was an intense hydrops of the sheath of the ninth nerve. The membranous inner ear, however, did not show any changes.

SARCOMA

It is usually very difficult to determine the exact origin of a sarcoma. It may either develop in the middle ear, growing into the inner ear, or it may derive from the periosteum of the temporal bone, or of the eustachian tube,

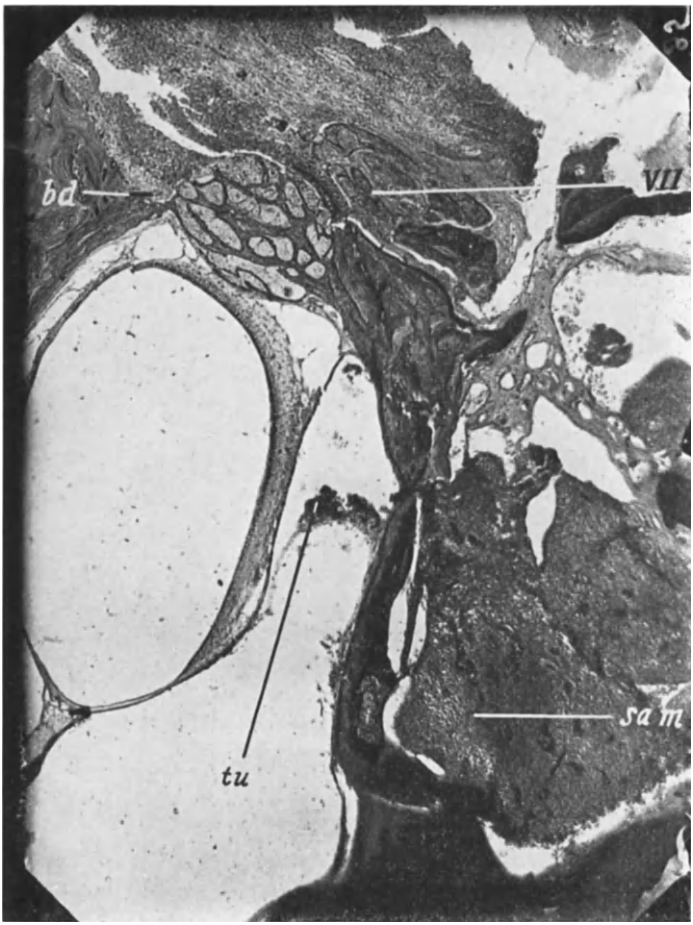


FIG. 68. PHOTOMICROGRAPH OF CASE OF SARCOMA OF PETROUS BONE: VERTICAL SECTION THROUGH MIDDLE AND INNER EAR

*sa m* = large sarcomatous masses in oval window and around facial nerve (*VII*); *bd* = destruction of bone of labyrinthine capsule in region of nervus utriculo-ampullaris; *tu* = accumulation of tumor cells in perilymphatic cistern between footplate of stapes and macula utriculi.

or of the carotic canal, etc. Myelogenic sarcomas originate in the spongious parts of the petrous bone. Most malignant of all the sarcomas is the round-cell type that shows diffuse infiltrations. I published the case of a 62-year-old woman who had a highly vascularized tumor of the petrous

bone. It consisted of large round cells with large nucleus and small protoplasm. The neoplasm had destroyed the tegmen tympani, invading the base of the middle cranial fossa. There was a marked bone destruction in the region of the canalis utriculo-ampullaris and in the canalis facialis. The perilymphatic cisterna contained many tumorous cell masses. The growth had further destroyed the bony labyrinthine capsula, thus penetrating the membranous cochlea.

Sarcomas of the petrous bone usually have no tendency to metastasize.

### CARCINOMA

Carcinoma practically always develops on the basis of a chronic otitis media. It is an amazing fact that these neoplasms usually are skin cancers, although the middle ear is lined by mucosa. The true columnar cell carcinomas occur very seldom. Efforts have been made to explain this fact. Some authors believe that a change of the normal epithelium takes place owing to the chronic inflammation (metaplasia theory). I investigated microscopically many cases of carcinoma and of cholesteatoma, since the latter was also considered as metaplasia. I was unable to find such a metaplasia in any of my cases. Other authors (Habermann) believe that dermic epithelium from the external ear grows into the middle ear, thus leading to a skin carcinoma. This theory, which is more plausible, I could corroborate, since I have noticed skin layers in the middle ear that obviously had passed the edges of the perforation of the drum. An inward growth of the epidermis from the external auditory meatus may cause one of the following changes: (*a*) the surface growth of the epidermis continues until a complete epithelization of the tympanic cavity has taken place—this in turn is identical with perfect healing; (*b*) the surface growth is replaced by proliferation of epidermis into the depth, leading to a formation of cholesteatoma pearls; (*c*) the surface growth is replaced by proliferation of epidermis into the depth, leading to a formation of cancroid pearls.

After many years of study on the pathogenesis of carcinoma and cholesteatoma, I addressed the New York Academy of Medicine in 1930 on the subject, "Analogy between Cholesteatoma and Skin Cancer," essentially as in the discussion that follows.

Before discussing the details of the pathology, it is necessary to understand the physiologic growth of the skin. The normal processes of dermic growth are characterized by two attributes: (1) marked tendency to proliferation, and (2) tendency to surface growth, which is not surprising when we remember that we have before us protective epithelium. This physiologic growth tendency is familiar to us and we make clinical use of it, for instance in radical operations where we cover the surgical wound with a skin flap in the expectation of thus stimulating epithelization and achieving ultimate recovery.

The pathologic change arises only after the protective epithelium has lost its physiologic function and grows downward. In this event digitate protuberances develop—so-called atypical epithelial formations—followed by the misplacement of the protective epithelium into the basal connective tissue, so-called epithelial heterotopia. In examining these sections under more powerful magnification, their close resemblance to early pavement carcinoma is striking (FIGS. 69, 70). The resemblance increases the more

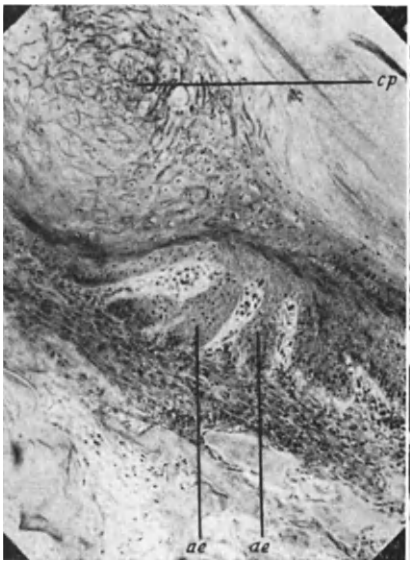


FIG. 69

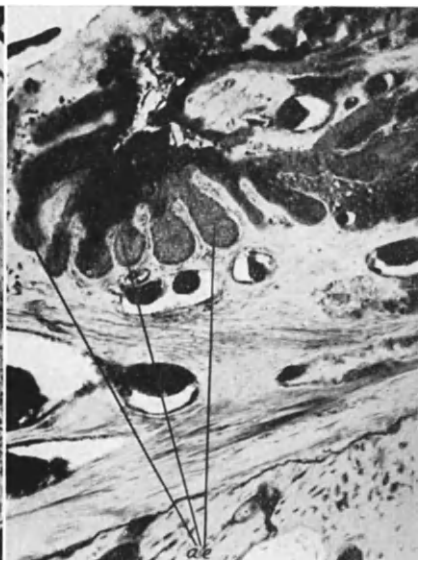


FIG. 70

FIG. 69. PHOTOMICROGRAPH OF CASE OF CARCINOMA OF MIDDLE EAR: REGION IN HYPOTYMPANUM UNDER HIGH MAGNIFICATION

*ae* = atypical epithelial formation in form of digitate protuberances; *cp* = cancroid pearl.

FIG. 70. PHOTOMICROGRAPH OF CASE OF CHOLESTEATOMA OTITIS MEDIA: REGION IN HYPOTYMPANUM UNDER HIGH MAGNIFICATION

*ae* = atypical epithelial formations in form of digitate protuberances.

these epithelial protuberances and islands invade the deeper tissues, particularly after formation of concentric layers of pavement epithelium exhibiting beginning or completed cornification in a central direction. The fact of this resemblance is expressed by designating the lamellar bodies in cholesteatoma as cholesteatoma pearls and in carcinoma as cancroid pearls.

What causes the loss of the physiologic surface growth of the protective epithelium and its migration to deeper tissues? Why does cholesteatoma result in one case and carcinoma in another? What is the cause of this

pathologic growth tendency? Histopathology does not satisfactorily explain these problems. In these growth anomalies we have before us microbiochemical cell processes. According to Cermak, three factors are chiefly involved in the physiologic growth process: (*a*) the endocrines; (*b*) the nervous system; and (*c*) the individual cell with its own growth energy. The undisturbed functioning of all three factors produces physiologic or surface growth. Pathologic growth results from disturbance of one of these factors. Disturbances may become manifest as incitation or transmission of the stimuli regulating growth. Or the cell itself may change, for instance, in sensory receptivity. Orth considered the transformation of epithelial into carcinoma cells the result of a long developmental series of progressive changes in the epithelial cell caused by injury of the cell's own inhibitory mechanism, with subsequently enormously increased ability of the epithelium to grow and proliferate.

For a clearer interpretation of these hypotheses I should like to recount the studies on the production of experimental carcinoma, or so-called tar carcinoma. The first experiments gave negative results. Only chronic dermatitis and hyperkeratosis were established in animal experimentation. Microscopically the sections demonstrated atypical epithelial formation and epithelial heterotopia. In later experiments continuous and prolonged tar painting produced typical skin lesions. How shall we explain this? The tar paintings produce persistent slight tissue lesions of the protective epithelium, which the organism tends to heal. Since regenerative processes remain, according to Sternberg, for a long time within normal limits, these persistently prolonged slight tissue lesions later stimulate regenerative ability beyond physiologic limits. Of course, the insult should be accurately graded (B. Fischer), otherwise the tissues become too severely injured and necrose, or induced regeneration is too weak, so that nothing happens.

Thus the stimulus must be of a definite intensity and prolonged duration. The inherent inhibitory mechanism of the cell is injured only by repeatedly stimulated regeneration, which produces irregular downward proliferation. Consequently the protective epithelium loses its physiologic growth tendency and produces atypical epithelial formation and epithelial heterotopia. In the deeper tissues concentric epithelial formation results in cholesteatoma or cancroid pearls, respectively.

For the etiologic factor tar in artificially produced carcinoma we substitute chronic middle-ear suppuration in cholesteatoma, or rather carcinoma of the ear. It represents the chronic persistent stimulus of graded intensity, neither too severe nor too weak, thus causing downward growth.

Up to this point the two processes are identical. In cholesteatoma as in carcinoma, chronic slight prolonged tissue insults, with subsequent cell

regeneration, are requisite. Both processes first produce atypical epithelial formation and epithelial heterotopia associated with the formation of epithelial pearls showing central cornification. Their further progress differs, however. Carcinoma exhibits asymmetric and pluripolar mitosis, including degeneration and loss of cell chromosomes, and displays greater variety in rapid growth. Cell proliferation in cholesteatoma is succeeded by rapid decay, thus preventing autonomous cell proliferation. The different structures of the dermic pearls can be histologically demonstrated. In cholesteatoma the matrix consists of proliferating living protective epithelium, and the other layers consist of dead cornified desquamated epithelium. In carcinoma, on the other hand, almost every layer of the cancroid pearls shows proliferating, more or less immature young cells with central cornification only.

A case of carcinoma of the petrous bone that I observed clinically and examined histopathologically was that of a 60-year-old man, who developed, on the basis of a chronic otitis media, a squamous cell carcinoma that caused destruction of almost the entire petrous pyramid. Microscopic examination revealed extensive carcinomatous epidermis proliferation in all spaces of the middle ear; the footplate of the stapes and the secondary typanic membrane were destroyed, which led to a free communication between the middle- and inner-ear spaces. Yet the inner ear contained no tumor cells, but debris and necrotic masses. The membranous inner ear, cochlea as well as labyrinth, was completely destroyed. It was amazing how much resistance the dura mater had shown. Although exposed by the extensive bone destructions and covered by tumorous masses, the dura was not perforated. The growth had further replaced the facial nerve, leaving but a few fibers intact. In spite of these large destructions of the petrous bone, there had not been any metastasis in any organ of the body.

The frequent involvement of the inner ear in the course of secondary malignant neoplasm has a threefold explanation: (*a*) there is a continuous extension into the inner ear; (*b*) there are changes in the inner ear due to disturbances of the flow of fluid (endo- and perilymph); (*c*) there are changes due to the action of toxins.

The *clinical diagnosis* of a neoplasm of the labyrinth often meets with great difficulties, since there are no characteristic symptoms. The slight cases often have no symptoms. In severe cases the elimination of cochlear and labyrinthine function gradually develops without any violent reaction. Hence the disclosure of such a tumor in the inner ear is usually accidently made by the autopsy or by the microscopic examination of the petrous bone. In the case of carcinoma described above, the patient was sent to me with the diagnosis of an external otitis. He showed a swelling

of the pinna, of the external meatus, and of the retro-auricular glands. Owing to the blockage of the canal lumen, the drum membrane was not visible. Irrigation of the ear by the use of an attic cannula brought out a milky emulsion containing fine particles of "bony sand." This led me to think of a tuberculous otitis media. On functional testing complete deafness and loss of labyrinthine excitability were found; X-ray examina-

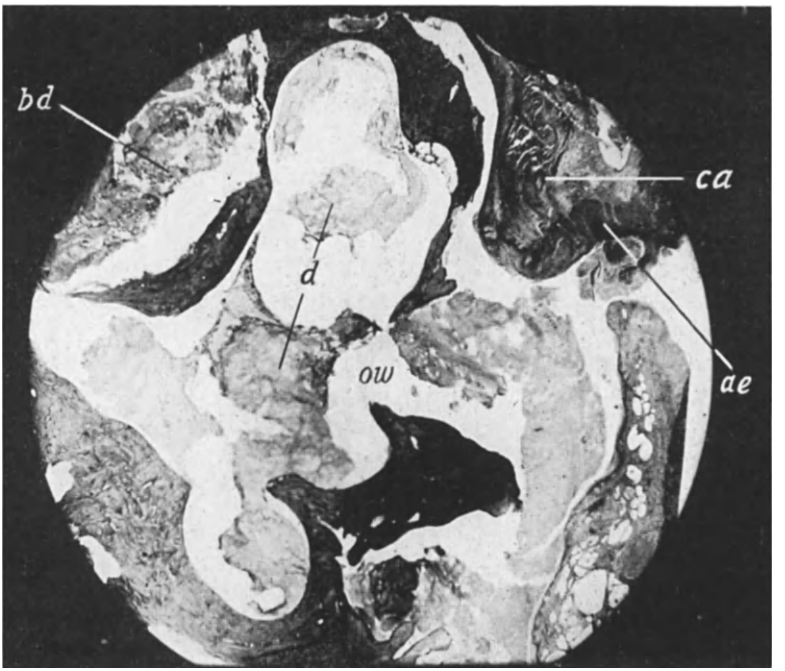


FIG. 71. PHOTOMICROGRAPH OF CASE OF CARCINOMA OF MIDDLE EAR: VERTICAL SECTION THROUGH MIDDLE AND INNER EAR

Tympanic cavity is partly filled with debris (*d*), partly with carcinomatous masses (*ca*): even under this low magnification, digitate epithelial formations (*ae*) can be seen. Inner-ear spaces are entirely filled with debris (*d*). Owing to destruction of stapes plate, oval window (*ow*) shows large communication. Labyrinthine capsule shows widespread bone destruction (*bd*).

tion showed large destruction of the pyramid. The real diagnosis, however, was furnished by the biopsy, which revealed a skin cancer.

Paralysis of the facial nerve is considered as a frequent symptom in malignant neoplasm. This I could not prove. A number of my cases showed no clinical signs, although microscopic examination revealed an infiltration with tumor cells in the facial nerve or in the geniculate ganglion. This shows that there is a certain resistance of the nerve tissue.

## 2. Neoplasms of the Cerebellopontile Angle

This term was applied by Henneberg and Koch to tumors located in the angle formed by the cerebellum, medulla oblongata, and pons. There is, however, no uniformity in the nomenclature, since some authors classify within this group only the true acoustic tumors (neurinoma), while others include all growths occurring in this space. Henschen suggests applying this term to tumors located in the space where the seventh and eighth nerves run in a horizontal and frontal direction. This space contains further the flocculus of the cerebellum, the posteromedial part of the lateral recessus, and the blood vessels going to the cerebellum. We must distinguish between (*a*) the true acoustic tumors and (*b*) the other cerebellopontile angle tumors.

### Acoustic Tumors (Neurinoma)

The great attention particularly given to this group of tumors can be explained by their clinical importance. The local diagnosis usually can be made easily because of the very characteristic initial symptoms. On the other hand their slow growth, relative benignancy, and tendency to stay well confined make them susceptible to favorable surgical intervention.

The *histogenesis* of these neoplasms is still in dispute. Some authors identify these growths as glioma, gliofibroma, fibrosarcoma, etc. Verocay regards their histologic structure as that of a neurogenic tissue derived from the ectodermal cells of the sheath of Schwan, and applied the term neurinoma. Henschen was the first who could demonstrate the origin of the tumor from the distal part of the vestibular nerve within the internal auditory meatus. From there the neoplasm usually grows toward the pontile angle. Cushing, who considers the neoplasm as a mixed tumor, uses the term fibroneuroma. In recent years Brunner suggested a new classification based upon a pathologic-anatomic and clinical point of view. He differentiates between lateral and medial acoustic tumors. The former develop in the fundus of the internal auditory meatus and penetrate the petrous bone, the latter develop in the free portion of the eighth nerve (located in the cisterna pontis lateralis) and penetrate the posterior cranial fossa.

The *pathologic-anatomic* changes in the inner ear are either caused by the expansive growth of the tumor or are the result of compression. There are often a marked enlargement of the internal auditory meatus and atrophic degenerations in the peripheral sense organ. The numerous, often contradictory findings reflected in the literature are summarized by Mann as follows: When the acoustic tumor has interrupted the continuity of the eighth nerve, the result will be a complete degeneration of the cochlear nerve, its peripheral distribution, and the hair cells of the organ of Corti.

The vestibular branch of the eighth nerve, however, shows the same changes only when the ganglion of Scarpa is also involved.

In contrast to the sensory cells, the supporting apparatus usually remains intact. Brunner distinguishes sharply between changes in the lateral and in the medial type of acoustic tumors. The former are associated with enlargement of the internal meatus and with degeneration of the cochlea and the labyrinthine nerve apparatus, the latter show rather a narrowing of the internal canal due to hyperostosis, and only slight degenerative changes of the cochlear apparatus.

For the *clinical diagnosis* of angle tumors, Eagleton, Cushing, Grant, Fisher, and Jones described a complex of symptoms termed "cerebellopontile angle lesion syndrome." It consists of a total abrogation of function of the cochlea, of the horizontal and vertical canals of the same side, and of the vertical canals of the opposite side. Grant and Fisher found this typical symptom complex occurring more often in true acoustic tumors while in other groups of angle tumors both the horizontal and the vertical canal of the opposite functioning ear tend to show impaired response. Winston, studying 34 cases of verified cerebellopontile angle tumors, arrives at the conclusion that one is not justified, from a vestibular examination alone, in differentiating between primary and secondary lesions of the angle. A number of otologists, particularly French authors, do not believe in the cerebellopontile lesion syndrome at all. The same view is taken by Maybaum in this country, who could not corroborate the clinical significance of such a symptom complex.

The clinical picture in the overwhelming majority of true acoustic tumors is very characteristic. This in turn makes the diagnosis very easy. There may, however, be some cases with an atypical course, so that difficulties arise as to the local diagnosis. Therefore it should be repeated, as already pointed out in chapter iv, that is not the task of the otologist to make a neurologic local diagnosis, even though he is tempted to do so in cases of acoustic tumors. He can give very important diagnostic information that the neurologist can combine with his other findings.

My studies on brain tumors in the Vienna surgical clinic included 52 cases of tumors of the posterior cranial fossa. Of these, 27 were acoustic tumors. In 24 cases, i.e., 90 per cent, the neurologist made the right local diagnosis supported by the otologic findings. Twenty-six out of the 27 acoustic tumors (96 per cent) were accompanied by *homolateral deafness.* In the only case with normal hearing, autopsy revealed a tumor of the size of a cherrystone. Cushing had among his cases of brain tumor 30 acoustic tumors. Twenty-five of the patients had disturbances of hearing. Some of them dated these disturbances many years back. Among the 26 cases of homolateral deafness among my patients, 7 cases showed also

disturbances of hearing in the contralateral ear; tuning fork tests revealed lesions in the sound-perceptive apparatus. In all these cases the acoustic tumors were of considerable size. Northington reported about 18 acoustic tumors with loss of hearing.

*Subjective noises* (tinnitus) must be considered as an important initial symptom. The patients usually complain of noises of high pitch (ringing, whistling, hissing, etc.). Henschen found tinnitus in 23 among 136 cases, while Cushing found it in 18 out of 30 cases. The latter author stresses further the clinical importance of an exact history with respect to the order and duration of the various symptoms. The question whether disturbances of the cochlear apparatus or of the labyrinth occurred first, may often be difficult to determine. Cushing himself states that patients, particularly those with severe symptoms of hydrocephalus, are not able to answer these questions satisfactorily. To this statement I would add that many patients with clear sensorium may also not notice a unilateral decrease of hearing as long as the other ear remains intact. I had some cases in which a complete unilateral deafness was revealed on functional test, while the patients were not aware of their deafness. Among my 27 cases of acoustic tumor, I found the cochlear apparatus first involved in 10 patients and the labyrinth first diseased in 6 cases, while a simultaneous involvement of both parts occurred in 5 patients. In the remaining 6 cases, the chronologic order could not be determined. According to Brunner, cochlear symptoms are found in the early stages of lateral acoustic tumors but occur very late in the medial types.

