THE EFFECT OF HEAD INJURY ON VESTIBULAR FUNCTION

AN ELECTRONYSTAGMOGRAPHIC STUDY

by

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WHERE THERE IS MUCH DESIRE TO LEARN
THERE OF NECESSITY WILL BE MUCH
ARGUING, MUCH WRITING AND MANY
OPINIONS: FOR OPINION IN GOOD MEN
IS BUT KNOWLEDGE IN THE MAKING

--- A speech of John Milton, 1644

A disturbed mind will seek a solution. The desire to learn the How's, What's and Why's of a problem prompted the author to search for an answer to this problem.

In recent years an increasing number of patients presenting vestibular symptoms, particularly postural vertigo, after head injury has been observed. Various opinions have been expressed regarding the frequency of occurrence of vestibular symptoms, the types of positional nystagmus and the probable sites of lesions resulting from head trauma. However, there has been no uniformity of opinion.

The purpose of the present investigation is to study the effects of head injury on vestibular function.

Chapter I

Anatomy and Physiology of the Labyrinth

I. Anatomy of the Labyrinth and Its Central Pathways

A. The Internal Ear

The internal ear is encased within a complex hollowed out space within the petrous portion of the temporal bone. It consists of three main parts : the otic or membranous labyrinth, a derivative of the ectoderm; the periotic labyrinth and the otic capsule or the osseous labyrinth, derived from the mesoderm. The structures of the internal ear can be briefly tabulated in the following outline[135].

1. Otic (Membranous) Labyrinth. This is lined with ectodermal epithelium and contains endolymph.
   a. Parts within the periotic vestibule (periotic cistern).
      1) The saccule and its macula.
      2) The utricle and its macula.
      3) The utricular duct.
      4) The saccular duct.
      5) The endolymphatic (otic) duct within the vestibular aqueduct.
      6) The ductus reuniens.
   b. Parts within the periotic semicircular canals.
      1) The otic semicircular ducts (posterior, anterior, and lateral or horizontal) and their ampullae.
   c. Parts within the cochlear periotic space.
      1) Scala media or cochlear duct.

a) Otic epithelium of the vestibular (Reissner's) membrane.

b) Glands of Shambaugh.

c) Organ of Corti

(1) Tectorial membrane

(2) Huschke's teeth of the limbus

(3) Epithelium of the internal spiral sulcus

(4) Epithelium of the external spiral sulcus

(5) Inner hair cells

(6) Inner pillar cells

(7) Tunnel of Corti

(8) Outer pillar cells

(9) Space of Nuel

(10) Cells of Deiters

(11) Outer hair cells

(12) Cells of Hensen

(13) Cells of Bottcher

(14) Cells of Claudius

d) Cul-de-sac of the scala media.

2. The Periotic Labyrinth.

a. The vestibule (periotic cistern)

1) Appendages of the periotic labyrinth

a) Short vestibular extension around the endolymphatic (otic) duct.

b) Fissula ante fenestram.

c) Fossula post fenestram.

d) Cul-de-sac of scala tympani

b. The semicircular canals (posterior, anterior, lateral).

c. Cochlea

1) Scala vestibuli

2) Scala tympani

a) Periotic (perilymphatic) duct

3) Basilar membrane excluding the otic epithelium.

4) Vestibular or Reissner's membrane excluding the otic epithelium.

a) Connective tissue of the limbus.

b) Spiral ligament.

c) The lining of the scala vestibuli and scala tympani.

d) Stria vascularis excluding the lining epithelium.

3. The Otic Capsule (Osseous Labyrinth).

a. Modiolus

1) Osseous spiral lamina.

2) Secondary osseous spiral lamina.

3) Osseous spiral septa between the turns of the cochlea.

b. Fenestrae

1) Vestibular fenestra (oval window)

2) Cochlear fenestra (round window)

c. Other openings

1) Internal acoustic meatus

2) Vestibular aqueduct

3) Cochlear aqueduct

B. The Membranous Labyrinth.

The membranous or otic labyrinth is a system of spaces and tubes lined with epithelium and filled with a clear fluid, endolymph or otic fluid (see Fig. 1). The membranous labyrinth is

for the most part surrounded by the periotic labyrinth and its supporting connective tissue. Both the membranous and the periotic labyrinth lie within the bony or osseous labyrinth but are separated from its wall by the internal periosteum.

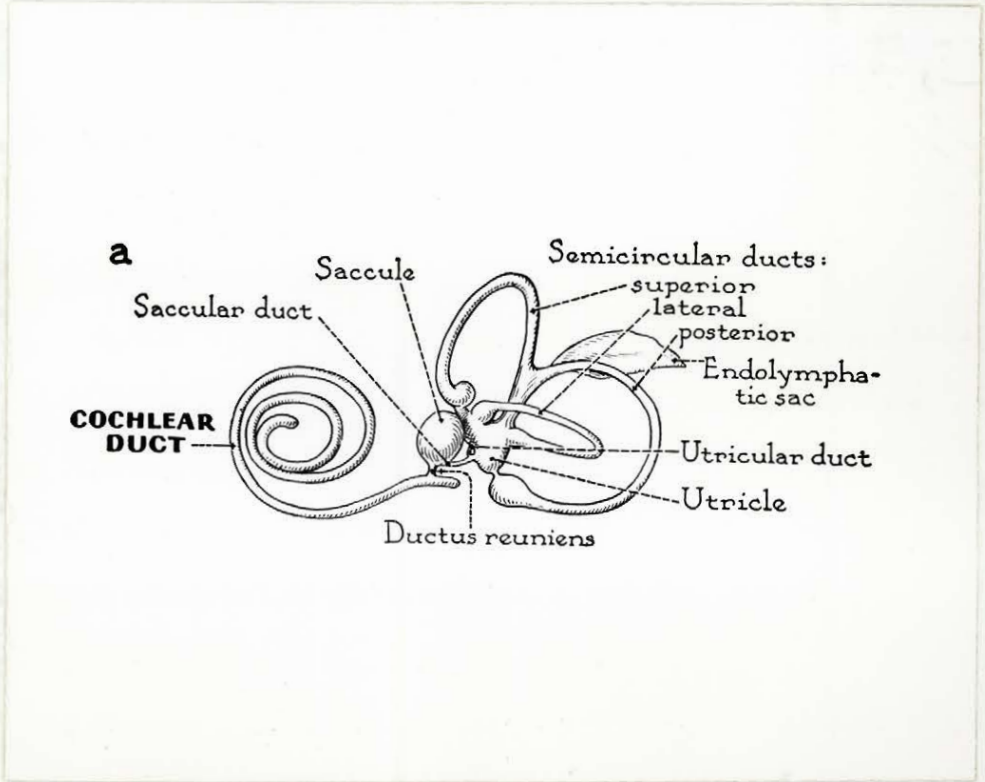


Fig. 1 The Membranous Labyrinth
(from Bast and Anson)

The main parts of the membranous or otic labyrinth are : three semicircular ducts and their ampullae, the utricle, the saccule, the cochlear duct, and the endolymphatic (otic) duct and sac. Smaller connecting channels are : the utricular duct, the saccular duct and the ductus reuniens.

The semicircular ducts, posterior, anterior and lateral, open into the utricle by five orifices. Each duct has a dilated end, the ampulla. The enlargement presents, on its convex side, a transverse

groove (sulcus ampullaris) where the ampullary nerve enters, and a sickle-shaped ledge (crista ampullaris) projects into the lumen.

The utricle is a somewhat tubular vesicle, lodged in an elliptical recess of the vestibule, fixed to the wall by connective tissue and by fibers of the utricular nerve. The nerve fibers terminate in a plaquelike, ovoid, thickening on the floor and part of the anterior surface of the utricle.

The saccule is a vesicle of flattened oval form, which lies in a spherical recess of the vestibule, attached thereto by connective tissue and fibers of the saccular portion of the acoustic nerve. The nerve terminates in the macula which lies in the medial wall of the saccule.

The cochlear duct begins as a blind sac (cul-de-sac of the scala media), lodged in the cochlear recess at the floor of the vestibule. The cochlear duct or scala media continues through the canal of the cochlea in spiral form to end blindly at the tip of the apical turn. The cochlear duct partially separates the two perilymphatic (periotic) scalae from each other; one of these, near Reissner's membrane is the scala vestibuli; the other near the basilar membrane is the scala tympani. In the apical turn of the cochlea, the cochlear duct, becoming flattened, extends beyond the hamulus of the spiral lamina. The blind end projects into the capsular cavity of the apical turn, there taking part in the formation of the helicotrema.

The otic or endolymphatic duct, which for its greater extent lies within a bony canal, the vestibular aqueduct, is surrounded directly by the periosteum and not by the periotic labyrinth. The distal end

of the endolymphatic duct communicates with the endolymphatic sac which lies outside of the otic capsule between the two layers of the dura. At the inner end of the vestibular aqueduct the endolymphatic duct enters the vestibule where it unites with the utricular and the saccular ducts in a Y-shaped formation. The ductus reuniens unites the saccule to the cochlear duct.

C. Functional Anatomy of the Vestibular Sense Organs of the Labyrinth.

1. Semicircular Ducts (ordinarily referred to as canals) and Cristae.

The semicircular ducts are arranged in three pairs, the posterior, the anterior and the lateral or horizontal, each pair lying in a plane approximately at right angles to the others (see Fig. 2). The dilated ends or ampullae of the two ducts in each pair are at opposite ends. Accelerative, rotatory movement of the head in any plane causes a fluid movement in the ducts or canals by virtue of the inertia of the contained fluid — endolymph. The effect is greatest in the pair of ducts closest to the plane of rotation but is rarely, if ever, limited to a single pair. The movement of the fluid is toward the ampulla, or ampullopetal, in one duct or canal and away from the ampulla, or ampullofugal, in the opposite duct or canal of any pair.

Each semicircular duct is connected with the utricle at both ends, the small ends of the posterior and the anterior semicircular ducts being fused to form a common connection (common crus) with the utricle. The dilated end or ampulla

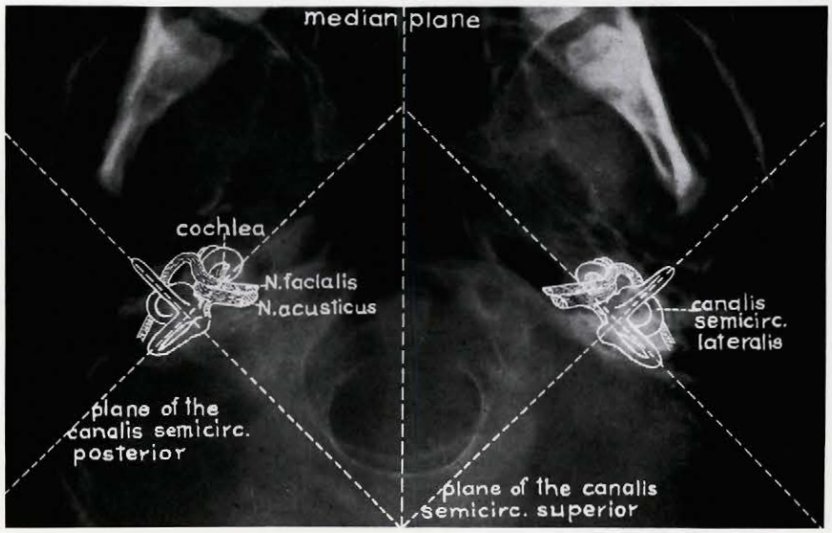


Fig. 2 The planes of the semicircular canals. (from Lindsay and Perlman)

of each duct contains : (1) a transverse ridge or crest, the crista, on the outer wall of which are the incoming nerves; (2) the sensory cells; and (3) the cupula with its supporting structure.

The crista ampullaris develops at the site of the entrance of the ampullary nerve, a branch of the vestibular nerve. The crista ampullaris is a ridgelike, elevated fold of neuroepithelium, containing the sensory cells which bear bristle-like hairs at their free margins, and the supporting cells. This sensory organ becomes elevated into a mound of acellular jelly-like mass, the cupula (see Fig. 3). In general, the plane of each crista lies at a right angle to the plane of each semi-

circular duct near the latter's point of entrance into the utricle. The posterior crista lies on the anterior wall chiefly, but slightly on the inferior and medial walls of the posterior ampulla. The anterior crista lies on the anterior and slightly on the ventro-lateral walls of the anterior ampulla. The lateral crista lies on the anterior wall and slightly on the superior wall of the lateral ampullæ.

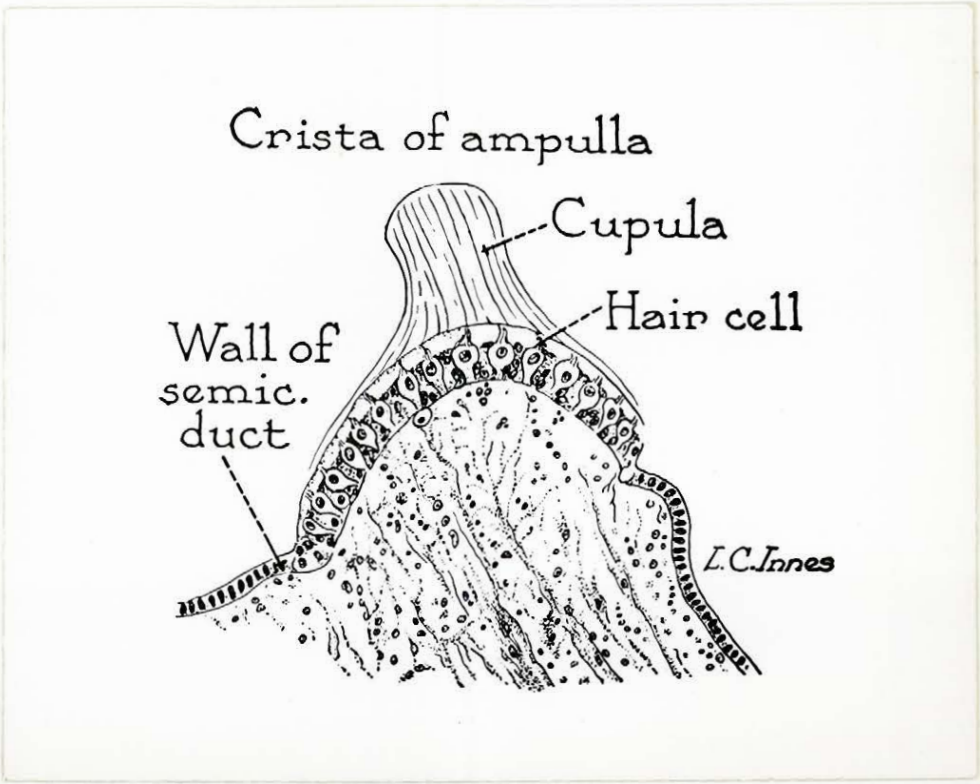


Fig. 3 The crista of an ampulla.
(from Bast and Anson)

The crista seems to be concerned with detecting those changes in the position of the head which are due to rotatory movements. Any rotatory movement of the head along the plane of a semicircular duct would cause an inertial flow of the fluid across the

cristal ridge and thus stimulate the sensory mechanism of the crista. Since the right and left cristae of the lateral semicircular ducts lie in the same plane, they seem to work together in lateral rotation, but in an antagonistic manner. For the vertical semicircular ducts a similar positional relationship exists between the left posterior and the right anterior ducts, or the right posterior and the left anterior ducts.

2. The Utricle, the Saccule and Their Maculae (Otolithic Organs).

The anatomy of the utricle and the saccule have been described. The sensory end organs of the utricle and the saccule are the maculae. The maculae develop from the lining epithelium which overlies the areas where the saccular and the utricular nerves enter the wall of the saccule and utricle. In these zones the epithelium becomes modified into a complex pseudostratified layer. Two types of cells are present, the sensory cells which bear bristle-like hairs at their free margin, and the supporting cells. The latter cells secrete a gelatinous substance which forms a cushion-like membrane, the otolithic membrane, which overlies the modified epithelium and bears superficial calcareous deposits, the otoconia or otolith. The macula of the utricle is essentially horizontal facing upwards. The maculae of the right and left utricles lie in essentially the same plane. With any movement, they should be stimulated alike. The macula of the saccule is essentially vertical facing outwards. The exact relation between the sensory cells and gelatinous mass is not clearly understood. Consequently, the mechanism of stimulation remains obscure. The otolithic organs respond directly to

the force of linear acceleration, e.g., force of gravity, centrifugal force, straight line acceleration in elevator, train or swing. In the absence of motion, the force of gravity will have a sustained effect on the otolith[87].

D. Circulation of the Endolymph or Otic Fluid.

The origin and circulation of the endolymph or otic fluid has not been settled with certainty [166]. Guild[58](1927) suggested that the probable origin of the endolymph is in the stria vascularis. It flows toward the basal end of the cochlear duct, from this through the canalis reuniens into the saccule, from the saccule through the saccular duct into the endolymphatic duct and the endolymphatic sac where absorption takes place. The endolymph is supposed to leave the membranous labyrinth by passing through the wall of the pars intermedia of the endolymphatic sac into numerous small blood vessels of this region.

Shambaugh[145](1908) attributed the formation of the otic fluid to some glandular structures occupying the deeper part of the sulcus externus of the cochlear duct. The endolymph is formed by secretion of these glands and not by filtration from the vessels. The work of Aldred, Hallpike and Ledoux[2](1940) on the osmotic pressure of the endolymph supports the position of Shambaugh that the endolymph is formed by secretion.

The site of absorption is likewise disputed. Lindsay[84](1947) has produced evidence that the vascular portion of the sac may not be the only area from which endolymph can be absorbed; apparent destruction of this area in monkeys failed to increase the endolymphatic pressure.

At the junction of the utricular duct and the utricle there is a valve-like fold of the wall referred to as the utriculo-endolymphatic valve[15](1928). Bast[16](1934) suggested that it is so placed that a sudden pressure disturbance in the utricle would close the valve and thus prevent endolymph from flowing out into the endolymphatic duct. He indicated that the usual flow of the endolymph is from the utricular duct into the utricle, but that a slow movement in the opposite direction should be possible.

E. Blood Supply of the Labyrinth.

1. Arterial Supply.

The chief artery of the internal ear is the internal auditory artery, usually given as a branch of the basilar artery in about 17% of cases or as a branch of the anterior inferior cerebellar artery[71] in the remaining 83% of cases. The internal auditory artery accompanies the seventh and the eight cranial nerves through the internal auditory meatus. Its main branches run in the inner periosteum of the bony labyrinth, and through the trabeculae between the membranous and the bony labyrinths are distributed to the membranous labyrinth (see Fig. 4). There are apparently no anastomoses between the vessels.

According to Shambaugh[146](1923) the internal auditory artery typically gives off the posterior vestibular (or vestibulo-cochlear) artery which supplies the macula of the sacculus, the ampulla and the entire length of the posterior canal, the common crus and adjacent posterior portion of the horizontal canal, and frequently, a branch to the base of the cochlea. After

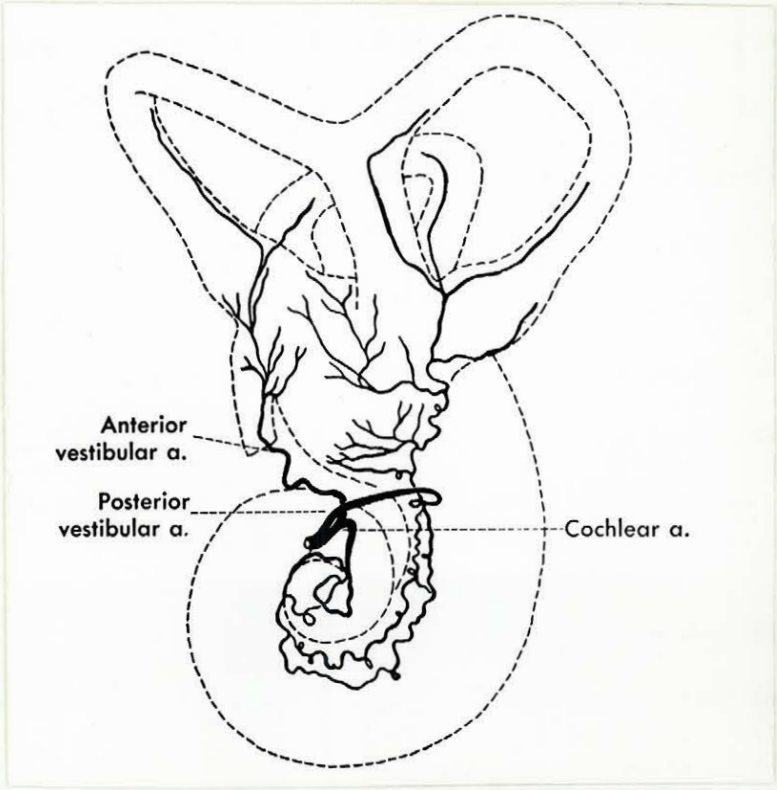


Fig. 4. Arterial Supply of the Labyrinth.
(from Hollinshead)

giving off its posterior vestibular branch the internal auditory artery then divides into the artery of the cochlea and the anterior vestibular artery. This latter artery supplies the macula of the utricle and the anterior portions of the anterior and horizontal canals, including the cristae.

2. Veins.

Shambaugh described only two veins as draining the labyrinth, one running by way of the cochlear aqueduct and the other, by way of the vestibular aqueduct. He stated that the vein of the cochlear aqueduct drains the entire cochlea and part of the vestibular apparatus, including the maculae of the saccule and utricle and the cristae of the anterior and horizontal canals,

while the vein of the vestibular aqueduct drains only a limited area, namely, the remainder of the anterior and horizontal canals, and the posterior canal including its crista.

The vein of the cochlear aqueduct joins the inferior petrosal sinus or the bulb of the internal jugular vein, while the vein of the vestibular aqueduct joins the superior petrosal sinus.

F. Acoustic Nerve, The Vestibular Division.

1. Special Somatic Afferent, Proprioceptive Component.

The vestibular nerve functions in the maintenance of equilibrium. It is therefore proprioceptive. The major fibers of the vestibular nerve are said to be of a special somatic afferent type[33].

The neurons of the first order for the vestibular nerve have as their cells of origin the vestibular ganglion (Scarpa's ganglion) in the lateral end of the internal auditory meatus. In this ganglion each part of the labyrinth has a definite localization[87]. The dendrites of these cells terminate in relation with the hair cells of the crista ampullaris of each semicircular canal, the macula sacculi and the macula utriculi. The axons of these cells forms the vestibular nerve proper. The vestibular nerve occupies the posterior part of the internal auditory meatus, associated with the cochlear division of the acoustic nerve, from which it is incompletely separated; the vestibular fibers lie ventral to the cochlear fibers. Peripherally, the vestibular nerve divides into two divisions, a

superior and an inferior; the superior division of the nerve is said to be distributed to the ampullae of the horizontal and anterior canals, and the maculae of both the utricle and the saccule. The inferior division of the nerve is distributed largely to the macula of the saccule and to the posterior ampulla (by a separate branch of the inferior division); it usually gives also a small division (Oort's nerve) to anastomose with the cochlear nerve (see Fig. 5). Another small anastomosing bundle of fibers was described by Hardy in 1934, known as the cochleosaccular branch which is closely related to the saccular nerve. The central processes from the vestibular ganglion pass medially and forward to enter the medulla

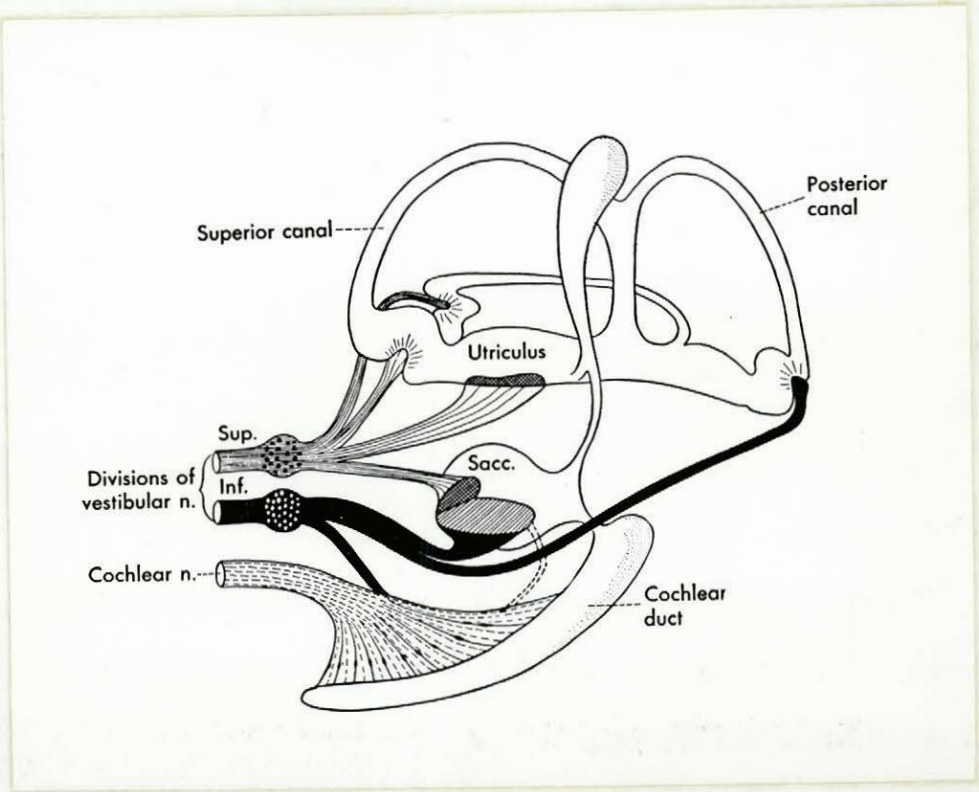


Fig. 5. Schema of the innervation of the labyrinth. (from Hardy)

oblongata medially and cephalad to the cochlear nerve. They enter chiefly at the inner aspect of the restiform body and terminate in the vestibular nuclei.