*Vertigo* is described either as typical turning sensations or as tactile vertigo with lateropulsion. Stewart and Holmes state that sham movements of objects are directed toward the sound side, those of the subject toward the diseased side. Many authors do not agree.

*Spontaneous disturbances of equilibrium and coordination* often occur. Cushing found positive Romberg reaction in 92 per cent of his patients. I observed it in 62 per cent of my cases. Falling occurred in the direction of the affected side regardless of the position of the head. Cerebellar gait and disturbances of coordination do not belong to the clinical picture of acoustic neurinoma. According to Cushing it usually takes a year before such disturbances develop.

*Spontaneous nystagmus* represents a very frequent symptom. It occurred in 55 per cent of my cases. This nystagmus is usually of first degree, associated and bilateral. Intensity and amplitude are more marked in the direction of the affected side. An amazing circumstance is the great changeability in form and intensity. Sometimes it is straight horizontal (→) or rotatory (↷), at other times it is mixed (⇄). At a given time it may be coarse and shortly thereafter fine.

*Disturbances of associated eye movements* do not belong to the picture of true acoustic tumors. Such positive symptoms may occur in the medial type of Brunner's classification or in the other forms of cerebellopontile angle tumors.

The *caloric examination* revealed in the majority of these cases a complete loss of labyrinthine excitability of the diseased side. Such results were obtained in 74 per cent of my patients. In 2 cases the caloric excitability was absent on both sides; 2 other patients showed only a marked decrease in the labyrinthine function of the diseased side.

The *turning chair examination* showed almost the same results as the caloric stimulation, i.e., a *homolateral loss of excitability.*

*Disproportion* between the various reactions has been observed. Cushing found in some of his cases a loss of function in the vertical semicircular canals, while the horizontal canals responded normally. This symptom has also been observed by Eagleton, Jones, Fisher, and Brent in cases with increased endocranial pressure. One of my patients had normal excitability to caloric stimuli but loss of function in relation to turning stimuli. Another case showed symptoms of vestibular disharmony (p. 175).

The *galvanic* excitability is usually absent on the diseased side. There may occasionally be a bilateral loss of function in relation to the galvanic stimulus.

*Past-pointing* occurred in 8 of my 27 cases of acoustic tumor. Three patients showed spontaneous past-pointing of the homolateral extremity toward the side of localization, whereas the other extremity pointed correctly.

### OTHER ANGLE TUMORS

These tumors are less frequent than the true acoustic neurinomas. Among the numerous growths that can be assigned to this group, the most common ones are as follows: (*a*) metastatic tumors (carcinoma, sarcoma); (*b*) granulation tumors (gumma, tubercle); (*c*) glioma; (*d*) meningeoma; (*e*) cysts; and, finally, (*f*) some of the "medial" acoustic tumors according to Brunner's classification. Because of the great variety of these tumors, one cannot expect to find such characteristic clinical symptoms as are observed in the true acoustic neurinomas. The clinical picture will depend largely upon the origin of the tumor, its size, its growing tendency, and the brain pressure symptoms. Therefore we shall find in some cases mainly cerebellar symptoms, such as disturbances of equilibrium and coordination, in other cases chiefly pontile symptoms (disturbances of associated eye movements, of speech, etc.). Other cases may be governed by the clinical symptoms and signs of the increased endo-

cranial pressure. The hearing function may or may not be intact, labyrinthine symptoms may or may not be present. Although the otologic findings may be negative, they still may render diagnostic help. When, for instance, all neurologic signs and symptoms point to a tumor in the cerebellopontile angle, while the otologic examination yields normal results or only slight disturbances, then the diagnosis seems probable that the tumor does not derive from the eighth nerve but instead is pressing upon the nerve stem.

### 3. Tumors of the Anterior and Middle Cranial Fossae: Congestive Inner Ear

Choking of the disk of the optic nerve is considered as one of the most important symptoms in diagnosing increased endocranial pressure. It is found in about 90 per cent of such cases. Efforts have been made by numerous otologists to reveal an otologic symptom analogous in its diagnostic significance to the choked disk. This problem, however, has not yet been definitely solved, as can be seen by the confusion in the terms applied, such as choked labyrinth, neuritis of the eighth nerve, stagnation dropsy, neuritis labyrinthica, and congestive inner ear. Many otologists deny absolutely that such changes take place. I therefore made large-scale examinations from a pathologic-anatomic as well as from a clinical point of view. My material comprised complete series of slides of 20 petrous bones of cases with increased endocranial pressure and 164 clinical cases of brain tumors.

By way of a summary, my histopathologic findings were classified in the following groups:

1. Transudation, exudation, and lymph congestion (dropsy and edematous infiltration) in the region of the membranous inner ear and the nerve ganglion apparatus (vaginal dropsy of the seventh, eighth, and ninth nerves).
2. Hyperemia, hemorrhage, changes in the blood vessels of the membranous inner ear and the petrous bone.
3. Formative changes (ectasia, stenosis, and collapse) of the ductus cochlearis, the ductus reuniens, and the saccus and ductus endolymphaticus and perilymphaticus.
4. Multiple hernias of the brain, especially on the anterior surface of the pyramids and in the middle fossa, accompanied by pressure erosion of the underlying bone.
5. Secondary (late) changes: pigmentation, adhesions, and degenerative atrophic processes of the sense organ and the nerve ganglion apparatus.

6. Destructive osseous changes (pressure atrophy).
7. Pathways by which the brain pressure is transmitted into the inner ear: the subarachnoid spaces of the aquaeductus cochleae, the nerve sheaths in the internal auditory meatus, the pacchionian granulations, the ligamentum spirale, the lymph channels of the bony labyrinthine capsule, and the blood vessels.

The *clinical symptoms and signs* of "congestive inner ear" were summarized as:

1. *Tinnitus* of high pitch (ringing, whistling, hissing, etc.) either continuous or alternating.
2. Impairment of *hearing*, gradually progressing with continuing pressure. The tuning fork test reveals a diminution of the upper limit of hearing, while the lower limit is not greatly diminished. In some cases with good hearing acuity the bone conduction is markedly reduced. There may often be a great changeability of the cochlear symptoms within a certain period of time. I therefore suggested repeated examinations as very important.
3. *Vertigo*, either of a slight degree—described as a vague feeling of uncertainty—or a turning vertigo, or a tactile vertigo with lateropulsion.
4. *Spontaneous nystagmus*, found very often. The nystagmus may either be the result of the increased endocranial pressure, or may represent a true focal symptom in tumors located within the labyrinthine reflex arc (for differentiation of the various types of nystagmus, see p. 110).
5. Pathologic change in *labyrinthine excitability* in frequent instances, usually with bilateral hyperexcitability, which can be considered only as an expression of increased endocranial pressure, i.e., a symptom of obstruction. No further localization, however, can be made. If the increase of pressure in the skull cavity is intensified very rapidly, or if such increased pressure persists for some time, the initial hyperexcitability may gradually diminish, resulting in a hypo-excitability. In some cases, sensation after turning may be disturbed. Eagleton found in cases of increased endocranial pressure a reduced excitability of the vertical canals.
6. Disturbance of *conjugate eye movements*, occasionally found. This symptom is significant of either a lesion in or a pressure upon the cortical centers or the pathways within the pons (for differentiation of the various forms of conjugate paresis and conjugate deviation, see p. 64).

## CONCLUSIONS

Histopathologic and clinical studies yield the result that an analogy can be drawn between choked disk and congestive inner ear.

Increased generalized brain pressure may exist over a certain period without damaging the delicate sensory epithelium and the nerve ganglion apparatus, even when extensive transudation or exudation is present.

Increased generalized brain pressure persisting over a long period of time produces secondary degenerative processes extending from the base of the cochlea to the top.

It is the duty of the otoneurologist by exact and periodically repeated examinations to discover the earliest stages of congestive inner ear and to give the indication for surgical intervention before irreparable changes occur in the delicate sense apparatus.

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# XII

# Vascular Lesions: Ménière's Syndrome

By *Louis E. Wolfson*

## 1. Classification

Ménière described in 1861 a case of a young girl who suddenly became sick with high fever, vertigo, tinnitus, and deafness. Death occurred five days later. On autopsy a diffuse hemorrhage was found within the inner-ear spaces. According to Alexander it must have been a leucemia, since he observed similar findings in his studies on leucemia. Politzer made a sharp differentiation between the apoplectiform type (Ménière's disease) and the cases in which vertigo, tinnitus, and deafness occur in patients with previous ear disease (Ménière symptom complex). Frankl-Hochwart distinguished between the apoplectiform type (Ménière's disease) and the paroxysmal type, in which vertigo, tinnitus, and deafness occur in patients who never had any ear disease (pseudo Ménière's disease). Dandy uses the term "pseudo Ménière" in a somewhat different sense as denoting a Ménière's disease minus deafness and tinnitus. Kobrak differentiates between apoplectiform insult and status labyrinthicus. Since he believes in a vascular origin of Ménière's disease, he calls it angiopathia labyrinthica.

Brunner classifies cases of Ménière syndrome in two groups, one in which the symptoms are due to certain diseases, such as chronic adhesive otitis media, otosclerosis, arteriosclerosis, neurolabyrinthitis, etc., the other in which the cause is unknown. The former type is called symptomatic Ménière syndrome, the latter idiopathic Ménière syndrome. Northington speaks of recurrent vestibular symptoms of obscure or unknown origin. To bring some order into the somewhat confusing terminology, I would suggest using only the term *idiopathic Ménière syndrome* (Brunner) and ruling out all other forms. In cases in which vertigo, tinnitus, and deafness represent symptoms of an underlying disease, it would be better to apply the term denoting the cause, as, for examples, traumatic inner-ear otitis, neuritis toxica, congestive inner ear, degenerative-atrophic inner-ear lesion, arteriosclerosis of the inner ear, labyrinthitis, etc.

## 2. Pathologic Anatomy

The pathologic anatomy is not clear as yet. Since the Ménière syndrome is not a lethal disease, autopsy findings are lacking. Microscopic

examination of the severed eighth nerve does not reveal any pathologic changes. In recent times 3 autopsy cases have been examined microscopically by Hallpike and Cairns. The authors found gross dilatation of the scalae mediae of the cochlea and the saccula, degenerative changes in Corti's organ and in the striae vasculares, changes in the connective tissue around the endolymphatic sac, and scattered albuminoid coagula in the vertical semicircular canals. They ascribe the primary changes to either an increased production of the endolymph or an alteration in its chemical constitution. This in turn might well lead to such dilatation of the endolymphatic system by osmotic attraction, through its membranous walls, of the water molecules from the surrounding perilymph.

The findings of Hallpike and Cairns are very important and very interesting, but not proof enough to explain the underlying pathology in the Ménière syndrome, or to build new theories upon. For such purposes not only a much greater material is necessary but also a carefully selected material. All cases in which inflammatory or suppurative processes in the middle-ear spaces are present should be ruled out. The same holds for cases in which there has been a surgical procedure, such as severing of the eighth nerve, with hemorrhages into the inner ear and into the subepithelial tissue. Further, an ideal good fixation and microscopic technic are needed to prevent postmortem or artificial changes. Findings such as that the organ of Corti or the membranous labyrinth is normal, except for postmortem changes, jeopardize the value of the pathologic changes. Wittmaack found, in 2 cases, obstruction of the cochlear aqueduct by a squeezed concretion.

### 3. Etiology

Great dissension exists with regard to the etiology and location of the disease. Some investigators find the site in the peripheral end organ (Crowe, Hallpike and Cairns, Fuerstenberg and co-workers, Brunner), others in the nerve stem (Dandy, McKenzie) or in the brain (Thornval, Skoog), or in a combined peripheral and central site (Grove). Dandy arrived at the conclusion that only a lesion in the sensory root of the vestibular branch can cause Ménière's disease, since both hearing and equilibrium are affected; a lesion in the end organ would be too diffuse not to be found. I do not think that many otologists will accept such an argument. Thornval gives a rather hypothetic explanation for the assumption of a central site of the disease. He believes that the nerve cells in the otolithic center of the brain stem are gradually charged with irritation; when the tension has reached a certain point, a sudden discharge occurs (Ménière attack). Grove directs attention to the persistence of tinnitus, which often can be found even after section of the eighth nerve. This leads him to the belief that pathologic processes, probably edema, exist not only in

the end organ but also in the region of the cochlear nuclei. Quix believes in an increased pressure in the cisterna pontis cerebellaris. The damage done to the cochlear nucleus is more severe than that done to the vestibular nucleus. The former is lateral and very superficially located, while the latter is medial and deep-seated.

### THEORIES OF CAUSATION

Some theories concerning the etiology are as follows:

1. *Water metabolism.* Mygind and Dederding see the disturbance in abnormalities in the water metabolism, not only in the labyrinth but also in other parts of the entire organism. Retention of fluids leads to extracellular edema. Since the labyrinth presents a cavity with stiff walls filled with fluid, the slightest cellular edema will increase the content of the labyrinth, thus raising the endolabyrinthine pressure. Although this theory sounds very logical, the presence of such a cellular edema in the labyrinth has not yet been proved microscopically.

2. *Sodium metabolism.* Fuerstenberg, Lashmet, and Lathrop see the disturbance of metabolism in the retention of sodium and not of water as claimed by Mygind and Dederding. They believe that the tissues responsible for the Ménière syndrome have been sensitized to or have an increased avidity for the sodium ion. Examination of the sodium contents of the blood have not corroborated the theory. In an effort to explain the negative laboratory tests, the authors refer to an analogy with Addison's disease. Although the cortex of the adrenal gland is alleged to be damaged, the sodium level of the blood may be found normal.

3. *Potassium metabolism.* Talbott and Brown found in cases of Ménière's syndrome an increased concentration of serum potassium during an attack, which they explain by the proportional decrease of the tissue potassium. They therefore suggest administration of potassium chloride in aqueous solutions.

4. *Allergy.* There are many reports in the literature of Ménière syndrome in patients with allergic manifestations. There are, further, experiments on animals for the purpose of eliciting the Ménière syndrome allergically, although the results are not yet very convincing. The main role is played by the abnormal capillary permeability and the vasomotor changes (Dean, Dohlman, Kobrak, Vogel, Meyer, Atkinson, etc.).

These four theories seem to contradict one another. It looks as though the reader should choose according to which of these theories he thinks is the most logical. As a matter of fact, there is not much difference. They all assume local extravascular accumulation of fluid within the tissue (local edema). In cases with allergic reaction, the permeability of the capillaries is supposed to be largely increased. This in turn leads to

seepage of fluid through the vessels into the surrounding tissue. The fluid, however, is not pure water, but a solution containing sodium, calcium, etc. Any treatment aimed to diminish the accumulation of fluid (edema) must restrict not only the water but also the sodium intake. The same holds true in regard to the potassium theory, since there is a reciprocity between sodium and potassium. Sodium increase in the blood causes a potassium decrease, and vice versa.

5. *Focal infection.* Wright believes that focal infection is one of the frequent causes of the Ménière syndrome (focal labyrinthitis). Brain regards the syndrome as the end result of a toxic-infectious process in the labyrinth. Shambaugh, Jr., states that in the majority of cases the disease begins with a serous labyrinthitis caused by a focal infection, resulting in increase of endolymphatic pressure and irritation of the labyrinth. Dandy rejects such a theory. If the Ménière syndrome were the result of an infection, it would be particularly prevalent in childhood, when these affections are common.

6. *Vasomotor internal otitis.* Brunner found, in his experiments on guinea pigs with traumatic injuries, perivascular infiltrations with lymphocytes, serous exudate in the perilymphatic spaces, and dilatation of the scalae mediae. He considers these pathologic changes—in contrast to the hydrops labyrinthi of Wittmaack—as an expression of a serous internal otitis of a special type, i.e., vasomotor internal otitis. Brunner assumes the same pathologic process for the Ménière syndrome. An acute Ménière attack corresponds to an attack of vasomotor internal otitis.

There are a number of other theories about the etiology of the Ménière syndrome, referring it to disturbance of endocrine glands, avitaminosis, disturbance of vasomotor sympathetic control, anatomic variations of the carotic artery, analogy with migraine, etc. In spite of all differences between the various theories, they all assume disturbance in the circulation, chemistry, or pressure of the labyrinthine fluids. This was already known to Knapp and Cheatle, who fifty years ago suggested that the lesion may be caused by increased pressure in the labyrinth, and compared the condition with glaucoma. The pressure can be increased by increased production of the endolymph, by decreased resorption, or by alterations in its physiochemical constitution as assumed by Crowe. In his recent studies on intralabyrinthine pressure, Hallpike states that the membranous labyrinth forms a closed fluid system; the perilymph reaches the surrounding spaces from the subarachnoid cisternae via the cochlear aqueduct. Any volume increase is answered by expansion of its membranes and by expulsion of a corresponding perilymph volume through the helicotrema and aqueduct. Any sudden volume increase of fluid must result in considerable pressure changes. Ménière attacks are due to rapidly

initiated bouts of asphyxia of the labyrinthine organs caused by extremely rapid rises of fluid pressure in response to relatively small volume increase of endolymph. Dilatation of the endolymph system to the limits of its bony walls is the indispensable condition for such an occurrence.

All these theories are very interesting, but it must be admitted that they still are unproved hypotheses. Not only are pathologic findings lacking, but there are also unknown factors in the physiology and chemistry of the labyrinthine fluids.

It is generally accepted that the endolymph is produced in the stria vascularis and that the endolymphatic spaces form a closed system. The perilymph is produced by diffusion from the blood vessels as a dialysate and is drained by the cochlear aqueduct leading into the subarachnoidal spaces. In case of increase of the perilymphatic pressure in the inner ear, there will be an outflow of perilymph due to the negative pressure in the cerebrospinal spaces. There is, however, great diversity of opinion in many respects, as shown in the following opinions. The endolymph is also produced in the organ of Corti and in the macula (Wittmaack). The outflow of endolymph takes place by osmosis through Reissner's membrane by channels through the walls of the endolymphatic sac or by the epithelium of the sac (Guild). There is no communication between the cerebrospinal and the perilymphatic fluid (Rejtoe). There are numerous pathways between the cerebrospinal fluid and the spaces in the inner ear, such as the cochlear aqueduct, the internal auditory meatus, the perineural and perivascular spaces, the lymph channels of the bone, the labyrinthine capsula, the spiral ligament, and the pacchionian granulations (J. Fischer). Perilymph, according to some authors, is cerebrospinal fluid; according to others, it is not cerebrospinal fluid but a product of the endolymphatic fluid by osmosis. This is only a scant illustration of the difficulties of the physiologic and physical problems involved.

Discussing the cellular edema of the labyrinth, Mygind and Dederding declare: "We must assume that they are changes of so transient a nature that they are destroyed by our ordinary preparation methods, and are, perhaps, also difficult to distinguish from the normal picture. We must therefore endeavor to find other means of clearing up the pathological process." Such statements illustrate best the hypothetic character of the etiologic explanations.

### 4. Symptomatology

The symptomatology of the *idiopathic* Ménière syndrome is usually very characteristic. There are cochlear, labyrinthine, and general manifestations. The classic symptoms are vertigo, tinnitus, and decrease in hearing. In addition, the following symptoms may be observed, though not invari-

ably: nystagmus, nausea, vomiting, diplacusis, hypo-excitability of the labyrinth, headaches, and vasomotor manifestations.

*Vertigo* usually occurs in a sudden violent apoplectiform attack with sham movements of the subject or of objects. Such an attack may last only a short time or may sometimes be extended over hours or days. It may be followed by a long interval of well-being or by an early repeated attack. The patient usually does not lose consciousness, even though the attack is very severe. Crowe, however, observed 15 cases out of 117 with temporary loss of consciousness. The present writers have never observed any loss of consciousness.

The *tinnitus* varies in character. It may be described as buzzing, hissing, ringing, etc. It may be intermittent or constant. Mostly it is persistent, bothering the patient sometimes even when the cochlear and labyrinthine functions have been destroyed.

*Decrease of hearing* follows the initial attack of vertigo and progresses with the further advance of the disease. Some authors consider the deafness as a sound-conductive lesion (Mygind and Dederding), others as a sound-perceptive one (Grove), while still others believe in a combined conductive-perceptive affection.

The *nystagmus* would be described more often if we could have opportunity of examining a patient during his attack. The form of nystagmus is either horizontal-rotatory or rotatory-horizontal, since the peripheral labyrinth must be considered as the site of the disease. The direction of the nystagmus, however, has no localizing significance.

*Nausea and vomiting* are combined with severe attack of vertigo.

*Diplacusis* has been found by Shambaugh, Jr., in nearly all his cases of Ménière syndrome. Such observations are rather surprising. He explains it by assuming distortion of the Corti organ, presumably as a result of edema of the vibrating membranes.

*Hypo-excitability* of the labyrinth has been reported by Brunner, while Mygind and Dederding consider such a reaction as extremely rare.

*Headaches* are one of the most frequent symptoms accompanying the Ménière syndrome (Mygind and Dederding, Cohen, Shelden, and Horton, etc.). They have the character of migraine and are usually on the affected side.

Vasomotor manifestations such as perspiration, pallor, etc., may occasionally be observed. While most of the symptoms are usually well marked during an acute attack, the patients may not show any signs during the interval period. The diagnosis can be made only from the case history, which reveals repeated attacks of vertigo, tinnitus, and decrease of hearing in the absence of any ear disease.

For differentiation of the *symptomatic* Ménière syndrome, the reader is

referred to the respective sections on brain tumors, labyrinthitis, trauma, etc.