Lorente de No[89](1933) in his study on the anatomy of the eighth nerve divided the vestibular nerve into five groups : Group I and II contain fibers from the semicircular canals ; Group IV fibers from the utricular macula; Group V, fibers from the saccular macula; it has not been determined whether Group III contains fibers from the central part of the cristae or from the utricular macula.

2. Efferent Component of the Vestibular Nerve.

Petroff[125](1955) gave the most convincing evidence of the presence of efferent fibers. He found that fine fibers in the vestibular rami disappeared following midline cuts in the floor of the fourth ventricle or eighth nerve section. The existence in the vestibular neuroepithelium of nerve endings possessing the properties of efferent fibers has been demonstrated by Wersall[163] in 1956 and by Dohlman, Farkashidy and Salonna[40] in 1958.

Oorts[121](1918) described the vestibulo-cochlear bundle which is associated with the saccular ramus to the cochlear nerve. He mentioned that it could conceivably carry the sympathetic fibers to the cochlea. He was not able to determine the peripheral extension of these fibers.

Hardy[64](1934) described the cochleo-saccular anastomosis or bundle. She observed only a portion of its peripheral extension. It is present in about 40% of cases. She believed that

this represented a true branch of the cochlear division, which supported the contention that the saccule has an auditory function.

Rasmussen[132, 133](1946) demonstrated that the so-called olivary peduncular fibers ultimately arrive in the cochlea via Oort's vestibulo-cochlear anastomosis. He termed it the superior olivo-cochlear fascicle. The morphological features of its neurons are suggestive of an autonomic function. He stressed the intimate relationship of Oort's and Hardy's bundles, and thought that both may have similar functional fibers.

Gacek[52](1960) demonstrated the efferent vestibular fibers which accompany more or less the efferent cochlear fibers in the vestibular nerve trunk as far as the saccular ganglion at which point they diverge at almost right angle to each other (see Fig. 6).

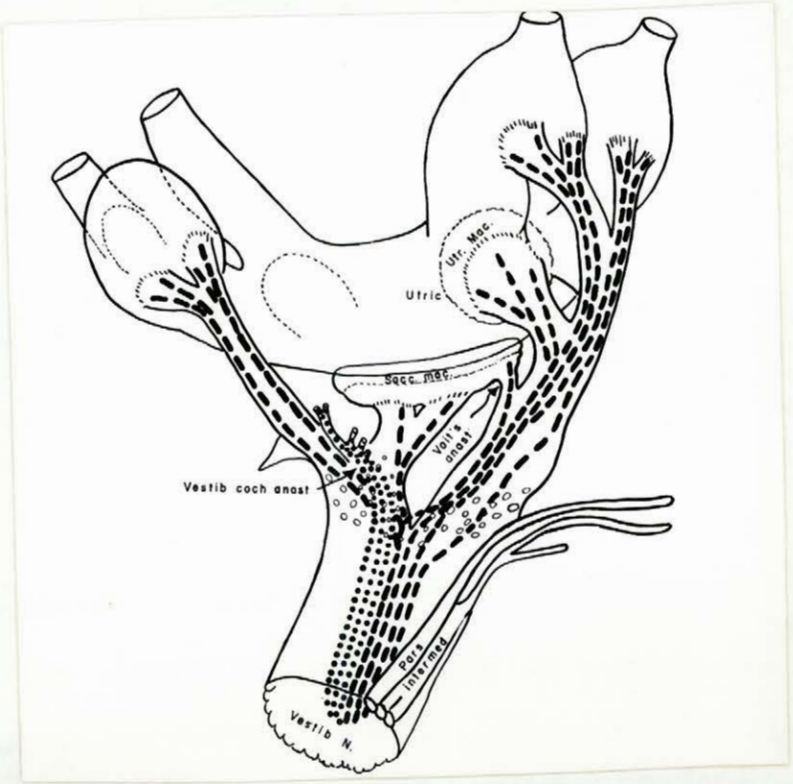


Fig. 6. Diagram of the vestibular efferent fibers. (from Gacek)

Rasmussen[131](1940) counted the nerve fibers in 37 vestibular nerves from persons with normal labyrinthine function. Apparently, a normal vestibular nerve contained from 14,200 to 24,000 fibers, averaging 18,500 fibers.

G. Vestibular Nuclei and Central Vestibular Pathways.

1. Vestibular Nuclei.

a. The Major Vestibular Nuclei.

The vestibular nuclei lie in the area acoustica in the brain stem near the floor of the fourth ventricle, medial to the inferior cerebellar peduncle, extending cephalocaudally from the caudal third of the rhomboid fossa to a point above the genu of the facial nerve[148].

The major vestibular nuclei are : a superior (nucleus of von Bechterew), a medial (principal, triangular or nucleus of Schwalbe), a lateral (nucleus of Deiter's) and an inferior (descending or spinal) vestibular nuclei[57, 130]. Brodal and Pompeiano[20] in 1957 have described these nuclei in detail in their study of the vestibular nuclei in the cat. A description of the cytoarchitecture and the topography of the vestibular nuclei is beyond the scope of this paper. The reader will please refer to the paper of Brodal and Pompeiano on the "Vestibular Nuclei in the Cat" in 1957 and to Chapter 17 of Brodal in "Neural Mechanism of the Auditory and Vestibular System", edited by Rasmussen and Windle, 1960.

b. The Minor Vestibular Nuclei.

Brodal[19] described certain minor vestibular cell groups

in the cat — group x, found lateral to the descending vestibular nucleus; group z, almost continuous with group x, extending caudally to the rostral pole of the nucleus gracilis; group f, caudoventral to the descending nucleus; and the interstitial nucleus of Ramon y Cajal. The function of these is not known ; however, he postulated that these minor vestibular nuclei might have something to do with the integration of the major vestibular nuclei. Crosby[33] and her associates described these as nucleus tangentialis. It is of no known importance in man, but it is large in some submamalian forms.

Although all vestibular nuclei receive vestibular root fibers, there is considerable evidence that a pattern is projected upon these nuclei which corresponds to the peripheral relationship of the vestibular fibers. From Lorente de No's studies[88, 89] (1933; 1933b) it appears that the fibers from the cristae of the semicircular ducts (his fiber groups I and II) terminate in the superior nucleus, in the lateralmost part of the descending nucleus, and in the medial nucleus, while they do not end in the nucleus of Deiter. The vestibular afferents to the nucleus of Deiter (its ventral region) are derived from the utricular macula (group IV). These fibers do not pass to the superior nucleus and apparently not to the medial nucleus while some end in the descending nucleus. The fibers from the saccular macula (group V) appear to pass chiefly to the dorsolateral part of the descending vestibular nucleus. Whether the lateral nucleus is supplied only by the utricular fibers, the superior only by fibers from the cristae, remains unsettled, because the origin of the fibers of group III, which end

in the nucleus of Deiter (and presumably also in the medial nucleus) could not be definitely established.

2. Efferent Vestibular Pathways.

a. Vestibulo-cerebellar Connections.

Secondary, as well as primary, vestibulo-cerebellar fibers have been traced to the nodulus, the flocculus, the adjacent part of the uvula, probably the lingula and the fastigial nucleus. The origin of the secondary vestibulo-cerebellar fibers, according to the work of Brodal and Torvik[21](1957), using the modified Gudden method, comes from the ventrolateral part of the descending or vestibulo-spinal nucleus and to some extent from the medial nucleus. Retrograde cellular changes are

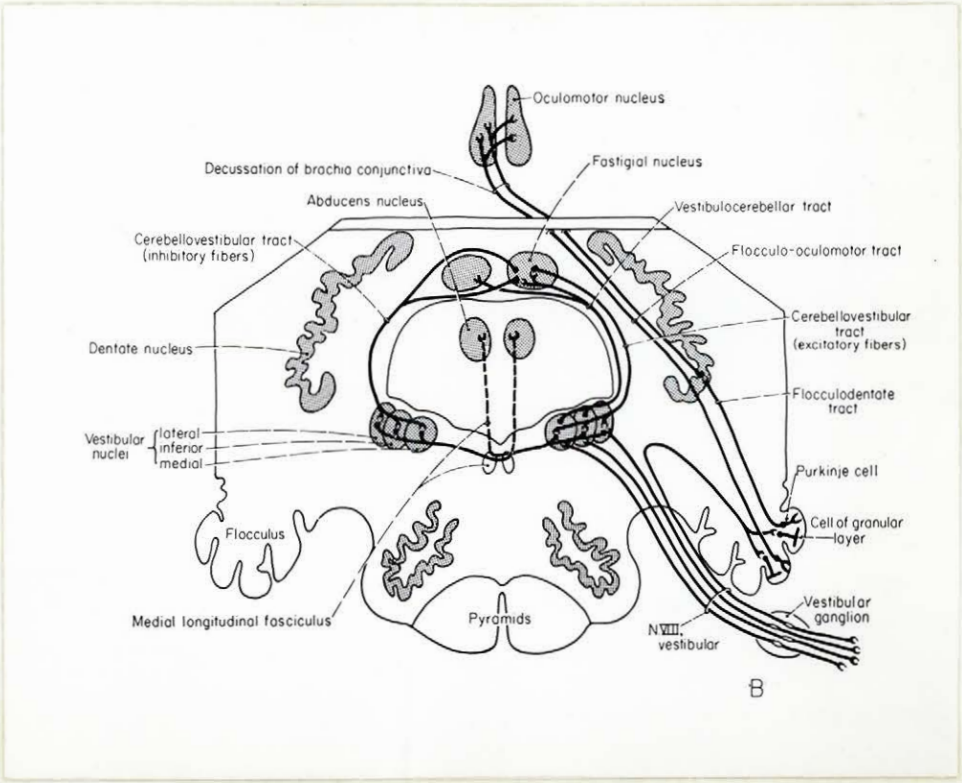


Fig. 7. Vestibulo-cerebellar connections. (from Crosby et al)

found also in the tangential nucleus, group x and group f. These fibers go to the cerebellar nuclei via the inferior cerebellar peduncle. Crosby et al[33] state that impulses from the medial, the superior, and perhaps the inferior vestibular nuclei (from only the medial and the inferior in the cat) are projected over the vestibulo-cerebellar fibers (see Fig. 7) to the homolateral flocculo-nodular lobe and to the homolateral and contralateral fastigial nuclei.

b. Medial Longitudinal Fasciculus.

1) Ascending -- vestibulo-mesencephalic fibers.

All four major vestibular nuclei send fibers to the ascending medial longitudinal fasciculus. From the superior vestibular nucleus, ascending fibers enter the homolateral medial longitudinal fasciculus to project to the trochlear and oculomotor nuclei, motor nuclei innervating the ocular muscles. From the rostral end of the inferior, from the medial and from the caudalmost parts of the lateral[22, 32, 130] vestibular nuclei, fibers cross the midline and ascend in the contralateral medial longitudinal fasciculus to terminate in the abducens nuclear complex, motor neurons innervating the lateral rectus muscle, and also in the smaller cells of the paraabducens nucleus. This latter nucleus, which is a reticular nuclear group lateral to and partly overlapping the abducens nucleus, discharges rostralward to the contralateral medial longitudinal fasciculus to contralateral oculomotor nuclear complex. As a result of this, there will be a conjugate deviation of the eyes (see Fig. 8). The termination of the ascending medial longitudinal fasciculus is not definite-

ly determined. Fibers have been traced to the interstitial nucleus of Ramon y Cajal, the nucleus of Darkschewitch and the nucleus of the posterior commissure, and some have suggested the presence of fibers to the colliculi, the medial geniculate body, the red nucleus, certain nuclei of the thalamus and even the hypothalamus.

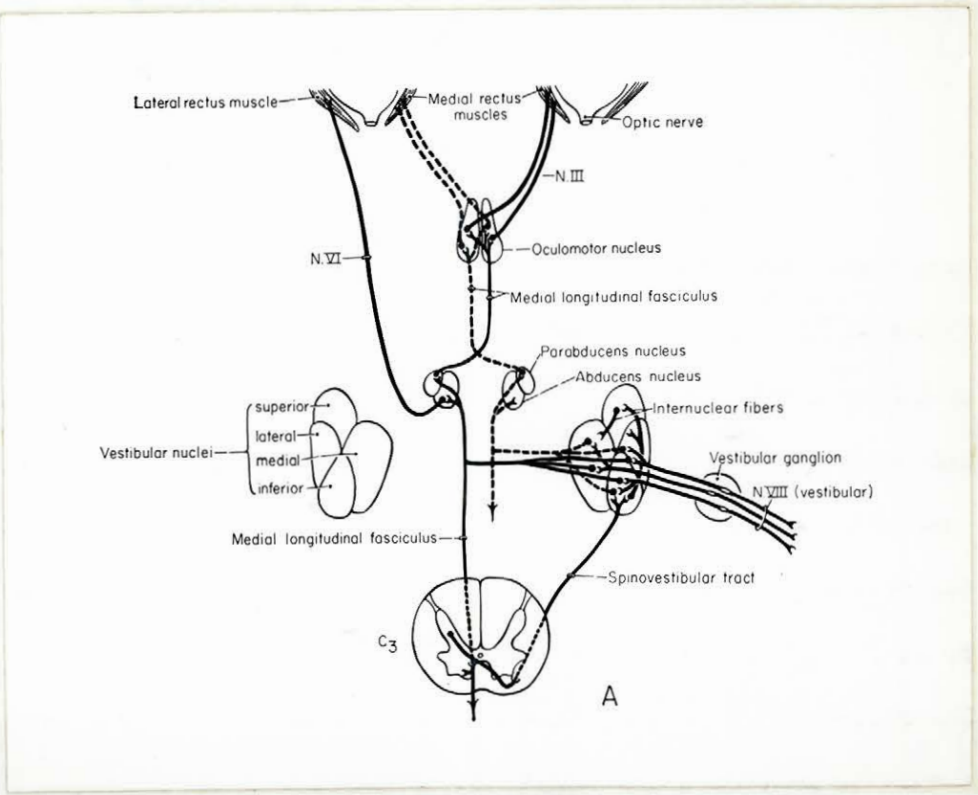


Fig. 8 A. Vestibular connections related to eye movements. (from Crosby et al)

2) Descending Medial Longitudinal Fasciculus.

Fibers from the medial vestibular nuclei and from the descending vestibular nuclei contribute to the descending medial longitudinal fasciculus, both contralateral and ipsilateral[33]. These descending fibers can be traced through the cervical cord levels. Often they are termed the medial vestibulo-spinal tract.

They terminate in relation to ventral horn motor neurons at cervical levels supplying the nuclei that turn the head and perhaps those that move the shoulder and the upper extremity. They are said not to pass lower than the cervical cord levels (see Fig. 8 B).

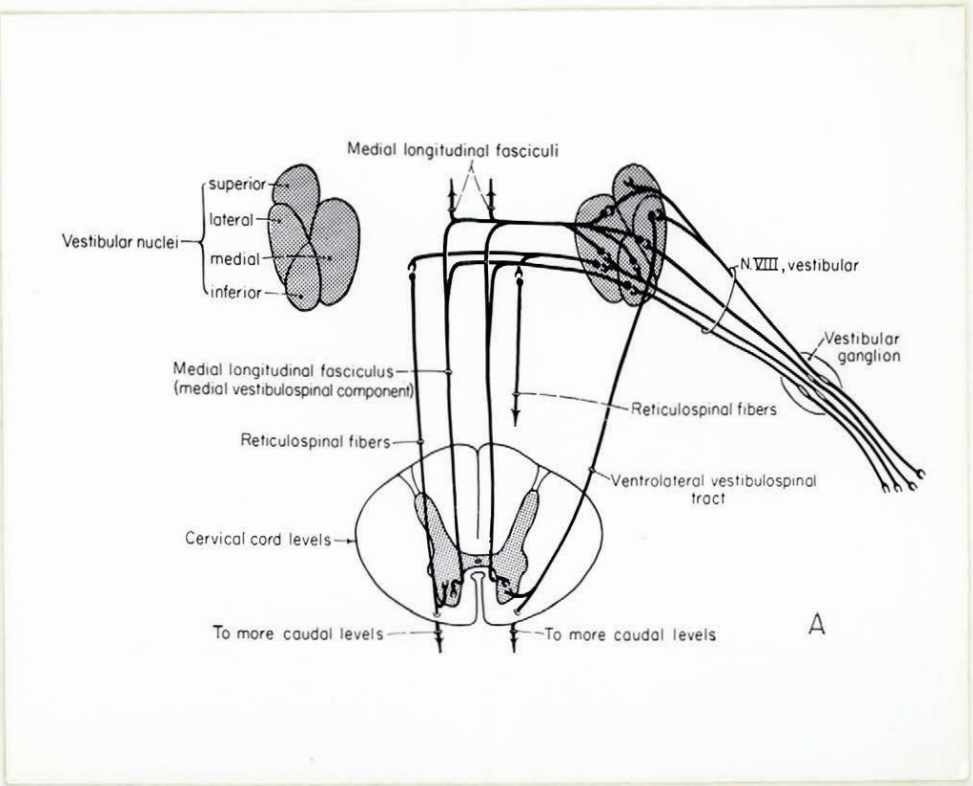


Fig. 8 B. Diagram of descending longitudinal fasciculus. (from Crosby et al)

c. Vestibulo-spinal Tract (Ventrolateral Vestibulo-spinal Tract).

Brodal[19] employing the modified Gudden method in cats showed by retrograde cellular degeneration of the vestibular nuclei after sectioning the spinal cord, that changes were mainly confined to the nucleus of Deiters on the site of the lesion. He was able to demonstrate a somatotopical pattern in the origin of the vestibulo-spinal fibers from the lateral vestibular nucleus (Deiters)

by sectioning the cord at different levels. However, this does not indicate that axons from other vestibular nuclei do not contribute to the vestibulo-spinal tract. This tract ends in relation to the motor neurons at various spinal cord levels. It is believed to reach the lumbar and perhaps sacral cord levels in the cat. It is a part of the neuron arcs concerned with maintaining the normal position of the body. If the vestibular ganglion or the vestibular root is destroyed, the animal falls toward the side of the destruction. If the lateral vestibular nucleus or the ventrolateral vestibulo-spinal tract is destroyed, then there is a deviation of the body toward the opposite side.

d. Vestibulo-reticular Connection.

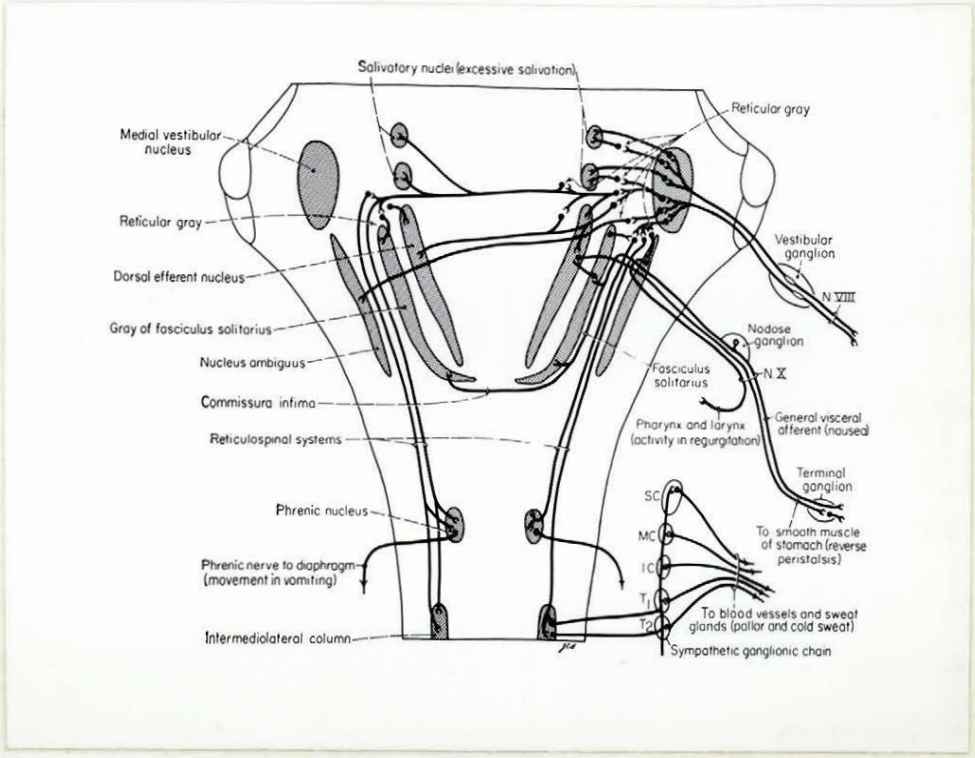


Fig. 9. Diagram of the vestibulo-reticular connections. (from Crosby et al)

The medial vestibular nucleus has connections with the ipsilateral and contralateral reticular grey formation of the brain stem and through this reticular grey formation with the dorsal efferent nucleus of the vagus. The connections with the reticular grey formation of the brain stem also make possible reticulo-spinal discharges in response to vestibular stimulations (see Fig. 9). This accounts for the vegetative symptoms when the vestibular system is stimulated.

e. Vestibular Projection to Thalamus and Cortex.

Secondary vestibular projections to the thalamus and to the cortex undoubtedly exist, but their anatomical pathways and location have not been definitely documented.

Physiological evidence for the projection of vestibular impulses to the thalamus in the neighborhood of the medial geniculate nucleus[111] and demonstration of a cortical vestibular center near the auditory cortex have been satisfactory.

Anderson and Germandt[5](1954) in their study on cats came to the conclusion that the vestibular projection to the cortex is principally contralateral, and it has been possible to demonstrate a specific localization in the cerebral cortex for the particular sensory end organs. Cortical responses to stimulation of the vestibular nerve branches concerned are evoked from an area comprising the anterior ectosylvian gyrus and a narrow strip of the posterior bank of the suprasylvian gyrus. Stimulations of the nerve from the utricle evoke responses particularly from the dorsal part of this area. Below and anterior to the latter focus,

responses to stimulation of the nerve from the lateral crista are recorded, and above it the cortical projection of the nerve from the anterior crista is found. These responsive areas are not only contiguous but to a considerable extent overlapping.

Penfield and Rasmussen[124](1950), by the method of electrical stimulation of the areas of the cerebral cortex in a patient undergoing surgery under local anesthesia, concluded that movement of the whole body in space is represented in the first temporal convolution. They added that ill-defined dizziness is caused by stimulation elsewhere in the hemisphere, especially in the subcortical areas and that occasionally illusions of movements are elicited from the parietal cortex.

Penfield[123](1957) in reviewing all his cases of craniotomies with electrical stimulation of the cortex of conscious patients concluded that the pathway of the vestibular sensations makes a detour from the thalamus out to the cortex where the vestibular area is next to the auditory area in the superior temporal convolution of both sides. From there the pathway probably returns to the higher brain stem where it enters the centrencephalic integrating system. Removal of the superior temporal convolution on either side is followed by no obvious defect of vestibular function.

3. Afferent Vestibular Pathways.

a. Cerebello-vestibular Connection.

Among the fibers from the cerebellum to the vestibular nuclei two components have to be distinguished, namely, those coming from the cerebellar cortex and those from the cerebel-

lar nuclei. Fibers from the vermis of the anterior lobe, the pyramis and uvula have been traced chiefly to the homolateral lateral vestibular nucleus; some appear to reach the superior nucleus. Fibers from the flocculus end in the superior and lateral nuclei. Fibers from the nodulus supply all four vestibular nuclei. Fibers from the fastigial nucleus supply the entire territory of the vestibular nuclei; fibers from the rostral third of the fastigial nucleus supply chiefly the homolateral or ipsilateral vestibular nuclei; fibers from the caudal third of the fastigial nucleus supply the contralateral vestibular nuclei[19].

b. Medial Longitudinal Fasciculus.

The afferents to the vestibular nuclei descending from higher levels of the brain have a restricted area of termination[127]. They end in the medial vestibular nucleus. They are found to descend in the medial longitudinal fasciculus.

There is no evidence obtained for fibers coming from the cerebral cortex, the basal ganglia or the colliculi.

c. Reticular Connection.

The reticular formation of the mesencephalon does not send fibers to the vestibular nuclei. The medullary and pontine reticular formation may contribute fibers to the vestibular nuclei[139].

d. Spino-vestibular Connection.

The spinovestibular fibers ascend with those of the dorsal spinocerebellar tract and end in the caudalmost part of the

descending vestibular nucleus and medial vestibular nucleus, but have their chief sites of termination in the lateral vestibular nucleus and in the groups x and z in a somatotopical pa ttern. They come from levels below L_3[126].

II. Physiology of the Vestibular Sense Organs of the Labyrinth.

A. Function of the Semicircular Ducts (Canals).

The sense of equilibrium or orientation in space depends on impulses from the labyrinths, from the retinae or eyes and from the muscles and joints, the so-called kinesthetic sensation. McNally[101](1947) stated "Whereas maintenance of equilibrium is an incidental function of the ocular mechanism and the kinesthetic mechanism, it is the main function of the vestibule or the labyrinth."

Although the vestibular part of the labyrinth, the semicircular canals and vestibule were known and described by the anatomist, Fallopio, in 1561, their function was not realized until the nineteenth century. Barany (1922) pointed out that Purkinje in 1825 reported the presence of nystagmus of the eyes and dizziness in persons who were subjected to rotation. Flourens, a physiologist, operated on the semicircular canals of pigeons and found that injury of a semicircular canal caused twitching movements of the head and eyes in the plane of that canal. Meniere in 1860 found that severe dizziness is frequently associated with deafness, and he suggested the possibility that lesions of the semicircular canals might be responsible for the dizziness. He found hemorrhagic exudate in the labyrinth.

Goltz in 1870 was the first to realize that the function of the labyrinth was the maintenance of equilibrium.

The experiments and observations of Mach in Prague, Breuer in Vienna, and Crum Brown in Edinburgh in 1873-1874 led each individually to the theory that stimulation of the semicircular canal is brought about by movement of the endolymph activating the hair cells in the ampulla. Later, Mach modified his theory to a mere pressure effect because he could not demonstrate gross movement of the fluid in the semicircular canals. He arrived at the conclusion that angular acceleration stimulates the six ampullae of the semicircular canals, which are arranged in opposite pairs. However, he could not express any opinion as to the receptors for positional, or gravitational, stimulation. (McNally and Stuart[103],1942)

Ewald in 1892 experimented with thermal, electrical and mechanical stimulation of the canals. He made an ingenious experiment on a semicircular canal of the pigeon in which he sealed off the distal part with a lead plug and inserted a pneumatic hammer between it and the ampulla. In this way he could exert positive and negative pressure within the membranous labyrinth to change the flow of the endolymph in the canal. Movements of the head and eyes took place in the plane of the canal stimulated. He concluded that in the horizontal canal of the pigeon, movement of endolymph towards the ampulla (ampullopetal) caused the greater stimulus and movement away from the ampulla (ampullo-fugal) caused a lesser stimulus. In the vertical canals, the condition was the reverse of the horizontal canal. Further, he concluded that when the horizontal canal or the vertical canals were maximally stimulated, there resulted a slow movement of the eyes to-

ward the opposite ear and a quick phase of nystagmus to its own side. When the semicircular canals were minimally stimulated, the quick phase of nystagmus was toward the opposite ear. These findings have since been known as "Ewald's Laws". The former (stimulation of the semicircular canal results in head and eye movements in the plane of the canal stimulated) becomes Ewald's First Law, and the latter "Ewald's Second Law".

The validity of Ewald's Second Law has been refuted. Maxwell[96] (1923), McNally and Tait[107](1925) and others found that a semicircular canal in the fish and the frog responds to stimulation only in one direction and that is, in the direction of the maximal stimulus for the canal. McNally, Stuart, Jamieson and Gaulton[104] (1948) carried out caloric tests (alternate hot and cold at 44°C and 30°C) in one hundred and one subjects (adults) in the face-up and face-down positions. They concluded that cold stimulation (cold water at 7° below body temperature) is a greater labyrinthine stimulus than hot water at 7° above body temperature; the horizontal canal in man is more responsive to caloric stimulation in the face-up position than it is in the face-down position; Ewald's Second Law is disproved if the caloric test is carried out in the face-up position, but if it is done in the face-down position, Ewald's law is confirmed. Hallpike[59](1961) reported that since 1941 he together with Fitzgerald and Cawthorne have observed that a cold stimulus elicited a greater labyrinthine response than did a hot stimulus (employing the usual Fitzgerald and Hallpike alternate cold and

hot stimulation) in normal subjects. This is against Ewald's Law. However, if one of the labyrinths was destroyed, "Ewald's effect" was observed in the remaining labyrinth. They attributed this to two mechanisms: the elimination of certain tonus action after the destruction of one of the labyrinths, leaving the opposite tonus action unopposed, causing at first spontaneous nystagmus to the unaffected side and a preponderance to the same side of evoked nystagmus; and second, the presence of a dual cupular mechanism, one for the tonic or resting discharge (action potential) and the other for the modulation of this resting discharge. They stated that since Ewald opened into the canal and packed its lumen with lead or amalgam, the resting discharge from the cupula must have been inevitably reduced. When Ewald then applied his hammer to the canal, the resting discharge being already reduced, could not be reduced much further. Violent stimulation could still, however, evoke a considerable increase in the response, and it did. Because of this evidence and the lack of proper electrophysiological study by Ewald, Hallpike favored the repeal of Ewald's 2nd law. During the Proceedings of the International Symposium on Problems in Otoneurology in May, 1960, Ewald's 2nd law was extensively discussed[129]. Opinions from leading authorities varied. However, the concensus of opinion suggested, on the basis of clinical observations and by electrophysiological studies, that Ewald's 2nd law is not valid in man.

Kubo in 1906 noted that the labyrinth of fish responded to thermal changes. He noted that the horizontal canals are more responsive than

the vertical canals and that heat is more effective than is cold.

Barany (1908) outlined the clinical importance of examining the vestibular apparatus. He pointed out that the clinical rotation test resulted from the experiments of many investigators, chiefly those of James and Kriedl (1893-1906) and of Wanner. Barany then developed the caloric test as a method of stimulating one labyrinth at a time as compared to the rotation test which affects both. He found that if a normal ear is irrigated with water above or below body temperature, nystagmus results. In analyzing the phenomenon he concluded that the nystagmus is the result of vestibular stimulation and that the semicircular canals are stimulated because of change of temperature in the endolymph. This causes a rise or fall of the endolymph, depending on whether the irrigating fluid is warm or cold. He stated that this new test is of value in the early detection of labyrinthine disease, either acute or chronic, suppurative or non-suppurative (McNally and Stuart[103], 1942).

Kobrak (1920-1923) described a modification of Barany's caloric stimulation of the labyrinth, which he called a minimal or threshold stimulation. He advocated the use of a small quantity of water, about 10 cc. at a temperature of about 27°C., in contradistinction to the large quantities used heretofore. The advantages he claimed for his modification are that the examiner can better gage the irritability of the labyrinthine mechanism, while at the same time the discomfort of the patient is greatly reduced. He considered at some length the mechanism of stimulation, whether the temperature difference affected the blood supply of the labyrinth or the endolymph. He expressed a preference for the former view[103].

Maxwell (1923) experimented on the dogfish. He exposed all the semicircular canals and stimulated their ampullae, individually and in pairs. He found that the stimulation of an ampulla elicited the same eye and fine movements as did a tilt or rotation in the plane of the same canal. Stimulation of the right anterior semicircular canal caused the right eye to move up and the left eye to move down. He then removed all the ampullae and rotated his animals around horizontal and vertical axes. He noted that there was no reaction to turning about a vertical axis but that turning about any horizontal axis elicited reactions which were practically normal. By removing the remainder of the labyrinth, he eliminated any compensatory reactions. He concluded that all compensatory movements except those in response to rotation in the horizontal plane can in the absence of the ampullae be brought about through the action of some other part of the labyrinth.

de Kleijn[34](1923) pointed out the different methods to study the function of the labyrinth in relation to the labyrinthine reflexes in posture. He suggested that by extirpation of the semicircular canals alone or of the otoliths alone, reflexes arising from the extirpated part of the labyrinth would be lacking and the reflexes arising from the remaining part would be present. He classified the different labyrinthine reflexes according to whether or not the reflexes resulted from the stimulation of the cristae of the semicircular canals or the maculae of the utricles.

Labyrinthine reflexes :

1. Reflexes responding to movement (reflexes of the semicircular canals).

   a. Rotatory reactions and after reactions (with nystagmus)

1) on head

2) on eyes

b. Progressive reactions

1) lift reaction

2) toe-spreading reflex

3) springing reflex

2. Reflexes resulting from position (tonic labyrinthine reflexes, reflexes of the otoliths)

a. Tonic labyrinthine reflexes on body musculature

b. Labyrinthine 'righting' reflexes

c. Compensatory eye positions.

Magnus[93](1926) made complete analysis of the labyrinthine and non-labyrinthine reflexes.

McNally and Tait's[99, 106,108, 155, 156](1925-1933) classic ablation experiments on frogs (ablation of individual ampulla of the semicircular canals) showed, by systematic examination of the de-ampullated animal to gravity, centrifugal force, linear acceleration, and angular acceleration about various axes, that (1) the semicircular canals are not involved in static reactions to gravity or centrifugal force, nor does their absence entail forced positions; (2) the horizontal semicircular canals respond only to angular acceleration; (3) any vertical semicircular canal responds to angular acceleration in its own plane either in a positive or in a negative direction; (4) effective stimulation of one of the horizontal semicircular canals brings the musculature on both sides of the body into play; (5) effective stimulation of each vertical semicircular canal is mainly concerned with the musculature of its own side; (6) the four vertical semicircular canals

form a functional group distinct from the horizontal semicircular canals; (7) any lesion of a single vertical semicircular canal is apt to affect the ultimate stationary posture of the frog; (8) the labyrinth is intimately concerned with the control of the muscle tone, posture and movement, the semicircular canals and the utricles all being essential to normal progressive movement and to normal resting posture; (9) the vertical semicircular canals and the utricles are both stimulated by any sudden tilting movements and their reactions being oppositely directed, the one exerting a modifying and controlling influence upon the other; and (10) each set of the semicircular canals in the frog responds to turning in one direction only.

Steinhaussen (1927-1935) devised a method whereby the ampulla of a semicircular canal can be examined in a living animal. He experimented with different animals and tried the effects of various dyes. He succeeded in bringing the different structures within the ampulla into contrast. He was able to make a window in the ampulla and photograph the movement of the cupula. From his microscopic studies and moving picture photographs, he was able to prove that the cupula is a relatively large structure, that it is attached to the top of the cristal ridge and that it extends to, and is in close contact with, the remaining circumference of the ampullary wall. He was able to study the return movements of the cupula after it had been displaced. He studied the reactions of the cupula during sudden and prolonged turning and during caloric stimulation. In the pike, he definitely proved that movement of the cupula in only one direction acts as a stimulus and elicits compensatory reactions. He also observed a definite relation between the duration of post-rotatory nystagmus, or after-nystagmus,

and the return of the cupula from a deflected to its resting position. During electrical stimulation of the labyrinth, he did not observe any movement of the cupula. He assumed therefore that electrical stimulation affects the nerves directly. He confirmed the results of Ewald's experiments, which showed that the direction of the endolymph movement for maximal stimulation differs in the horizontal and the vertical canals (McNally and Stuart, 1942).

Ross[137](1936) recorded action potentials from individual nerve fibers in the anterior and posterior branch of the eighth nerve. He found that the labyrinthine receptors could be divided into two groups, the first responding to crude mechanical vibrations and the second responding to gross tilting movements but not to vibration. The latter group may be divided into two parts, the first is a gravity group responding to simple tilting movements and to linear acceleration, the main receptor being the utricular macula. The second of this group is the rotation group proper, members of which are stimulated by rotatory acceleration. This group contains the three semicircular canals.

Lowenstein and Sand[92](1940) performed electrophysiological analysis of the activity in the ampullae of the three semicircular canals of thornback ray. They showed that there is a spontaneous discharge of sensory impulses from each ampulla when the labyrinth is at rest. During angular displacement in the appropriate direction the discharge of impulses is increased or inhibited. They found that the horizontal canal responds to rotation about the vertical primary axis and is not affected by rotations about the two horizontal primary axes; the anterior and the posterior vertical semicircular canals responds to

rotation about all three primary axes. During rotation about the horizontal longitudinal axis (tilting sidewise) the four vertical canals are laterally synergic and during rotation about the horizontal transverse axis they are transversely synergic. During rotation about the vertical axis the four vertical canals are diagonally synergic . They also observed the occurrence of an after-discharge following inhibitory rotation, which was in agreement with Steinhaussen's observations on the physical properties of the cupula in the pike, in that the duration of the discharge on stopping rotation conformed to the time required for the cupula to return to its normal position.

The experiments of Cawthorne, Fitzgerald and Hallpike[27](1942) on unilateral labyrinthectomy subjects was in favor of bi-directional function of the semicircular canal in contradistinction to others who were in favor of uni-directional function of the semicircular canals.

Dohlman[38](1944) injected China-ink through a glass capillary into the semicircular duct and observed clearly the deformation of the cupula. The cupula moved in a semicircle like radius occluding the lumen so well that it scarcely let any of the endolymph or the ink pass through. This confirmed the findings of Steinhaussen.

Adrian[1](1943) was the first to use a higher mammal, the cat, for recording the discharge following varying stimulation of the labyrinth. The activity was recorded from the vestibular nuclei. The results obtained have not shown any marked difference between the vestibular apparatus of the cat and that of the frog or the fish. He could also differentiate two main types of discharge in the single unit, gravity controlled and rotation controlled.

Van Egmond, Groen and Jongkees[43](1949) from an experimental basis

stated that the semicircular canal with its cupula-endolymph system is best considered as a torsion pendulum with a high degree of damping. There is always a slight leak (not a tight fitting cupula) between the cupula and ampulla, which causes deviations from the theoretical behaviour described by the solution of the differential equation of the torsion pendulum. It is possible to calculate the movements of the cupula, taking the leak into account, and the results then correspond to those obtained from insensitive subjects. Their calculations show that the semicircular canal acts as an "impulsometer". For physiological movements it is the most appropriate instrument, comparable to a fluxmeter used for measurement of electric charges. It normally causes after sensation after turning. They attributed this to the restoration of the cupula which has been driven in the opposite direction. The process of restoration begins immediately after the deformation.

B. Function of the Otolithic Organs.

1. The Utricle.

Breuer (1874-1891) was the first to distinguish between the function of the semicircular canals, on the one hand, and that of the otoliths, on the other. He suggested that the stimulus for the otolithic organ was linear acceleration.

In 1875, Mach described the sensation produced by linear acceleration in man. Kriedl[80] (1893) succeeded in substituting iron fillings for sand in the otoliths of the prawn, and then applying magnetic force. He demonstrated marked disturbance of the animal's equilibrium.

Maxwell[96] (1923) succeeded in moving the otolith directly in

fish and noted counter rolling of the eyes with no nystagmus comparable to that seen in tipping.

Magnus and de Kleyn[94](1923) investigated the whole subject of postural reflexes including those from the otolithic organs. They divided all these into : (1) tonic labyrinthine reflexes on the body, limbs and neck; (2) compensatory eye position reflexes; and (3) righting reflexes.

Versteegh's[161](1927) experiment in severing the nerve to the utricle in the rabbit demonstrated that the utricle was the site of origin of those static reflexes previously attributed to both the utricle and the saccule.

McNally and Tait[107](1925) thoroughly studied the otolithic function of the frog by selective ablation of the nerve supply to the various functional units. They found that in unilateral utricular injury, there is apparent loss of muscular tone on that side; in bilateral utricular injury, the forced position is symmetrical and all gravity responses on the tilt table are absent. They came to the conclusion that the utricle is the apparatus for static equilibrium, the most important gravity organ in the labyrinth. Tait and McNally[108](1933) and Ross[137](1936) all indicated that the utricles are not stimulated by rotation about a vertical axis.

According to Tait and McNally[156](1934) the reflexes initiated by otolithic stimulation are designed to produce compensating movements of the body and limbs in order to right the head and body when it is displaced. On exposure to linear acceleration outwards as in a centrifuge, the utricle of a frog initiates reflex muscular movements that produce a leaning of the animal towards the center of rotation. A slow tilt also initiates compensatory reflexes from the utricular otolith to

maintain the head and trunk level. Tait and McNally concluded that the organs of the labyrinth concerned in reflex adaptation of bodily posture to the field of gravity are the utricular maculae. They also stated that the reflexes from the vertical semicircular canals of the frog tend to oppose the reactions from the utricle, as manifested by tilting the animal.

Summary. The utricular macula is an organ of static equilibrium. It is affected by any change of position of the head -- response to gravity. It is also affected by any steady force acting upon the animal in a direction other than the pull of gravity, such as centrifugal force. The utricle influences the tone of the muscles of all four limbs symmetrically. It affects the tone of the muscles of the opposite side of the neck. It is also the seat of origin of the labyrinthine righting reflexes and labyrinthine conpensatory eye positions[98].

2. The Saccule.

McNally and Tait[106](1925) and Versteegh (1927) were among the first to perform experiments by sectioning the nerve to the saccule and they found no disturbances to the equilibrium.

Ashcroft and Hallpike[11](1934) made electrical recordings of single nerve-fiber preparations from the saccule of the frog and showed that the saccule does not respond to movement but to vibration. Their finding was confirmed by Ledoux in 1949.

Owada and Shizu[122](1960) made experiments in rabbits by sectioning the saccular nerve. They concluded from their experimental results that the compensatory deviation of the eyeballs is one of the functions of the saccule.

C. Mode of Hair Cell Stimulation.

The conventional explanation of hair cell stimulation is dependent upon the bending or a pulling of the hairs of the sensory cells. The mechanism of such an action is quite obscure.

Wersall[164](1961) stated " The receptor cells in the sensory epithelia of the labyrinth are mechanical transducers with ability to transform mechanical energy into electrical impulses. The manner in which energy is transformed from the endolymph to the hair cells is of considerable interest. The forces acting on the receptors apparently stimulate the hair cells through the hair bundle protruding from the top of each hair cell. These bundles contain up to around 70 sensory hairs, most of which are of sterociliar type.

According to Vilstrup and Jansen[162](1961) both the cochlea and the vestibular labyrinth are stimulated by mechanical energy. This type of energy is transformed into electrical energy. How does the transformation take place? These authors stated that the site of energy transformation must be in the regions of the sensory hairs of the labyrinth. Acid mucopolysaccharides are present in the human cupula. These are extremely high molecular substances, the potassium salts of which when dissolved in water form viscous solutions or gelatinous substances. With proteins, they form compounds. Because of physicochemical properties of these acid mucopolysaccharides, displacement of such a fluid might be accompanied by a generation of chemo-electric potential. This was proven experimentally.

Lowenstein[91](1961) likewise agreed that the hair cells of the labyrinth are a transducer for mechanical stimuli. However, what happens electro-physiologically at or in the hair cells before, during and after deformation of the hair processes, according to the

author, is not known. He postulated the existence of a potential boundary between the cupula and the hair cells. Cupula displacement leads to changes in the potential -- depolarization or hyperpolarization, according to the direction of the cupula displacement towards or away from the utricle respectively.

Chapter II

Effects of Stimulation of the Vestibular Sense Organs of the Labyrinth

The clinical evaluation of the vestibular sense organs of the labyrinth is mainly determined by the stimulation of the semicircular canals. There is a widespread effect on the whole body mechanism on stimulating these semicircular canals. The vestibular portion of the labyrinth together with the eye and proprioceptive mechanism from the muscles, joints, tendon and body surfaces are concerned with the maintenance of equilibrium or orientation of the body in space. Of all these, the most important is the labyrinth. The effects of labyrinthine stimulation are manifested by nystagmus, alteration in the pattern of the body musculature, changes in the autonomic nervous system, and subjective sensation of turning or falling (vertigo). For the most part, however, im clinical practice the observations of labyrinthine reactions are restricted to eye movements -- nystagmus. Exceptions to this are the occasional testing of past pointing of the arms, the recording of the sensation of dizziness, the presence of pallor and perspiration, and, very rarely, blood pressure studies (McNally and Stuart[106], 1953).

A. Nystagmus.

1. Definition. Nystagmus is a rhythmic to and fro movement of the eyes. There is usually a slow motion in one direction (slow phase or slow component) and a quick recoil (quick phase or fast component) in the opposite direction.

2. Classification.

a. As to origin of the nystagmus.

1) Peripheral Labyrinthine Nystagmus.

Labyrinthine nystagmus is usually of the horizontal ro-

tatory type, of small amplitude, moderate frequency and of short duration; the eye movements are associated. In this type of nystagmus, the slow phase is the primary movement and the quick phase, secondary. This is seen in diseases of the inner ear or the vestibular nerve. During the stage of labyrinthine irritation the fast component is to the side of the lesion. As soon as the labyrinth has been overcome by disease, the quick phase or fast component is toward the healthy side and is produced by the unopposed healthy labyrinth. This second stage will not last more than a few weeks, so that a spontaneous nystagmus of labyrinthine origin is relatively of short duration and is usually accompanied by vertigo and hearing changes.

Fixation of gaze has an inhibiting effect, while gaze in the direction of the quick component increases the intensity of the nystagmus.

2) Ocular Nystagmus.

Ocular nystagmus is usually of long duration. It tends to be irregular and may be characterized by to-and-fro oscillation rather than by slow and fast components. Ocular nystagmus is generally horizontal in character; there is usually an associated eye lesion.

The following types of ocular nystagmus may be differentiated :

a) Nystagmus of the Blind. This is characterized by both wandering and to-and-fro movements. There are not sufficient retinal stimuli to allow fixation.

b) Nystagmus in Amblyopia. This has a distinguishing features of pendular movement which is due to fixation that results from congenital defect with deficient central vision.

c) Miner's Nystagmus. This may develop in people with good vision after prolonged exposure to darkness, as in the case of coal miners.

d) Hereditary Nystagmus. This is familial and is associated with

amblyopia.

e) Musculoparetic Nystagmus. This may appear in the presence of paresis of an eye muscle on looking to the side of the paresis. This is due to the inability of the paretic muscle to maintain the lateral fixation position.

f) Gaze Nystagmus (Deviational Nystagmus, Nystagmus of Eccentric Fixation or Fixation Nystagmus, End Position Nystagmus). Gaze nystagmus is present in 60% of normal subjects (when the eyes are directed toward an object in the extreme lateral position). Unilateral gaze nystagmus is usually pathological.

g) Opto-kinetic Nystagmus. Optokinetic or optomotor nystagmus is a reflex response excited by movement of images across the retina. It occurs during movement of either the subject or the environment or both; this is exemplified when the subject is rotated or looks out of the window of a rapidly moving conveyance, or when the environment moves rapidly past the subject, as a train passing the window, for example, nystagmus of this form is actually an oculocerebral reflex.

3) Central Nystagmus.

Central nystagmus is due to a disturbance of the central vestibular nuclei or their secondary pathways. It is fairly constant and may be of long duration. It may be horizontal, vertical or diagonal in direction. The eye movement may be dissociated. There are usually other signs of intracranial involvement to assist in the diagnosis. This type of nystagmus is similar to ocular nystagmus in the direction of gaze, in that the quick movement is primary and the slow phase of the nystagmus is secondary.

b. As to Intensity of the Nystagmus.

The intensity of a nystagmus of vestibular origin has been graded as to (by Alexander) :

1) 1st degree -- when the nystagmus is only visible during gaze in the direction of the fast component.

2) 2nd degree -- when the nystagmus is present or visible on the primary position or forward gaze.

3) 3rd degree -- when the nystagmus is present or visible also in the direction of the slow component.

c. Other Classifications.

1) Spontaneous Nystagmus.

This should be restricted to a nystagmus which occurs without any apparent provokation. It may be caused by disease or irritation of any of the structures normally concerned in the production of nystagmus.

2) Provocation Nystagmus.

This type of nystagmus was described by Frenzel in 1955[50]. By this is meant nystagmus present after repeated posturing of the head, or nystagmus induced by the stimulation of neuro-otological investigation such as results from caloric or rotation tests.

3) Positional Nystagmus.

Positional nystagmus is defined as a spontaneous nystagmus, which is not constantly present, but develops when the head takes a certain position in space[53, 120]. It may be tonic or transitory, and it is frequently associated with dizziness.

a) Types of Positional Nystagmus[120].

(1) Direction-changing positional nystagmus.

This is characterized by the nystagmus changing direction

in different, most often opposite, position of the head. (Nylen's Type I Positional Nystagmus)

(2) Direction-fixed positional nystagmus.

This is recognized by the nystagmus always in the same direction when it arises regardless of changes of position of the head. (Nylen's Type II Positional Nystagmus)

(3) Irregular positional nystagmus.

This designates that nystagmus which is characterized by variations in its behaviour. (Nylen's Type III or Transitory Form of Positional Nystagmus)

Type II is found in both peripheral and central disorders. Types I and III indicate a central disorder when no labyrinthine fistula is present[119].

b) Classification of Positional Nystagmus.

Spector[152](1961) classified positional nystagmus as peripheral or central type according to its characteristics.

(1) Peripheral Type.