## 5. Treatment

It is obvious that the great diversity of opinion concerning the etiology and pathology of the Ménière syndrome will be reflected in the treatment. Before discussing the various therapeutic measures in detail, it may be interesting to give a brief survey of the statistical figures concerning the percentage of cured cases.

Mygind and Dederding report on 157 cases, 151 of which lost vertigo and nystagmus after treatment with restricted fluid intake. These authors therefore obtained 96 per cent of healing. Fuerstenberg and Lathrop observed satisfactory results in approximately 85 per cent of their cases, using a salt-free diet to limit the intake of sodium. Bárány administered monotrean in 23 cases and had success in 21 cases, i.e., 91 per cent. Sheldon and Horton treated 15 cases with histamine and had no failure, i.e., 100 per cent success. Wright had 88 per cent of success with his method of elimination of a focal source. Walsh and Adson severed the eighth nerve (Dandy method) in 13 cases, with only one failure, i.e., 92 per cent of success, while according to Dandy all his patients lost vertigo. Woodman operated on 11 patients by the method of Portmann and had only one failure, i.e., 91 per cent of recovery, while Mollison reports about 90 per cent of healing with alcohol injection into the semicircular canal. One wonders why so many cases of the Ménière syndrome still appear and desire treatment.

The various forms of treatment can be classified according as medical or surgical procedures are used.

### Medical Therapy

*Salt-free treatment* (Fuerstenberg and co-workers). Diet is the most important factor. The patient should be hospitalized in order to be carefully controlled. He is given 6 capsules of 7.5 grains of ammonium chloride each, three times a day (with each meal) through three days. Then the capsules are omitted for two days and again given for three days.

The diet is regulated as follows: low sodium content—i.e., no salt, soda, or baking powder; animal protein (except milk) and cereal products unrestricted; vegetables, fruits, and milk limited; calories sufficient to maintain a desirable weight; water unrestricted.

This strict regimen is maintained for a period of six weeks. If the patient then remains asymptomatic, the ammonium chloride can be discontinued; otherwise treatment should be given for another period of six weeks.

*Water restriction* (Mygind and Dederding). The daily fluid intake must be restricted to 700 cc., occasionally still less. In order to promote diuresis, diet with low salt content is given. Reduction diet is used for patients with overweight. In order to stimulate vasomotor tone, exercise, massage, light therapy, etc., are recommended.

*Histamine treatment.* Sheldon and Horton use histamine diphosphate intravenously and subcutaneously. In cases of severe acute Ménière syndrome, 2.75 mg. of histamine diphosphate in a 250 cc. physiologic saline solution is injected intravenously. Since histamine is a powerful vasodilatator, the administration should be very slow; the injection should usually take one and a half hours. If there is no prompt effect, the intravenous injection may be repeated on successive days.

For prevention of future attacks, a maintainance dose of 0.1 to 0.2 mg. should be given subcutaneously two to four times a week. Crowe recommends subcutaneous injections with diluted solution. A concentrated solution of histamine phosphate comes in 1 cc. vials containing 1 mg. per cubic centimeter. A maximal dose of this concentration is 0.2 cc. of this vial. Treatment is begun by diluting the concentrated solution with sterile normal salt solution in a ratio of 1 to 10. This 1:10 solution of histamine phosphate is injected twice daily, beginning with 0.1 cc. The second day 0.15 cc. is given morning and evening, and the dose is increased by 0.1 cc. daily until the 0.9 cc. dose is reached. After this the patient's tolerance is maintained by giving 0.3 to 0.4 cc. of the concentrated vial once a week for an indefinite period.

*Other methods.* Talbott and Brown administer daily 6 to 10 Gm. of potassium chloride in aqueous solution. They do not prescribe any dietary measures. The treatment is maintained for one to sixteen months. Bárány used the drug monotrean, which is a combination of quinine and papaverine hydrochloride. It acts upon the smooth muscles, diminishing the tonus; this in turn relieves the angiospasm in the labyrinth. The patient is given 3 tablets daily after each meal for a period of six weeks. If, however, the results are not satisfactory, the course of treatment should be repeated two or three times. Other forms of medical treatment are sedatives, atropine, adrenaline, pilocarpine, quinine, acetyl choline, etc.

Morsch advocates administration of vitamins for cases with vitamin deficiency.

Physiotherapy includes faradization, high-frequency diathermy, radiotherapy, etc.

### SURGICAL THERAPY

Operative procedures are undertaken either to destroy the peripheral labyrinth or to sever the eighth nerve. Some of the technics are as follows:

Dandy: Ménière's disease can be permanently cured by division of the auditory nerve. This procedure carries almost no risk to life.

A vertical incision is made along the mastoid, and at the tip the incision is curved mesially and downward; only a small cut is made through the insertion of the trapezius muscle. The muscle-skin flap is reflected mesially. A circular area of bone (size of 50-cent piece) is removed with rongeurs. After incision into the dura, the cisterna magna is exposed and its contents evacuated, thus providing ample room to retract the cerebellum safely. The cisterna lateralis is then opened and the fluid contents aspirated. The eighth nerve is elevated with a small hook. The handle of the hook is touched with the electrocautery and the nerve is severed. If partial section of the nerve is desired in order to save the hearing, the hook passes under the anterior border of the nerve to the point of division and the cautery is applied in the same manner. The dura is then carefully sutured.

Portmann: The mastoid is opened and the dura of the posterior fossa is exposed in front of the lateral sinus. In order to reach the endolymphatic sac—which lies halfway between the sinus and the internal auditory meatus—the dura is lifted from the posterior surface of the petrous bone. After severing of the adhesions around the sac, a small drop of clear fluid occurs. In cases in which the endolymphatic sac cannot be found, the dura is elevated until the internal auditory meatus is reached. The wound is sutured.

Mollison: A simple antrotomy is performed, with exposure of the horizontal semicircular canal. The canal is then opened with a narrow gouge, and through its anterior end a few drops of dehydrated alcohol are injected into the vestibule. The wound is then sutured.

Berggren: An incision is made in the inferoposterior quadrant of the drum close to the annulus, and the flap is turned upward and forward, thus exposing the promontory. After scarification of the mucous lining, the promontory is perforated with a round burr. A few drops of the fluid are then aspirated with a needle, and some drops of alcohol are injected. After the flap is replaced in normal position, an iodoform gauze strip is introduced into the external canal.

Putnam: An incision is made from the zygoma upward in the temporal region. The squamous portion of the temporal bone is removed, in a section about 4 cm. in diameter. The dura is elevated from the petrous portion, and by percussion the superior vertical canal is located. One or two burr holes are made, until the eburneous texture of the bony labyrinth is felt. The superior vertical canal is then opened. A wire connected with a coagulating electrode is introduced into the anterior end of the semicircular canal for about five seconds. The dura is put back into place

and the wound is closed. The advantage of this method of approach is that it keeps one well away from the large vessels, the cranial nerves, and the brain stem. It furthermore entails less danger of producing deafness.

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# XIII

# War Trauma

By *Louis E. Wolfson*

## 1. Skull Fractures

If the skull is subjected to a sudden blow, compression of the bony capsule occurs. If the acting force exceeds the limits of elasticity of the bone, a "bursting fracture" results. The direction of the fracture line is identical with that of the acting force. If, therefore, the skull is compressed transversely, the fracture line shows a transverse course, i.e., paralleling the upper margin of the pyramid. Although the mechanism of skull fractures is determined by numerous factors, such as the violence and direction of the blow, the position of the head etc., a certain regularity can nevertheless be observed as regards the type of the temporal bone lesion. This can be explained on the basis of the architecture of the temporal bone. Three main types of fractures can be distinguished: (1) longitudinal, (2) transverse, and (3) combined fractures.

*Longitudinal fractures* are the most common (about 80 per cent), owing to the fact that weak bone areas lie close to the compact labyrinthine capsule. The typical fracture line is in front of the upper margin of the pyramid, i.e., in the middle cranial fossa (Fig. 72). It involves the roof of the middle ear (tegmen tympani and antri), extending medially to the region of the gasserian ganglion and laterally to the mastoid and squama or through the external auditory meatus.

Figure 72 shows a longitudinal fracture through both temporal bones of a 42-year-old white male. The fracture line courses on the right from the lacerate foramen along the roof of the eustachian tube and the tympanic cavity to the mastoid. On the left the fracture line runs from the region of the gasserian ganglion along the tube and the middle ear to the external auditory meatus. The darkened areas represent the sites of hemorrhages.

Involvement of the middle and the external ear may lead to dislocation of the ossicles and usually results in a rupture of the drum membrane with hemorrhages. Fraser described cases of longitudinal fracture with dislocation of the incus.

*Transverse fractures* occur vertical to the longitudinal axis of the pyramid. The fracture line runs through the vestibule and the facial canal to the internal auditory meatus, i.e., the compact bone of the cochlea is spared, while the thin bony walls of the vestibule are fractured. Brunner stressed

the clinical importance of determining whether or not the lateral labyrinthine wall is involved. Grove distinguishes between an external and an internal type of transverse fracture. The former passes through the entire inner ear, the cochlea, the vestibule, and the fallopian canal, the latter passes through the internal auditory meatus and shatters the anterior and medial portions of the cochlea. Transverse fractures lead to lacerations of the membranous inner ear with consequent hemorrhages. The drum membrane and the external auditory meatus remain intact.

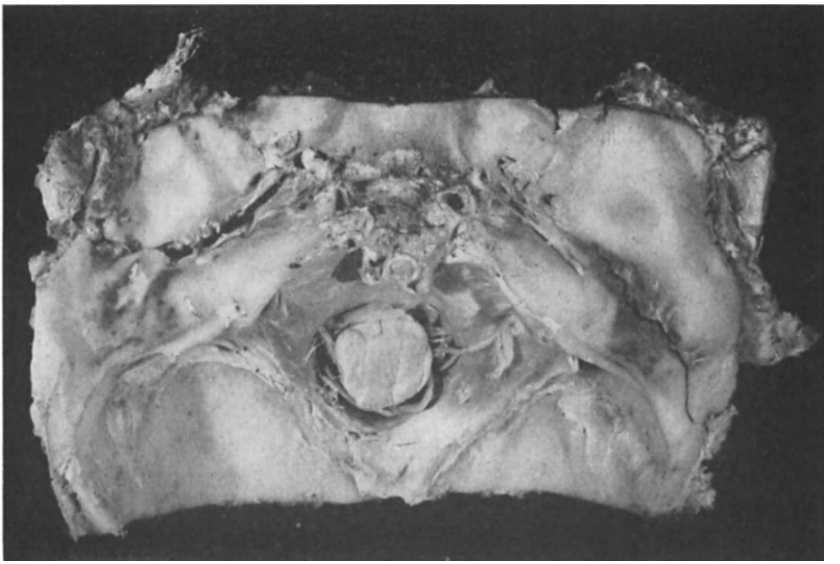

FIG. 72. PHOTOGRAPH OF BASE OF SKULL WITH REGION OF MIDDLE AND POSTERIOR CRANIAL FOSSAE: LONGITUDINAL FRACTURE THROUGH BOTH TEMPORAL BONES

Fracture line on right side extends from apex of pyramid through eustachian tube and antrum into mastoid. On left side, fracture line courses through eustachian tube and antrum into mastoid. On left side, fracture line courses through eustachian tube, antrum, and attic to external auditory meatus. Discolored areas represent hemorrhages into middle-ear cavities.

*Combined fractures* are very rare. They lead to damage of the external, the middle, and the inner ear. This in turn causes a wide communication between all the cavities of the ear organ. The few cases that survive such severe injuries are very liable to secondary infection, with rapid spread into the cranial cavity.

Numerous other classifications of fractures of the temporal bone occur in the literature. Following are some examples: Voss distinguishes eight groups: (*a*) longitudinal fracture, (*b*) transverse fracture, (*c*) combined

oblique fracture of the posterior fossa and transverse fracture of the pyramid, (*d*) combined oblique and longitudinal fracture of the posterior fossa, (*e*) combined longitudinal and transverse fracture of the petrous bone, (*f*) partial or (*g*) complete avulsion of the mastoid process, and (*h*) isolated rupture of the tegmen tympani. Brunner differentiates between

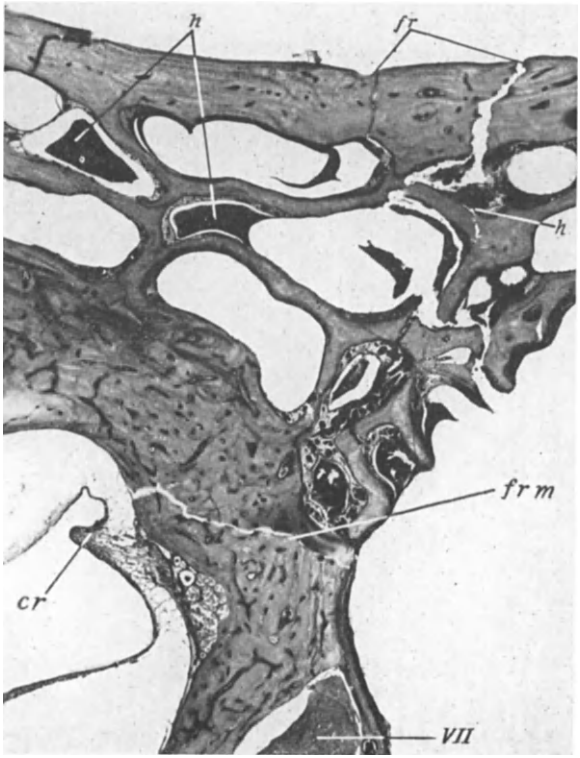


FIG. 73. MICROPHOTOGRAPH OF CASE OF LONGITUDINAL FRACTURE OF TEMPORAL BONE

*fr* = fracture through roof of middle ear; *fr m* = microscopic fracture line crossing bony labyrinthine capsule; *h* = hemorrhages in middle ear and in perilabyrinthine spaces; *VII* = facial nerve; *cr* = crista of horizontal semicircular canal.

the longitudinal and transverse fractures and avulsion of the apex of the pyramid. Klingenberg, Grove, etc. describe isolated fractures of the cochlea, etc.

Besides these macroscopic fractures of the temporal bone, visible at operation or on autopsy, a number of fine *microscopic* fractures or fissures have been described (Scheibe, Nager, etc.) on the basis of histologic examinations. Brunner and Ulrich state that in longitudinal fractures such micro-

scopic fissures usually extend into the external meatus or into the squamous portion but not into the labyrinthine capsule. However, FIGURE 73 does not corroborate such a statement. The photomicrograph represents the case of a 28-year-old white male who died after a violent skull injury. Besides the longitudinal fracture through the roof of the middle ear, a

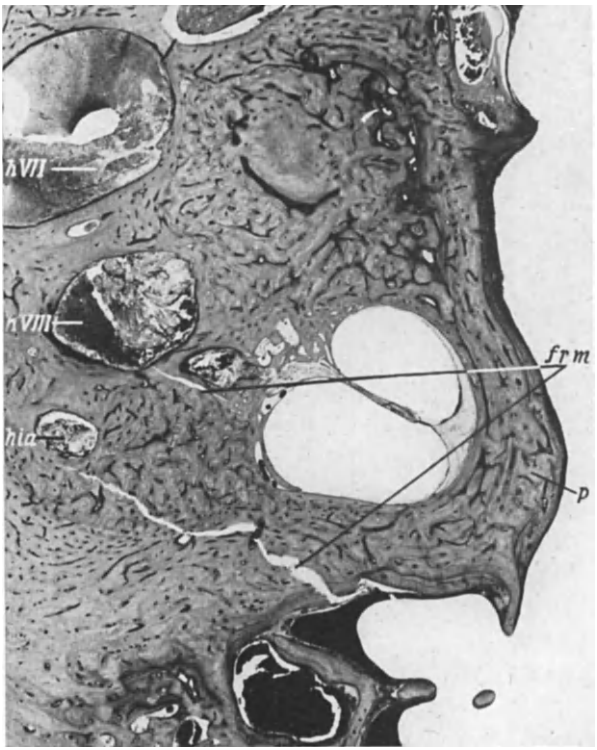


FIG. 74. MICROPHOTOGRAPH OF CASE OF LONGITUDINAL FRACTURE OF TEMPORAL BONE

*fr m* = microscopic fractures in bony capsule; *hVIII* = hemorrhages in internal auditory meatus (cochlear nerve); *hVII* = hemorrhage in internal meatus (facial nerve); *hia* = hemorrhage in inferior ampullar nerve; *p* = promontory.

microscopic fracture is seen. Its line crosses the labyrinthine capsule in a transverse direction, starting above the fallopian canal and extending up to the endostial layer of the horizontal semicircular canal.

Such fissures may develop at the weakest part of the labyrinthine capsule, such as the oval or the round window, at the footplate of the stapes, or at the secondary membrane. FIGURE 74 shows two such microscopic fractures, in the case of a 32-year-old white female who had a macroscopic

longitudinal fracture through the roof of the antrum. One fracture line starts in the region of the round window and crosses the otic capsule in a transverse direction, extending almost up to the bony canal of the inferior ampullar nerve. The other line runs parallel to it from the basal turn of the cochlea to the internal auditory meatus.

In discussion of the *pathologic anatomy* of the fractures of the temporal bone, one has to distinguish between fresh injuries and late stages.

In *fresh injury* the changes include—besides the above discussed macroscopic and microscopic fractures—also lacerations of tissue and extensive hemorrhages.

In longitudinal fractures hemorrhages occur not only in the region of the fracture line (middle and external ear) but also in the inner ear, as in the internal auditory meatus, in the bony nerve channels, in the cochlear aqueduct, and in the perilymphatic spaces of the cochlea and labyrinth. There is great diversity of opinion as to the etiology of these hemorrhages. A number of authors (Linck, Lange, Ulrich) consider the bleeding in the internal meatus as a result of stretching of blood vessels and nerves. The acting force of the blow causes a twisting movement of the pyramid that in turn leads to a stretching of blood vessels and nerves in the internal auditory meatus. It is generally believed that in longitudinal fractures the hemorrhages occur in the perilymphatic spaces, while the endolymphatic cavities are spared. FIGURE 75, however, shows a longitudinal fracture through the antrum and the external auditory meatus with rupture of the drum membrane. There is an intense hemorrhage in the perilabyrinthine cells and in the endolymphatic duct, while the perilymphatic spaces are free of hemorrhage. Other authors (Alexander, Brunner) consider the hemorrhages in the inner ear as bleedings *in extremis*, without any connection with the head trauma.

The pathologic picture of the *late stages* of temporal bone fractures is not very clear. The main difficulty in the interpretation is due to secondary infections following the trauma. Even the slightest infection may influence the pathologic picture to a great extent. Manasse distinguishes three groups of pathologic changes: direct traumatic changes (fracture, fissure); new formations of connective tissue and bone in the perilymphatic cavities (periostitis ossificans); degenerative processes. Alexander differentiates two groups: results of the head trauma (labyrinthitis ossificans); nontraumatic changes (alteration in the endolymphatic spaces).

A study of the *healing processes* in temporal bone fractures reveals that although regeneration processes are found very early, a complete healing with production of callus does not occur. Alexander examined histologically a case in which the skull fracture occurred thirty-one years before and found only connective tissue in the fracture region. Ulrich observed, in

late stages of fractures, bony callus in the endostal and the periostal part of the labyrinthine capsule, but could not find any bone formation in the enchondral part. Such findings can be explained only on the basis of the peculiar ossification processes of the otic capsule. Histologic studies by Mayer, Bast, Nager, etc., have shown that the ossification processes in the enchondral part of the capsule stop at the age of 2 years. There are no major metabolic processes after this age period. Similar results were ob-

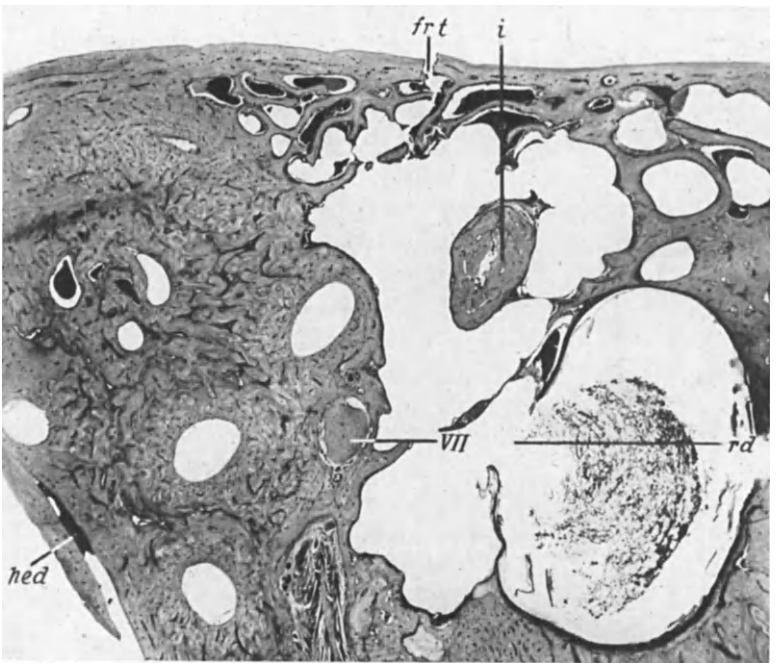


FIG. 75. MICROPHOTOGRAPH OF CASE OF LONGITUDINAL FRACTURE OF TEMPORAL BONE: VERTICAL SECTION THROUGH EXTERNAL, MIDDLE, AND INNER EAR

*fr t* = fracture of tegmen antri; *rd* = rupture of drum membrane; *i* = incus; *VII* = facial nerve; *hed* = hemorrhage in endolymphatic duct.

tained by Perlman in his experimental investigations concerning the ossification processes after injuries.

The other reparation processes in the ear organ (hemorrhages, tearings) lead to extensive formation of connective tissue and bone in the perilymphatic spaces, thus filling the cavities almost completely with the newly formed masses. Such cases have been described by Alexander, Nager, and Manasse. Nager investigated a case of a 64-year-old deaf-mute who suffered a severe head injury at the age of 4. He found all the perilymphatic spaces of the cochlea and labyrinth entirely filled with

connective tissue and bone, but could not find any fracture in the temporal bone. Lange and Brunner consider these new formations as a product of inflammatory processes.

FIGURE 76 illustrates similar regeneration processes as described by Nager. The photomicrograph is from the case of a 25-year-old deaf-mute who had suffered a severe head blow at 2 years of age, with temporary loss of consciousness. The basal coil of the cochlea is entirely filled with

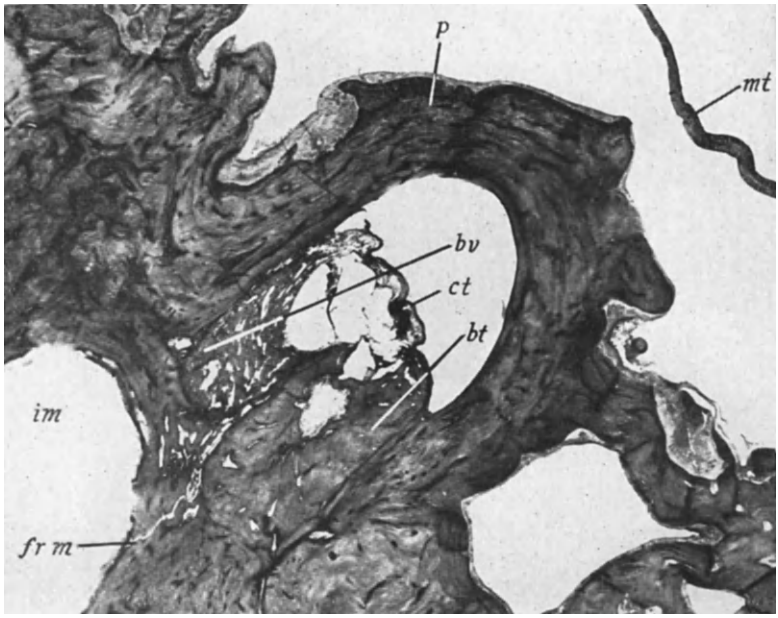


FIG. 76. MICROPHOTOGRAPH OF HEALED CASE OF TRAUMATIC DEAFNESS: SECTION OF BASAL TURN OF COCHLEA

*bt* = bone formation in scala tympani; *bv* = bone formation in scala vestibuli; *ct* = connective tissue; *fr m* = microscopic fracture extending into internal auditory meatus (*im*); *p* = promontory; *mt* = membrana tympani.

newly formed bone and some connective tissue; the nerve and sensory organ are completely destroyed. There is no major fracture in the temporal bone, but a fine microscopic fissure running from the bony scar to the internal auditory meatus. The pathologic picture can be interpreted in three ways: (1) complete healing of the fracture occurred, and for that reason no fracture can be detected—such a result is possible, since the trauma dated back to a time in which the enchondral ossification in the otic capsule was still in progress; (2) the formation of bone and connective tissue is the result of secondary infections; (3) the pathologic changes repre-

sent end stages of a concussion of the labyrinth as described by Brunner (p. 356).

### SYMPTOMATOLOGY

The clinical picture of a fracture of the temporal bone depends upon (1) the involvement of the brain, (2) the time of the otologic examination, (3) the type of fracture, and (4) presence or absence of secondary infection.

1. *Involvement of the brain* is manifested as concussion, contusion, or increased pressure of the brain. The *concussion* is characterized by a temporary physiologic disturbance of the cortical function without detectable organic changes of brain tissue. The most common clinical symptoms are: transient loss of consciousness; retrograde amnesia in relation to all events during the occurrence of the injury and the period of time immediately preceeding and succeeding the trauma; headaches, dizziness, nausea, vomiting, and changes of pulse rate. The most significant feature of concussion lies in the fact that all symptoms subside after a short period of time, regardless of how severe the concussion has been.

*Contusion* of the brain usually produces mechanical injuries of brain tissue with development of focal signs. There is often an elevated temperature. The loss of consciousness lasts much longer, and the clinical symptoms subside very slowly.

*Increased pressure* is due to damage of the brain by the fracture or to injury of extra- or intradural vessels with subsequent hemorrhages. There is often an interval free of symptoms between subsiding concussion and development of increased pressure due to hemorrhage. Very significant is the conjugate deviation of the eyes, which is directed to the contralateral side during the stage of irritation and to the homolateral side during the stage of paralysis. In contrast to the course in concussion of the brain, all the clinical symptoms increase with the further development of increased pressure. The hemorrhage usually occurs immediately after the trauma, but may occasionally develop in a later stage. Grove distinguishes five types of hemorrhages: extradural hemorrhage, usually due to rupture of the middle meningeal artery; subdural hemorrhage, usually due to a tearing of the sinuses; subarachnoid hemorrhage, in which the blood spreads out over the cortex, leading to pachymeningitis; subcortical hemorrhage, usually petechial; and intraventricular hemorrhage.

2. The *time of otologic examination* influences the clinical picture, since in the early stages all otologic symptoms and signs may be overshadowed by the brain lesion. Grove distinguishes three stages of the brain lesion: stage of shock, stage of increasing intracranial pressure, and stage of medullary failure. The phase of shock shows the usual symptoms accompanying surgical shock; the phase of increased pressure is manifested by a rising

blood pressure and a falling pulse; the stage of medullary failure is characterized by a falling blood pressure and a rising pulse.

Furthermore, it is not irrelevant whether the otologic examination is performed shortly after the brain symptoms have subsided or many years later. The greater the interval between the trauma and the examination, the more marked are the regenerative and degenerative changes (see above) determining the function of the ear organ.

3. The *type of fracture* plays a significant role in the otologic symptomatology. It has already been demonstrated that longitudinal fractures involve the middle and the external ear, whereas transverse fractures damage the inner ear.

The diagnosis of fracture of the temporal bone is based upon the otoscopic examination, the functional test of cochlea and labyrinth, the X-ray findings, and the neurologic investigation.

It must be emphasized that otoscopic examination in early stages, particularly of longitudinal fractures, should be performed with the greatest precaution to avoid secondary infection. Any manipulation, such as irrigation, instillation, removal of crusts, probing of the canal, etc., is strictly contra-indicated. If there has not been any infection up to the time when the patient is seen by the physician, it should not be introduced by the examiner. This may happen in a case in which the physician is eager to visualize the drum by any means in order to give a report to the court or insurance company or to the military authority. Every physician has to realize that it is not a deficiency in technic not to visualize the drum under the circumstances. He should postpone his report, plug the canal with sterile cotton, and apply a dressing.

The examination of *longitudinal fractures* may yield the following findings: escape of blood or cerebrospinal fluid from ear or nose, blood crusts or clots in the external auditory meatus or on the drum, blood in the tympanic cavity (hematotympanum), a steplike appearance of the external auditory canal, and rupture of the drum membrane. Hemorrhages from the ear occurring immediately after the head trauma originate, according to Mellinger, from the tympanic plexus, the sigmoid sinus, the superior petrosal sinus, the jugular bulb, and from the middle meningeal artery. The escape of cerebrospinal fluid may occur immediately after the trauma, twenty-four hours later, occasionally much later. Grove described a case in which it occurred fifteen days after injury. Voss considers such escape of fluid as indicative of transverse fracture, whereas Grove and Brunner regard it as a sign of longitudinal fracture. Voss applies the term "liquor tympanum" to cases in which cerebrospinal fluid is found in the tympanic cavity without any rupture of the drum membrane.

The functional test reveals a decrease of hearing acuity, spontaneous

nystagmus of a low degree, and occasionally a hyperexcitability of the labyrinth. The impairment of hearing is, according to Brunner, in 30.4 per cent of cases due to a lesion of the inner ear. Grove found a hearing loss in 63.4 per cent of his cases, particularly of the inner-ear type. Ulrich observed in 55 per cent of his subjects moderate deafness, in 28 per cent marked deafness, and in 17 per cent total loss of function of the inner ear.

X-ray investigation in longitudinal fractures may often fail to delineate the fracture, in contrast to the transverse lesions, which can be demonstrated in almost every case (Alberti).

The examination of *transverse fractures* gives a more uniform result. The drum membrane is usually intact, occasionally shows some petechiae. The external ear is normal. The functional test reveals homolateral total or subtotal loss of hearing and of labyrinthine excitability. The facial nerve may or may not be affected. On X-ray examination the fracture lines can easily be demonstrated, and even fine fissures may become visible.

4. The possibility of *secondary infection* is always present. In longitudinal fractures with rupture of the drum, infection may spread via the external canal or eustachian tube into the tympanic cavity, with extension into the middle cranial fossa. In other cases an otitis media that has existed before the head trauma may be the source of infection. In transverse fractures with involvement of the lateral labyrinthine wall, i.e., the medial wall of the middle ear, the infection may spread from the middle ear into the inner ear and via the internal auditory meatus or the aqueduct into the posterior cranial fossa.

In spite of all the theoretic possibilities, the clinical incidence of posttraumatic meningitis is relatively low. This fact should play a great role in the indication for surgical intervention (see below). Cases of late meningitis following skull fractures have been described by Politzer, Scheibe, Voss, Klestadt, Nager, Ramadier, etc.

### TREATMENT

In the early stages of fracture of the temporal bone, all attention should be focused on the brain symptoms. In the event of shock, the usual surgical shock treatment should be the first to be undertaken. Signs of hemorrhage indicate immediate surgical intervention, which should preferably be left to the neurosurgeon. When all the brain symptoms have subsided, attention should be directed to the ear. In longitudinal fractures with rupture of the drum and escape of blood and cerebrospinal fluid, any active treatment is strictly contra-indicated. As previously instanced in regard to the otoscopic examination, the ear should be plugged with sterile cotton and a dressing applied. Bed rest and sedatives, exclusive of morphine, are important.

The main danger is always an impending secondary infection. While the majority of otologists adhere to a conservative treatment and operate only in cases of absolute necessity, some authors (Voss, Linck, Grossmann) advocate surgical intervention for prophylactic reasons to prevent infection. Voss states that all recent and old infective processes, whether located in the external or in the middle ear, in the mastoid, or in the labyrinth, must be eliminated surgically when there is a fracture of the temporal bone. This viewpoint does not seem justified. In fact, in my own experience in numerous cases of fracture of the skull with hemorrhage from the ear, there was only one case that required mastoid surgery. This was a case in which there was an acute otitis media at the time of injury; four days later a facial paralysis developed on the side of the otitis media, the bloody discharge having become suppurated in the meantime. A mastoid operation, with radical exposure of the area involved, resulted in disappearance of the facial paralysis and perfect healing. Brunner divides all cases of temporal bone fracture into three groups with respect to the treatment. The first comprises the cases of chronic otitis media that would not require an operation were it not for the fracture. There is no indication for surgery in longitudinal fractures, but it may arise in the case of some transverse lesions. The second group includes the cases of chronic otitis that become surgical even without regard to the fracture. In these cases the operation frequently cannot be avoided. The third group includes the cases that acquire an acute otitis media after the trauma. If the fracture is transverse, an operation is absolutely indicated; in the case of longitudinal lesions, however, the surgical intervention follows the usual indication for mastoid operation.

Grove outlines the treatment of fractures of the temporal bone as follows. Should otitis media intervene, a simple mastoid operation will produce adequate drainage. Intense chemotherapy is advised. Should the fracture run through a chronically suppurating ear, radical exposure should be made as soon as the period of shock is over, the fracture line being uncovered as far as possible. If post-traumatic otitis develops in fractures involving the labyrinth, radical surgery plus chemotherapy should be employed. Facial paralysis occurring immediately after the head trauma usually originates internally to the knee and is not amenable to mastoid surgery.

### 2. Concussion of the Inner Ear

The fact that ear symptoms may occur even in cases with a slight or medium degree of head trauma, short of fracture of the temporal bone, was already known to the older otologists (Politzer, Passow, Schwartze). Politzer explained this post-traumatic impairment of hearing on the basis of sudden changes in the structure of the nerve endings due to a "concussion

of the inner ear." Rhese in 1906 examined a great number of concussion cases and gave an exhaustive description of the clinical picture. He found changes not only in the cochlear apparatus but also in the labyrinth.

There has always been an intense dispute as to whether there is such a condition as a concussion of the inner ear. A number of authors (Ulrich, Uffenorde, Nager, Berberich, etc.) deny the existence of such a lesion. They base their arguments upon the failure of proof of the histologic evidence. The lack of pathologic examination is understandable, since such light injuries do not cause sudden death. A microscopic examination performed many years later loses much of its value because of secondary infections that make interpretation of the changes difficult. There are only 2 cases in the literature that can be referred to concerning concussion of the inner ear. One case is described by Theodore, who found a degenerative atrophy in the organ of Corti, in the spiral ganglion, and in the nerve endings of the basal coil and of the labyrinth. Another case was observed by Wittmaack, who described a destruction of the organ of Corti with subsequent degeneration and atrophy of the cochlear nerve and tearing of the sacculus. The rarity of these cases explains why otologists tried another approach to the problem of concussion, i.e., animal experiments. Stenger (1909) subjected rats to blows on the head with a hammer, with various degrees of force. Histologic examinations were made on those animals in which the head trauma did not lead to a fracture of the temporal bone. He observed in mild cases hemorrhages into the cochlea, particularly in the vicinity of the round window. After more severe blows he found hemorrhages also in the ampullae and between the fibers of the acoustic nerve, and alterations of the nerve and its endings and of the ganglion cells.

Brunner in 1925 made experimental investigations on guinea pigs, exposing them to hammer blows. He found hemorrhages by diapedesis, and lymphocytes and transudate in the perivascular spaces and in the peri- and endolymphatic spaces. He stresses the point that concussion of the labyrinth results in acutely increased pressure, with damage to the saccus endolymphaticus and the lymph channels. He concludes that all the changes are due to two factors: one is the pushing wave of the endo- and perilymph, the other the disturbance of the blood circulation of the inner ear. With respect to the important role of the vasomotor circulatory disturbances, he applies the term "vasomotory internal otitis."

FIGURE 77 illustrates the case of a 48-year-old white male who incurred a head trauma fifteen years before death. The trauma led to a temporary loss of consciousness, but no fracture of the head could be found. The slide shows a complete destruction of the organ of Corti and of the basal coil; the endolymphatic space is entirely filled with connective tissue.

There is further a complete atrophy of the cochlear nerve branch and of the spiral ganglion. Connective tissue and bone trabeculae are found in the scala tympani and the scala vestibuli. It is difficult to determine whether these pathologic changes represent an end stage of the vasomotory internal otitis as described by Brunner in its initial phase, or whether the changes are the result of healed secondary infections.

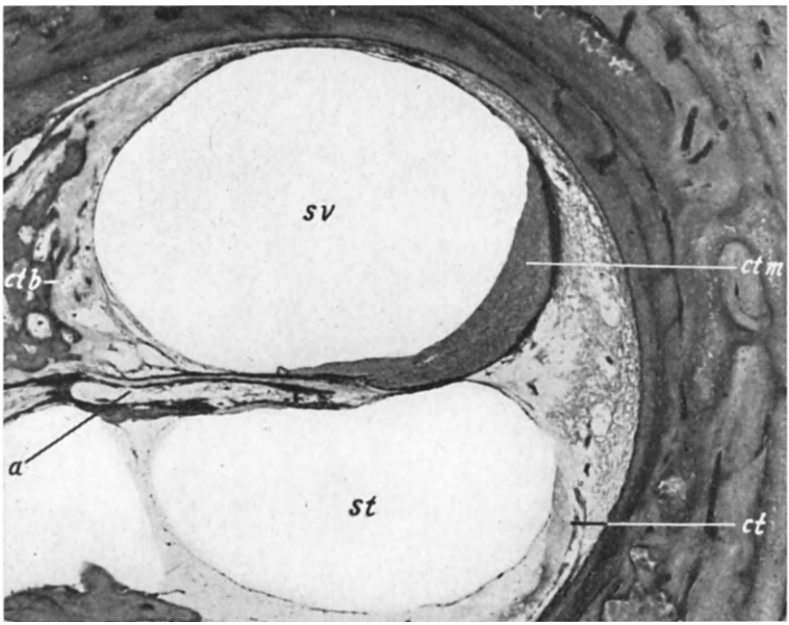


FIG. 77. MICROPHOTOGRAPH OF CASE OF TRAUMATIC DEAFNESS

Section through basal turn of cochlea, showing organ of Corti destroyed and replaced by connective tissue filling entire scala media (*ct m*); scala tympani (*st*) also containing connective tissue (*ct*); network of connective tissue and bone (*ct b*) in scala vestibuli (*sv*); complete atrophy (*a*) of spiral nerve.

Wittmaack, on the basis of his experiments, expounded the following theory. The head trauma elicits a sudden pushing wave, which in turn leads to destruction of the organ of Corti, with subsequent degeneration and atrophy of the cochlear nerve. The hemorrhages into the perilymphatic spaces are negligible. The occurrence of disturbances of cochlear and labyrinthine functions does not depend upon the question of whether the injury is a plain concussion or a fracture of the temporal bone. More important is the site on the skull where the blow was inflicted, or the site from which the pushing wave originated. He further found a greater resistance of the macula utriculi and the cristae to the trauma than of the macula sacculi and the organ of Corti. The author recommends the use

of the term "traumatic degeneration of the labyrinth" as a substitute for "concussion of the inner ear."

### SYMPTOMATOLOGY

For the discussion of the clinical picture, it is advisable to follow the classification of Brunner. In view of the fact that in every case of concussion of the inner ear a concussion of the brain has been found, he divides the cases following head trauma into two groups: (*a*) plain postconcussion syndrome, and (*b*) postconcussion syndrome combined with concussion of the inner ear. To the former group belong the cases with ear symptoms but without any organic changes in the inner ear; the latter group includes cases with ear symptoms of central as well as peripheral origin (inner ear).

*Plain postconcussion syndrome.* It is beyond the scope of our book to discuss all the numerous theories about the mechanism and pathology of brain concussion. Here it is sufficient to mention only the experimental investigations of Jacob, who found, in such cases, hemorrhages in the meninges with obliteration of the meningeal spaces, circulatory disturbances of the cerebrospinal fluid, and degenerative changes in the various parts of the brain. Friedmann (1903) assumed a disturbance of the vasoregulatory center leading to marked degeneration of the small vessels. He observed the following symptoms: headaches, vertigo, tinnitus, photophobia, hypersensitivity to noises, flashes, tachycardia, tenderness to pressure over the sensory nerves, concentric narrowing of the visual field, emotional hyperexcitability. The otologist is mainly interested in symptoms and signs such as tinnitus, impairment of hearing, vertigo, spontaneous nystagmus, and those relating to labyrinthine excitability.

*Tinnitus and impaired hearing* are not so frequently found as is generally believed. Alexander and Scholl described disturbance of hearing in only 1.7 per cent of their cases. Brunner made similar observations.

*Vertigo* is the most common symptom of this group. Osneta and Giliberti observed it in 57 per cent of cases, Glaser in 75 per cent, and Linthicum and Rand in 90 per cent. The latter authors distinguish two types of vertigo, i.e., true or systematized vertigo with sham movements of surrounding objects, and false or nonsystematized vertigo manifested in a feeling of uncertainty or confusion. Glaser differentiates between true vertigo, postural dizziness, general dizziness, and a combination of general and postural dizziness. Brunner distinguishes between general vertigo and labyrinthine vertigo. For a better understanding of the various forms of dizziness, the reader is referred to chapters iii and iv.

The reports concerning *spontaneous nystagmus* vary to a great extent. Grove found it in 70 per cent of his cases, Linthicum and Rand in 30 per cent, and Glaser in only 4 per cent. These variations can be explained

only on the basis of differences in technic and interpretation in regard to spontaneous nystagmus.

The same can be said in regard to *labyrinthine excitability*, which is described as normal, increased, decreased, and even lost. According to Rhese, a normal hearing acuity with hypo-excitability of the labyrinth in relation to caloric and galvanic stimuli is significant of a central lesion. Brunner believes that in plain concussion syndrome the labyrinthine function is either normal or increased, whereas a hypo-excitability or a loss of function bespeaks rather a postconcussion syndrome complicated with concussion of the inner ear. Koch recommends bilateral calorization for the purpose of revealing central tonus differences (p. 131). In cases in which a caloric examination cannot be made, the bipolar galvanic test (p. 143) should be used.

*Summarizing*, it can be stated that the otologic symptoms in this group of post-traumatic disturbances mostly concern the *vestibular region*, whereas the cochlear apparatus remains spared. According to Fletcher and Grove, the spontaneous signs of a deranged vestibular function are more significant than the induced labyrinthine reactions.

*Postconcussion syndrome combined with concussion of the inner ear.* Here we refer to the exhaustive description of Rhese, who, in 1906, summarized the clinical picture as follows: engorgement of the blood vessels of the superior wall of the external meatus; decrease of bone conduction, particularly as concerns medium tones (c-2); peculiar form of the audiogram, showing strikingly similar involvement of both ears, although of different intensity; marked decrease of hearing as regards the sound of a watch (more marked than in regard to speech); fatigue symptom in tuning fork tests; positive Rinne reaction; spontaneous nystagmus toward the non-affected side; homolateral hypo-excitability of the labyrinth; and vasomotor instability.

Rhese distinguishes two groups of head injuries. The first comprises cases with deafness or subtotal loss of hearing, loss of labyrinthine excitability under caloric stimuli, and normal labyrinthine function in relation to galvanic current. The other group includes cases with impaired hearing acuity but loss or hypo-excitability in relation to caloric and galvanic stimuli. The first type represents a lesion in the peripheral ear organ (inner ear), the second a central lesion.