Peripheral type of positional nystagmus is far more frequent than the central type (about 90% of all incidence). It is characterized by the presence of a latent period, a horizontal and rotatory type of nystagmus, which is easily fatigable, usually disappears in 5-30 seconds, and a non-reproducibility (not re-produceable) within a short period of time. It is usually associated with vertigo.

(2) Central Type.

Central type of positional nystagmus is characterized by the absence of any latent period (it appears as soon as the head is placed in that critical position), continuation (prolonged) of

the nystagmus as long as the head is in that position, and re-appearance of the nystagmus everytime the position is resumed. The associated vertigo is slight or absent.

c) Mechanism of Positional Nystagmus.

Barany (1906-1921) was the first to make a close study of positional nystagmus as an oto-neurological symptom. He was the first to engage in research into positional nystagmus in connection with alcohol[12] (Bergstedt, 1961).

Barany regarded the cause of positional nystagmus to be a pathologically functioning vestibular apparatus, the phenomenon possibly arising in the otolithic organ but he left open the possibility of its arising exclusively inthe central nervous system.

Nylen, from 1922 onwards, has carried out studies on the principles of classification and the clinical aspect of the pathological oto-neurological phenomenon of positional nystagmus (Bergstedt[17], 1961). Nylen[118, 120](1931, 1950) stated "Positional nystagmus must be considered to be one of the surest, most objective and most easily demonstrated signs of disturbance in the human vestibular system." He stated further "The probability is that both the peripheral and the central systems are capable of giving rise to positional nystagmus." In the explanation of the occurrence of positional nystagmus, he said that the phenomenon originates from the semicircular canals via disturbances in the otolithic organ. Nylen (1950) maintained that the pathogenesis of positional nystagmus can be explained uniformly by the assumption of pathologically altered tonus either in the entire vestibular system or in parts of it. Moreover, it can arise in connection with changes in circulation, intracranial pressure, thrombosis, hyper-or hypotonia, arteriosclerosis, etc. With

peripheral affections it is conceivable that positional nystagmus arises through deficient interplay of the otoliths and the cupulae. With central affections, his opinion is that morbid changes disturb the flow of stimuli from the two normal labyrinths to the vestibular centers. Under these conditions a central tonus difference arises which can in turn lead to positional nystagmus. The central point in Nylen's conception is that nystagmus is dependent on disturbed tonus conditions in the vestibular centers.

De Weese[36](1952) pointed out the lack of distinction between positional nystagmus and postural vertigo. He stated "Since we know from experimental work that specific injury to the otolith membrane can produce positional nystagmus, that postural vertigo and positional nystagmus both occur in various dysfunctions of the central nervous system, and that there are no hard and fast rules by which we can interpret postural vertigo and positional nystagmus, I am convinced we are still in a dilemma. I do not believe that the demonstration of positional nystagmus and its accompanying vertigo has been of real value in making a diagnosis. It is my opinion that eliciting postural vertigo and positional nystagmus by positional tests merely indicates that a reaction has been induced in some portion of the stakinetic system. We do not know exactly where or why the reaction is produced".

Lindsay[85](1951) pointed out the different methods of doing postural testing and commented on the head hanging position. He stated that in the head hanging position, one introduces possible factors of tension on the neck muscles and circulatory interference. He added that an optokinetic nystagmus is always visible during the change in position. He demonstrated the frequency of postural vertigo and positional nystagmus in certain diseases of both central and peripheral localization. He also

pointed out that head trauma is a common cause of postural dizziness in the subsequent months. Type I positional nystagmus is the most frequent form and suggests a disturbance in the vicinity of the vestibular nuclei. The pathogenesis of the peripheral type of positional nystagmus in inflammatory conditions may be due to physico-chemical or pharmacological changes in the labyrinthine fluid. Central type of positional nystagmus may be produced by lesions of various parts of the central vestibular system. He stated that the vestibular centers, primarily the nuclei, may be considered as normally in a state of equilibrium. He agreed with Nylen that the presence of a diseased state affecting one side or both sides unequally might result in an abnormal response to a normal peripheral stimulus.

Jongkees[75](1949) carried out experiments in rabbits and concluded that direction-changing positional nystagmus may be caused by damage to the pars inferior labyrinthi, even though the function of the semicircular canals appears normal. He also showed that vertical positional nystagmus may be caused by lesions of the peripheral labyrinth.

Cawthorne[25](1954) gave a synopsis of his views about positional nystagmus. He carried out the practical tests in the sitting and supine positions with the head back, as well as to one or the other side. His finding is that increasing the number of possible positions produces little further information. He does not discuss the causal mechanism of positional nystagmus.

Gerlings[53](1948) reported a series of peripheral labyrinthine diseases with positional nystagmus. He stated that positional nystagmus of peripheral origin occurs and is very important in the diagnosis of labyrinthitis. He added that the most frequent form in his series is the so-called direction-changing positional nystagmus.

Lindsay and Hemenway[86](1956) presented a group of cases with postural vertigo due to unilateral partial loss of vestibular function as a result of vascular accidents. Histopathology revealed occlusion (thrombosis) of the vestibular artery, degeneration of Scarpa's ganglion and superior division of the vestibular nerve (nerve to the utricle, the horizontal canal and the anterior canal). They commented that positional nystagmus, if constant in direction, tends to indicate a lesion in the contralateral side. They pointed out that the assumption that the otoliths were responsible has some objections -- failure to produce nystagmus on physiological stimulation of the otolithic organ.

Citron and Hallpike[31](1956) reported cases of positional nystagmus of the "benign paroxysmal type" and they believed that the pathological basis is to be found in an organic lesion of the otolithic apparatus within the labyrinth (utricle).

Cawthorne and Hallpike[29](1957) described a similar case of benign paroxysmal nystagmus and pathological changes in the utricular macula. However, it was found out later that more pathological changes were present in the central nervous system of this case which could account for the positional nystagmus.

Stahle[150](1956) presented studies on sixteen cases of paroxysmal positional nystagmus. It seemed to him that the otolithic apparatus and its connections were involved.

McNally[100, 102](1944, 1956) stated "If the otoliths are not signaling when the head is at rest or is held steady in one position, any other part of the labyrinth might be equally responsible for a positional nystagmus", in other words, the otolithic organs have no connection with positional nystagmus. He added further that there are no confirmed experi-

ments reported to prove that stimulation of the utricle produces nystagmus.

Aschan, Bergstedt, Drettner, Nylen and Stahle[10](1957) have studied positional nystagmus in man using an electrically driven table. The investigation indicated that in persistent forms of positional nystagmus the position of the head was the determining factor. In transitory and transitional forms, on the other hand, movements of the head also play a significant part in the releasing mechanism. With both forms of nystagmus the authors maintained that it is conceivable that the influence of gravity and linear acceleration on the otolithic organ can give rise to nystagmus.

Morimoto[114](1955) stated "Although the canal organ is not stimulated in an abnormal position, yet in a normal state the otolith system has a controlling effect, like brake, upon the canal system, and if the balance of such an interaction has been broken, then the canal system is spontaneously charged to elicit a nystagmus. In other words, the positional nystagmus should be due to a disharmony of the interaction between the canal and the otolith system, and the degree of imbalance is under the threshold in a normal head position, while it is above the threshold in an abnormal position, so that a nystagmus may appear."

Fernandez, Lindsay and Alzate[47](1960) reported that destruction of the nodulus of the cerebellum in cats resulted in nystagmus in certain positions of the head. Bilateral cocaine anesthesia of the labyrinth caused temporary cessation, while destruction of the labyrinths produced a complete cessation of this positional nystagmus.

Fernandez, Lindsay and Alzate[45, 46](1959) carried out experiments in cats for positional nystagmus. When one of the utricular nerves was sec-

tioned, spontaneous nystagmus to the unaffected ear developed, followed in 24 hours later by direction-changing positional nystagmus and later by a benign paroxysmal type of positional nystagmus. All these disappeared in seven days. Unilateral labyrinthectomy gives rise to spontaneous nystagmus for 3 days and is not followed by postural nystagmus. These observations support the theory that a lesion in the otolithic organ may be accompanied by postural nystagmus.

Bergstedt[17](1961) carried out extensive studies on positional nystagmus in a human centrifuge on normal healthy subjects before and after alcohol intoxication and on subjects with labyrinthine diseases. He found spontaneous or positional nystagmus in 20-30% of the normal healthy subjects. He came to the conclusion that the intensity of the positional nystagmus stands in relation to variations in the strength or direction of the gravitational field. This relation shows so marked a connection and so strong an agreement with known facts of the function of the otolithic organ that this organ can be reasonably considered to be the primary release mechanism for positional nystagmus.

Cawthorne and Hinchcliffe[30](1961) reported 6 cases of subtentorial lesions (metastatic carcinoma) with obvious manifestation of the so-called central type of positional nystagmus.

Spector[152](1961) described the characteristics of the peripheral type and the central type of positional nystagmus. He stated that positional nystagmus is usually overlooked in the routine examination. He added that in head traumas positional nystagmus and postural vertigo may be the only signs of organic pathologic change. Usually, injuries to the head cause the direction-fixed peripheral type of nystagmus and vertigo, not the central type.

Hallpike and Pfaltz[60] (1961) stated that positional nystagmus seemed to follow and might be the only recognizable sequela of a head injury.

Schuknecht[142] (1961) stated that a benign paroxysmal type of positional vertigo is a common symptom in the post-concussion syndrome (head trauma). The attack is usually elicited when the affected ear is undermost in the test position. He tried to explain this by the gravitational effects of the loose statoconia (macula of the utricle) following head blows or injuries on the cupula of the posterior canal ampulla. The hypothesis assumes that the statoconia come to have a close relationship to the cupula of the posterior canal when the head is turned over; the downward movement of the statoconia creates a sharp deflection of the cupula (ampullopetal deflection).

Seiferth[144] (quoted by Nylen) stated that positional nystagmus is constantly present after skull trauma. He added that a direction-changing or irregular types of positional nystagmus, indicating central trauma, are very common. He pointed out that under all circumstances a posture-test should be included in a neurological examination in connection with cranial injury, more especially as the symptom is now usually considered to be an expression of more or less serious damage in the vicinity of the vestibular nuclei in the brain stem, and is seldom thought to depend on a so-called commotio labyrinthidis.

3. Mechanism of Labyrinthine Nystagmus.

Vestibular nystagmus is a to-and-fro movement of the eyes with a slow shifting of the eyes in one direction, the slow phase or slow component of the nystagmus, and a quick return to the center,

the quick phase or fast component of the nystagmus. The slow phase is due quite certainly to stimulation of the vestibular arcs. There are various opinions as to what produces the quick phase. This will be discussed later.

The work of Szentagothai[154](1950) on dogs and cats by employing a modified Ewald's technique to determine the vestibulo-ocular reflex arc is very interesting. He completely agreed with Ewald that the effectiveness of the artificial movement of fluid was found to depend largely on its direction. The current toward the ampulla was effective in the horizontal canal, and the current away from the ampulla was effective in the vertical canals. The current in the opposite direction had no results in most cases.

His findings were :

a. Posterior semicircular duct. An artificial current away from the ampulla yielded contractive response of the superior oblique muscle (action of the muscle is to depress and abduct the eyeball) on the ipsilateral eye and of the inferior rectus on the contralateral eye. Inhibition could occur in several muscles — but most frequently in the superior rectus and inferior oblique muscles on both sides.

b. Anterior semicircular duct. Currents away from the ampulla elicit contractions of ipsilateral superior rectus muscle and the contralateral inferior oblique muscle (action of this muscle is to elevate and adduct the eyeball). Inhibition could occur in several muscles, most frequently in the inferior rectus and superior oblique muscles on both sides.

c. Horizontal semicircular duct. The response to currents towards the ampulla was a contraction of the contralateral lateral rectus

and of the ipsilateral medial rectus. Inhibition was most frequently found in the respective antagonist.

d. Response after transection of the medial (posterior) longitudinal fasciculus. The posterior longitudinal fasciculus was transected in order to destroy the three-neuron connections between the labyrinth and extra-ocular muscles. The reticular substance remained intact. After complete transection of the posterior longitudinal fasciculus, contractive responses of these muscles were not produced; inhibitory responses were less affected or did not change at all.

e. Responses after large transverse lesions of the pons, leaving the posterior longitudinal fasciculus intact. By this procedure, the typical contractive responses to the flow of endolymph could always be elicited. The reciprocal inhibition of the antagonist generally disappeared.

These results point out to the fact that the typical contractive responses to appropriate currents are mainly produced by way of the posterior or medial longitudinal fasciculus and that no other vestibulo-ocular pathways are needed to bring them forth. All other responses seem to be abolished; inhibitory ones are especially affected by transection of the reticular pathways.

Lorente de No[90](1933) demonstrated (besides this three-neuron vestibulo-ocular arc) numerous arcs, most of them crossed through the reticular formation, by way of which the impulses from any labyrinthine receptor may be brought into connection with any one of the extra-ocular muscles.

Spiegel and Sommer[148](1944) explained the conjugate deviation of both eyes when each labyrinth is stimulated by cold and hot water by

the crossed and the uncrossed fibers of the medial or posterior longitudinal fasciculus. With cold water the uncrossed fibers will be stimulated, and with hot water the crossed fibers will be stimulated.

Ordinarily, the direction of the nystagmus is usually designated by its fast component. This is due to the fact that the quick phase of the nystagmus is the one most readily detectable clinically. Actually, the quick phase of the nystagmus is not, strictly speaking, labyrinthine.

Spiegel and Price[147](1939) put forth several theories in an attempt to localize the origin of the fast component of the nystagmus. They worked on cats to prove the theory.

a. The cerebral theory. This theory assumes that the central nervous system above the midbrain is the origin of the fast component. The cerebral theory is refuted by the failure of the elimination of the prosencephalon and the diencephalon to prevent the appearance of nystagmus on labyrinthine stimulation.

b. Theories assuming that the origin of the rhythm is in parts of the vestibulo-ocular reflex arc. There are numerous theories in this group:

   (1) The proprioceptor theory : This assumes that the rhythmic reaction is due to proprioceptive impulses from the ocular muscles. This theory is refuted by the production of nystagmus when the proprioceptors have been paralyzed by the injection of procaine hydrochloride into the ocular muscles.

   (2) The ocular muscle nuclei theory : This locates the origin of the rhythm in the nuclei of the motor nerves to the ocular muscles. This theory is refuted by the failure of the internal rectus muscle, innervated by the opposite third (oculomotor) nucleus, to contract rhythmically on labyrinthine stimulation,

when only one oculomotor nucleus rema ins connected with the vestibular nuclei. This shows that the mutual connections of the two oculomotor nuclei are unable to produce the rhythm.

(3) The labyrinthine theory : This attempts to localize the origin of the rhythm in the labyrinth. This theory is also refuted by the failure of elimination of both labyrinths to prevent nystagmus of central origin.

(4) The reticular substance theory : This localize the origin of the rhythm in the substantia reticularis. This theory is refuted because destruction of the reticular substance fails to prevent nystagmus on labyrinthine stimulations.

(5) The vestibular nuclei theory. This attempts to localize the rhythm in the vestibular nuclei. Since all other possibilities are excluded, it remains that the vestibular nuclei are the most possible origin of the fast component of the nystagmus.

B. Labyrinthine Falling Reactions and Past Pointing.

Falling usually accompanies labyrinthine nystagmus, which is associated with pronounced vertigo. The direction of falling in peripheral labyrinthine nystagmus is opposite to the direction of the nystagmus, i.e., it is in the same direction as the slow component, and changes direction when the position of the head is changed. This is a compensatory reaction to labyrinthine stimulation. This is also true with past pointing which is always in the direction of the slow phase of the nystagmus. Physiologically, the labyrinth is stimulated by movement and normally these compensatory reactions serve a useful purpose in adjusting the individual's balance. When the stimulation of the labyrinth is due to disease or to the caloric test these compensatory reactions are called forth to adjust for an

apparent movement, and the result may actually be upsetting to the individual.

C. Vegetative Symptoms.

Stimulation of the vestibular apparatus elicits a series of vegetative manifestations — pallor, perspiration (cold sweats), nausea, vomiting, bradycardia or tachycardia and alteration of the blood pressure.

Through the reticular gray formation, connections between the vestibular nuclei (specifically, the medial vestibular nucleus) and the autonomic nervous system and between the former and the motor neurons responsible for the act of vomiting are established. Thus visceral reflexes are elicited by vestibular stimulation (see Fig. 9, Chapter I, page 24).

D. Vertigo.

Vertigo and dizziness are words used to describe a very common symptom. It is sometimes very difficult to interpret what is meant by the patient when he says he is dizzy (McNally and Stuart, 1953). It is the result of a conflicting information from the sense organs concerned with equilibrium. This confusion in consciousness gives rise to the sensation of dizziness or vertigo. The sensation of dizziness or vertigo varies in different individuals. Some individuals will feel a rotatory type of dizziness, others a feeling of falling, and still others a feeling of going to faint, a swimming sensation or feeling of weakness. The sensation of turning following labyrinthine stimulation is usually in the direction of the quick phase of the nystagmus (McNally and Stuart[106], 1953).

The definite pathway of vertigo is not yet determined. Several authors were of the opinion that the pathway for vertigo is via the

vermis of the cerebellum before it is projected to the cerebral cortex through the superior cerebellar peduncle. However, Hydman[72](1939) stated "I am not convinced that the pathways mediating vertigo and past pointing course only through the vermis of the cerebellum. I have removed a solid astrocytoma from the vermis in 2 cases in which there are complete or almost total destruction of the vermis and certainly injury to the roof nuclei. The labyrinthine functional tests were normally performed with respect to past pointing and the presence of vertigo, before and after the operation."

Penfield[123](1957) in reviewing over 700 cases of craniotomies under local anesthesia concluded that the pathway of vestibular sensations makes a detour from the thalamus out to the cortex where the vestibular area is next to the auditory area in the superior temporal convolution of both sides. He quoted Gerebtzoff that the vestibulocerebral pathway passed upwards in the medial or posterior longitudinal fasciculus of the ipsilateral side.

Cahpter III

CLINICAL METHODS OF TESTING THE VESTIBULAR LABYRINTH

A. Rotation Test.

Rotation test is one of the first labyrinthine tests. This was systematized and popularized as a clinical test by Barany. He designed a special chair for carrying out the test. This rotating chair is so designed that it can be stopped suddenly. The positive acceleration is usually disregarded and the subject's after rotation reaction is observed. The head of the subject is placed in different positions depending on which of the semicircular canals are to be stimulated. The subject's eyes should be closed during the rotation to prevent any optokinetic or optomotor effects. The usual speed of the rotation is ten turns in twenty seconds. The objection to this test is that individual labyrinth cannot be properly evaluated.

B. Cupulometry.

Van Egmond, Groen and Jongkees[42](1948) developed another method of rotation test which they termed cupulometry. They objected to Barany's rotation test for two reasons : (1) Barany's rotation test applies too severe a stimulus to the labyrinth, and (2) the stimulation is too complex. These authors advocated the use of slower rotation starting at a subthreshold level of stimulation and gradually increasing the rate of rotation but never over 90 degrees per second. During the test, extraneous stimuli are excluded as much as possible. These authors believed that by using minimal stimuli they are able to study the actual deflections of the cupula. The after-turning sensations of the subject and the after-nystagmus were recorded separately on a graph, which they called a cupulogram.

C. Electrical Stimulation -- Galvanic Test.

If a galvanic current flows transversely through the skull, the electrodes being placed on the mastoid processes or in the external auditory meatuses (binaural stimulation), there appears, under a current from 1 to 2 milliamperes, rolling of the eyeballs in the direction of the anode, and under a current of from 2 to 4 milliamperes, horizontal-rotatory nystagmus with the fast component in the direction of the cathode. On reversal of the direction of the current, the direction of the nystagmus is also reversed. When the current stops, the nystagmus may continue in the same direction as during the flow of current, or it may beat in the opposite direction. On monaural stimulation -- with the stimulating electrode on the mastoid process, the diffuse electrode anywhere on the body -- the threshold values ma y be much higher, up to 10 milliamperes. The nystagmus beats toward the side of the stimulated ear if the cathode is applied to the mastoid process, and away from this side on anodic stimulation.

Mechanism of Galvanic Stimulation. There are three possibilities : the galvanic current may be acting upon (1) the labyrinth, (2) the central nervous system, or (3) the Scarpa's ganglion of the vestibular nerve. Different authors have observed that on galvanic stimulation of the labyrinth, there is no deflection of the cupula. Bilateral sectioning of the eighth nerves prevents the galvanic reaction in man, therefore, the galvanic current is not acting on the central nervous system. Thus, it implies that the galvanic current acts on the peripheral vestibular neuron -- Scarpa's ganglion[148].

D. Fistula Test.

Fistula test is applied clinically to determine any presence of erosion through the bony wall of the labyrinth in any suppurating

middle ear disease or to determine the patency of an artificial labyrinthine fistula after a fenestration operation. Fistula test is carried out with the Siegel's speculum or with the Politzer bulb and olive tip. When air is forced into the middle ear, and there is a fistula through the bony wall of the labyrinth, the patient will experience a sudden onset of dizziness, associated with nystagmus. This is known as a positive fistula test. If the fistula test is negative, it does not mean that there is no fistula present; the fistula may be completely obstructed by cholesteatoma or granulation tissue, or the labyrinth may be dead as a result of the disease process and therefore not reacting.

E. Caloric Test.

Barany developed the caloric test in 1908 through his keen observation in patients after syringing the ears for cerumen. These patients developed dizziness and nystagmus. After much investigation and consideration, he came to the conclusion that the labyrinth is stimulated by a difference of temperature between the body and the water being used, either hot or cold. He applied the mass irrigation technic (mass caloric test) by employing about 200 cc of water for each irrigation using either cold or hot water. As a rule, nystagmus may be elicited if the temperature of the water is only a few degrees below or above that of the body.

The position of the head is of great importance. If mass irrigation is applied, the patient's head is usually held in the upright position, so that an endolymph flow is produced in the horizontal semicircular canal and also in the vertical semicircular canals, a rotatory-horizontal nystagmus results. If the head is inclined 30° forwards so that the horizontal canals are exactly horizontal, the

the results is a rotatory nystagmus. If the head is inclined 60° backwards, the horizontal canals are now vertical, horizontal nystagmus results. When the head is inclined 90° forward so that the ampulla lies in the inferior position, cold water irrigation is followed by a nystagmus to the same side; i.e., the endolymph flow is now reversed in direction with respect to the ampulla. This nystagmus is best seen in a mirror held in front of the patient's face. This is also the observation of McNally, Stuart, Jamieson, and Gaulton[104](1948).

Unlike the rotation test, the caloric test has the advantage of evaluating the labyrinths individually.

In 1923 Kobrak introduced the idea of using a small quantity of solution just sufficiently above or below body temperature to cause a mild labyrinthine reaction but below the stage of nausea and vomiting. This is also known as Kobrak's minimal caloric test. There followed a host of modifications of Kobrak's minimal caloric test.

McNally and Stuart[106](1953) advocate a very practical minimal caloric test which can be applied conveniently in the office. They advocate the use of 3 cc of ice water (ice water has a constant temperature) introduced into the ear with a Luer's syringe and an 18-20 gauge needle for a period of 20 seconds. The discomfort in this procedure is negligible. The reaction after the stimulation is usually minimal.

Cawthorne, Fitzgerald and Hallpike[27,49](1942) described an alternate hot and cold caloric test for the horizontal canals. The test is carried out alternately with cold water at 30°C (7 degrees below body temperature) and hot water at 44°C (7 degrees above body temperature)

with an interval of 5 minutes between each irrigation. Eight ounces of fluid is delivered through a nozzle of 4 mm. caliber, from a can elevated 2 feet above the patient's head; this permits 8 ounces of water to flood the ear canal in 40 seconds. The duration of the nystagmus is recorded from the time the water starts to flow. The latent period is included in the duration of nystagmus. The patient lies on his back, with the head elevated 30 degrees, to place the horizontal canal in a vertical plane. The examiner can compare the duration of nystagmus by both hot and cold stimulation and can demonstrate directional preponderance (nystagmusbereitschaft).

Glorig and Fowler[54](1947) modified the above test, suggesting that thermos jugs be used as the source of the irrigating fluid and that a thermometer should be inserted in the apparatus near the nozzle so that it might be possible to record more accurately the temperature of the water entering the ear canal.

Cold Air Test. Dundas Grant employed a stream of cold air for the caloric test in cases of dry perforated ear drum. This method is used to prevent reactivation of quiescent suppurative middle ear disease. Dundas Grant's instrument consists of a simple coiled copper tube covered with cloth mesh. One end of this tube is to be inserted into the ear canal, and on the other end a compressed air mechanism is attached. Ethyl chloride is sprayed on the cloth covering and serves to cool the air which passes through the tube.

1. Mechanism of Caloric Nystagmus.

Barany in 1908 analyzed the mechanism of the caloric stimulation of the labyrinth and came to the conclusion that the vestibular response is a result of the change of temperature in the endolymph, which causes a rise or fall of the endolymph depending on whether

the irrigating fluid is warm or cold.