Koch examined 47 cases of concussion of the inner ear short of fracture of the temporal bone. Of these, 45 cases showed ear symptoms. The cochlear apparatus was involved in 39 cases, while 16 had vestibular symptoms. He found total loss of hearing in 7 cases, impairment of hearing of the inner-ear type in 20 cases, and a combined sound conductive–sound perceptive lesion in 12 cases. The labyrinthine test revealed a marked

decrease or a total loss of function. In some cases the labyrinthine excitability was normal under turning stimuli, but there was no response to caloric irritation. Grove, Brunner, Fletcher, etc., described cases of bilateral involvement of the cochlear apparatus. One side, however, always showed a more marked impairment than the other. *Summarizing*, it can be said that the changes in this group involve particularly the *cochlear* apparatus, in contrast to the cases of the previous group. Another differentiation lies in the bad prognosis as to the function, which is not surprising if one considers the pathologic basis. In the plain concussion group, the inner ear does not show any organic changes.

This discussion shows that although the pathology of concussion of the inner ear is not clear as yet, a lesion in the ear does exist following head injuries short of fracture. It is therefore unnecessary and even unjust to the patient to assume a priori a functional genesis such as neurosis, hysteria, aggravation, or malingering.

### 3. Trauma Due to Explosions

In the first world war many injuries of the ear organ were observed that were due to the blast of high explosives. The great development in technical warfare, particularly the invention of the various forms of aerial bombs and depth charges, has led to a marked increase in the incidence of such cases. Since the present war involves the civilian population to almost the same extent as it does soldiers, such injuries, very rare in peace times, become more and more common. Damage due to blast can be caused by explosions of guns, high-explosive shells from artillery and trench mortars, grenades, bombs, torpedo mines, and munition dumps.

The damaging effect of guns on the ear organ is limited to close range. The high-explosive shells have a thick iron casing enclosing violent explosives; of the same type is shrapnel, which consists of a steel cylinder containing a number of round lead balls. The grenades are similar in principle, though they are hand-thrown or fired from a rifle. Aerial bombs have thin walls but are filled with a maximum of high explosives; thus the bursting bombs break into numberless tiny fragments that travel with a tremendous velocity. Mines are either terrestrial or marine; the former type consist of high explosives buried in the ground and released by an electric spark, the latter are put beneath the surface of the water or are sent down as depth charges.

The main factor as regards all the various explosives is the sudden great increase of pressure in the air within the focus of explosion. This sudden increased pressure produces a wave of compression (arhythmic wave) that, with increasing distance from the focus, gradually continues as a

common sound wave (rhythmic wave). There is great diversity of opinion as to the damaging role played by the two different waves. The damaging effect of the compression wave upon the ear organ is easily understood. The sound wave, however, which represents an adequate stimulus of the ear organ, may lead to injury only—according to Lange—when its intensity far exceeds physiologic limits. Mauthner does not believe in the damaging effect of the sound wave in relation to the blast of high explosives; the compression wave has usually reached and already struck the ear before the arrival of the sound wave.

Wolf describes the mechanism of explosion as follows. At the site of contact between highly compressed explosive gases and the surrounding medium (air), a compression wave is produced, followed immediately by a suction wave. Both follow the physical rules of longitudinal waves. The compression component is short, sudden, powerful, and travels with a tremendous velocity away from the focus of explosion (positive phase). The suction component is weaker, of longer duration, and is directed toward the focus (negative phase). The same holds true, for instance, in the case of a missile passing close by the ear, exerting a positive as well as a negative pressure.

Blast injuries resulting from the detonation of high explosives under water are called "immersion blasts." The critical distance from the center of detonation is approximately four times greater than it is in the air.

The damaging effect on the ear organ depends on a number of factors, such as the kind and volume of the explosives, the distance of the focus of explosion from the ear, the rapidity of increase and decrease of air pressure, the position of the ear in relation to the oncoming wave, and the obstacles between the focus of explosion and the inner ear. The obstacles can be either of external or internal kind: the former comprise the walls of the explosive shell, buildings, protective walls, etc; the latter include the anatomic and pathologic changes in the ear organ.

A very important factor lies in the rapidity of changes of air pressure. A relatively slow change, such as is observed in caisson work (see p. 393) or in aviation during ascent or descent, may lead only to transient lesions of the drum or the tympanic cavity (see p. 393). In blast from high explosives, however, the increase or decrease of air pressure takes place with a tremendous velocity, hence damage to the inner ear can often be observed. In order to understand the various ear symptoms due to explosions, we suggest the following classification, according as they are due to (*a*) a sudden blast (acute trauma); (*b*) repeated short explosions (chronic trauma); (*c*) concussion of the brain; (*d*) concussion of the brain combined with concussion of the inner ear; (*e*) brain concussion combined with fracture; (*f*) psychogenic causes.

### EAR SYMPTOMS DUE TO A SUDDEN BLAST (ACUTE TRAUMA)

A review of the literature reveals the complete absence of histologic examinations of the human ear organ that could illustrate the pathologic-anatomic basis of such injuries. The trauma has either been so violent that the body was torn to pieces or else has not led to immediate death. The lack of pathologic examinations forced the otologist to look for other ways and means of research, such as the animal experiment. Wittmaack, in 1908, was the first to undertake such investigations. He subjected animals to the blast of gunshots fired close to the ear. One group was examined just after the first trauma, the other several days later after repeated shots. In the first group he observed rupture in the drum and hemorrhage in the middle ear, but none in the inner ear; the sensory and supporting cells of the organ of Corti were jumbled promiscuously. In the other group he found advancing destruction of sensory and nerve cells and nerve fibers. Wittmaack applied the term "detonation neuritis" to these changes. Similar results have been obtained by Yoshii, who further observed more intense changes in the inner ear when the drum membrane remained intact. He believed that in cases with rupture of the drum the compression wave travels via the round window, thus diminishing the effects of damage. This is in contrast to the assumption of the older otologists (Politzer, Passow) that the drum plays a protective role in blast injuries of the inner ear.

Hoessli was able to show that damage to the cochlea occurred always in the same place—regardless of the quality of the shot explosion—i.e., in the superior part of the first turn and in the beginning of the second, extending up to the middle portion of this coil. Popoff found changes also in the labyrinth, but he employed more violent forces. Prenant and Castex subjected rabbits and guinea pigs to detonations of heavy cannon of modern type. They found, histologically, dislocation of the cochlear duct, with destructive changes in the organ of Corti; effusion of blood in the scala tympani, due to rupture of minute vessels; atrophy of the organ of Corti and of ganglion cells; and occasionally degenerative changes in central fibers of the cochlear nerve. The labyrinth did not show any pathology.

The *mechanism* of blast injuries of the inner ear has been studied by Mauthner, who arrived at the following conclusions:

The compression wave reaches the inner ear via the external and the middle ear. The labyrinthine windows are considered as the actual sites of the effect of compression or suction following blasts. The extent of the damage is proportional to the kind and volume of the explosives, the distance of the focus of explosion from the ear, and the obstacles between the focus of explosion and the inner ear.

The wave of negative pressure (suction) may lead to a diminution of the pressure of the vessels, thus damaging the inner ear. Another possibility

of damage to vessels or nerves of the inner ear may arise from the poisoning effect of certain gases released by the explosion.

The *clinical picture* will depend largely upon the extent of the damage to the inner ear. There are numerous cases described in the literature. The first systematic examinations of the inner ear in connection with a disastrous explosion were performed by Hofer and Mauthner in 1912, when an ammunition plant near Vienna containing 350,000 pounds of gunpowder exploded. The investigators classified the persons involved in the disaster into three groups: the first was made up of those who had been on the scene of the explosion or in close vicinity to the stored ammunition; the second comprised persons who had been within a range of 200 meters, while the third group included all the persons who had been within a range of 200 to 1,200 meters.

On the first group no examination could be performed, since all these persons were torn to pieces.

The second group contained many cases of inner-ear damage, showing the following symptoms and signs: bilateral impairment of hearing, although one side was more involved than the other; decrease of bone conduction, with lowering of the upper tone limit (the hearing loss for high tones amounted to 40 per cent); and slight impairment of hearing for low tones. Spontaneous nystagmus could be observed in all the cases immediately after the trauma.

In the third group the subjects usually complained of headaches, earaches, sensations of fulness, tinnitus, and vertigo. Functional testing revealed a slight impairment of hearing of the inner-ear type. The farther from the explosion the person had been, the more likely was the ear lesion to be unilateral. Spontaneous nystagmus occurred in 20 per cent of the cases.

Castastrophic explosions of such extent are fortunately rare. Mauthner in his capacity as an army otologic expert had plenty of occasion to study blast injuries. In spite of the great variety of symptoms, he was able to outline a main type of inner-ear lesion due to sudden explosions, the signs of which are unilateral impairment of hearing and lowering of the upper tone limit owing to injury of the basal coil. The shortening of duration for high tones does not so much involve c-5 or c-4 as might be expected, but affects the tone scale from c to c-2. The changes in the upper tone range are independent of a coincident rupture of the drum membrane. Spontaneous nystagmus is almost always present in the first week after the trauma. There is occasionally a hypo-excitability of the labyrinth in the early stages.

Streit observed spontaneous nystagmus in 47 cases out of 240. Tobeck studied 200 cases of ear lesions in the first world war and found the drum

membrane ruptured owing to exploding grenades in 49 per cent of cases, and owing to exploding mines in about 20 per cent. The most frequent sign of auditory disturbances was marked reduction of auditory perception of whispering, with consequent mixed hearing impairment. I observed rupture of the drum in approximately 30 per cent of cases of blast injury.

Collier, Colledge, and Negus reported on war injuries of the ear in the Spanish civil war. The authors observed 175 injuries by blast against 33 by bomb fragments. Negus found, among 622 civil air raid casualties, 193 with injuries of head and neck, 129 with craniocerebral lesions, and 24 with damage in the ear.

### EAR SYMPTOMS DUE TO REPEATED SHORT EXPLOSIONS (CHRONIC TRAUMA)

If the trauma is slight either because the amount of explosive was small or because of great distance from the focus of explosion, the inner ear will not show any pathology. If, however, such slight insults are repeated often over a certain period of time, damage to the inner ear will occur. The pathologic changes in such chronic trauma are exactly the same as those first described by Habermann in cases of occupational deafness. The main changes occur in the upper parts of the cochlea, in contrast to the changes in acute blast injuries, which particularly involve the basal coil. Wittmaack stresses further the difference in mechanism of the two lesions. Whereas in the case of acute blast the compression wave travels via air conduction, in the chronic injuries the main role is played by the bone conduction. The clinical picture is very characteristic. Such cases show progressive impairment of hearing, marked decrease of bone conduction, lowering of the upper tone limit, and tinnitus of a high-pitched character. This lesion is usually found in professional soldiers, particularly artillerists, machine gunners, bombers, and also pilots of combat planes.

### EAR SYMPTOMS DUE TO CONCUSSION OF THE BRAIN

The blast of high explosives may either directly cause a so-called "shell concussion of the brain" or may lead to a blow on the head that indirectly results in a concussion. Although there are no pathologic changes in the inner ear, the patient shows clinically the so-called postconcussion syndrome (see p. 358). There are usually spontaneous signs of a deranged vestibular function, while the cochlear apparatus seems to be intact.

### EAR SYMPTOMS DUE TO CONCUSSION OF THE BRAIN AND OF THE INNER EAR

This group includes cases in which the inner ear is damaged by the effect of blast as well as by the effect of trauma of the head. The cochlear apparatus is severely damaged, with consequent deafness, while the labyrinthine function may still be present.

### EAR SYMPTOMS DUE TO BRAIN CONCUSSION AND FRACTURE OF THE SKULL

This group includes the cases with the most severe injuries of the ear organ. In transverse fractures a complete loss of cochlear and labyrinthine function will be found. But the longitudinal fractures also will show a more severe course, since to the usual damage of the middle ear the blast injuries of the inner ear must be added. It is obvious that these pathologic changes make the prognosis for the inner-ear function unfavorable even in longitudinal fractures.

### EAR SYMPTOMS OF PSYCHOGENIC NATURE

In the first world war all the army physicians were amazed by the great number of soldiers affected with bilateral deafness or deaf-mutism following an explosion of high shells. The patients usually had no visible wounds or signs of a skull fracture. Because of the lack of organic changes, the physicians considered such patients as hysteric cases or malingerers. However, there were a number of patients who had neither any signs of hysteria nor any tendency to malinger. Since the mechanical trauma had elicited a psychic trauma that in turn led to the sudden deafness, the psychogenic basis is obvious. The main role in the pathogenesis of such a lesion is played by the severe sudden fright; therefore it was referred to as "fright neurosis." It was interesting to note that bilateral deafness did not occur in cases of direct blows on the head with subsequent loss of consciousness. Muck considers the bilateral deafness or deaf-mutism following detonation of high-explosive shells as a "psychic elimination of the sense of hearing" due to the psychic trauma. It shows a great resemblance to hysteric deafness, but is not identical.

Examination of such patients reveals a complete loss of hearing, while the labyrinthine function remains intact. In spite of a complete bilateral deafness, the auropalpebral reflex is elicitable in both ears; when a c-4 tuning fork is struck close to the patient's ear, an eyelid reflex occurs. It is further interesting to note how quickly the patients are able to lip-read, a faculty that usually requires many years of special training. If, however, the patient's ear canals are tightly plugged, this capacity of lip-reading is lost. This would prove that such patients are capable of hearing, although subconsciously. Hurst directs attention to the fact that hearing does not consist merely of the perception of impulses conveyed to the brain when the ear is stimulated. In order to hear sounds, the individual must listen. Impulses directed to the cortical centers of hearing are interrupted by inattention, in consequence of which the synopses in the auditory path are unswitched, possibly as a result of retraction of the dendrites. Further examinations reveal an analgesia of the external ear or even of the entire body surface, but the sense of touch remains intact. Whereas in

some cases the deafness subsides after few hours or days, many other patients show a permanent loss of function.

In cases of *partial impairment* of hearing following explosions, we may assume a combination of an organic lesion with a psychogenic factor. It is often very difficult or even impossible to determine to what extent of involvement each factor participates. This may be accomplished if the physician has opportunity to observe the patient for a long period of time. Repeated functional examinations may reveal some lack of agreement in the results.

### 4. Gunshot Injuries

The literature contains numerous articles concerning gunshot injuries of the ear. However, a thorough study of the papers from the various countries reveals the fact that there is no characteristic complex of symptoms that can be used for diagnostic purposes. In all the various war injuries, such as skull fractures, concussions, and explosions, a certain regularity has been observed. However, this does not apply to gunshot injuries. The variance of symptoms can be explained in the light of a number of factors, such as the impossibility of determining the direction of the missile, the kind of gun used, and whether the case represents direct injury by the projectile, or indirect damage to the inner ear due to fractures or fissures of the bony labyrinth capsule. Furthermore, extensive hemorrhages into the vestibule, the semicircular canals, or the cochlea may also occur along with laceration of the nerves. It is further important whether the ear organ is the sole site of injury or whether there is concomitant brain involvement.

Nevertheless, efforts have been made to classify gunshot injuries of the inner ear into groups, such as (*a*) *direct* injury caused by a straight hit of the projectile itself or of accompanying foreign bodies, and (*b*) *indirect* injuries due to fractures, fissures, and splinters of the otic capsule, to hemorrhages and dislocation of tissue, to the transmission of the impact, and finally to the compression wave of the blast. Another classification is according as the damages are due to shots at long or short range, to tangential, segmental, perforating, ricocheting, penetrating, or skimming shots.

In a *tangential shot*, the direction of the projectile is almost parallel to the surface of the skull. The bone of the capsule sustains a damage in the form of a groove with radial fine fissures.

In a *segmental shot* the projectile penetrates the skull at an acute angle; the wound of exit is often pretty close to the wound of entry.

In a *penetrating shot* the missile becomes lodged within the skull.

In a *perforating shot* there is a hole of entry and a hole of exit.

In a *ricocheting shot* the projectile, rebounding from a flat surface, has lost most of its force by the time it hits the skull.

In a *skimming shot* the missile traverses only the soft tissues and does not show any visible injury of the bony skull. Although no damage on the outside of the bony skull occurs, there is very often a splintering of the internal lamina (lamina vitrea), with subsequent injury of the dura or brain.

This classification, however, has a minor diagnostic value, since for instance a slight skimming shot often causes a homolateral deafness accompanied by a contralateral impairment of hearing. On the other hand, a severe perforating shot need not necessarily affect the inner-ear function at all, particularly if it pierces the skull in a sagittal direction, entering by the occiput and issuing via the mouth or face.

Large-scale investigations of gunshot injuries of the ear have been carried out by Alexander and Urbantschitsch, who saw 2,500 such cases during the first world war. Their statistics show that out of 1,000 cases of war trauma, 225 cases (22.5 per cent) represented direct gunshot injuries, while 602 (60.2 per cent) showed indirect damage of the inner ear; this gives a ratio of 1:3. Hinsberg observed 8 direct injuries among 100 cases.

*Direct injuries* of the inner ear show a high mortality rate, since they usually are combined with mortal damage to adjacent vital parts (brain, carotid artery). Particularly dangerous are—according to Alexander—diagonal shots through the skull or perforating shots with vertical direction, for instance with entry on the parietal bone. Those cases of direct injury that survive have an unfavorable prognosis with regard to function. Usually complete deafness and loss of labyrinthine excitability ensue. Another danger in cases of direct injury of the inner ear lies in the possibility of secondary infection: a purulent meningitis may immediately follow the trauma or may even occur weeks or months later (Politzer, Klestadt). The incidence of meningitis amounts to 50 per cent, according to Haymann.

*Indirect injuries* of the inner ear are more common. Alexander found the inner ear involved in 86 per cent of his cases of gunshot injury of the middle ear. He further observed impairment of hearing in an excessively high percentage of cases due to skimming shots, where the bony capsule of the skull showed no visible changes. The hearing defect was of the inner-ear type, but showed a disproportionate marked decrease of the bone conduction. Alexander explained this phenomenon on the basis of an increased endocranial pressure due to the meningeal trauma.

Mauthner observed indirect damages to the inner ear particularly in those cases in which the gunshot injury was combined with a fracture of the jaw. Ruttin found the inner ear always involved in cases due to shots of sagittal direction when the face was hit.

The *clinical picture* of the direct as well as of the indirect injuries must therefore be manifold. It is governed either by the brain symptoms or by

signs and symptoms of a fracture of the skull. In other cases, ear symptoms may occur owing to concussion of the inner ear or to the blast injury. In addition to these, there may be signs and symptoms of the various forms of traumatic neurosis, such as fear neurosis, concussion neurosis, hysteria, neurasthenia, etc. This shows clearly that a complex of symptoms characteristic for gunshot injuries of the inner ear does not exist.

### TREATMENT

The treatment depends upon the kind of gunshot injury and the factor of whether or not the brain is involved.

*Tangential shots* usually require immediate surgery of the skull.

*Perforating shots* should be treated conservatively. Only in those cases in which the bone is splintered to a large extent are operative measures indicated.

*Penetrating shots* should be let alone in the absence of any symptoms; if the projectile is easily amenable, removal may be tried. Operation is indicated for cases with intracranial symptoms.

*Ricocheting shots* should be treated conservatively unless the bone shows extensive splintering.

Generally it can be said that the treatment of gunshot injuries must be a gentle surgical one. Alexander outlined the treatment of gunshot injuries of the ear as follows: (*a*) exposure of the dura for clear, thorough inspection; (*b*) elimination of all necrotic particles or of such as may cause necrosis; (*c*) early surgical care and definitive rest, avoiding any unnecessary transportation of the patient.

Bárány advocated during the first world war a new method of treatment for gunshot injuries of the skull, consisting of a primary closure of the dura that produced excellent results. Alexander, however, preferred the method of endodural drainage, arguing that the primary closure can be performed only if the brain is completely normal; the slightest thrombosis of dural vessels could easily lead to the development of a brain abscess. He opposed the primary closure also in cases in which the patients had to be transported after the operation.

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# XIV

# The Role of the Inner Ear in Aeronautics

By *Louis E. Wolfson*

Man's sensory organs have for thousands of years proved adequate to all his activities in his earth atmosphere environment. The organs serving for spatial orientation are the labyrinth, the eye, and the proprioceptors. The labyrinth, however, does not play such an important role in man as it does in animals, particularly in those living in water and the air. It has been shown in chapter ii, for example, that in fish and amphibia the body equilibrium is controlled solely by the labyrinth, while in man the labyrinth function is assisted by other mechanisms. While destruction of the labyrinth in lower animals means annihilation of life, such destruction in man is soon overcome by compensation. The labyrinth is particularly well developed in fish and in birds that must move in two elements. In the latter the three semicircular canals are very long and very high and narrow, and have a special arrangement of the vertical canals; the sacculus is exceptionally small. It is therefore no wonder that the human labyrinth does not function properly in the new environment of the air. The labyrinth is not able to distinguish between gravity—in accordance with its function on the earth's surface—and the sense reactions produced by centrifugal forces during movements in an aeroplane.

In order to understand the problems of aviation, it is necessary to consider the following conditions: (*a*) the physiology of the cochlea and labyrinth in the usual environment; (*b*) the physiology and pathology of the cochlea and labyrinth in the unusual environment (air); (*c*) functional tests and aviation fitness.

## I. Physiology of the Cochlea and Labyrinth in the Usual Environment

### Cochlea

The physical elements involved in sound production are the adequate stimuli for the organ of hearing. Sound—in a physical sense—is the result of vibration that takes place in a body disturbed by shock or friction. We differentiate between sound and noise. The former has a smooth, steady,

periodic course, while the latter is irregular, unsteady, and rough. A sound produced by a simple pendulum vibration is called a tone. The characteristics of a tone are its pitch, intensity (volume), and quality (timbre). The pitch depends upon the number of vibrations per the time unit, the intensity upon the force and amplitude of the vibrations, and the quality upon the form of the vibrations (material of vibrating substance).

The organ of hearing serves two functions: one is the conduction of sounds, the other the analysis of sounds. The apparatus for conduction consists of parts able to vibrate (external, middle, and inner ear). The apparatus for analysis comprises the cochlear nerve and its distributions The sound reaches the inner ear by two ways, i.e., the air or the bony skull. The latter can be subdivided into a direct way (cranial conduction) and an indirect way (craniotympanic conduction).

*The apparatus for sound conduction.* According to the commonly accepted theory, sound waves striking upon the tympanic membrane are by it communicated to the ossicular chain, by which in turn they are transmitted through the footplate of the stapes to the labyrinthine fluids. The excursion of the stapes in either direction is accompanied by a corresponding movement of displacement of the fluid in the vestibule, inaugurating fluid waves that finally impress the terminal filaments of the auditory nerve. For the execution of these passive movements, the tympanic membrane and ossicles constitute a perfectly adapted mechanism. The handle of the malleus is longer than the long arm of the incus; that is, with each inward excursion of the hammer handle, the long arm of the incus moves forward, carrying the stapes with it through a shorter distance, but with greater changes. This is of especial importance in the transmission of the lower musical tones, which are produced by vibrations of large amplitude but little force. Apparently the ossicular chain is most essential in transmitting the lower tones, which are transformed by the ossicles into vibrations of smaller amplitude but greater force. This explains impairment of hearing for low tones in connection with any severe lesion of the conducting mechanism.

*The apparatus for sound analysis.* The cochlear apparatus consists of an incompressible fluid and of elements capable of vibrating (radial fibers of basilar membrane). The vibrations are finally converted to inward and outward movements of the stapes footplate that in turn lead to changes in the endolabyrinthine pressure. Since, however, the fluid cannot be compressed, an escape becomes necessary. We find it in the form of the secondary tympanic membrane in the round window.

The numerous theories of hearing can be classified into three groups, representing the following standpoints:

1. The peripheral end organ of the cochlea is directly stimulated by periodic-mechanical vibrations and is capable of perceiving them as tones (Koenig, Taminoga).

2. Resonating elements transmit the stimuli to the nerve endings. The cochlea breaks up the sound vibrations into their simple components. The analysis of tone occurs in the cochlea. The nerve impulses from each sector of the basilar membrane are transmitted to the brain.

This resonance theory (Helmholtz) is the most popular. There is, however, great diversity of opinion as to the resonating part of the cochlea. Helmholtz considers the basilar membrane as the actual resonating element. It is 33½ mm. in length and consists of 25,000 fibers that increase in length from the base of the cochlea to the top in a ratio of 1:12. The membrane shows a great tension in a radial direction but only little tension in its longitudinal axis. An inward movement of the footplate of the stapes increases the pressure in the scala vestibuli, which in turn pushes the basilar membrane down into the scala tympani. An outward movement of the stapes has the opposite effect. It is clear that the organ of Corti, based upon the basilar membrane, will show the same up-and-down motions. This leads to attachment and detachment of the hairs of the ciliated cells with respect to the tectorial membrane. The fibers of the basilar membrane vibrate in sympathy with the sound waves, i.e., certain fibers or groups of fibers vibrate to certain tones. Since the fibers are short at the base of the cochlea, the high tones are localized there, while the apex of the cochlea is the site of the low tones.

Shambaugh opposes the Helmholtz theory that the basilar membrane constitutes the resonator of the cochlea. He believes that the tectorial membrane is the vibrating element. He further points to the analogy with the nerve endings in the labyrinth: there is the same histologic structure in the maculae, the cristae, and the organ of Corti.

3. The analysis of tone takes place in the central organ. Rutherford believes that the sound vibrations are directly transmitted to the nerve and are differentiated in the auditory center in the brain. He assumes the same mechanism as in telephony, where sound waves are converted into electricity (telephone theory).

The Weaver-Bray experiments, in which electrodes are attached to an amplifier placed on the exposed eighth nerve, brought new theories of hearing based upon the idea of a central analysis of tones.

Although the Helmholtz theory cannot explain all the problems involved, it is still worth considering and cannot yet be replaced by any of the "new theories" of hearing. There is now, on the contrary, a tendency to return to the old theory of Helmholtz (Crowe, Guild). Recent authors, such as Davis, Derbyshire, and Lurie, after working on the Weaver-Bray experi-

ments, arrived at the conclusion that neuro-anatomic and physiologic evidence is in favor of the resonance theory. Lurie finds the pitch perception definitely localized in the basilar membrane of the cochlea. The external hair cells are responsible for detection of very faint sounds, while the internal hair cells are concerned with the fine discrimination of pitch. The chief cause of perception deafness is a degeneration of the external hair cells.

### LABYRINTH

The problems concerning the physiology of the labyrinth have been discussed in detail in chapters ii and iii. Before exploring the subject of aviation medicine, a short review seems to be advisable. The manifold functions of the labyrinth can be summarized as follows:

Control of angular movements;
Control of linear movements;
Control of position;
Control of the eye muscles;
Control of the skeletal musculature (tone);
Control of equilibrium within the frame of the other centripetal mechanisms;
Kinetovisual function;
Influence upon the autonomic system.

The semicircular canals react only to movements with *angular acceleration*, but do not respond to movements with permanent velocity. If the head or the body is rotated, only the beginning or the end of the motion or a change in the speed will cause a sudden push on the endolymphatic fluid, which in turn leads to deflection of the cupula and the hairs of the sensory cells. In order to stimulate the cristae, the change in direction or speed must be of a certain amount over a certain period of time (physiologic threshold), as for example: a magnitude of 2 to 3 degree seconds over a period of 14 to 16 seconds (Mach); a magnitude of 72 degree seconds over a period of 1/45 second (von Rossem); a magnitude of 2 degree seconds over a period of 0.8 second (Mulder). The various reactions elicited on turning a person can be classified into subjective reactions (vertigo, turning sensations, nausea, etc.) and objective reflexes (nystagmus, falling, pastpointing, etc.).

The *control of linear movements* is still under debate. Some authors consider the otolith organs as the site of stimulation (Mach-Breuer-Brown, Schubert), others make the semicircular canals responsible (Magnus, Lorente de Nó), while De Kleyn believes in a combined crista and macula function. The physiologic threshold for vertical linear movements is 12 cm. sec.$^2$ (Mach) or 4 cm. sec.$^2$ (Bourdon); for horizontal linear movements figures between 2 to 20 cm. sec.$^2$ are given.

In the *control of position* the stimulus acts upon the otoliths of the maculae. The driving force is the gravity. There is a diversity of opinion as to whether pressure or traction of the hairs of the sense cells is responsible for stimulation. In any position of the head, a constant definite stimulus is produced by the otolithic organ, eliciting a reflex that persists as long as this position is maintained (postural reflex of Magnus and De Kleyn).

The *control of the eye muscles* was first recognized by Hoegyes (1881). He assumed that equal impulses are sent from both labyrinths to the eyeballs, so that a certain balanced position of the eyes is maintained. Bartels believed that each labyrinth—in exerting an influence upon the tonus of the eye muscles—shows a continuous preparedness to elicit nystagmus toward its own side.

The theory that the labyrinth *controls the tonus of the skeletal musculature* was founded by Ewald. He proved experimentally that each labyrinth is concerned with the tonus of the homolateral extensors and abductors and with that of the contralateral flexors and adductors. Resection of one labyrinth has a crossed effect upon the muscles that move head and spine. Impulses emanating from the labyrinths are transmitted to the centers and central pathways, thus reaching the muscles.

The function of *human equilibrium* represents a complicated muscular activity. It is concerned not only with the maintenance of the static equilibrium but also with the ability to perform skilfully complicated actions (coordination). All these mechanisms are rooted in the eye, the labyrinth, and in the sum total of proprioceptive factors (skin, muscles, tendons, viscera). The role of the individual components in this cooperation is still the subject of controversy. Some authors believe that the labyrinth is the most important of the three factors, since the slightest disturbance is sufficient to upset the statics. The proprioceptive component is the least important, as even severe disturbances do not produce dizziness. Others take an opposite viewpoint. Foerster uses the anatomic pathways as the basis of his argument. Whereas the labyrinth influences the cerebrum and cerebellum, the proprioceptive influence extends to the cerebrum and cerebellum and also to the spinal cord. Hence the proprioceptive component is the most important one.

The *kinetovisual function* (*Arellano*) of the labyrinth is of great importance for clear perception in vision of moving objects during movements of the head. The nystagmus elicited by stimulation of the semicircular canals obliges the eyes to follow the movements of the different sectors of the panorama, which in turn leads to an immobilization of the optic image on the retina. By these means vision is accomplished in the same manner as in the case of still objects.

*The influence upon the autonomous system* can be seen in changes of the

respiratory curve, of the blood pressure and pulse rate, of the pupillary reaction, etc. Stimulation of the labyrinth leads to inhibition of the central mechanism of the respiratory regulators and to vascular dilatation in the region of the splanchnic nerves. An irritation of the labyrinth may therefore elicit symptoms such as nausea, vomiting, palpitation of the heart, sweat, pallor, etc.

## 2. Physiology and Pathology of the Cochlea and Labyrinth in the Unusual Environment (Air)

### COCHLEA

Among all the sensory organs that come into play in aviation, the ear is the most common site of involvement. In particular, the cochlear apparatus is exposed to numerous minor or major acoustic traumas. For our knowledge of this subject credit must be given to the investigations of Gradenigo, Scott, Bauer, Foges, Troina, Myrick, Balla, Bunch, Armstrong and Heim, Firestone, Green, Dickson, Ewing and Littler, Campbell and Hargreaves, etc.

The main sources of damage to the ear organ are the noises of the engine exhaust, of the propeller, and of the wind (slip stream), and the vibrations of the structure inside of the cabin. Other sources arise from the sounds and noises of the earphones of the aircraft radios to which the modern pilot is exposed. The fact that damage to the ear occurs is generally recognized, but there is great diversity of opinion as to the percentage of incidence, as to the kind of ear lesion, and as to the interpretation of the hearing curves.

Scott, Bauer, and Foges found a temporary loss of hearing following airplane flights, while Myrick could not observe such a decrease in his cases. Armstrong examined pilots of the American Air Corps and found in 76 per cent a normal hearing. He concludes that deafness in pilots is not as extensive as was formerly believed. Myrick found a permanent decrease of only 1.5 per cent in hearing in flyers as compared to nonflyers. Bunch considers the impairment of hearing as a nerve deafness of the occupational type, with an abrupt dip at 2,048 or 4,096 double vibrations. Green made large-scale audiometric studies of pilots that resulted in a striking absence of findings of serious occupational deafness: he found the average air conduction loss to be 5.5 per cent and the average bone conduction loss 9.7 per cent. Dickson, Ewing, and Littler made audiometric tests on flyers of the R.A.F. and found a high tone deafness after a few hundred hours of flying without a protective helmet. Believing that the auditory defect is at first temporary but gradually becomes permanent, they state that aviator's deafness is a potentially serious and lifelong disability.

While the injuries due to noises and sounds can easily be understood, the role played by the *vibrations* is not yet clear. Firestone offers a new theory of auditory defects in aviators, built on effects of vibrations on the human organism during flight. He quotes an article of Koelsch, who studied the effects of vibration and noise on a certain group of manufacturing workers and who found a vasoneurotic diathesis and productive changes in the region of the capsules and the tendons of the joints. Firestone assumes analogous changes in the aviator's bony labyrinthine capsule and even some productive changes in the oval window region as results of noise and the violent vibrations of his entire musculoskeletal system. Functional tests of all his pilots revealed a deterioration in bone conduction. He therefore suggests applying to aviator's deafness the name *aero-otosclerosis*. This term, however, is not justified and it must be hoped that it will not be accepted. Otosclerosis represents a disease of unknown etiology. The diagnosis can be made either tentatively on the basis of certain clinical symptoms or definitely from microscopic slides post mortem. In the former case, the characteristic symptoms are a positive family history, normal drums, patent eustachian tube, *prolonged* bone conduction, elevated low tone limit with almost normal high tone limit, and negative Gellé test. Another argument against such a name is the absence of any proof that otosclerosis is caused by traumatic vibrations.

The role played by *fatigue* of the cochlea is well known. A continuous stimulation of a definite tone site in one ear will lead to deafness for that specific tone in that ear. The same tone that is not heard by the fatigued ear will be normally heard by the other ear. Also, the ear deafened to that particular tone will be normal for other tones. For that reason, when making hearing tests with tuning forks, otologists are careful not to hold the tuning fork continually against the ear, but to take it away and bring it back to the ear every few seconds.

Campbell and Hargreaves divide the auditory defects of flyers into two main groups, i.e., perceptive deafness and conductive deafness. The first includes acute and chronic fatigue of the end organs of hearing and related structures, the second comprises the changes in middle-ear pressure and the effects of faulty ventilation.

The foregoing discussion indicates that aviator's deafness is partly due to an inner-ear nerve lesion (occupational type) and partly to an affection of the sound-conductive apparatus.

Animal experiments have shown that a noise level exceeding the value of 120 decibels and of higher frequencies leads to a damage of the organ of Corti. The latter type will be discussed in the next chapter (aero-otitis, Armstrong).

Summarizing, it can be said that nerve deafness usually starts with an

abrupt dip in the 4,096 double vibration range, involving the air conduction as well as the bony conduction, no doubt owing to the proximity of the cochlear basal tone site to the middle ear. The frequencies below and above remain intact for a certain period of time. In this initial stage the patient is not aware of his impaired hearing. If, however, the patient is exposed to the traumatic effects of noise and vibration for a longer period of time, an involvement of other frequencies, such as 2,048 or 1,024 double vibrations, may be observed. Such a loss of hearing can then not only be recognized by means of an audiometric investigation, but can also be noticed by examination of the spoken voice. It is known that speech comprises certain frequencies and intensities. The vowels consist of relatively low frequencies but high intensities, while the converse is true for the consonants. Hence a patient in such a stage of hearing loss has difficulties in understanding words with high consonants.

The factors influencing the occurrence of aviator's deafness are: the total of flying hours; the length of time of each flight; the type of aircraft; the flyer's occupation; his susceptibility; deficiency of protection.

Audiometric investigations on flyers with many years of experience have not yielded substantiated results. It has been shown that a number of pilots with more than 800 flying hours had a normal hearing, while others with much less flying time revealed a marked impairment of hearing. Those pilots who show loss of hearing after years of flying usually have a dip in the range of 1,024 to 4,096 double vibrations or higher.

The cumulative effect of *repeated single flights* has been illustrated by the investigations of Campbell and Hargreaves. The experiments consisted of six one-hour periods of flight on different days, six two-hour periods of flight, and four four-hour periods of flight. The greatest loss of hearing occurred after the four four-hour periods, amounting to 25 decibels for 4,096 double vibrations, with a widening of the gap extending from 1,024 to 8,192 double vibrations. In each of their experiments, complete recovery followed a period approaching the square of time during which the fatiguing force was applied. These examinations show clearly that complete recovery can be expected if the rest period is adequate.

Damage of the cochlear function depends to a certain extent upon the *type of aircraft.* Factors influencing noise and vibration are the number of engines, their horsepower, the number of revolutions, the length of the propeller, the arrangement of the exhaust stack, the position of the cockpit, etc. Campbell and Hargreaves found a relatively high loss of hearing by flight in the type BT-2 aircraft used by the American Air Force.

The incidence and intensity of aviator's deafness may further depend upon the flyer's *occupation,* i.e., whether he is a pilot, radio man, gunner, or common crew member. Foges does not believe that the noise and vibra-

tion of the engines are a cause of avaiator's deafness. He makes the intensity of the signal tones responsible for it, and urges that every aircraft should be equipped with an instrument that indicates the allowed maximum for the signal tone. Carson also believes that headset noises are much more disturbing in aircraft than any other noise.

The *susceptibility* of the flyer's ear organ plays a great role, although we are not able to explain this fact. Whether or not anatomic anomalies of the inner ear are an expression of a constitutional inferiority of the ear organ has not yet been proved. Previous ear diseases may also account for a certain susceptibility to acoustic trauma.

One of my patients who is now in the air service easily passed his physical examination. After several hundred hours' flying, his hearing became much poorer. He has been changed from the position of pilot to that of gunner, in which hearing is not quite so essential. His case is interesting, because his father and paternal uncle and aunt have otosclerosis.

For avoiding acoustic injuries in aviators there are three possibilities: (1) reducing the sources of noise, as in the propeller and engine exhaust; (2) placing the flyer in a soundproof cabin; and (3) plugging the aviator's ear canals.

The first way has not yet brought any practical results. Armstrong explains the lack of success as follows. If several noises reach the same point with equal intensity, the combined noise level will be only a few decibels above that of any one component. If, for example, the engine and propeller each make the same amount of noise, the two together do not make twice as much noise as either one alone, but an amount only 3 decibels louder. If, therefore, the engine exhaust and the propeller together make a sound equal to 120 decibels, complete elimination of the exhaust sound will reduce the noise level only to 117 decibels.

The second way, that of placing the flyer in a soundproof cabin, has achieved some success. The third method, that of plugging the aviator's ear canals with cotton, wax, rubber, etc., is more or less impracticable, since this makes radio and telephone communication impossible. Flyers of the British Royal Air Force are equipped with a flying helmet with telephones attached, thus giving full protection without interference with the intelligibility of speech and signal sounds.

## LABYRINTH

The various movements and positions of an aircraft during flight can be classified as linear motions (straight flight), angular motions (turns), and inclinations. The angular movements comprise not only the ordinary turns but also more complicated maneuvers, such as rotation over a horizontal axis parallel with the transverse axis of the plane (looping), over a

horizontal axis parallel to the sagittal axis of the plane (tonneux), and over a vertical axis parallel to the sagittal axis of the plane (spinning). Inclination of the aircraft may be either over a transverse axis (ascent, descent) or over a longitudinal axis (banking).

Studying the physiology of the labyrinth, one would expect a stimulation of the cristae in angular movements of the plane, of the maculae or cristae in linear motions (according to Mach-Breuer-Brown or Magnus and De Kleyn), and a stimulation of the otolithic apparatus in changes of the position of the plane. Amazingly enough, this usually does not happen. A number of authors have been able to demonstrate that flyers are not aware of changes in the direction or position of the aircraft if their eyes are shut. Wulften-Palthe investigated the pilots of the Dutch army air corps, using four groups of persons: men without any flying experience, persons with little experience, pilot pupils, and finally experienced pilots with more than 800 flying hours. Before blindfolding the flyers, he informed them that changes in direction and position would occur; yet only 28 per cent

TABLE 5

| | Persons without Flying Experience | Experienced Flyers |
|---|---|---|
| Elevation | 17° | 6° |
| Declination | 9° | 4° |
| Tilt | 35° | 5°–9° |

of the subjects made correct reports. The experiments proved the inadequacy of the labyrinth in the unusual environment (air). There was furthermore no evidence of any influence of training, since the group with the best estimates comprised the men with only little flying experience.

The fact that angular movements of the aircraft are chiefly perceived by the eye and not by the labyrinth can further be illustrated by the following observation. If one looks—during a sharp turn—along the lower wings down to the earth, one gets the impression of a very slow turning motion of the plane, while if one looks along the upper wings the plane seems to be turning with rapid speed.

In order to test the perception of *changes in position* of the aircraft, large-scale investigations have been made by the American Air Service Medical Research. Some figures indicating the threshold for inclinations are given in Table 5. The experiments have revealed that the threshold is higher than is found in examinations on the tilt table.

It is generally known that three factors are responsible for the maintenance of equilibrium, i.e., the labyrinth, the eye, and the proprioceptive impulses; but there is still dispute as to which component plays the main

role as regards equilibrium on the ground. There is still more dissension in regard to maintenance of equilibrium *in the air* and spatial orientation. Investigations have been made with elimination of one or two of the three components—for example, blindfolding deaf-mutes who lacked labyrinthine function, blindfolding persons who had diseases of the proprioceptive apparatus (tabes), etc. The American Air Service Medical Research examined five groups of persons: normal subjects, deaf-mutes lacking vestibular perception, deaf-mutes possessing vestibular perception below normal, tabetics with impaired deep sensibility, and whirling (ballet) dancers. The findings were as follows:

Tactile and deep sensibility participate respectively in the composite of general motion perception in flying, but are not capable individually, or in combination, of correctly interpreting changes of position and hence of providing orientation in the air.

Normal persons suffer an illusion of reversal of motion during deceleration of vertical or rotary motion, while deaf-mutes with subnormal or absent vestibular perception usually do not.

Normal persons are more accurate than deaf-mutes in detecting tilts, banks, and turns in flight when blindfolded.

Normal pilots when coming out of a spin suffer an illusion of reversal of motion and as a consequence frequently fall into a second spin, with usually fatal results.

Professional ballet dancers with normal vestibular reactions suffer dizziness and nausea when whirling, unless they jerk their heads around rapidly and keep their eyes fixed on a definite point of reference at all times except for the instant when the head is turning.

Garten approached the problem in a different way. In order to reduce the superficial and deep sensibility, he cooled the gluteal region in the persons to be examined or placed them in tubs full of water. He observed in such persons a loss of spatial orientation, although the labyrinthine functions were intact. On the other hand, deaf-mutes lacking labyrinthine function showed only slight disturbances of orientation. He therefore arrived at the conclusion that only the muscle-joint-tendon sense is responsible for orientation.