Jongkees[77](1948,b) in reviewing the literature on the mechanism of caloric stimulation agreed with Barany that a convection current (the result of change of temperature of the endolymph) in the endolymph causes the stimulation of the sensory end organ through the deflection of the cupula.

Dohlman's experiments have shown that a considerable amount of the temperature stimulus is used up to overcome the heat resistance of the air and bone surrounding the membranous labyrinth. The labyrinth itself will respond to a change of temperature of as little as 0.1 to 0.2 of a degree. He also found that the latent period of the reaction is decreased according to the difference between the temperature of the stimulus and the temperature of the body. The intensity of the reaction varies with the length of time the stimulus is applied to the labyrinth. Dohlman also found that the less a particular membranous canal is separated by bone from the middle ear, the more it is affected by the temperature change in the external ear canal. The horizontal semicircular canal is most affected, the anterior vertical next, and the posterior vertical canal is least affected by the temperature change (McNally, Stuart, Jamieson and Gaulton, 1948).

Cawthorne and Cobb[26](1954) recorded the temperature changes in the labyrinth induced by caloric stimulation in the canal of the cadaver and of living beings respectively. They found that there is a temperature change of 0.8°C in the horizontal canal when the water in the external ear canal is 7°C above body temperature. When the stimulus is 7°C below body temperature, the mean fall within the semicircular canal was 0.67°C.

Arslan[6](1955) in reviewing the methodology for the stimulation of the vestibular apparatus stated that there are several factors which determine the physical variation of the thermic stimulation of the vestibular apparatus. These factors are : (1) temperature of water; (2) quantity of water; (3) velocity of irrigation; (4) site of tympanic surface on which the jet of water is directed; (5) size and pneumatization of the bony bridge between the tympanum and the external wall of the lateral semicircular canal; and (6) velocity of the blood stream. Not all these factors have the same modifying power in eliciting a physical variation in the stimulation process, but all play an important role. There are factors which one can modify as to their values, like temperature, quantity of water, velocity of irrigation, and site of tympanic surface ; however, there are other factors which one is not able to modify and for which one must attempt to find the way to minimize their power of influence on the mechanism of the stimulation. He added further that the temperature is transmitted by bony tissue six times faster than by air, and this fact explains the importance of the thickness and pneumatization of the bony bridge between the tympanic annulus and the lateral canal. The airfilled middle ear cavity has therefore very little importance.

2. Laws of Interpretation of the Direction of Nystagmus.

The two "Ewald's Laws" have been described in Chapter I. The interpretation of the clinical findings following labyrinthine stimulation can be based upon these rules.

3. Directional Preponderance.

Directional preponderance (nystagmusbereitschaft) is a term used to designate a phenomenon occurring after induced alternate hot and

cold caloric stimulations of the labyrinths, whereby the right-beating nystagmus or the left-beating nystagmus is very much prolonged. If the right-beating nystagmus is prolonged, it is known as a directional preponderance to the right; and if the left-beating nystagmus is prolonged, it is known as a directional preponderance to the left.

This phenomenon was described by Dusser de Barenne and de Kleyn in 1923. They termed this "nystagmusbereitschaft".

Fitzgerald and Hallpike[49](1942) called this phenomenon, directional preponderance, which was found in a series of temporal lobe lesions. The directional preponderance with optic fixation is to the side of the lesion. They used only the duration of the nystagmus (40 seconds difference) as the criterion for the directional preponderance. Cawthorne, Fitzgerald and Hallpike[27, 28](1942) reported that directional preponderance occurred in 21 per cent of 100 cases of Meniere's syndrome. They attributed this to an utricular lesion (paresis); the directional preponderance being opposite to the diseased utricle. In labyrinthectomy cases, the directional preponderance observed is to the side of the healthy labyrinth.

Jongkees[77](1948) examined a series of 125 normal subjects and reported 17% of this series had directional preponderance to one or the other side. He used 20% difference in duration of the nystagmus as the criterion for directional preponderance.

McNally, Stuart, McKercher and Lockhart[105](1949) did not find directional preponderance of any diagnostic significance in a series of 200 cases of vertigo.

Kirsten and Preber[81](1954) employed encephalographic studies and caloric tests in 68 cases with various cerebral lesions. Directional

preponderance was found in 29 cases. They came to the conclusion that directional ponderance has no localizing value in a cerebral lesion.

Hamersma[63](1957) employed electronystagmographic studies in a series of normal subjects. He found directional preponderance in 55% of the subjects. He applied the 20% rule of Jongkees in the interpretation of the records.

Stahle[151](1958) employing electronystagmographic studies found directional preponderance in 29 out of 35 cases of peripheral labyrinthine diseases (Meniere's disease, vestibular neuronitis, etc.). Lesions of the central vestibular system likewise showed directional preponderance. He used 60 seconds difference in duration as a criterion for the directional preponderance. He stated that duration of the nystagmus alone could not be used to evaluate directional preponderance in peripheral labyrinthine lesions; however, it was adequate for the evaluation of directional preponderance of central origin.

Sandberg and Zilstorff-Pedersen[138](1961) carried out caloric tests in a series of subjects with unilateral temporal lobe lesions. They employed the technique used by Carmichael, Dix and Hallpike (1954) with optic fixation maintained, and confirmed the occurrence of directional preponderance to the side of the lesion.

Carmichael, Dix and Hallpike[24](1961) repeated caloric tests in 10 cases of temporal lobe lesions with optic fixation and without optic fixation. They found that with optic fixation the directional preponderance is to the side of the lesion, whereas without optic fixation the directional preponderance is reversed in direction or absent.

Summary. Directional preponderance is observed in normal subjects, in labyrinthine diseases, and in intracranial lesions, especially supratentorial lesions. The exact mechanism of its production is not yet determined.

4. Habituation.

The term "habituation" is used when a behavioral event tends to disappear when the stimulus is presented repeatedly. The characteristics of the phenomenon are similar to, although not identical to, those of learning. The locus of the process is in the central nervous system, but the neurophysiological bases underlying it are unknown (Fernandez and Schmidt[48], 1962). The term "habituation" is used to indicate the phenomenon of progressive reduction of the nystagmic response (response decline) to either repetitive rotatory or caloric tests.

The investigations of Mowrer[115](1934) and Halstead[61](1935) emphasized three characteristics of the phenomenon : acquisition, retention, and transfer. "Aquisition" stands for the progressive decrement of the nystagmic response. "Retention" is used to indicate the persistence of the habituation by the neural process; without retention, acquisition can not occur. The term "transfer" indicates that a nystagmus habituated in one direction by repetitive stimulation of one ear exhibits similar characteristics when elicited in the same direction by adequate stimulation of the opposite ear. Fernandez and Schmidt[48](1962) stated "The fact that transfer occurs is important for supporting the notion that habituation is not due to adaptation or fatique of the peripheral sense organs, but to some mechanism located in the central nervous system.

Most of the investigators postulated that habituation was a central nervous system phenomenon. The experimental evidence of Halstead, Yacorzynski and Fearing[62](1937) suggested strongly that the process is partially associated with functional changes in the cerebellum.

MaCabe[97](1960) observed that in figure skaters not only the vestibulo-ocular reflex arc is suppressed but also the postural reflexes. The suppression is central and not peripheral.

The work of Henriksson, Kohut and Fernandez[68](1961) on cats demonstrated well all the characteristics of the phenomenon of habituation. They agreed with Mowrer and others that habituation is not due to fatique or adaptation of the vestibular receptors but it is due to a central process, the nature and locus of which were not yet known.

Fernandez and Schmidt[46](1962) studied "habituation" in cats and demonstrated a response decline of all parameters of nystagmus. It is often found that amplitude and duration may or may not be affected. The eye-speed of the slow phase, the frequency and the total number of beats are consistently reduced with repetitive stimulation. The locus of habituation was also studied. Unilateral and bilateral ablation of the temporal lobes did not have any effect on habituation. Hemi-decortication and total ablation of the neocortex did not interfere with habituation. Extensive lesions in the midbrain likewise did not interfere with the acquisition or retention of habituation. Ablation of the nodulus resulted in retarded acquisition of habituation until the animal compensated from the cerebellar deficiency. They concluded that the locus of habituation is in the structures operating the vestibulo-ocular reflex arc, that is, the vestibular nuclei and reticular formation.

Chapter IV

NYSTAGMOGRAPHY

A. Brief Survey of Nystagmographic Methods.

The demonstration of the presence or absence of spontaneous nystagmus is of valuable clinical importance. Induced nystagmus by the different clinical methods of labyrinthine stimulation can give valuable diagnostic data. In all kinds of nystagmus the movements of the eyes may be so rapid that the examiner has difficulty in studying the details of frequency, duration, amplitude and the speed of the slow component. He may therefore be unable to clearly visualize the course of the reaction. These are the reasons behind the development of the different methods of recording nystagmus.

There are three fundamental methods by which eye movements can be objectively recorded (Duke-Elder [41], 1938). These three methods are: (1) mechanical, (2) optical, and (3) electrical.

1. Mechanical Method.

Berlin[18](1891) was the first to make attempts to record nystagmus. He succeeded in tracing a curve on a watch glass by means of a "pin" attached to an ivory shell fitted onto the cornea.

Buys[23](1924) described a pneumographic method for recording the movements of nystagmus. He allowed the cornea of the moving eye to act upon a membrane whereby the pressure in a closed air-filled system increased and decreased with the movement of the eyes. By registering the variation in the pressure in the system he obtained a good measure of the movement of the eyes.

The mechanical method is not being used anymore. It has been supplanted by the other two methods.

2. Optical Method.

Dodge and Cline[37](1901) photographed the light reflex thrown back by the cornea and could thereby record nystagmus.

Dohlman[39](1925) used mechanical and optical principles in combination. With the aid of a mirror attached to the cornea by means of a rubber sucker he was able to record the movement of nystagmus.

Torok and Guillemin[158](1951) described an optical-electrical method. They utilized the difference in the intensity of reflexion from the sclera and iris for registering the movements of nystagmus with a photocell.

3. Electrical Method of Recording Nystagmus -- Electronystagmography.

This method of recording the nystagmus has been gaining ground in the last few years. It is also known as electro-oculography. It is based on the difference of potentials between the cornea and the retina. Corneoretinal potential was discovered by duBois-Reymond in 1849. The cornea is positive in relation to the retina of the eyeball which is negative.

The standing potential, although originating in the eye, is not limited to it but spreads outwards into the adjacent tissues. If the electrodes are placed just nasal and temporal to the palpebral fissure, each electrode is approximately equidistant from the positive corneal pole and the negative posterior pole of the eyeball. The net effect, assuming that the surrounding tissues conduct uniformly, is that no potential from the eye is measured. If the eye should be turned inward, say as far as it can go, the nasal elec-

trode will be much nearer the positive corneal pole than the negative posterior pole. On the other hand, the temporal electrode will be much nearer the negative posterior pole than the positive corneal pole. Hence, the nasal electrode will be positive and the temporal electrode negative. Therefore, the greater the angle of lateral rotation of the eye from its straight-ahead position (primary position of the eye), the greater will be the component of the standing potential which is picked up.

Dewar[35](1877) first observed that eye movement could give changes in potential in human eyes.

Schott[140](1922) with the aid of a string galvanometer first succeeded in recording the difference of potential during eye movements.

Meyers[110](1929) used an electrocardiograph to study the movements of the nystagmus. He regarded the potentials as action potentials from the ocular muscles.

Jacobson[78](1930) was the first to use a vacuum tube amplifier in taking the electro-oculogram. He believed that he was recording the action potentials from the extraocular muscles.

Mowrer, Ruch and Miller[116](1936) were the first to suggest that the potentials recorded in the angles of the eye were related to the corneo-retinal potential difference. They also found that the difference in potential between the angles of the eye increased with the deviation of the eye. They used a D.C. amplifier and a ballistic galvanometer and led off the potentials with silver chloride electrodes in isotonic saline.

Hoffman, Wellman and Carmichael[70](1939) showed that the greatest voltage was recorded when the potentials were led off bitemporally.

Miles[112](1939) measured potential differences with ocular deviations of 30 degrees in either direction and found variations of 0.3 - 2.5 millivolts for each eye. He also found that on induction of emotional states, changes in potentials can occur rapidly and sometimes reach a level of several millivolts because of psychogalvanic reflex.

Glorig and Mauro[55](1950) recorded especially caloric nystagmus by means of an amplifier and a portable electrocardiograph.

Marg[95](1951) gave a detail review of electro-oculography and suggested that it could be used not only for the recording of nystagmus but also in other fields of research -- visual research and neuro-psychiatric research.

Hertz and Riskaer[69](1953) used an A.C. amplifier with three channels, which were connected to three pens of an electro-encephalograph. In this way they recorded the horizontal and vertical movements of both eyes.

Electronystagmography has since been extensively used and widely adopted in different otologic centers in conjunction with posture testing, caloric and rotatory stimulations for various labyrinthine and oto-neurological conditions. It renders a better assessment of the labyrinthine function.

In general, the apparatus used for electronystagmography is either a modified electrocardiograph or electroencephalograph with A.C. or D.C. pre-amplifiers.

B. The Principle of Electronystagmography.

The principle of electronystagmography has been described. It is based on the difference of potential between the cornea and the posterior pole of the eyeball (see Fig. 10). Rotation of the eye in one or the

other direction gives displacement to the potential, and this is recorded via the amplifier and the writing system.

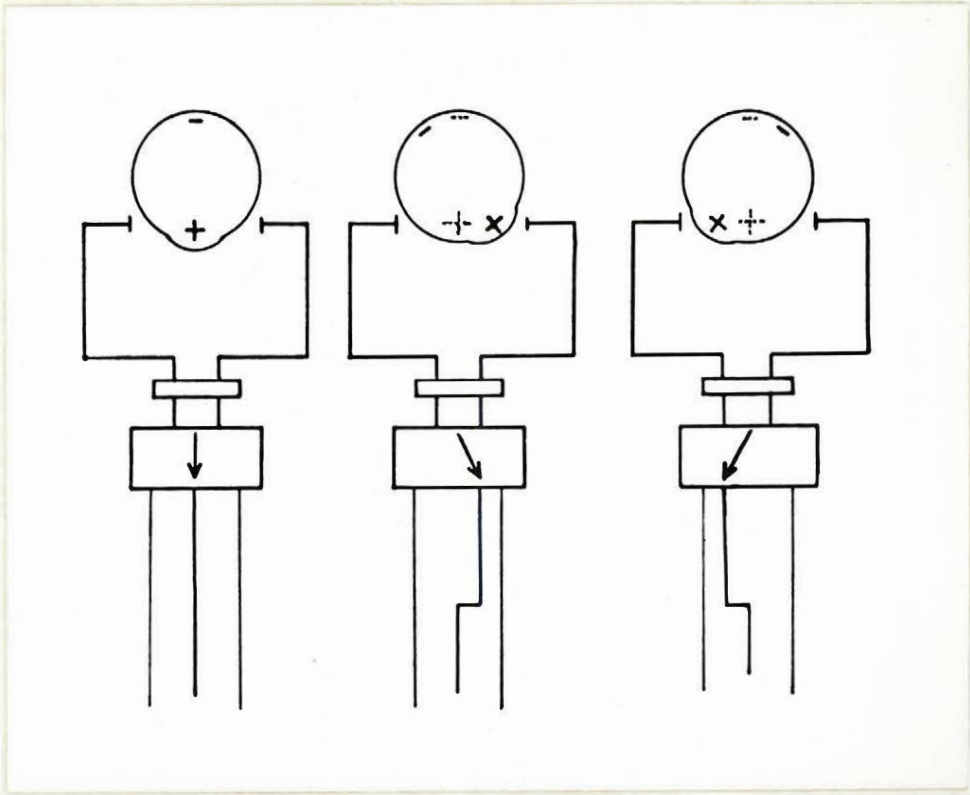


Fig. 10. Diagramatic representation of the difference of corneo-retinal potentials.

C. The Evaluation of the Magnitude of Nystagmus (Duration and Intensity of Nystagmus) Following Caloric Stimulations with Electronystagmography.

The caloric reaction should be assessed or evaluated with regard to its duration and three other characteristics of nystagmus --- number of beats, total amplitude of the nystagmus and maximum eye-speed in the slow phase of nystagmus (maximum intensity). The latter three characteristics of the reaction are grouped as "intensity". Maximum intensity refers only to the maximum eye-speed of the slow phase of nystagmus.

1. Duration of Nystagmus.

The duration of the nystagmus is the conventional method of evaluating the nystagmus. The duration of a response may be determined either by simple observation of the nystagmus, with or without Frenzel's spectacles, or by nystagmography. The duration of a reaction is influenced by fixation. In electronystagmography, which permits recording of nystagmus in darkness or with the subject's eyes closed, the durations obtained are greater than conventional method (Stahle[151], 1958). Fitzgerald and Hallpike[49], who used simple observation, reported a mean duration of about 120 seconds for the cold-water reaction. Henriksson[67](1956) reported a duration of about 155 seconds in nystagmography with open eyes in a dark room. Stahle[149](1956) found the mean duration of 177 seconds if the recording was done with the subject's eyes shut.

Mittermaier[113] (quoted by Stahle) found that the duration varied only to a minor degree with temperature variations between 17°C and 47°C, whereas the number of beats and the total amplitude clearly reflected the magnitude of the stimulus. Aschan[7] (1955) demonstrated that the speed of the slow phase of the nystagmus is considerably more sensitive than is the duration to temperature variations. Henriksson[67](1956) syringed mastoid cavities after radical operations. The duration of the resulting nystagmus was the same as that elicited from the normal side, but the maximum eye speed was appreciably greater.

Mittermaier[113] found in a few cases that the duration gave no indication of a labyrinthine lesion which was primarily reflected in the total number of beats and the total amplitude. Similar

findings were reported by Aschan, Bergstedt and Stahle[9](1956).

The inflexibility of the duration of nystagmus to temperature variations was attributed by Henriksson[67] to the time taken for the blood flow through the temporal bone to restore the temperature to normal after calorization.

2. The Total Number of Beats.

The total number of beats of nystagmus can easily be determined by nystagmography. However, it can also be determined, though with difficulty, by visual assessment. The total number of beats per unit of time is known as the frequency of the nystagmus. Jongkees[76](1949) stated that frequency is too variable to be of significance in the evaluation of the intensity of nystagmus. Torok[157](1961) measured the frequency of the nystagmus during a 10-second period at its maximal response, which he termed the culmination phenomenon, to assess the vestibular sensitivity. This culmination phenomenon varied only slightly in normal subjects; however, it might vary extremely under pathologic conditions. An electronic device was constructed by Torok[159](1962) to compute the frequency. He called it a frequency meter.

3. The Total Amplitude of Nystagmus.

The total amplitude of the nystagmus is the sum of all the amplitudes of the fast component of each nystagmus. It is expressed in degrees of eyeball rotation[151]. The total amplitude, however, is extremely variable; normally it varies between 1000° and 4500°, and most of the clinical cases fall within these limits (Stahle[151], 1958). Mittermaier et al[113] considered the total amplitude to be the best indicator of labyrinthine function.

He preferred to express the magnitude of the reaction by the total amplitude and the total number of beats. Aschan et al[9] also found the total amplitude quite variable.

4. The Maximum Eye-speed of the Slow Phase of Nystagmus (Maximal Intensity).

Buys (1924) and Dohlman (1925) showed that the speed of the eye in the slow phase of nystagmus is a direct expression of the cupular deviation and may be taken as a measure of the labyrinthine function (Henriksson[66], 1955). Henriksson (1955) devised an electrical derivation method for the computation or analysis of the maximal speed of the slow phase of the nystagmus.

Stahle[149](1956) stated that the eye-speed varies during the course of a response, and in order to establish a satisfactory expression for it, it has been suggested that the maximum eye-speed be employed. This is termed as the "maximal intensity", which denotes the mean eye-speed during a 10-second period at the peak of the reaction. The mean eye-speed of the slow phase is obtained by dividing the sum of the total amplitudes in a 10-second period by the time, and is expressed, taking the calibration into consideration, in degrees per second.

The studies of Stahle[151](1958) in a series of 104 nystagmographic investigations (normal subjects, patients with labyrinthine diseases, and patients with various intracranial lesions) showed that caloric reaction should be assessed with regard to its duration and one of the three intensity factors (number of beats, total amplitude, and maximal intensity). He stated that this is necessary because in peripheral lesions the duration cannot always

be relied upon to reflect diminished labyrinthine function, whereas it is plainly revealed by the intensity (number of beats, total amplitude, and maximal intensity), which is therefore of greater diagnostic significance in such cases. He added that the number of beats, the total amplitude, and the maximal intensity give largely similar results. The investigations can therefore be simplified by estimating only one of these three intensity factors.

D. Clinical Assessment (Electronystagmography) of the Caloric Reaction (Alternate Hot and Cold Labyrinthine Stimulation).

The clinical assessment of the caloric reaction should be based on :

1. Difference of Right/Left Vestibular Sensitivity.

The values of the magnitude of nystagmus evaluated from the duration and the intensity (number of beats, total amplitude and maximal speed of the slow phase) resulting from cold and hot stimulations of the right and the left labyrinths are summed up individually. The difference between the magnitudes of response from the right labyrinth and from the left labyrinth can be obtained, i.e., (right hot / right cold) - (left hot / left cold). The sensitivity of the individual labyrinths can then be properly assessed.

2. Difference between Right-beating Nystagmus and Left-beating Nystagmus.

The magnitudes of response from hot stimulation of the right labyrinth and the cold stimulation of the left labyrinth minus the magnitudes of response from hot stimulation of the left labyrinth and cold stimulation of the right labyrinth, i.e.,

(right hot / left cold) - (left hot / right cold), gives the difference of the right-beating nystagmus and the left-beating nystagmus. This gives a proper evaluation of the presence of a crossed asymmetry of the vestibular sensitivity or directional preponderance.

Stahle[151](1958) stated that caloric directional preponderance is often found in cases with peripheral vestibular diseases, and is usually associated with diminished vestibular sensitivity. It is commonly reflected in both duration and intensity. In cases with central lesions the duration seems to reflect directional preponderance more commonly than does the intensity. He also emphasized that if the electronystagmographic findings are to be of maximal usefulness, they should be correlated with the clinical data.

Chapter V

THE EFFECT OF HEAD INJURY ON VESTIBULAR FUNCTION

Review of Literature

In recent years increasing attention has been devoted to the vestibulo-cochlear symptoms following head injuries, commonly known as post-concussional syndrome. The common symptoms are : dizziness, particularly postural dizziness, hearing loss, and headache.

Rhese[134](1906) and Barany[13](1907) were the first to point out that vestibular disturbances very often occurred after head traumas (quoted by Lange and Kornhuber, 1957). Barany pointed out that a sudden bending backwards or turning of the head might in some post-traumatic patients give rise to nystagmus with vertigo, lasting for 30 seconds. He attributed this to either a peripheral or a central lesion of the vestibular system. However, in 1921, Barany[14] stated that the cause might be due to damage of the otolithic organs, since he was able to demonstrate that it was the position of the head in space and not the actual turning, i.e., a static and not a dynamic effect, that produced the vertigo.

Trotter[160](1924) postulated the presence of diffuse punctate hemorrhages in the central nervous system for the protracted and troublesome post-traumatic symptoms.

Mygind[117](1933) found 47% of head injury cases had vestibular symptoms; these patients were examined within 3 months after the injury. Koch[79](1933) reported 49% of head injury cases with vestibular symptoms.

Seiferth[144](1936) stated that positional nystagmus was constantly present after skull trauma. He added that a direction-changing or

irregular type of positional nystagmus, indicating central trauma, was very common. He pointed out that under all circumstances a posture test should be included in a neurological examination in connection with cranial injury. He attributed the cause to the damage in the vicinity of the vestibular nuclei in the brain stem and not to labyrinthine concussion.

In a reasonably large series of head trauma cases, Alexander and Scholl[3](1938) found that 23% of these cases complained of vestibular disturbance and 15% had hearing impairment. The severity of the symptoms increased with the severity of the injury. They stated that ¾ of these cases fully recovered without any subsequent complaints.

Symmonds[153](1942) attributed the cause of dizziness following head injuries to a defect of vasomotor control resulting from medullary concussion.

Friedman, Brenner and Denny-Brown[51](1945) in a series of 200 cases of head injuries stated that dizziness following head injury was always intermittent in character. Each attack was of variable duration, usually lasting for a few minutes. The severity and frequency of the vertiginous attacks were likewise quite variable. The outstanding precipitating factor was change in posture, whether sudden or otherwise. In this series 51% presented dizziness immediately or shortly after the trauma. In 54% of cases dizziness disappeared within 12 hours after the injury. If the dizziness should persist for longer than 12 hours, there was a high incidence of prolonged dizziness. They added that direct damage to the bony labyrinth by a cranial fracture was an obvious possible cause of prolonged vestibular disorders; however, it was remarkable that when there was clear

evidence of such damage, dizziness was remarkably infrequent. They found little support for the thesis that post-traumatic dizziness was derived from the labyrinthine trauma. However, they expressed the opinion that it was difficult to exclude the possibility of labyrinthine concussion. They stated that labyrinthine concussion should exhibit the characteristics of concussion of other nervous structures, namely, an initial maximal disturbance of function followed by progressive lessening of the disorders. A tilt table was used for the positional testing to exclude vasomotor changes, and they concluded that vasomotor disorder or instability following head trauma was not the cause of dizziness as suggested by others. They came to the conclusion that in cases of prolonged dizziness (more than 2 months) both physical trauma and psychological factors played an important role. There was little evidence to support that post-traumatic dizziness was related to damage to the vestibular end-organs.