All the various investigations have yielded no answer to the question as to why we are not able to recognize changes in direction and position of an aircraft. Motions with constant velocity cannot be perceived even on the ground. In order to sense movements with acceleration or deceleration, a certain magnitude and a certain period of time are required (threshold). The respective figures for motions on the ground have been given (p. 374). The changes of direction or position of a plane in the air are usually not very great, or, should they be so in the modern speedy and powerful

aircraft, the period of time is very short. In other words, it can be said that most of the changes in direction and position of the planes represent stimuli *beyond the physiologic threshold* of the labyrinth. In addition to this, the threshold for perception of movements in the air seems to be somewhat higher than on the ground. This explains why the labyrinth appears inadequate for equilibrium and spatial orientation. Whether or not the human labyrinth can ever adapt its function to the new environment of the air remains to be seen. Statements that repeated training can influence the labyrinth lack any substantial proof.

Nevertheless, there are a number of circumstances in which the labyrinth is stimulated and gives sudden rise to the most disturbing effects. This can happen when the labyrinth is hypersensitive, when to the passive movements of the plane an active head motion of the pilot is added, when linear movements occur with great acceleration in a vertical direction, when in turbulent air there is unusual rolling and pitching of the aircraft, and finally in blind flight and in combat flight.

Vertigo and turning sensations may endanger the life of the pilot and the crew. The vertical vertigo is more dangerous than the horizontal. Well known is the example of the flyer who may be caught in continuous spinning motions. On recovery from a right spin, for example, the pilot has the sensation of being turned in a frontal plane to the left, and in an effort to counteract this supposed motion he may send the aircraft again in the direction of the same spiral turn. In order to avoid such dangerous mistakes, Wulften-Palthe advises the pilot to bend his head forward at an angle of 90° during the spin and to get back to his upright position as soon as he has finished the spiral movement.

A motion of the head while turning produces movements of the endolymphatic fluid in the canals that may differ markedly from those produced by the head motion alone. This produces additional sensation that in a rapid spin at a high rate of rotation may be falsely sensed as a tipping over.

*Blind flight.* When all visual reference to the earth is absent, as in darkness, clouds, fogs, etc., the expression "blind flight" is used. This situation may lead to a loss of orientation. However, even more dangerous than complete loss is the false sensation of orientation. For the maintenance of equilibrium and orientation on the ground, sensory impressions on the eyes, the labyrinth, and the deep sensibility are sent to the brain center in complete harmony. The forces of gravity act as effective stimuli. On angular movements of the aircraft, additional forces (accelerative and centrifugal) become effective. The resultant of the various forces elicits a complex of sensations. Neither the labyrinth nor the muscle-joint-tendon sense is capable of analyzing the components of the resultant of the forces. Sensations sent to the brain by the labyrinth may be contradictory to those sent

by the other sensory organs. This leads to a faulty operation of the human equilibrium mechanism, which in turn may often be the source of many false conceptions during blind flying. If, for instance, an aircraft entering a cloud in a horizontal position has slowly tipped to one side, the pilot is unaware of the change of position. Upon coming out from the cloud he sees the earth obliquely tipped. Since he has not observed any change of position of his plane, he is convinced that the earth has changed its position. If during a blind flight a pilot performs an unnecessary steep turn, the resultant of the forces acts upon the pilot's body in the direction from head to spine, giving him an impression of ascending instead of turning.

Efforts have been made to devise instruments that will inform the pilot of all the changes of direction and position of the plane, in order to make him independent of his deceptive sensation. Such instruments are the turn-and-bank indicator, the rate-of-climb indicator, the alimeter, the artificial horizon, the revolution indicator, and the air-speed indicator. The first experiments were not very successful, because the false sensations were so convincing to the pilot that they shook his belief in his instruments. Diringhofen lists the disturbing deceptions during blind flying as follows: the feeling of ascent while turning; the feeling of sinking on recovering from a turn; the feeling of a tilt to the opposite side during a turn; the feeling of tilting when flying between two cloud banks of different slope; the feeling of turning during an even flight; the feeling, due to movements of the head during too sharp a turn, that the aircraft is tipping.

Great progress in the problem of blind flight was made by the investigations of Ocker and Crane and D. Myers. Ocker constructed a view box containing a flashlight and a bank-and-turn indicator. The view box was held in such a manner that only the interior of the box and the instrument could be seen. When he was to be turned in the Bárány chair, he put the bank-and-turn indicator into action and turned the flashlight on. By completely disregarding his own sensations and relying entirely on the instrument, he was able to orient himself completely at all times. This simple but ingenious device proved beyond doubt that blind flight is possible.

The ability to disregard all sensations of position and motion as given by the labyrinth, or at least to put them into the background of his mind, is often much more easily acquired by the inexperienced pupil pilot than by a fully experienced pilot who has developed a marked "flying sense." This sense was always considered a proof of the aviator's special ability. In blind flight, however, it is more disturbing than helping. The pilot must direct his course by the instruments and hold it in this position. This means that he has to fly his instruments and not his aircraft. The instructions for aviators in blind flying are: The sensations may deceive, but the

instruments tell the truth. Since blind flying plays an important role in modern aeronautics, the pilots of the American Air Corps receive a considerable amount of training for it. For this purpose the "link trainer" is used. This is a mechanical device covered with a hood to exclude exterior vision, and a panel with usual controls by means of which blind flying can be practiced on the ground. The ability to do blind flying varies in different individuals and depends upon their labyrinthine sensitivity and the frequency and amount of blind flying done. Armstrong describes a certain type of labyrinthine reaction during blind flight that he terms "static vertigo."

*Combat flight.* Maneuvers during combat flights performed with the modern speedy and powerful aircraft elicit forces so great that they may damage the sensory function and lead to loss of consciousness. The speed of such modern combat planes is from 300 to 500 miles per hour. The enormous power of the centrifugal forces produced by rapid changes in direction and sudden acceleration or deceleration puts a tremendous strain upon the pilot's body. When a plane changes its direction, the crew are pressed into their seats by the centrifugal force, and when flying upside down they are pressed into the shoulder harness. The strongest centrifugal forces are experienced in the course of dive bombing and air combat. In a bombing attack the bombs are often released in an almost vertical dive at a speed that may be 310 miles per hour, and when the plane is only from 2,200 to 1,900 feet above the ground. The bomber ascends immediately to avoid the ground, for the diameter of the curve that must be made to zoom from a vertical dive at 310 miles per hour must be at least 1,600 feet. This elicits forces that press the pilot into his seat with a force of eight times his own weight.

These forces may lead to loss of vision and unconsciousness. The most common effect arises from the tendency of the blood mass to move in the direction of the centrifugal force. The tremendous speed of pulling out from a dive results in a shifting of the blood away from the head of the pilot, thus producing cerebral anemia.

*Airsickness.* Symptoms such as vertigo, nausea, vomiting, pallor, sweat, fear or anxiety, feeling of faintness, etc., may be observed not only in persons on their first trip in a plane but also in experienced professional flyers. The term airsickness has been applied to emphasize the analogy with seasickness. It is generally assumed that seasickness is caused by the various movements of a ship (pitching, rolling, bobbing motion (p. 102)). These unpatterned movements, imparted to the passenger, elicit not the normal counteractions usual for the maintenance of equilibrium, but rather bizarre and unusual reactions. The combined motion of pitching and rolling of the ship are the main factors responsible for these symptoms. The

similarity of the vertigo to that produced by lesions of the peripheral labyrinth led to the belief that the vestibular apparatus was chiefly involved. This theory was supported by the observations of W. James that deaf-mutes without labyrinthine function were not subject to seasickness. Kreidl showed experimentally that the symptoms of seasickness were alleviated after extirpation of the labyrinth or section of the vestibular nerve. However, there is one main symptom missing in seasickness, namely, spontaneous nystagmus, which is characteristic for peripheral labyrinthine lesions. The same absence of nystagmus is observed in airsickness.

In order to study the problem of vertigo, it seems advisable to consider again the classification of Alexander (p. 100). He distinguishes between physiologic vertigo, experimental vertigo, and pathologic vertigo. Physiologic vertigo represents a protective measure, aimed to maintain the equilibrium. A sudden movement resulting in a shift of the gravity line may cause a vertigo apprising the body of the pending danger of falling, thus eliciting certain countermovements. Experimental vertigo, such as is produced in the turning chair, is elicited by stimuli that far exceed the physiologic threshold. Such an overstimulation must lead to disturbances in the equilibrial mechanism, thus producing confusion and disorientation. Pathologic vertigo due to diseases of the labyrinth does not produce any counteracting reactions, but instead favors the falling of the patient. An overstimulation of the labyrinth may therefore be produced when the labyrinth is hypersensitive, when unaccustomed motions of the aircraft occur over a certain period of time, or when the flight is in very rough and bumpy weather.

Airsickness due to unaccustomed motion is often overcome by a physiologic adaptation after repeated flights. It is usually easier for the pilot to adapt himself than it is for other occupants of a plane. The pilot sits close to the center of rotation and he is also able instantly to counteract the movement of the aircraft by anticipation; furthermore his attention is taken up below the level of consciousness. In cases of hypersensitivity of the labyrinth, the physiologic adaptation may occasionally not occur at all. In prolonged flights made in exceptionally bumpy weather, airsickness may be observed even in experienced personnel. The physiologic factors, such as fear of falling down, play a great role in airsickness. Armstrong gives the following definition: "Airsickness (or vertigo) may be looked upon as a true temporary functional neurosis. The primary cause may either be accelerations which bring conflicting sensory impressions of orientation and equilibration to the mind, or it may be caused directly by the fear of high places. The secondary cause is a psychogenetic state in which there is a conflict between the instinct of self-preservation and the situation."

## 3. Functional Tests for Aviation Fitness

### COCHLEA

Good hearing is one of the most important requirements for present-day pilots. It is essential for the complicated service required in modern warfare. The aviator has to communicate by wireless telephony and telegraphy with other planes or with the various ground stations, and by house telephony with the other members of the crew. He must hear and interpret auditory signals, although exposed to all the noises and vibrations of the engines. He must further be able to hear and recognize any unusual noise of the engines indicating an eventual disturbance of their function. The well trained pilot uses the noise of the plane for the finer control of speed, distinguishing every change of pitch and character of sounds. It enables him to estimate the flying speed in case of landing. The important role of the ear organ can best be seen in so-called beam flights.

It has been shown above that the continuous noises and vibrations can damage the hearing acuity of fliers, particularly when slight impairment or a certain weakness in the ear organ already exists. It is therefore of profound importance to test the hearing function very thoroughly before admitting the applicant to the aeronautic service.

The common hearing tests are made by voice, tuning forks, and audiometers.

The British otologist Fry does not regard the hearing tests as commonly used to be sufficient for the selection of flyers. Hearing acuity alone is not enough. Ability to interpret signals depends on both hearing and intelligence, i.e., ability to guess the right answer. The audiometric tests do not give any clue to quickness of interpretation of hearing. In addition, the flyer must be able to receive signals against a noisy background and must understand sentences, not isolated sounds or syllables. Fry uses the following technic of testing:

The subject is placed in a high-level noise field from which his ears are protected by the standard service flying helmet. The articulation tests (list of words and sentences) are then conveyed to him through a pair of telephone receivers carried in his helmet. In a later modification of the test, both the background noise and the speech signals are delivered into the candidate's telephones. Gramophone records are made in which the test words and sentences are spoken against a noise background. This has the advantage that the acoustic conditions for testing can be reasonably standardized and that a number of candidates can be examined at the same time. These tests, however, are still in the laboratory stage and have not yet found their way into the routine examination.

*American Naval Air Corps requirements.* Applicants must have normal

external canals and no perforation of the drum. Evidence of serious past inflammation disqualifies. The presence of a small scar, caused by trouble several years before that has not recurred and with which there is no deficiency of hearing and no evidence of other inflammation, does not disqualify. Marked retraction of a drum membrane, following chronic ear disease, disqualifies. Hearing test results should be normal for each ear. The whispered voice, the coin click, and watch tests are used. In the whispered voice test each ear is tested for whisper at 15 feet. In the watch test an ordinary Ingersoll watch is used, which should be heard at about 40 inches. In the coin click test, in which the ear not under examination is closed, the examiner, who is 20 feet back of the examinee, clicks two coins softly together and the examinee is directed to count aloud the number of clicks each time. Hearing by this test should be equivalent to 20/20. If the examiner is convinced from the results of the several tests that definite impairment of hearing exists, he will reject the examinee as an applicant for aviation training. However, in the case of a qualified flyer, due allowance will be made.

*American Army Air Corps requirements.* The essential qualifications are the same as for the naval air service. The Army has three classes of flying personnel, with requirements as follows. In eligibles for the first class, hearing must be 20/20 in each ear for low conversational voice. The average loss must be not more than 20 decibels (audiometer). For the second class of service, hearing for low conversational voice must not fall below 15/20. For the third class, hearing for low conversational voice must not fall below 8/20.

## LABYRINTH

In contrast to the exact methods of testing the hearing function by means of voice, tuning fork, and audiometer, great difficulties arise if the labyrinthine function has to be tested. With all the various methods, it is not the peripheral sense organ that is directly examined, but rather a reflex (nystagmus) elicited by stimulation. This reflex depends not only on conditions in the peripheral labyrinth or nerve, but also on those in the central vestibular apparatus as well as in the eye nuclei and eye muscles. Furthermore, the functional tests as used in practice are very rough, since they employ stimuli that far exceed the physiologic threshold of the labyrinth. Finally, we must admit that we do not know the exact figures for normal excitability. No wonder that the clinical examinations as used in aviation medicine in the various countries differ largely not only with respect to method but also with regard to evaluation of the resulting figures. For a full understanding of this complex matter, the reader is referred to chapters ii, iii, and iv.

The first question to be answered is whether the rotation test or the caloric test should be applied in aviation medicine. From a physiologic point of view, the turning reaction has much more significance, since turning acts as the only adequate stimulus for the semicircular canals. It further enables one to stimulate the canals in the different planes, thus eliciting horizontal, rotatory, and vertical nystagmus. The caloric test, on the other hand, makes it possible to examine the labyrinth of each side separately. It further has the advantage of employing minimum stimuli.

The turning test, as used in Bárány's chair, has often been subject to criticism. Besides the contention that it is too rough and too inexact, it is also claimed that the stimuli interfere with each other. On turning a subject, two forces are effective, i.e., the angular acceleration in the beginning of turning (positive) and the angular deceleration or retardation at the moment of stopping (negative). In the usual turning test, the first stimulus is still present when the second stimulus (counterstimulus) is applied by stopping.

Efforts have therefore been made to find functional tests in which the maximum stimuli are replaced by weak, i.e., minimum stimuli, so that the physiologic threshold can be determined. Attempts have further been made to differentiate between peripheral labyrinthine irritability and central sensitivity. Such tests were found for the caloric examination (5 cc. test) as well as for the turning reaction (electric turning chair).

The caloric minimum test was first described by Kobrak for scientific purposes and was then introduced with various modifications into the clinical examination. The ear canal is irrigated with 5 cc. of water at 55 F. Latent time, duration, and intensity of nystagmus are then determined. Average figures for normal excitability range between 15 to 30 seconds for latent time and from 60 to 120 seconds for duration. Shortening of latent time below 10 seconds, lengthening of duration time above 2 minutes, and intensity of nystagmus amounting to second and third degree indicate hyperexcitability. Subjective reactions such as vertigo, nausea, vomiting, pallor, and sweating, although usually proportional to the degree of hyperexcitability, cannot be used for diagnostic purposes, since they may or may not occur. However, in aviation medicine, they should be given more attention because of the airsickness that occurs more often in persons with vasomotor instability. In order to distinguish between peripheral and central excitability, special attention has been given to the latent time, which is supposed to express the peripheral irritability, while the duration of the nystagmus relates to the central sensitivity. It has been claimed that a latent period consists of two factors: one, the physical component, concerns cooling off of the various tissues, while the other, the physiologic component, represents the true physiologic latent reaction. Efforts have

been made to separate the two from each other in order to improve the exactness of the tests (see p. 76).

For quantitative determination of the labyrinthine reaction to *turning* stimuli, the tests are made by use of an electric turning chair. It enables the investigator to begin with very slow turns (0.3 degree second) and to increase gradually until the physiologic threshold is reached; it usually lies between 1 to 2 degree seconds. From then on turning is continued with a steady speed (no acceleration) over a period of 3 minutes, after which turning is suddenly stopped. In this way interference of stimuli is eliminated, and the duration of the nystagmus or the number of eye jerks per the time unit can be determined exactly. None of the quantitative tests except the caloric minimum test has as yet found its way into the clinical examination.

*American Air Corps Requirements.* The vestibular tests include the Bárány *chair test* in which nystagmus and falling after turning are tested, and the *nystagmus test.* The examinee's head is inclined 30° forward, so that the tragus of the ear is on a horizontal line with the external canthus of the eye. The examinee is then asked to fix his eyes on a distant point and the chair is turned slowly from side to side, in order to note whether or not spontaneous nystagmus is present. Then the examinee, with eyes closed, is turned to the right ten times in exactly 20 seconds. The instant the chair is stopped, the stop watch is clicked. The examinee opens his eyes and looks straight ahead at some distant point. A horizontal nystagmus to the left, of 26 seconds' duration, should occur. The examinee then closes his eyes and is turned to the left. A horizontal nystagmus to the right, of 26 seconds' duration, should occur. A variation of 10 seconds more or 12 seconds less is allowable.

In the *falling test,* the examinee's head is inclined 90° forward, his forehead resting on the upper part of his fists, which are placed one above the other on his knees, these being brought close together. He is turned to the right five times in 10 seconds. On stopping, the examinee raises his head and should fall to the right. This tests the vertical semicircular canals. He is then turned to the left, with the head inclined forward 90°; on stopping, he raises his head and should fall to the left.

In the *balancing test,* the applicant stands erect without shoes on, with heels and toes touching. He then flexes one knee to a right angle—being careful not to support it against the other leg—closes his eyes, and endeavors to maintain this position for 15 seconds. The test is then repeated for the other leg. The performances are recorded as "steady," "fairly steady," "unsteady," or "failed." The applicant should be instructed that this is the equilibrium test. There is no objection to his aiding his balance by moving and bending back and forth. Inability to pass the equilibrium test satisfactorily constitutes cause for rejection.

*In summary*, it may be said that in aviation medicine the old Bárány test (forced turning method) is still playing the main role. The functional tests for the labyrinth and the interpretation of the results differ in the various countries.

It must be admitted that for testing of pilots we should by guided only by practical viewpoints. Examinations for scientific or research purposes cannot be made in war times. On the other hand, there is now a chance for the otologist, when thousands and thousands of persons are being examined, to try one or the other quantitative method besides the ordinary Bárány chair test. A comparison of the results with such a tremendous material may be of great value, and may help convert one or the other method from its laboratory stage into a routine clinical test.

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# XV

# Effects of Atmospheric Pressure Changes on the Ear

By *Louis E. Wolfson*

The fact that the ear organ is a common site of involvement during changes in atmospheric pressure is not surprising. The adequate stimuli for the organ of hearing are represented by sound waves caused by minute differences in air pressure. The proper conduction of sounds depends upon a definite tension of the drum membrane and the chain of ossicles regulated by the function of the eustachian tube (ventilation). Changes in atmospheric pressure that far exceed the physiologic limits, combined with an improper function of the eustachian tube, must therefore lead to disturbances of the air equilibrium.

Modern mechanisms of warfare, such as fast-climbing aircraft, dive bombers, stratosphere planes, submarines, etc., produce such a tremendous degree of change of atmospheric pressure that considerable stress is placed upon the ear and the upper respiratory tract. One would therefore expect to find modern aviation medicine confronted with entirely new problems. A study of the literature, however, reveals the astonishing fact that the older authors had a thorough knowledge of the effects of changes in atmospheric pressure upon the ear.

Boyle (1670) and Hoppe-Seyler (1857) assumed that in high altitude gas bubbles in the blood may occur, owing to a reduction in atmospheric pressure. P. Bert (1873) was able to show experimentally that the bubbles are composed of nitrogen liberated from the blood. He must be considered as the founder of the theory of aero-embolism, which plays such an important role in modern aviation. Jourdanet (1862) believed that a reduced carbon dioxide tension of the air at an altitude of 9,000 feet may lead to a deficiency in oxyhemoglobin. He applied to it the term anoxyhemia. P. Bert recommended the use of oxygen for flights in high altitude and emphasized the importance of the early introduction of oxygen before symptoms occur. Schroetter was the first to invent a device for administering oxygen to airmen by means of a mask covering both nose and mouth. Smith (1873) examined the caisson workers of the Brooklyn Bridge in New York and found impairment of hearing, vertigo, nausea, and pains in the lower extremities (bends). Schroetter and his co-workers (1897) made large-scale investigations on workers subjected to compressed air and gave a thorough

description of the resultant damages of the ear organ, as follows: otalgia, hyperemia, ecchymoses, hemorrhages, inflammation of the drum membrane, hemorrhages in the tympanic cavity, acute otitis media, and abscess of the middle ear. They considered these lesions as a result of pressure differences between the middle-ear and the external atmosphere, which are particularly marked in cases of impaired function of the eustachian tube.

This short review of the old literature is very impressive. For thorough study of the entire problem, the following factors must be considered: (1) the effects of increased atmospheric pressure; (2) the effects of decreased atmospheric pressure; (3) the effects of sudden changes in atmospheric pressure.

### 1. Effects of Increased Atmospheric Pressure

The earliest studies were made on deep-sea divers and caisson workers. The literature contains numerous incidental observations reported in various countries. The first systematic investigations on a large scale were made by Heller, Mager, and Schroetter. These authors report in a monograph of 1,230 pages on the effects of air pressure. They based their studies on clinical, postmortem, and animal experimental investigations. The clinical material comprised 675 persons engaged in caisson work in Vienna in the years from 1897 to 1900. Of these, 320 workers—48.9 per cent—suffered minor or major injuries. The various lesions were classified into two groups: (*a*) direct lesions due to pressure differences as between the middle-ear and the outside atmosphere, and (*b*) indirect lesions due to decompression.

The clinical symptoms of the *direct lesions* were summarized as follows: sense of fulness in the ears or terrific earache, tinnitus, headaches, transient deafness, and vertigo. Otoscopic examination revealed retraction of the drum with loss of the light cone, and injections of the vessels behind the handle of the malleolus or in the peripheral parts of the drum. In other cases the drum membrane appeared pink or intensely red and bulging. In severe cases ecchymoses, hemorrhage, and rupture of the drum could be observed. Occasionally a bloody-serous discharge from the middle ear or an acute otitis media was seen. In the latter case, the incidence of mastoiditis was relatively high; this is not surprising, since we know that traumatic otitis media always has a more perilous course than the usual form. The authors explain all these symptoms as a result of the negative pressure in the tympanic cavity and the eustachian tube.

*The indirect lesions* due to compression are the results of disturbances in the exchange of gases between the blood and the respiratory air. On *compression*, saturation with nitrogen takes place. This lasts from seven to ten hours, or until complete saturation of the body is reached. On sudden

*decompression*, the nitrogen is liberated from the blood in the form of bubbles.

The clinical manifestations can be classified into lesions of the central nervous system and of the cardiovascular system. The symptoms are itching, myalgias and arthralgias (bends), monoplegias, pareses, paraplegia, asphyxia, aphasia, and Ménière's syndrome. All these symptoms can be observed either immediately after decompression or somewhat later, usually thirty minutes later. The critical threshold for the pathologic changes starts at a pressure of 1.4 atmospheres.

The authors further observed certain changes in speech involving the timbre, while pitch and intensity were not affected. At 2.5 atmospheres the speech sounds metallic. Speaking under air compression is more strenuous and leads quickly to fatigue. Whistling is not possible at all. Laryngoscopic examination, however, did not reveal any pathology in the larynx or vocal cords.

In experimental investigations, the authors could show gas bubbles in the blood system in animals kept for fifteen minutes under a pressure of 2.5 atmospheres. Even after a decompression time of thirty minutes, the bubbles could still be seen. They further found hemorrhages in the cochlea and labyrinth and necroses in the spinal cord.

Microscopic examinations of petrous bones were made by Panse, who found hemorrhages in the middle ear, perivascular exudation in the labyrinth, the modiolus, and the scalae, extending to the organ of Corti. Large-scale investigations on caisson workers were further made by Thost, who examined 800 cases. He could fully corroborate the interesting findings of Schroetter and his co-workers. He explained the cases of deafness as a result of gas embolism in the vessels in the inner ear.

Recent audiometric studies on deep-sea divers have been reported by Behnke. The audiograms of 19 divers subjected to pressure trauma over a period of from five to fifteen years revealed a diminution of hearing in the range of 4,096 double vibrations. Investigations by Shilling and Everley showed that exposure to Diesel engine noise in submarines resulted in the greatest loss in the higher frequencies in the first hour and in much less proportional loss over two, three, or four hours more, but recovery was longer retarded in cases of exposure for a longer time.

### 2. Effects of Decreased Atmospheric Pressure

The air mantle surrounding the earth surface is called troposphere up to an altitude of from 38,000 to 40,000 feet, and stratosphere above that level. The air is composed four fifths of dry nitrogen and one fifth of oxygen, carbon dioxide, water vapor, and some rare gases. The figures are: 79 per cent nitrogen, 20.96 per cent oxygen, and 0.04 per cent carbon dioxide.

Although with increasing height the atmospheric pressure decreases and the number of gas molecules in every cubic centimeter diminishes correspondingly, the quantitative relation in the air mixture, i.e., 4:1, remains unchanged. The ratios of altitude and pressure are shown in Table 6.

The influence of reduced atmospheric pressure upon the organism was brought to light as a result of aeronautics and mountain climbing. It would go beyond the scope of this book to describe the development of aviation since the epic flight of Montgolfière in 1783. The first physician who undertook a flight for medical research was Petrard, who in 1873 ascended to an altitude of 30,000 feet at a temperature of −10 C. He observed a lowering of the pulse rate (10 beats less than normal) and a 60 per cent increase in the normal respiratory rate.

The influence of reduced pressure upon animals was studied in low-pressure chambers. Hoppe-Seyler (1857) observed death in animals during convulsions—in rats and cats when kept at a pressure rate of 40 to 50 mm. of

TABLE 6

| Altitude (ft.) | Pressure (mm.Hg) | Altitude (ft.) | Pressure (mm.Hg) |
|---|---|---|---|
| 0 | 760.0 | 20,000 | 349.2 |
| 1,000 | 733.0 | 30,000 | 225.6 |
| 2,000 | 706.6 | 40,000 | 140.7 |
| 5,000 | 632.4 | 60,000 | 54.1 |
| 10,000 | 522.6 | | |

mercury, in guinea pigs at 75 to 80 mm., and in pigeons at 120 to 150 mm. Bert (1873) could show that the real danger lies in the reduction of the oxygen content of the air and not in the reduction of the air pressure. To prove it, he put himself into a pressure chamber at 248 mm. of mercury, in which, however, he was breathing an air of normal oxygen content. He had no discomfort at all. In order to study the formation of nitrogen bubbles, he subjected animals to high atmospheric pressure followed by sudden decompression. He stated that atmospheric nitrogen is dissolved in the body tissues during increased atmospheric pressure and is liberated in the form of bubbles during decompression. He found nitrogen bubbles in the vessels of the spinal cord as late as four days after decompression. He assumed that the bubbles lead to formation of emboli that are carried throughout the body by way of the blood stream. In order to prove that the bubbles are composed of nitrogen and not of air, he exposed animals in a high-pressure chamber to a mixture very poor in nitrogen, and could not find any bubble formation after decompression.

Mosso (1882) believed not in an oxygen deficiency but rather in a carbon dioxide deficiency, which he called acapnia in contrast to asphyxia. Cyon

assumed a stimulation of the vasomotor and respiratory centers owing to lack of carbon dioxide. A further step forward was made when Loewy (1895) directed attention to the fact that it is not the oxygen content of the external air but the oxygen tension within the alveoli of the lungs that plays the main role. As long as the intra-alveolar oxygen contents amount to approximately 9 per cent, compensation of the oxygen deficiency takes place and no change of the respiratory quotient will be seen. A reduction to 7 per cent makes the compensation difficult, while a further decline to 5 per cent leads to a complete lack of compensation.

This review of the old literature shows that most of the altitude effects are due to scarcity of oxygen or loss of carbon dioxide and to formation of nitrogen bubbles. Modern aviation medicine corroborated these facts and made practical use of them. The deficiency of oxygen (anoxyhemia of Jourdanet) is now called anoxia and anoxemia. The former denotes oxygen scarcity in the body tissues, while the latter applies to oxygen deficiency in the blood. Barcroft differentiates three groups of anoxemia: (*a*) anoxic anoxemia, i.e., oxygen deficiency of the blood due to oxygen deficiency of the air; (*b*) anemic anoxemia, i.e., oxygen deficiency of the blood due to ill-functioning hemoglobin; and (*c*) ischemic anoxemia, i.e., oxygen deficiency due to circulatory disturbances.

It can generally be said that normal persons (at rest) usually tolerate a reduction in oxygen pressure of about 33 per cent. A pressure of 100 mm. of mercury being normal, an alveolar oxygen pressure of 60 mm. of mercury, which is equivalent to an altitude of 11,000 feet, produces an oxyhemoglobin saturation of about 85 per cent. This altitude is called the "critical altitude." With regard to the effects of altitude, three zones can be distinguished, according to Strughold:

1. Indifferent zone: Up to 10,000 feet.
2. Zone of adjustment: The readaptation of respiration and circulation is adequate to compensate for the effects of height if no physical work is done. The transition to this zone is called the "threshold of reaction."
3. Zone of incomplete compensation: This extends from 13,000 to 16,000 feet, and is bounded by the "critical threshold," which leads into the zone of death, usually above 22,900 feet.

Most of the experimental work has been done in pressure chambers and in laboratories on high mountains. Armstrong and Heim could demonstrate that, for aviation medicine, a number of other factors besides altitude have to be taken into consideration—as rate of ascent, duration of exposure, frequency of exposure, etc. Instead of "mountain sickness," the term "altitude sickness" has been introduced by Schneider into aviation medicine. Armstrong distinguishes between acute and chronic alti-

tude sickness. He defined the former as a condition occurring as a result of the decrease in partial pressure of oxygen in the inspired air during an aircraft flight at high altitude, which is marked by acute anoxia and its accompanying symptom complex. Chronic altitude sickness occurs under the same conditions during frequently repeated flights with cumulative effect.

In the United States, military aircraft must be provided with gaseous oxygen equipment, and the personnel are required to utilize oxygen at all times while participating in flight above 15,000 feet, when remaining at an altitude below 15,000 feet but above 12,000 feet for periods of two hours or longer, and when participating in flight below 12,000 feet but at or above 10,000 feet for periods of six hours or longer.

### 3. Effects of Sudden Changes in Atmospheric Pressure

The ill effects of increased and of decreased pressure are the result either of direct pressure differences between the middle-ear and the external atmosphere or of indirect lesions due to the formation of nitrogen bubbles in the body. The greatest danger, however, lies not so much in the amount of atmospheric pressure as in the period of time in which the changes occur. The shorter the time interval, the more marked are the ill effects. This fact has long been known, as can be shown by the strict regulations for persons engaged in work under high pressures. Table 7 shows, for example, some of the regulations for caisson workers in Vienna (1897) regarding the period of time required for compression as well as for decompression.

These figures reflect an average of 0.1 atmosphere per minute for compression, and an average of 0.1 atmosphere for every two minutes of decompression, and show the slowness necessary, particularly for the change from higher to lower atmospheric pressure. It is understandable that efforts have been made to shorten the time for practical work. Haldane and his co-workers (1907) found that the volume of nitrogen that would be liberated would be the same when the total pressure was halved, whether the pressure was low or high. They worked out a new system of "stage decompression" in which the elimination of nitrogen is accomplished much faster than with the old uniform method. For practical use they set up decompression tables based on depth and duration of exposure. Although the time for decompression was cut down to half, the procedure was still very slow. To illustrate this fact a comparison between diving and altitude flying will serve. When a diver ascends from 100 feet of depth (4 atmospheres) to sea level (1 atmosphere), the time required is eighty minutes according to the old method of decompression, and forty minutes with the new system of stage decompression. A modern aircraft is capable from the start (1 atmosphere) of going to an altitude of 34,000 feet (¼ atmosphere), which

represents the same difference in pressure, in approximately seven minutes. This shows the great danger of modern aviation. Modern research workers have tried to find the rate of ascent and descent within the range of safety. Armstrong found that up to an altitude of 30,000 feet, which he called the critical height for liberation of nitrogen bubbles, the rate of ascent should not exceed 200 feet per minute. The higher the altitude, the slower must be the rate of ascent. Lieut. Col. R. P. Todd, with whom I have been in communication, states: "As to the maximum safe rate of ascent or descent, I doubt if anyone has the answer. I believe that at present the highest performance in climb would not be over 4,000 feet per minute. The dive, of course, might reach around 600 miles an hour straight down, or over 50,000 feet per minute. I have made vertical dives of over 10,000 feet at approximately half that speed in open-cockpit airplanes without noticing any great physical discomfort or effect on my ears." With the installation

TABLE 7

| Compression to (Atmospheres) | Minimum Time (Minutes) | Decompression to (Atmospheres) | Minimum Time (Minutes) |
|---|---|---|---|
| 0.5 | 5 | 0.5 | 10 |
| 1.5 | 10 | 1.0 | 20 |
| 2.5 | 15 | 2.0 | 40 |
| 3.5 | 20 | 3.0 | 60 |
| 5.0 | 30 | 4.0 | 80 |
| | | 5.0 | 100 |

of low-pressure chambers and oxygen supply in aircraft, the dangers of anoxia and nitrogen bubble formation have been greatly reduced. The rate of ascent in military aviation is at present between 500 and 1,000 feet per minute. Armstrong and Heim made experimental studies of altitude tolerance, i.e., ability to endure anoxia over a period of time. They found the optimum tolerance at a rate of 500 feet per minute. The more *rapid* the ascent, the greater the effect on the central nervous system; the *slower* the ascent to the same height, the greater the strain on the cardiovascular system.

After this discussion of the indirect lesions, we may turn to the effects of direct differential pressure as between middle-ear and outside atmosphere. The enormous pressure exerted upon the drum membrane at given depths is shown by the following figures:

| Atmospheres | Pressure (Grams) |
|---|---|
| 1 | 550 |
| 2 | 1,100 |
| 3 | 2,000 |

The great importance of a normally functioning eustachian tube in withstanding the severe strain of sudden pressure changes was known to the older otologists. Hartmann in 1877 made experimental investigations on the function of the eustachian tube in pressure chambers. He arrived at the conclusion that the tubes act as valves that are closed during rest of their muscles, and open during swallowing. Increase of pressure in the nasopharynx presses the membranous walls of the tubes toward the cartilage. This in turn leads to such a tight closure that air, even under a pressure of 200 mm. of mercury, cannot be brought into the tympanic cavity. In contrast to Hartmann, Schroetter and his co-workers believe that the closure of the eustachian tube is not caused by the increased pressure in the nasopharynx, but must be considered as a result of suction due to the negative pressure in the tympanic cavity. In this connection, it is interesting to recall another paragraph of the regulations for caisson workers that excludes all persons with catarrhal conditions of the nose and throat from work in compressed air for the duration of their disease.

Recent studies have been made by McMyn and by McGibbon. The former arrived at the conclusion that the tube is opened by the tensor palati muscle, which causes relaxation of its membranous wall. Other muscles, such as the levator palati, salpingopharyngeus, and superior constrictor may act synergically. McGibbon believes that the tube is opened in a sinuous manner by the combined action of the sphincter of the pharyngonasal isthmus and of the upper part of the superior constrictor muscle, which drags the posteromedial cartilaginous wall inward, while simultaneously the tensor palati muscle braces the anterolateral wall against the medial pterygoid lamina and draws down the hamular process of the cartilage to relax the membranous wall.

The effects of differential pressure from the standpoint of *aviation medicine* have been studied by Armstrong and Heim (1937). The authors described the phenomena as a new clinical entity to which they applied the term "aero-otitis media." They defined the disease as an acute or chronic traumatic inflammation of the middle ear caused by a pressure difference between the air in the tympanic cavity and that of the surrounding atmosphere. It must be considered as the result of inadequate ventilation of the middle ear during ascent or descent in flight. Such inadequate ventilation may be due to either failure or inability voluntarily to open the eustachian tube. The characteristic features of aero-otitis media are congestion, inflammation, and discomfort and pain in the middle ear, occasionally followed by a temporary or permanent impairment of hearing. The symptoms depend upon the rates of ascent and descent, which in turn cause the pressure differences. The greater the difference between the pressures of the middle-ear and the outside atmosphere, the more severe are the clini-

cal manifestations. At a negative pressure of about 60 mm. of mercury in the middle ear, the patient has the same symptoms as in acute otitis media, i.e., terrific earaches, tinnitus, impaired hearing, etc. At from 60 to 80 mm. negative pressure, the pain is very severe and radiates from the ear to the temporal region, the parotid gland, and the cheek, and there is marked deafness and vertigo. At a pressure of between 100 and 500 mm. of mercury the drum membrane ruptures; with rupture of the membrane the acute pain quickly subsides, but a dull ache persists for from twelve to forty-eight hours. The otoscopic examination reveals in mild cases a slight retraction or bulging of the drum. In more severe cases the drum may be retracted or bulging; it is also inflamed, and its color varies from a slight pink tinge to an intense red. There is also injection of the blood vessels along the handle and around the drum periphery. In very severe cases, there are traumatic ruptures that are usually linear and quite extensive, with red blood spots on the margin and blood crusts in the external auditory canal.

Besides the acute form of aero-otitis media, Armstrong and Heim described a chronic type resulting from frequently repeated traumatic insults. In such cases the patients complain of a "full and stuffy" feeling in the ears and of tinnitus. The drum membrane is dull and slightly thickened.

It is generally agreed that the eustachian tube plays a vital role in the effects of pressure changes. Adequate ventilation is of first importance. The changes in atmospheric pressure experienced under normal conditions in the usual environment are slight. In the air, however, the pressure is greater in the middle ear than in the external ear in ascent, and the reverse on descent. Small differences can easily be adjusted by opening the eustachian tube. Ascent provides no difficulties, since air can easily be blown out through the tube. On descent, the air cannot be forced through the tube, which is tightly closed, owing to the negative pressure in the tympanic cavity. In *ascent* the air pressure is equalized at an altitude of about 500 feet by opening of the tube. Above 500 feet, the tubes open at intervals of approximately 425 feet, up to an altitude of 35,000 feet. During descent the tubes are tightly closed, with a differential pressure of 80 to 90 mm. of mercury. The difficulties in adjustment to pressure changes depend upon the altitude and the rate of ascent or descent per minute. In ascent of 200 feet per minute, the ventilation is adequate; with a change to 500 feet per minute, it may be slightly impaired; while at a rate of 1,000 feet per minute symptoms of congestion of the middle ear will occur. During descent a rate as low as 300 feet per minute can lead to a tight closure of the tubes with marked pressure changes in the ear.

*Failure* to open the tubes during changes in altitude in aircraft flights

is, according to Armstrong and Heim, due to the following factors: ignorance of the necessity of doing so, carelessness, being asleep, and the influence of analgesics or anesthetics. *Inability* to ventilate the middle ear voluntarily may be caused by acute or chronic inflammations of the upper respiratory tract, nasal obstructions, growths, and paralysis of the soft palate.

Behnke and Willmar observed symptoms of congestion in the middle ear appearing eighteen hours after prolonged inhalation of oxygen at high altitudes, which they explained as negative pressure effects brought about by the absorption of oxygen from the middle-ear spaces during sleep, when voluntary opening of the auditory tubes is not effected. Upon awakening in the morning, the individual experiences sensations of fulness accompanied by pain and tenderness in the ears, tinnitus, and decreased hearing. About six hours later, the symptoms and findings usually diminish without therapy, and they disappear in from twenty-four to forty-eight hours. These authors recommend that surrounding air of only 21 per cent oxygen be introduced during the last stages of descent from 10,000 feet.

The effect of pressure changes upon the *paranasal cavities* has been studied by Campbell. He found that normal sinuses in normal tissue surroundings are unaffected by variations in pressure. The presence of fluid or of redundant tissue in such a position that it may come in contact with the ostium of a sinus produces mechanical phenomena that may lead to pathologic change and symptoms. He applied to such cases the term "aerosinusitis."

The various efforts to control the ill effects of pressure changes can be classified as prophylactic and therapeutic measures. The former include instruction of flyers regarding the normal function of the eustachian tube and advice on how and when to ventilate the tubes voluntarily. Ventilation may be accomplished by swallowing, yawning, yelling, and chewing. Some commercial aviation lines pass out chewing gum to the passengers as a preventive measure. Other methods are air inflation by the technic of Valsalva, or forced expiration. Lamport recommends the following maneuver. After maximal expiration in the upright position, the lips and nostrils are held shut and the flyer produces as much nasal suction as possible. Then, while at the peak of this effort, he voluntarily ventilates the eustachian tube, following immediately with release of suction. Lovelace, Mayo, and Boothly have used helium oxygen mixtures and obtained faster reduction of tympanic vacuum. Armstrong recommends a return to higher altitudes when discomfort and pain occur during descent. The therapeutic measures consist of Politzer inflation or catheterization after shrinkage of the pharyngeal ostium. For the relief of pain, heat should be applied and analgesics administered.

## 4. Regulations for Examination of Flyers

### EUSTACHIAN TUBE PATENCY

A drum membrane with normal light reflex, and not retracted, can be interpreted as indicating patency of the eustachian tube on the given side. The patency of the eustachian tubes is to be tested in all doubtful cases by use of the Politzer bag. This can readily be done by placing the tip of the bag in one nostril, closing the other nostril with the finger, and inflating the bag as the examinee swallows. The patency of each tube is determined by observing whether or not the drum membrane is deflected outward. With the bag removed, a subsequent swallow should cause the drum to return to its normal position.

### NOSE AND PARANASAL SINUSES

The following conditions should be looked for: acute or chronic infections, deviation of the nasal septum, polyps, tumors, or other conditions causing irritation or obstruction. A perforated nasal septum should raise the suspicion of syphilitic infection.

Following examination of the nose proper, the antrums and the frontal sinuses should be viewed in the dark room by transillumination for evidence of acute or chronic inflammation.

Any acute or chronic inflammatory processes of the nasal passages or sinuses should be disqualifying. An obstruction of any of the sinus openings or an obstruction of 25 per cent or more in either nasal passage should disqualify.

### THROAT

The tonsils, if normal in size and not infected, may be disregarded. If infected or hypertrophied to such an extent that they encroach on the lumen of the throat, they should be removed and the candidate should be re-examined at a later date.

Adenoids should not be present to an extent sufficient to obstruct nasal breathing in any degree or to have caused habitual mouth breathing.

The pharyngeal orifices of the eustachian tubes should be inspected to determine whether or not they are obstructed by tumors, growths, scar tissue, inflammatory processes, or tonsillar tissue.

With regard to the regulations for examination of flyers, I feel that the great stress given to slight deviations of the septum is definitely wrong. Since December 7, 1941, I have had occasion to examine a number of applicants for pilot work in both the army and the navy who had been turned down because of a slight deviation of the septum, although there

were no past history of infection and no evidence of any pathology whatsoever. It was compulsory for these men to have a submucous resection performed before they could be accepted. This was done to carry out the necessary regulations, but with the feeling that their nasal condition was often not quite as good after the operation as before it.

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