Evans and Scheinker[44](1945) did histologic studies of the brain following head trauma. They classified the degrees of closed brain injury into : cerebral concussion, cerebral edema, cerebral contusion, intracranial hematoma, cerebral laceration, massive infarction of the cerebral lobes, and later atrophic changes. They stated that cerebral concussion was a purely physiologic state, not accompanied by demonstrable histological changes, and it was interpreted as a temporary arrest of neuronal function, causing loss of consciousness and interference with brain stem activity. They could not demonstrate any functional vascular changes histologically to account for the concept that "functional vascular changes" were the underlying mechanism for the post-traumatic symptoms.

Jasper, Kershman and Elvidge[73](1945) stressed the point that electroencephalography was a sensitive indicator of the existence of damaged nervous tissue following injury to the head. The degree of abnormalities shown by the electroencephalogram varied according to the severity of the injury. In milder forms of injury, there might be minimal electroencephalographic changes or normal tracings. The abnormalities might be generalized or focal. Electroencephalography might also serve to localize the site of injury to the cerebral tissue. They emphasized that electroencephalography should be done within 24-48 hours after the injury, because the electroencephalogram might return to normal within 24 hours. Jasper and Penfield[74](1943) did repeat electroencephalography in a series of 86 cases of chronic epileptic patients following head injuries and showed that in 90 % of these cases the abnormalities were not altered.

Schuknecht[141](1950) reported 17 cases of hearing loss following head injuries. He divided the cases into 3 groups : (1) hearing loss with longitudinal fracture of the petrous temporal bone, (2) hearing loss with transverse fracture of the petrous temporal bone, and (3) hearing loss without any roentgenological evidence of fracture. In the longitudinal fracture of the petrous temporal bone, the hearing loss is a combined conductive hearing loss and neuro-sensory (perceptive) hearing loss. The conductive element was attributed to injury to the middle ear structures which was usually reversible either completely or partially. In transverse fracture of the petrous temporal bone, a total hearing loss and total loss of vestibular function were observed, if the fracture line extended through the labyrinth. In the 3rd group, the hearing loss was neuro-sensory type and was most severe for the high tones, particularly the 4000 cps frequency.

He added that this type of hearing loss was frequently seen in the ear opposite to the side of the petrous temporal bone fracture.

Lindsay[85](1951) pointed out that head trauma was recognized as a common cause of dizziness in subsequent months. The dizziness was characteristically postural and varied in severity. The most frequent type of positional nystagmus elicited by the postural tests was the direction-changing nystagmus which suggested a disturbance in the vicinity of the vestibular nuclei.

Gordon[56](1954) stressed the occurrence of the so-called benign paroxysmal type of positional nystagmus and vertigo following head injuries. He associated this to the damage of the otolithic organs.

Andersen, Japsen and Kristiansen[4](1954) in a series of 64 patients with head injuries (employing the Fitzgerald-Hallpike technique of caloric test) found that 40% of these patients showed directional preponderance to the affected side of injury. They stated "Directional preponderance has been previously described by many investigators as a sign of frequent occurrence in truamatic brain lesions, and a great importance is attached to this sign as a manifestation of an organic brain lesion." They added further that it was claimed that unilateral cortical lesions were accompanied by ipsilateral directional preponderance, whereas the direction of preponderance in bulbar lesions was to the opposite side. However, their investigation seemed to confirm the former assumption but could not prove or disprove the latter view.

Aschan[8](1955) referred to the sequelae of head injuries as encephalopathia traumatica (traumatic encephalopathy). According to him the diagnostic problem in these cases was to determine whether the patient's complaints were of organic or of psychogenic origin ,

especially when insurance assessment of the condition was concerned. In a series of 102 cases of head injuries and another series of non-traumatic psychiatric cases, he did complete oto-neurological studies which included audiograms, postural tests, cupulograms and caloric examinations (using the Fitzgerald-Hallpike method) with electro-nystagmographic recordings and electroencephalograms. Generally, the head injured patients were examined at least a month or more after the injury. He found that 50-58% of the head injury cases showed abnormal or pathological electroencephalograms (EEG) and 41-47% of the non-traumatic psychiatric cases showed pathological EEGs. However, 78% of the head injury cases with pathological EEGs gave abnormal vestibular findings, whereas only 21% of the non-traumatic cases showed certain abnormal vestibular findings. In the series of the head injury cases 52% showed abnormal or pathological vestibular findings. The nystagmus present was often of the positional type; this was regarded as pathological. The cochlear symptoms were dependent on the nature of the injury, i.e., whether the temporal bones were affected. He came to the conclusion that the combination of a pathological EEG and a pathological vestibular status should be of diagnostic value in the evaluation of the disease state in encephalopathia traumatica.

Harrison[65](1956) investigated a series of 123 cases of positional nystagmus. In 104 out of the 123 cases, the positional nystagmus was of the benign paroxysmal type (as defined by Dix and Hallpike). In 19 of the 104 cases (18.2%) exhibiting the positional nystagmus of the benign paroxysmal type, there was a clear history of head injury. Among these 19 head injury cases, 7 had fractures of the skull. In another series of 108 head injury cases, he found 15% with positional

nystagmus of the benign paroxysmal type. He observed that the rapid phase of the nystagmus was directed towards the affected ear when the latter was underneath in the postural tests. The nystagmus increased rapidly, associated with vertigo, and died away within a minute with relief of vertigo. On sitting up there might be a burst of nystagmus, opposite in direction to that observed in the supine position. On repetition the nystagmic response was either reduced or abolished altogether. He concluded that the nystagmus was an irritative phenomenon arising from a lesion of the otolith system, towards which when undermost, nystagmus was directed.

Schuknecht and Davison[143](1956) classified head injury cases with auditory and vestibular symptoms into : (1) longitudinal fracture of the temporal bone, (2) transverse fracture of the temporal bone, and (3) labyrinthine concussion. Longitudinal fracture of the temporal bone constitutes 80% of all temporal bone fractures and commonly occurs from blows to the parietal and temporal regions. Characteristically, the fracture line traverses the annulus tympanicus, creating a laceration of the tympanic membrane and bleeding from the ear. Facial weakness or paralysis occurs in less than 25% of cases and usually is temporary. Cerebrospinal otorrhea occurs in some cases. Subluxation of the incudostapedial joint occurs in some cases. Hearing loss is of the conductive type with occasional high-tone perceptive loss. Vertiginous attacks may persist for a period of days or months after the injury.

Transverse fracture of the temporal bone usually results from blows to the occiput. The fracture occurs perpendicular to the long axis of the petrous pyramid and traverses the vestibule of the inner ear, causing extensive destruction of the membranous labyrinth

and usually complete loss of cochlear and vestibular functions. Severe vertigo may persist for a few days and spontaneous nystagmus to the opposite ear may last for months. In about 50% of cases, the facial nerve is lacerated, and the resultant facial palsy sometimes is permanent. Bleeding from the ear is rare; hemotympanum is a frequent finding. Cerebrospinal fluid may continue to fill the middle ear after the blood is absorbed. Some cases of incomplete loss of the vestibular and auditory functions following transverse fracture of the temporal bone are observed.

Schuknecht and Davison described labyrinthine concussion as perceptive deafness and vertigo resulting from head injury without fracture of the bony labyrinthine capsule. They stated that labyrinthine concussion was commonly seen : (1) in the ear of a longitudinal temporal bone fracture, (2) in the ear opposite a temporal bone fracture, and (3) in head injury without any evidence of skull fracture. They added that to produce labyrinthine concussion a head injury must be severe enough to cause loss of consciousness. The hearing loss is severest for the high frequencies and the peak loss is usually at 4000 cps. Loudness recruitment is characteristically present and supports the concept that damage is to the sense organ. The vertiginous attacks are of the postural type, and positional nystagmus may persist for months after the injury. Positional nystagmus following head injury may occur independently of the hearing loss; usually it is characterized by transient attacks of vertigo and nystagmus when certain head positions are assumed. The underlying pathological change responsible for the positional nystagmus is thought to be injury to the otolithic organs.

Animal experiments (cats) were used by Schuknecht and Davison to demonstrate the pathological changes in the labyrinth after head blows.

Preber and Silfverskiold[128](1957) investigated a series of 42 cases of positional vertigo following head injury with complete oto-neurological examination (EEG, skull x-rays, otoscopy, audiometry, posture tests, caloric tests and cupulometry with electronystagmography) and found that 18 cases had paroxysmal type of positional nystagmus. They stated that in one-third of the cases of paroxysmal positional nystagmus both caloric and rotational tests showed a so-called central vestibular tonus difference, which suggested a central origin of the nystagmus.

Wilhagen[165](1960) in a series of 60 cases of post-traumatic head injury showed that 30% of these cases had vestibular symptoms. All of these latter cases showed pathological or abnormal vestibular findings. About 50% showed central tonus difference (central disturbance of the vestibular system) which was suggestive of a central origin for the vestibular symptoms.

Lange and Kornhuber[82](1961) stated that positional nystagmus and postural vertigo were the usual symptoms following head trauma. In the acute phase of trauma, often the central type of positional nystagmus was found; however, this did not last long. Usually, there was complete recovery. They explained the central type of positional nystagmus in the acute phase of trauma by the contusion or edema of the brain stem. Prolonged appearance of positional nystagmus and postural vertigo was due to a peripheral labyrinthine lesion as a sequela of the trauma. Usually, the position-changing positional

nystagmus and postural vertigo was observed in a fairly large series of head injury cases. The positional nystagmus was transitory. The fast component was directed towards the underneath ear on postural tests, and changed in direction on sitting up position. They found 60-77% of spontaneous or positional nystagmus after trauma to be due to peripheral labyrinthine lesions. Directional preponderance was observed in 17% of cases; however, there were no correlation between the directional preponderance and the side of the brain lesion after the trauma.

Chapter VI

PRESENT INVESTIGATION

THE EFFECT OF HEAD INJURY ON VESTIBULAR FUNCTION

I. Methods.

The methods of investigation include (1) a complete neurological examination, (2) roentgenological examination of the skull, (3) roentgenological examination of the cervical spine in some cases, (4) electroencephalography (some cases), and (5) an oto-neurological examination : an ear, nose and throat examination, audiometric studies, and vestibular tests which include testing for spontaneous nystagmus, postural tests, and caloric tests (modified Fitzgerald-Hallpike method) with electronystagmography, the subject's eyes closed to eliminate inhibiting factors.

A. Description of the Apparatus.

1. Recording Equipment.

A Model 5 Grass four-channel Polygraph Machine with P 1 low level AC pre-amplifiers with a pen writing oscillograph is used for the recording of the nystagmus (see Figs. 11 A and B). The original built-in AC pre-amplifiers have a time constant of 0.8 second. Special capacitors are inserted to give a time constant of 4.5 seconds.

For the nystagmograph ordinary EEG paper is used, and the paper speed is set at 15 mm. per second. Channels 2 and 4 are used for recording the horizontal and the vertical nystagmus respectively. An electronic timef is connected to channel 1 to mark the beginning and the end of the stimulus. An attempt has been made to utilize the channel 3 to record the

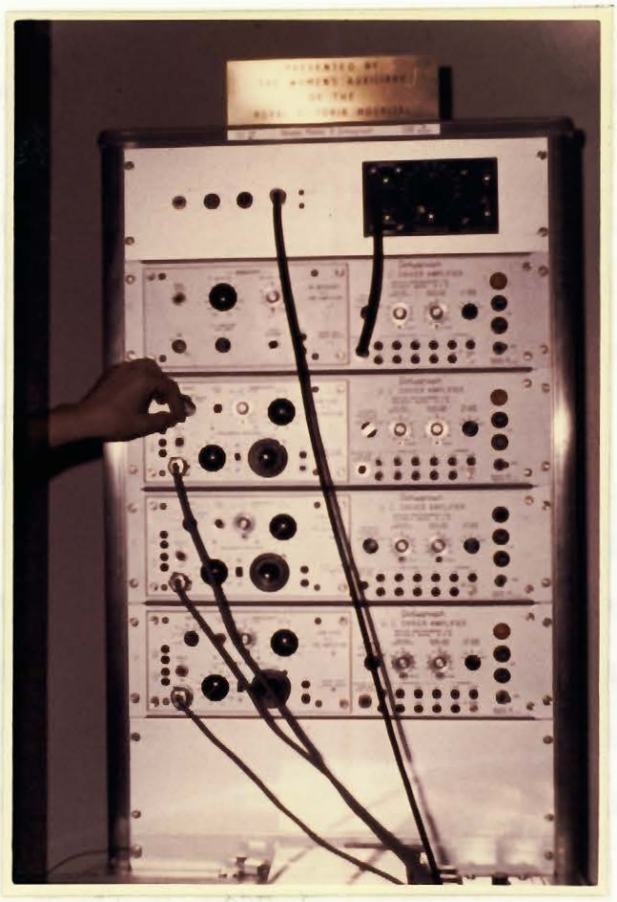


Fig. 11 A. Model 5 Grass Polygraph Machine.

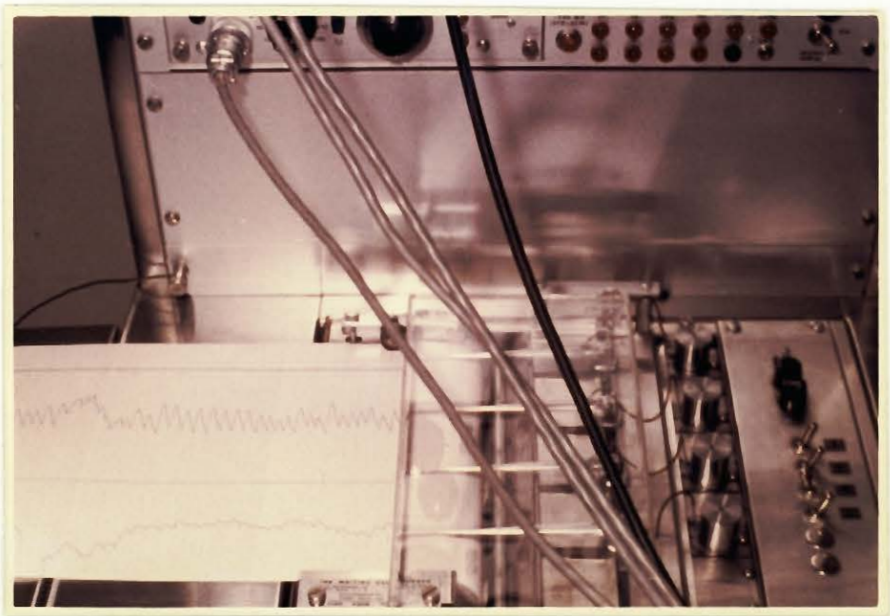


Fig. 11 B. The four channels of the machine. A sample of nystagmograph is demonstrated.

beginning and the end of vertigo; however, this has not been very successful, because most of the subjects fail to press the button when they get dizzy.

The pre-amplifier sensitivity is usually set at 0.05 mv per cm. However, this setting depends on the calibration of the machine . If a 10^{o} deviation of the eyes caused a deflection of the pen more than 2 cms., a lesser sensitivity of 0.1 mv per cm. is needed.

Following the usual routine, the polarity of the oscillograph recording the nystagmus is such that the eye deviation to the right produces a deflection in the upward direction, and the eye deviation to the left in the downward direction(see Figs. 12 A and B). Similarly, in the vertical channel, upward and downward deviations of the eye produce a deflection of the pen upwards and downwards respectively.

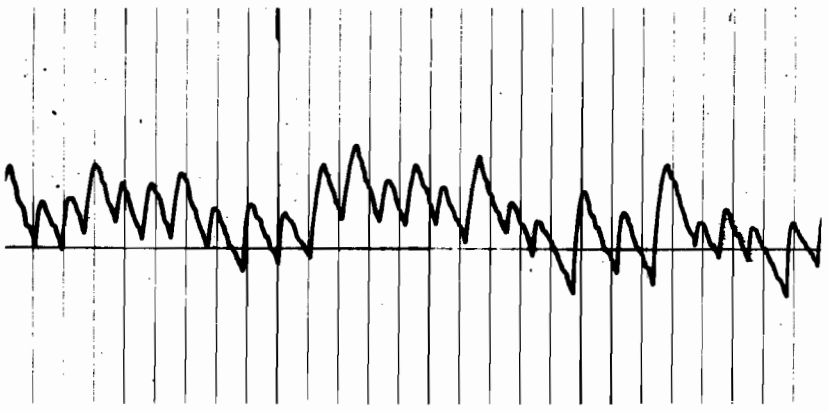


Fig. 12 A. Nystagmograph showing the eye deflection to the right.

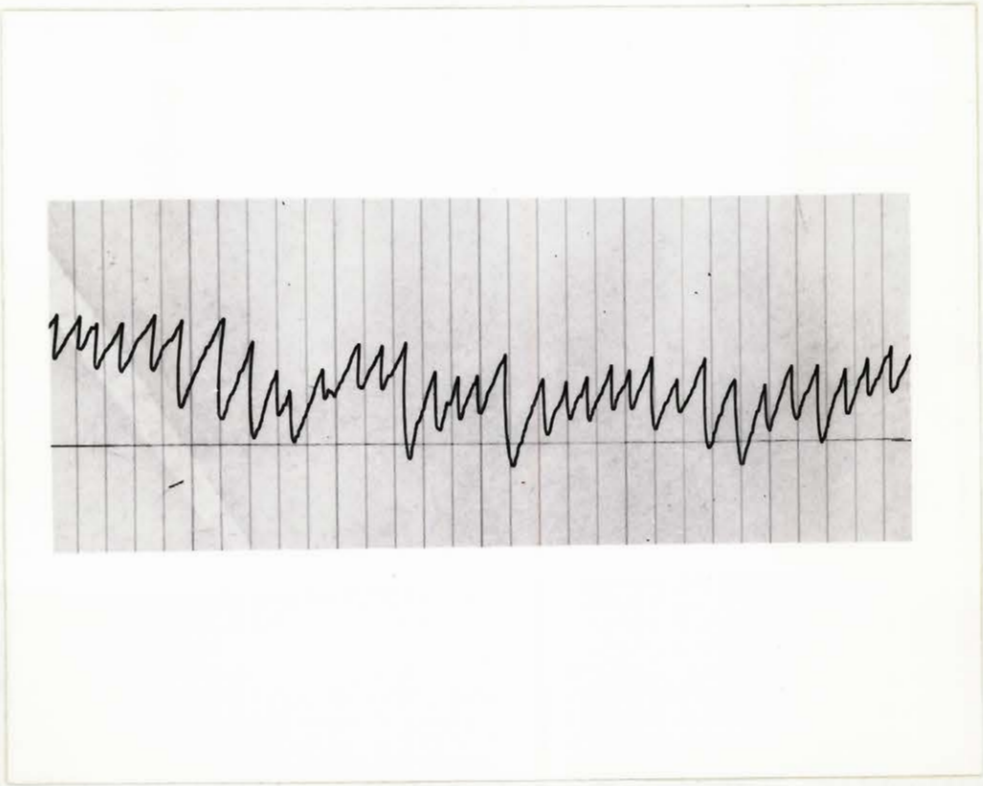


Fig. 12 B. Nystagmograph showing the eye deflection to the left.

2. Calibration of Eye Deviation.

The calibration is carried out by getting the subject to look alternately at a cross-bar with flashing lights, in both horizontal and vertical planes 10° apart, at a distance of 1.5 meters from the eye. Calibration is usually done before the start of the caloric test and at the end of the caloric test, both the right and the left sides of the horizontal plane, and then upward and downward on the vertical plane (see Fig. 13).

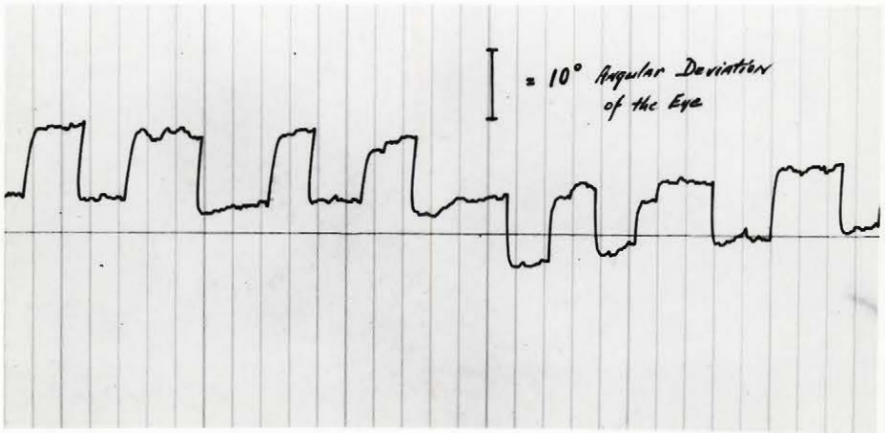


Fig. 13. Calibration of a 10° angular deviation of the eye in the horizontal plane.

3. Apparatus for the Application of Thermic Stimulus.

Two water baths (Haake Thermostat Model F) are used for the application of the thermic stimulus (see Fig. 14). One is set at 30°C and the other at 44°C. Water is continuously circulating

through outlet and inlet rubber tubings. At the end a Y-tube connection is used, and a small rubber tube is connected to the third limb of the Y-tube. At the tip of the rubber tube is fixed a nozzle of such a size that the amount of water ejected by the pump of the water bath is 250 cc in 40 seconds. The thermo-regulator of the water bath is set to insure that the water ejected at the nozzle tip is at the required temperature. This thermostatic control is frequently checked for accuracy. The body temperature of the subject is not taken into account in regulating the thermostat, as it would not make much difference in the overall results of the four tests.

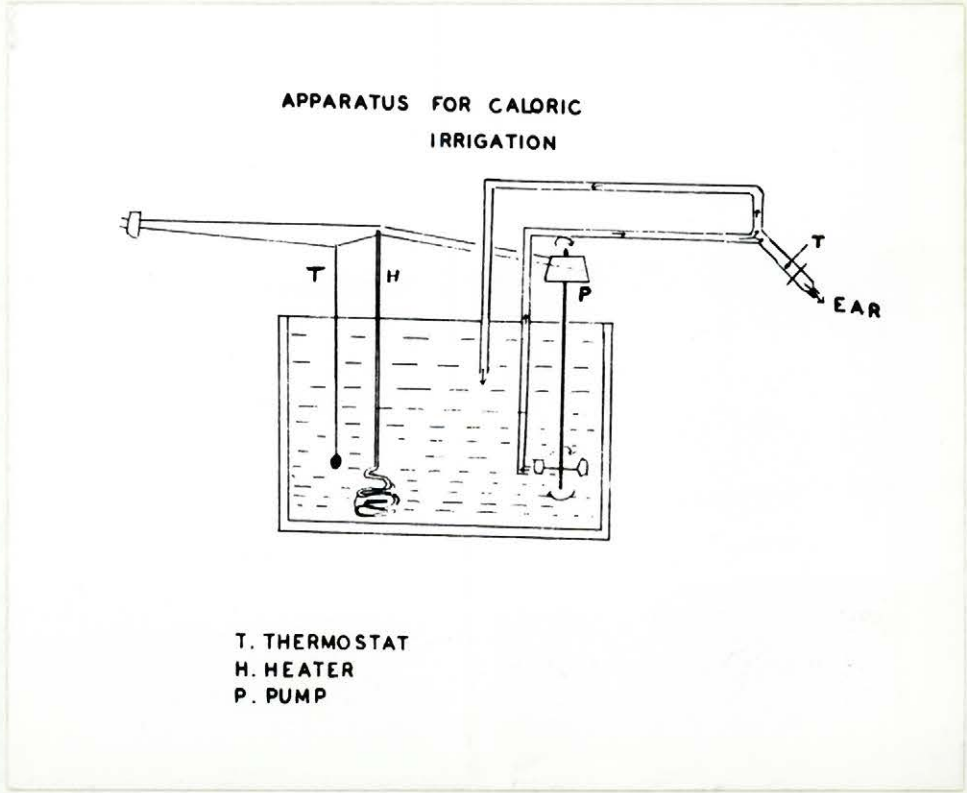


Fig. 14. Diagramatic representation of the water bath.

B. Procedure.

All subjects are tested with their eyes closed in a quiet, semi-darkened room in the following procedures.

1. Application of Electrodes.

The subject is lying on an examining table with the head elevated 30° upwards. Five electrodes are applied on each subject; two of these are bitemporal for the recording of the horizontal deviations of the eyes; two electrodes for the recording of the vertical nystagmus are applied, using the pupil of the left eye as the central point, one above the left eyebrow and one on the left infraorbital region; the 5th electrode is applied at the midline on the frontal region as the ground electrode (see Fig. 15 A and B).

The electrodes used are silver discs of about 1 cm. in diameter and 1.5 mm. in thickness. Before these electrodes are applied the skin is carefully cleaned with soap and water and alcohol to remove the grease. Ordinary ECG electrode jelly is used to make contact with the skin. The electrodes are fixed to the skin with adhesive tape.

These electrodes are connected through tinsel covered wires to the 2nd and 4th channels of the Model 5 Grass Polygraph apparatus for the nystagmograph.

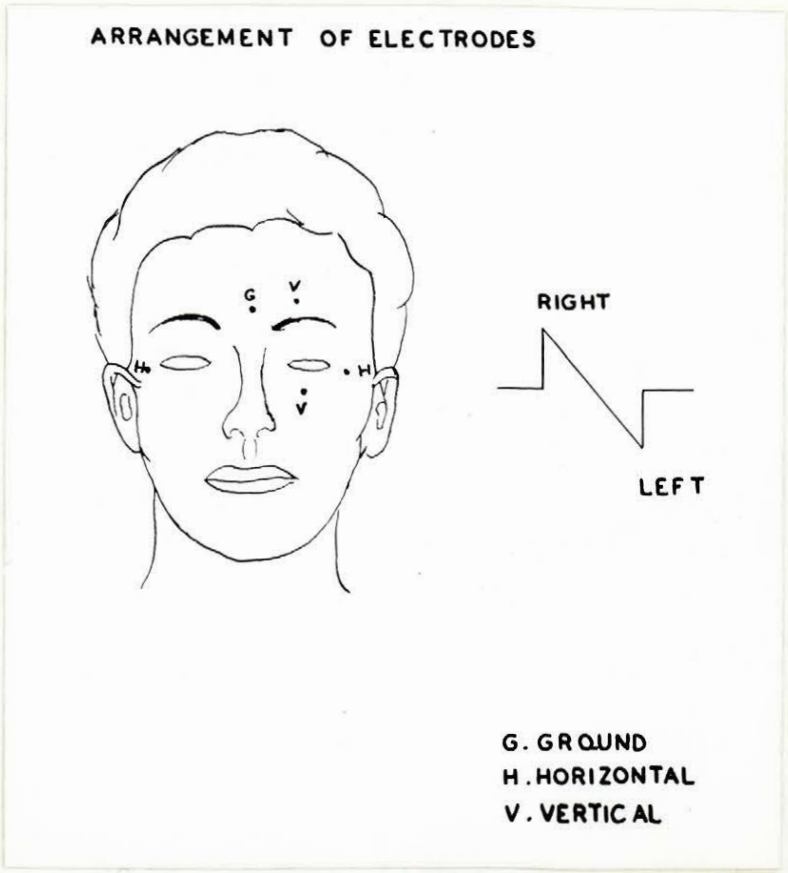


Fig. 15 A. Diagramatic Arrangement of Electrodes. (from Mehra)

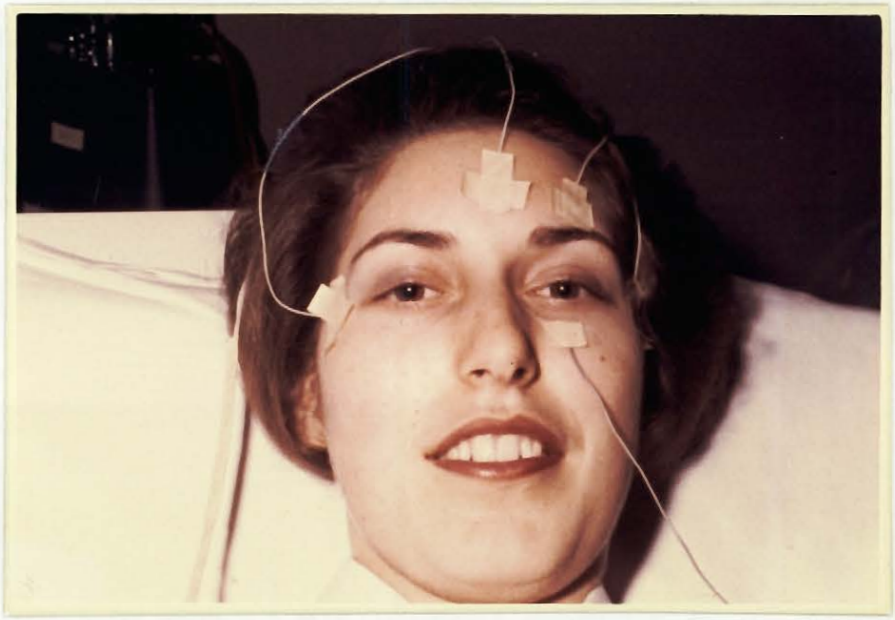


Fig. 15 B. Arrangement of Electrodes.

2. Testing for Spontaneous Nystagmus.

With the subject's eyes closed in the supine position with the head elevated 30° forwards, an initial nystagmograph is recorded for a period of 30-40 seconds. If spontaneous nystagmus were present, it would be recorded not only in the supine position but also in all the other positions being tested for positional nystagmus.

3. Postural Tests.

A modification of Cawthorne's postural tests is used. With the subject's eyes closed and the electrodes attached for the recording of nystagmus, postural tests are carried out in the following manner:

a. Supine position with the head forwards and elevated 30° upwards.
b. Supine position with the head slowly turning to the right.
c. Supine position with the head slowly turning to the left.
d. Sitting up position with the head forwards.
e. Sitting up position with the head slowly turning to the right.
f. Sitting up position with the head slowly turning to the left.
g. Head-hanging position (shoulder of the subject is at the edge of the examining table) with the head forwards.
h. Head-hanging position with the head turning to the right.
i. Head-hanging position with the head turning to the left.

Each position is maintained for about 20-30 seconds and occasionally longer if necessary. Before the procedure, each subject is instructed to tell the examiner the beginning and

the end of any vertigo or dizziness which may be present, and this will be marked on the record.

The main reason for turning the head instead of the subject's body in the lateral position is to prevent the slipping of the electrodes during the procedure. Any vascular disturbance is minimized by slow turning of the head. Subjects with evidence of cervical diseases are excluded from postural tests. Further discussion will be given in the later part of this chapter.

4. Caloric Stimulations.

In the present study, a slight modification of the Fitzgerald-Hallpike method of caloric stimulation is employed. Each subject is stimulated alternately with cold water at 30°C and hot water at 44°C for 40 seconds. The water is ejected by the pump of the water bath (Haake Thermostat Model F) through a nozzle which allows 250 cc to flow through in 40 seconds. The temperature of the water is controlled by the thermo-regulator of the water bath which is constant. The caloric stimulation is carried out alternately in the following order : right cold, left cold, right hot, and left hot. At least 10 minutes is allowed between each stimulation to avoid as much as possible the phenomenon of habituation.

C. Methods of Analysis.

Each subject's nystagmogram is carefully analyzed for the presence of spontaneous nystagmus or positional nystagmus and its characteristics; the caloric reactions are evaluated by the following parameters:

1. Duration of Nystagmus.

The duration of the nystagmus is taken from the beginning

of the stimulation to the last nystagmus beat in the same direction. The duration is expressed in seconds of the caloric reaction.

2. Latent Period of the Reaction.

The latent period of the reaction is the time (in seconds) elapsed after the beginning of the stimulation to the time of onset of the nystagmus.

3. Total Number of Nystagmus Beats.

The total number of beats of nystagmus is counted from the onset of nystagmus to the very last of the nystagmus beat during each stimulation with a hand counter.

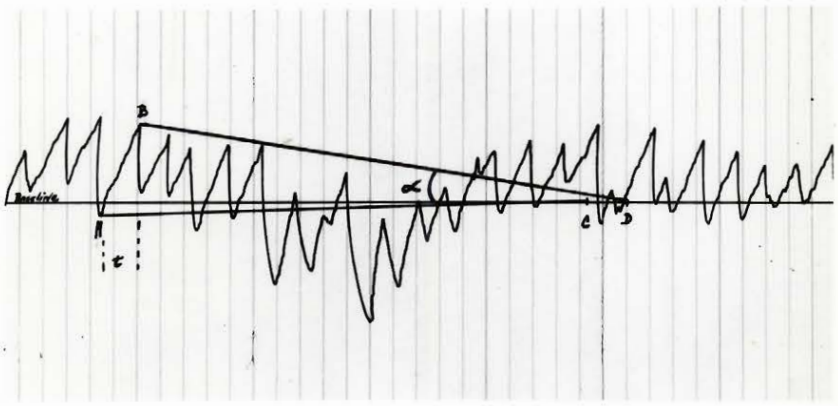
4. Total Amplitude of the Reaction.

The total amplitude of the reaction is the sum of the amplitudes of the fast components of the nystagmus beats. This is computed with the aid of a map measure. The total distance indicated by the map measure is then converted into degrees of eye rotation or deviation, taking the calibration into consideration.

5. Maximal Intensity or Maximum Speed of the Slow Component.

The maximal intensity or the maximum speed of the slow component of the nystagmus is taken from a 10-second period at the peak of the response. Taking the calibration into consideration, the maximal intensity is expressed in degrees of eye deviation per second.

The method of calculation of the maximum speed of the slow component is shown below :



The pen describes an angular movement proportional to the potential of the eye. While moving from A to B, the paper moves from C to D, AC or BD being the length of the pen arm. The rate of change of potential is angle alpha/t, t = time in seconds expressed in mm. of paper movement.

The calibration relates the change of potential to a 10° angular deviation of the eye. The paper speed is negligible compared to the fast component of the nystagmus. Thus, the change of potential can be expressed in angular deviation of the eye.

II. Discussion of the Method and Analysis.

A. Errors of Method.

1. Postural Tests.

We are aware of the various factors affecting the pro-

duction of positional nystagmus and vertigo by turning the head and by the head-hanging position during the postural tests. These factors may be briefly enumerated : tension on the neck muscles (neck reflexes), vascular interference (temporary occlusion of the vertebral artery), optokinetic nystagmus (this usually occurs with fast turning of the head and with eyes opened), degenerative diseases of the cervical spines, particularly C_5-C_7 (the so-called Barre-Lieou syndrome) and possible disturbance of the cervical sympathetics. Optokinetic nystagmus is avoided by doing the postural tests with the eyes closed and by changing the head position slowly. However, we have attempted to minimize these disturbances as much as possible by turning the head or changing the head position slowly. Nevertheless, we can not eliminate all these factors. The only possible solution is doing the postural tests on a posture table. In our present series, there was no clinical evidence of degenerative diseases of the cervical spine. Most of the subjects belonged to the younger age group. At the slightest suspicion of cervical trauma, radiological examination of the neck including the cervical spine was done.

2. Recording of Positional Nystagmus.

The electronystagmographic recording of the positional nystagmus occasionally is cut short in some cases, because of discomfort and the associated vertigo the patients refused to remain in those critical positions. Consequently, in a few cases it was difficult to determine whether the positional nystagmus was sustained or transitory.

B. Errors of Analysis.

1. Post-caloric Dysrhthymia.

Post-caloric dysrhythmia or dysrhythmia post-caloric nystagmus (see Fig. 16) is a phenomenon observed during the procedure of caloric stimulation with electronystagmography by the appearance of fine "irregular" oscillation of the eyes for a certain period of time between intervals of regular rhythm of nystagmus. This phenomenon is observed in normal subjects and in patients with peripheral and/or central disturbances of the vestibular system.

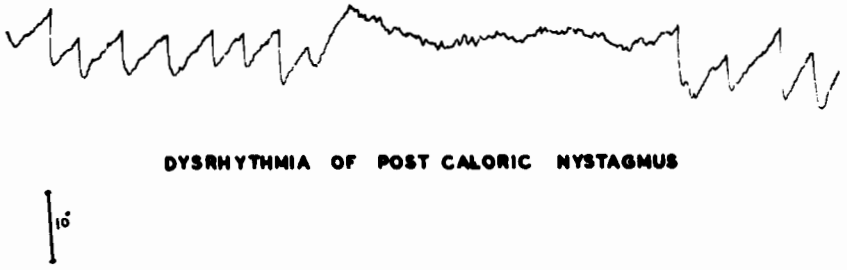


Fig. 16. Post-caloric Dysrhythmia.

Aschan, Bergstedt and Stahle[9] (1956) stated that post-caloric dysrhythmia was often seen in cases of suspected cerebral lesions and rarely in peripheral lesions.

Riesco-MacClure and Stroud[136](1960) attributed this phenomenon to cerebellar lesions which disturbed the cerebello-vestibular interrelationship.

In the present study, post-caloric dysrhythmia was frequently observed both in the healthy normal subjects and in the post-traumatic cases. This was also the observation of Mehra[109](1961) and Wong[167](1962). After observing this phenomenon so frequently both in clinical cases and in the present investigation, it is my personal opinion that this phenomenon may be a manifestation of habituation, because it is frequently manifested towards the latter part of the caloric stimulation, particularly the hot water ($44^{\circ}C$) stimulations.

Lidvall[83](1961) in his study of response decline after repeated calorizations reported that dysrhythmia increased in frequency and in grades as the number of calorizations was increased.

Post-caloric dysrhythmia, especially when it appears over a prolonged period of time and occurs at frequent intervals, interferes or disrupts the final analysis of the electronystagmograph in all the parameters.

2. Eye Rolling.

Eye rolling is occasionally a problem in doing electronystagmography with the eyes closed. It is frequently observed in the normal subjects as well as in the patients with central or peripheral disturbances of the vestibular system. The rolling of the eyes interferes particularly with two parameters of the nystagmus, namely, the amplitude and the maximal intensity of the nystagmus. Occasionally, all the parameters -- the duration of

the nystagmus, the total number of the nystagmus beats, the total amplitude, and the maximal intensity, are affected, especially in cases with constantly marked rolling of the eyes. In some cases the nystagmus is completely abolished. If the rolling of the eyes is towards the direction of the fast component of the nystagmus, the frequency of the nystagmus is observed to be increased, and the amplitude and the maximal intensity of the nystagmus are invariably affected, and vice versa. The etiology of this is not yet established.

3. Muscle Potential.

Muscle potentials are not infrequently recorded in cases of multiple sclerosis and also during the act of swallowing. These muscle potentials may interfere with the regular rhythm of the nystagmus.

4. Vertical Nystagmus.

The evaluation of the vertical nystagmus is rendered very difficult by the blinking of the eyelids. Very often it is extremely difficult to differentiate between the two on the electronystagmogram.

5. Spontaneous Nystagmus.

Spontaneous nystagmus appearing on the electronystagmograph should be carefully analyzed with regard to its intensity -- the amplitude, the frequency and the maximal speed of the slow component -- in order to evaluate properly the magnitudes of the caloric reactions. However, this was not done in the present investigation.

6. Pen Drift.

Pen drifting is a frequent occurrence in electronystagmography,

especially when intense caloric reactions are encountered, in spite of all efforts to eliminate the factors which will prevent a proper contact between the electrodes and the skin of the subject. This pen drifting, if prolonged, interferes to some extent with the proper assessment of the nystagmogram.

III. Materials.

A. Controls.

The control group is composed of 19 normal healthy young adults, most of them are student nurses of the Royal Victoria Hospital. An otoscopic examination, a pure tone audiometric examination, testing for spontaneous nystagmus, postural tests and caloric tests (under the same conditions and procedures previously described) with electronystagmography are performed on these subjects.

B. Patients.

Most of the patients were admitted to the Montreal Neurological Institute after head injuries, and the oto-neurological examination were performed while they were in the hospital. Some of these patients were seen in the Otolaryngology Clinic of the Royal Victoria Hospital presenting vestibular symptoms after head injuries and were submitted to oto-neurological examinations. A total of 18 cases was examined. These cases will be presented individually.

Case No. 1 M.R., 48 years of age, white, single, female, jobless. She was involved in a train accident on May 18, 1962. There was no loss of consciousness. She was confined in a hospital for 9 days. Physical examination re-

vealed some bruises in the neck and the chest; other physical findings were essentially negative. Radiological findings were likewise negative. Two weeks after the accident, she started to have intermittent tinnitus in the right ear and postural vertigo on lying down on her right side. There was no dizziness in any other position. Her hearing was not affected. She was seen in the Otolaryngology Clinic on the 5th of July, 1962. ENT examination was negative. Audiogram revealed slight neuro-sensory loss, 15 db, in the right ear and a sudden drop of hearing, neuro-sensory, at 3000 cps, 4000 cps and 8000 cps in the left ear (see Fig. 17). Electronystagmography revealed spontaneous nystagmus to the

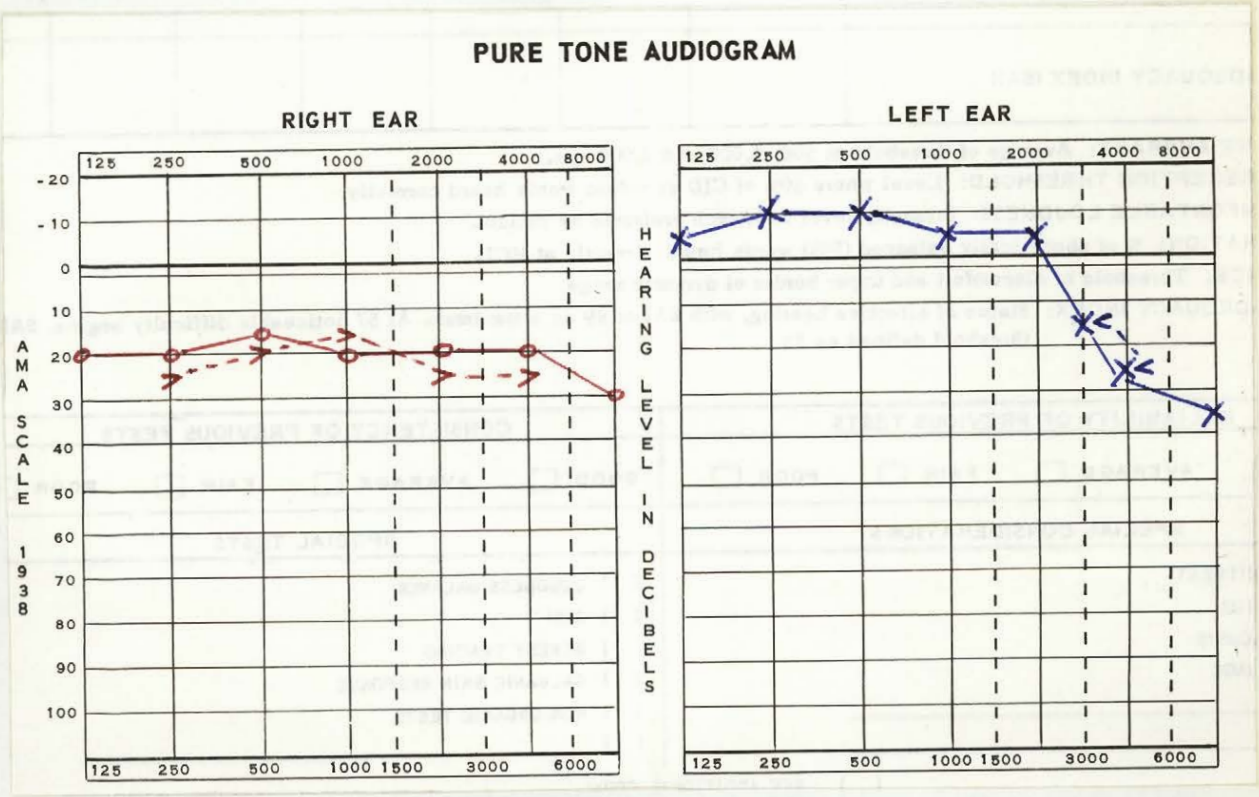


Fig. 17

left and a questionable sustained positional nystagmus, direction-fixed, to the left in the head-hanging position, associated with marked dizziness; alternate cold and hot caloric tests showed marked directional preponderance to the left, otherwise both labyrinths appeared intact.

Skull x-ray and x-ray of the internal auditory meati were negative. A complete neurological examination was negative. Serology was likewise negative.

Case No. 2. R. L., 15 years old, white, single, male , student. While riding a bicycle on the 23rd of August, 1960, he struck a light post and fell. Following the trauma he was unconscious for an undetermined time. He was admitted to the Montreal Neurological Institute 2½ hours after the accident. When he was seen, he was conscious but confused and disoriented as to time, place and person. He could not remember the accident. He vomited profusely several times. Complete neurological examinations were otherwise negative. Skull x-ray was negative. Conservative management for closed head injury was given. He was discharged on the 26th of August, 1960 without any neurological deficit except for amnesia for the time of the accident. He first noted hearing loss in the right ear when he could not hear the telephone on the 29th of August, 1960 (6 days after the trauma). He also had intermittent tinnitus in the right ear and was dizzy intermittently. He was first seen in the Otolaryngology Clinic on the 1st of September, 1960. He denied having mumps, measles, scarlet fever, etc. There was no history of administration of any ototoxic agent. ENT examinations revealed a slightly retracted right tympanic membrane, otherwise the findings were not remarkable. Minimal caloric tests with 3 cc of ice water elicited normal response from the left ear and no response from the right ear. Audiogram revealed normal hearing in the left ear and no hearing in the right ear (see Fig. 18). Upon recommendation from school, he came back to the Otolaryngology Clinic on the 29th of November, 1962, i.e., more than 2 years and 3 months after the accident, for

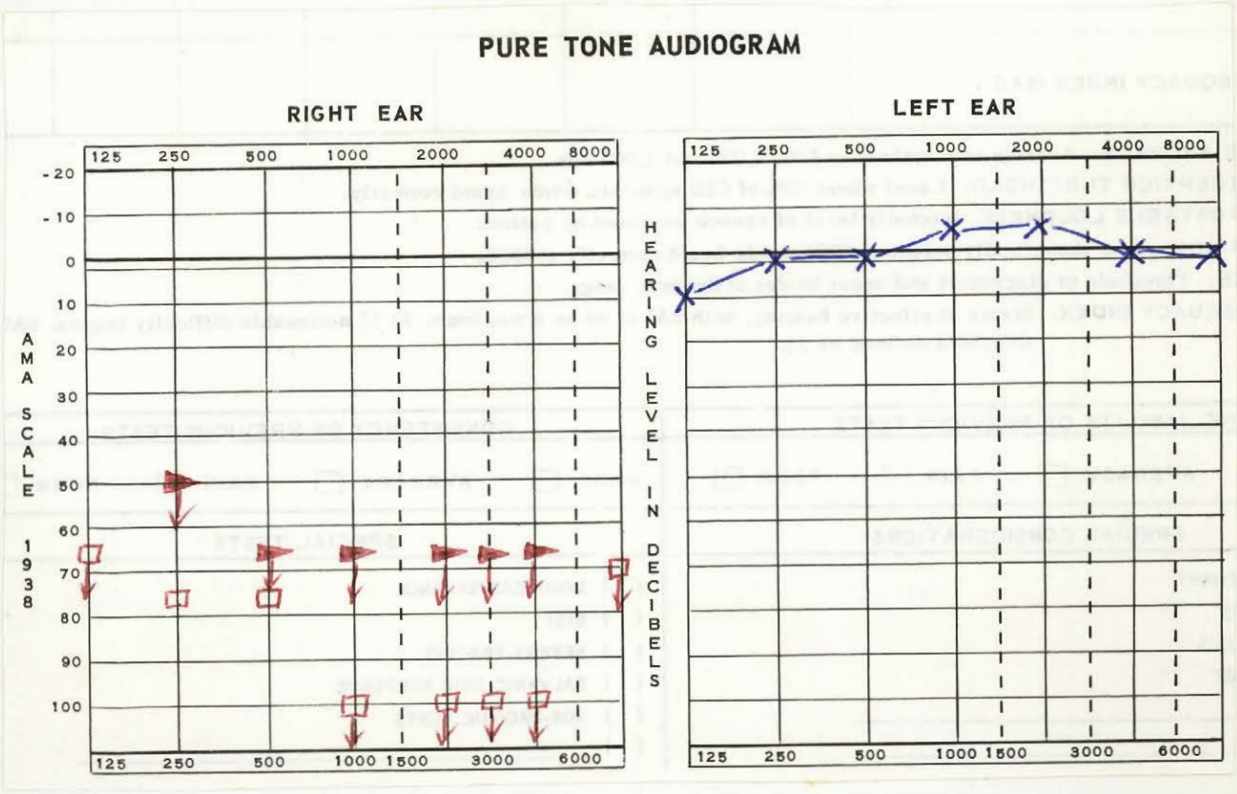


Fig. 18.

re-evaluation of his hearing. ENT examinations showed practically the same findings. Ice water caloric tests were repeated; both labyrinths responded fairly well. Audiogram revealed still profound neuro-sensory hearing loss in the right ear and normal hearing in the left ear; however, the hearing in the right ear appeared to have improved (see Fig. 19). Electronystagmography showed moderate spontaneous, direction-fixed, nystagmus to the left, moderate to marked directional preponderance to the left (see Tables IV - X); both labyrinths were otherwise intact.

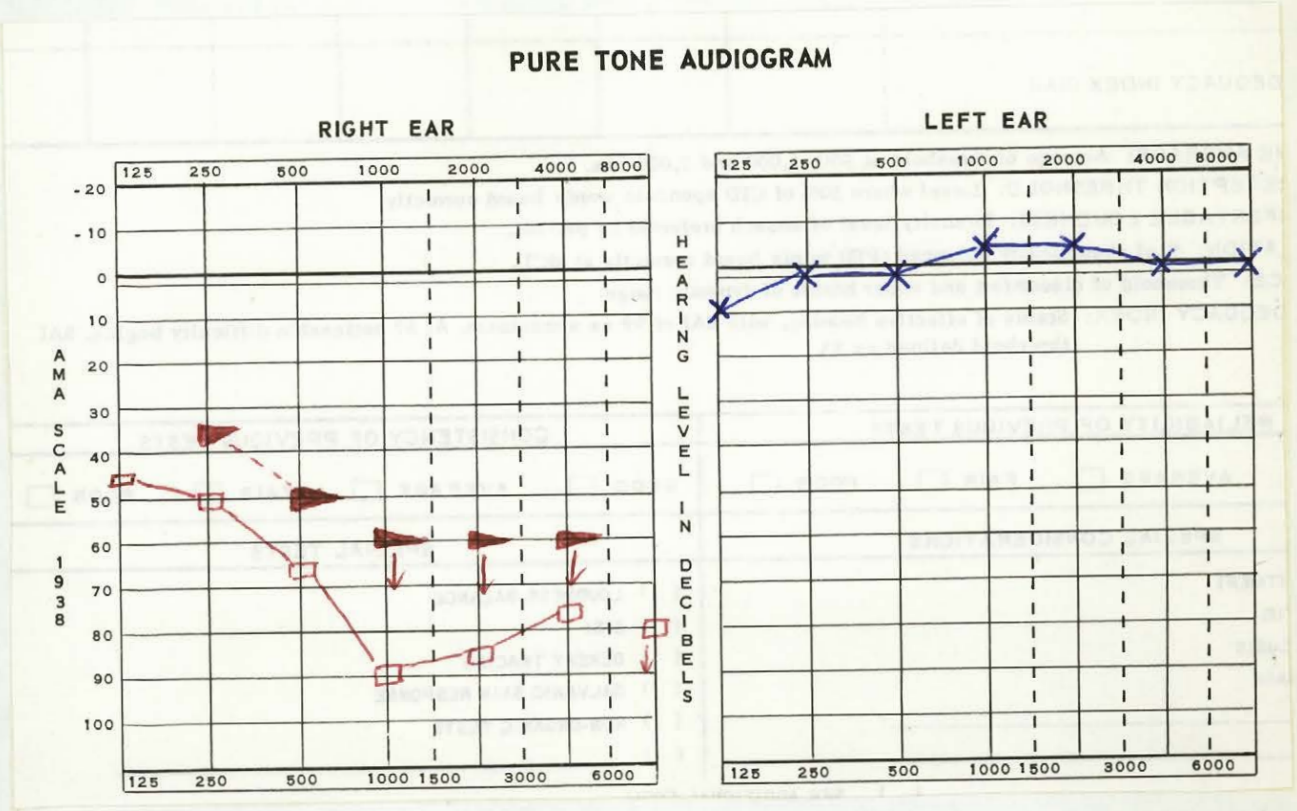


Fig. 19

Case No. 3. L.B., 29 years old, single, white, male, rigger. He fell from a height of 15 feet on the 31th of May, 1962 and landed on the right side of his body. He was unconscious for 5 minutes, and after that he was confused and restless. His speech was incoherent and irrelevant. He was admitted to the Montreal Neurological Institute. On admission, his vital signs were stable. Physical examination revealed bruises and contusion over the right fronto-parietal area, hematoma around the right orbit, slight bleeding from the nose and left ear, right hemotympanum, fracture of the right humerus and dislocation of the right shoulder joint. Skull x-ray revealed extensive fracture running through the right orbit, right frontal sinus, right fronto-parietal and right temporal bones. The fracture appeared to run through the right side of the middle fossa

with clouding of the sphenoid sinus. Chest x-ray revealed multiple fractures of the ribs of the right hemithorax and contusion of the right lung parenchyma. Other neurological findings were normal. An emergency tracheotomy was performed. All other laboratory findings including blood serology were normal. Conservative management was given to his skull fractures and cerebral concussion. On the 7th hospital day, an open reduction for dislocation and fracture of the right humerus was performed. He improved remarkably well and was discharged on the 15th of June, 1962. Since then he has tinnitus, intermittent attacks of dizziness and hearing loss in the right ear. He had no history of ear, nose and throat problems prior to the accident. There was no history of any ototoxic drugs being administered. He came to the Otolaryngology Clinic on the 9th of August, 1962 (more than 2 months after the accident). ENT examinations were not remarkable. Ice water caloric tests showed no response

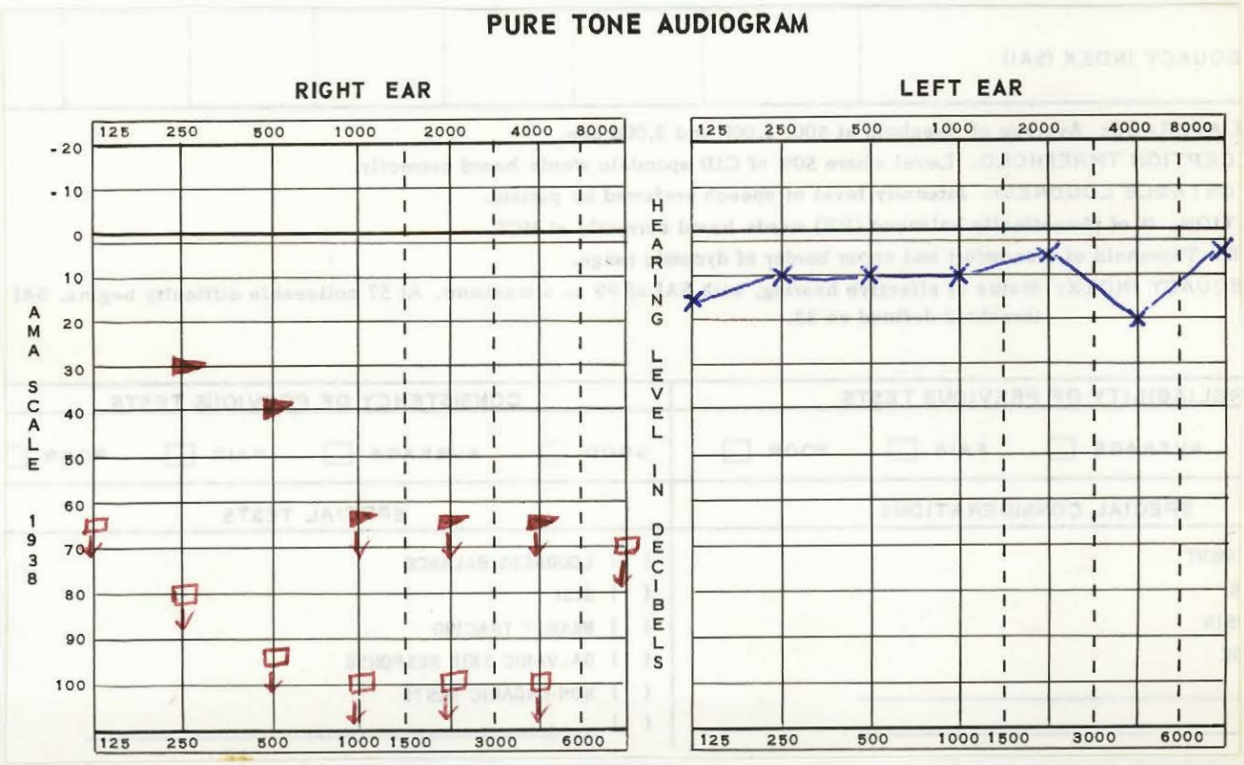


Fig. 20.

from the right labyrinth and fairly normal response from the left labyrinth. Audiogram revealed total hearing loss in the right ear and fairly normal hearing in the left ear (see Fig. 20). Electronystagmography at this time showed marked spontaneous nystagmus to the left; alternate cold and hot caloric stimulations revealed no response from the right labyrinth and fairly normal response from the left labyrinth (see Tables IV - X).

Case No. 4. G.R., 40 years old, single, white, female. She was beaten by a man in 1953 when she was 4½ months pregnant. She received several severe blows in the jaw, which resulted in subsequent deformity of the jaw. She was not unconscious. She had no headache or other abnormal neurological signs, but since then her hearing in both ears gradually deteriorated. She had continuous tinnitus in the left ear. There was no vertigo. She was wearing a hearing aid in her right ear since 1955 which helped her very much. She never had any ear, nose and throat troubles previously. There was no family history of deafness. She had no contagious diseases prior to the onset of the hearing impairment. She never received any ototoxic drugs. She came to the Otolaryngology Clinic on the 4th of October, 1962, complaining of progressive hearing deterioration. ENT examinations were not remarkable. Ice water, 3 cc, caloric tests revealed no response from her left labyrinth and markedly hypoactive response from the right labyrinth. Audiogram revealed total hearing loss in the left ear and severe neuro-sensory hearing loss in the right ear (see Fig. 21). Electronystagmography did not reveal any spontaneous or positional

nystagmus; alternate cold and hot caloric stimulations did not elicit any response from her left labyrinth and marked canal paresis of the right labyrinth (see Tables IV - X). X-ray of the skull, mastoids and internal auditory meati were normal. Blood serology was negative. Neurological examination was negative.

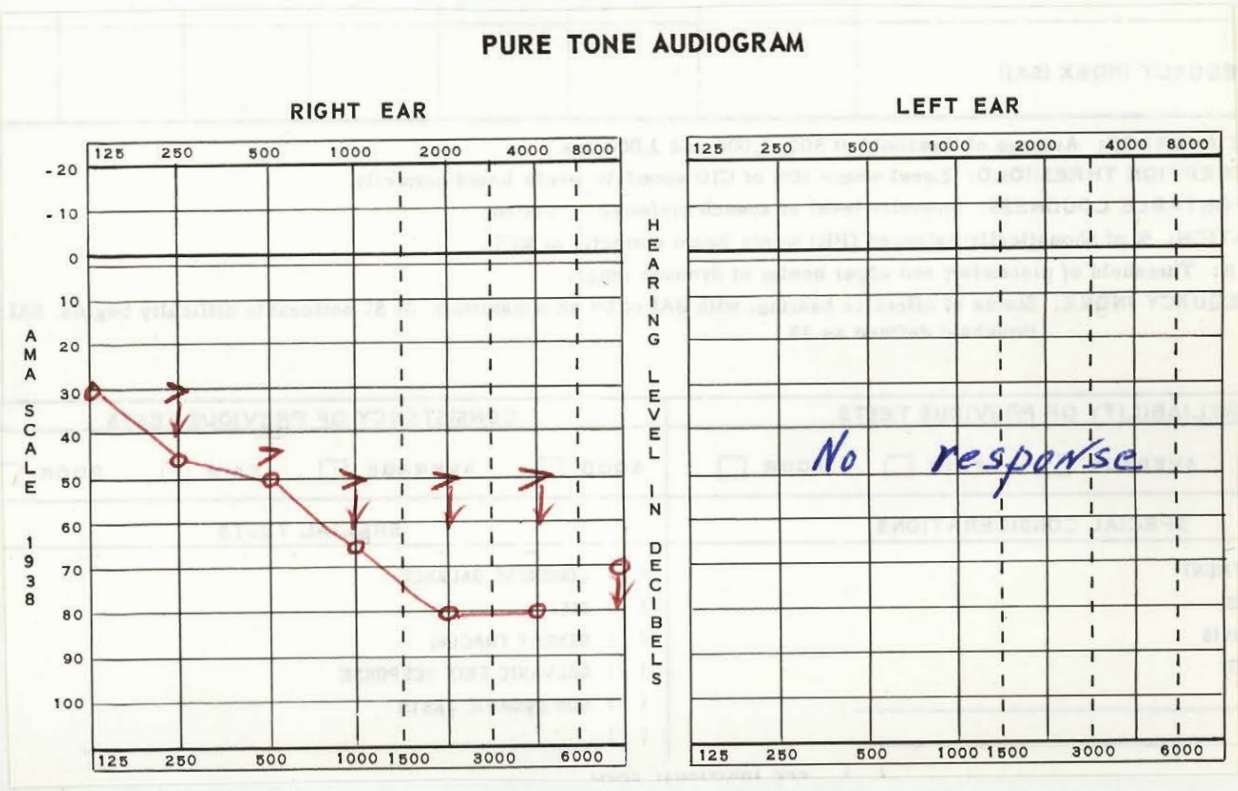


Fig. 21.

Case No. 5. J.G., 19 years old, single, white, male, student. This 19 year-old football player was injured in a foot ball game on the 27th of September, 1962. He was not unconscious but suffered a laceration of the left orbital region. He developed slight headache and dizziness with nausea. Dizziness was worse when he lay on the left lateral position or with the left ear down. He had no tinnitus or hearing loss. There was some tenderness on the left side of the neck. He was admitted to the Montreal Neurological

Institute on the same date. On examination, his vital signs were stable. He had a laceration on the left orbital region with ecchymosis of the left eye. The vision was not impaired. There was slight tenderness at the back of his neck. He had marked spontaneous nystagmus to the right which changed direction on turning to the right lateral position. There was slight tendency to fall to the left. Other neurological findings were normal. X-ray of the skull, cervical spines and chest was not remarkable. Conservative management of the closed head injury was given. Blood serology was negative. Routine laboratory examinations were normal. Otolaryngology was consulted. ENT findings were not remarkable. Audiogram revealed a minimal high tone (1000 cps, 2000 cps, and 4000 cps) neuro-sensory loss in the left ear and a slight drop at 4000 cps in the right ear (see Fig. 22). Electronystagmography

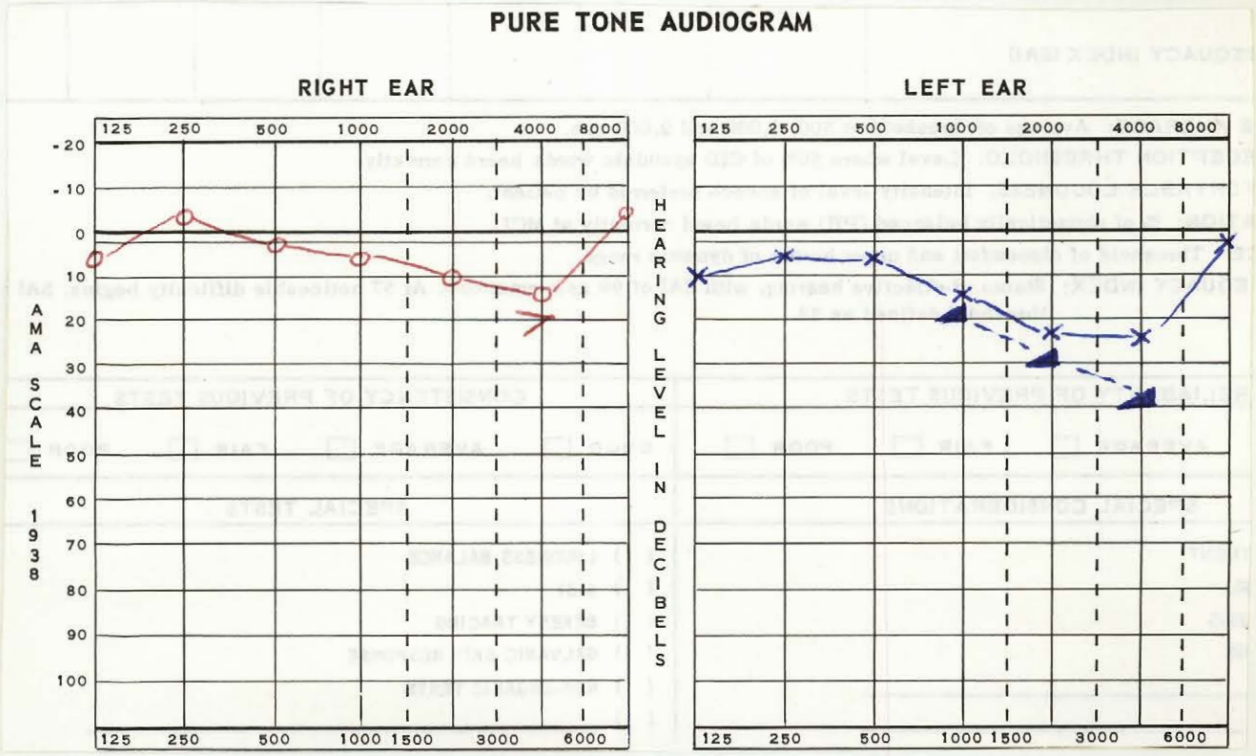


Fig. 22.

revealed marked position-changing, unsustained, positional nystagmus in the supine position and on sitting up position, associated with marked dizziness; alternate cold and hot caloric stimulations revealed normal response from the right labyrinth and hypoactive response from the left labyrinth; marked directional preponderance to the right was demonstrated. The subsequent course in the hospital was uneventful; the spontaneous nystagmus and dizziness gradually subsided. Repeat audiogram did not show any change in the hearing; SISI test was positive for recruitment for the 2000 cps and 4000 cps in the left ear.

Case No. 6. J.M., 22 years old, white, single, female, student. She had a car accident when she was 2 years old, and since then she had lost her hearing in both ears. Prior to the accident, she had good speech. However, her speech deteriorated after the accident. She had continuous tinnitus in the left ear and occasional episodes of tinnitus in the right ear. She never complained of dizziness or disturbance in her balance. She had been attending lip-reading training since 10 years of age. Her speech at present was quite good although it was monotonous and high pitched. She had otherwise no ear, nose and throat problems. She had a deformed, atrophic and paretic left arm and shoulder since the accident. She was healthy otherwise. There was no history of deafness in the family. She never received any ototoxic agent. She came to the Otolaryngology Clinic on November 22, 1962 because of her hearing problem. Physical examination showed a very intelligent, young, healthy, white female with atrophic slightly deformed left upper extremity; other physical findings were not remarkable. ENT exa-

minations were not remarkable. There was no neurological deficit. Her speech was monotonous and high pitched. She had to lip-read otherwise she could not understand. Ice water caloric tests showed minimal response from both labyrinths. Audiogram revealed severe neuro-sensory hearing loss in both ears (see Fig. 23). X-rays of the skull and mastoids were normal. Electronystagmography revealed questionable sustained, direction-fixed positional nystagmus to the left, associated with marked dizziness, in the supine position with the head turning to the left, sitting up position with the head turning to the left and in the head-hanging position; alternate cold and hot caloric stimulations showed profound bilateral canal paresis (see Tables IV - X).

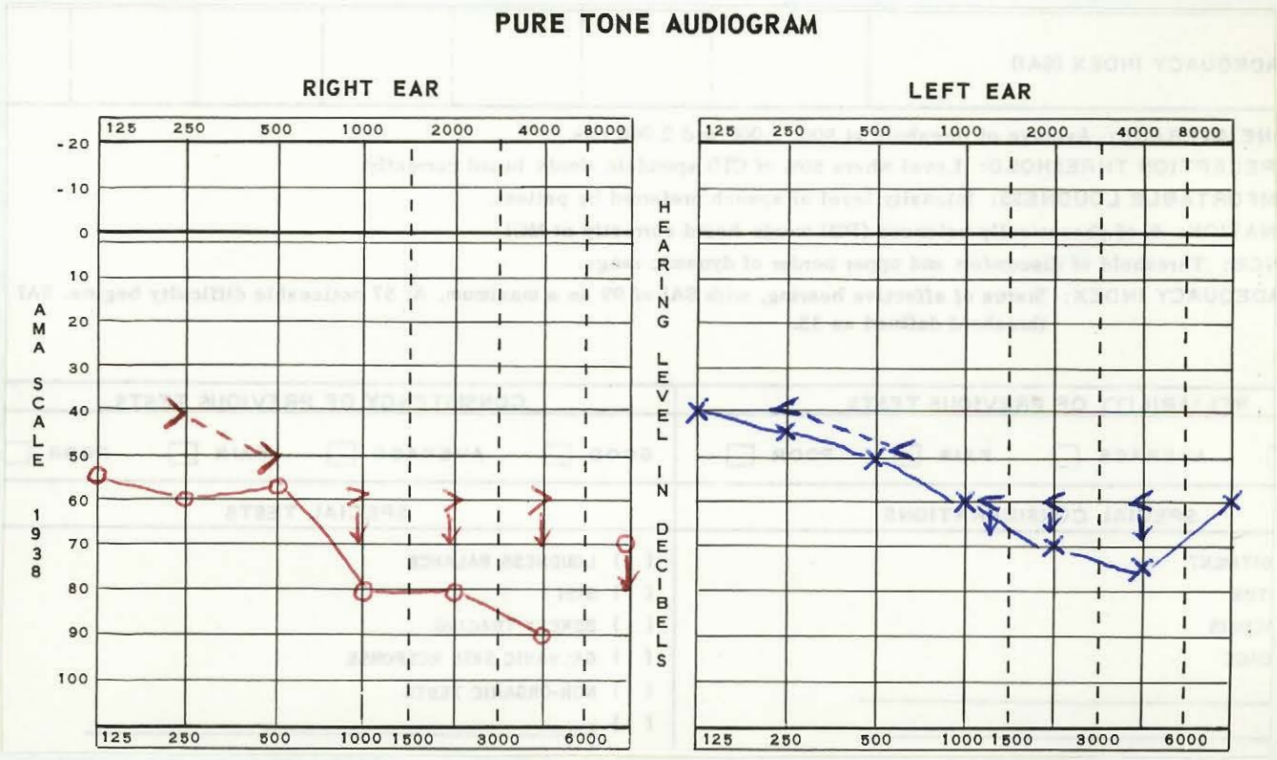


Fig. 23.

Case No. 7. E.G., 32 years old, married, white, male, laborer. This 32 year-old truck driver was working in a chemical fertilizer plant. He was involved in an accident on the 28th of December, 1962. Apparently, a large heap of frozen chemical fertilizer fell on him and buried him up to his neck. He did not lose his consciousness and was able to free himself in about 10 minutes. Following this injury he complained of pain in the chest and pain in the mid-thoracic region of the spine. He was taken to a hospital, and within a few hours he developed difficulty with his speech, dysarthria; he had also difficulty swallowing. He noticed numbness of the left arm which progressed to a complete hemi-anesthesia within 24 hours. He was transferred to Montreal Neurological Institute on the 1st of January, 1963. At that time a left facial weakness was reported in addition to the hemianesthesia. Physical examination revealed : an alert, well oriented, cooperative, 32 year-old male with stable vital signs; subcutaneous emphysema over left antero-lateral chest wall; fractured 7th and 8th ribs; multiple contusions over the anterior and posterior chest wall; tenderness over left side of the neck; left pneumothorax; and slight hematoma over the 9th and 10th T-vertebral bodies. Neurological examination revealed : no evidence of stiff neck; no anosmia; good vision; no diplopia; visual fields were full; EOM were not impaired; few nystagmoid jerks on the left lateral gaze; pupils were equal and reacted to light; left peripheral type of facial weakness; underactivity of the soft palate on the left; deviation of the tongue to the left; diminished strength of the left sterno-mastoid and trapezius muscles; drifting and impairment of the left arm; diminished abdominal reflexes;

downgoing plantars; complete hemianesthesia for touch, pinprick as well as position sense and vibration sense on the left side; and gross cerebellar ataxia of the left arm and to a lesser degree of the left leg. An impression of acute compression injury of the chest with multiple fractured ribs and pneumothorax and multiple brain stem contusions was made.

Skull x-ray did not reveal any evidence of fracture nor signs of intracranial lesion. X-ray of the cervical spine did not show any evidence of fracture or dislocation. X-ray of the thoracic spine and ribs revealed no evidence of fracture or dislocation of the thoracic spine but fracture of the left 6th and 7th ribs was demonstrated. Chest x-ray revealed a pneumothorax localized to the lateral, inferior and medial aspects of the left base with secondary partial collapse of the left lower lobe. Pneumoencephalogram showed no obstruction to cerebro-spinal fluid circulation and no evidence of any space occupying lesion. Electroencephalogram showed abnormality in the right centro-temporo-parietal regions which was consistent with the existence of slight cerebral damage to this area.

Routine hematologic examination, urinalysis, blood chemistry, blood serology and cerebro-spinal fluid examination were normal.

Otolaryngology was consulted. Except for the above findings, ENT findings were not remarkable. Audiogram revealed hearing to be normal in the speech range and a sudden drop of hearing at 4000 cps in the left ear, about 40 db neuro-sensory loss, and a slight drop of about 10 db at 4000 cps in the right ear (see Fig. 24). Electronystagmography revealed spontaneous nystagmus to theleft; alternate cold and hot caloric stimulations showed bilateral hyperactive labyrinths with marked directional preponderance to the left (see Tables IV - X).

A conservative management was given while he was in the hospital. His condition improved remarkably. Dysarthria gradually disappeared; left facial weakness gradually recovered; cerebellar ataxia disappeared; sensation in the face was the first to return. He was discharged on the 27th of January, 1963.

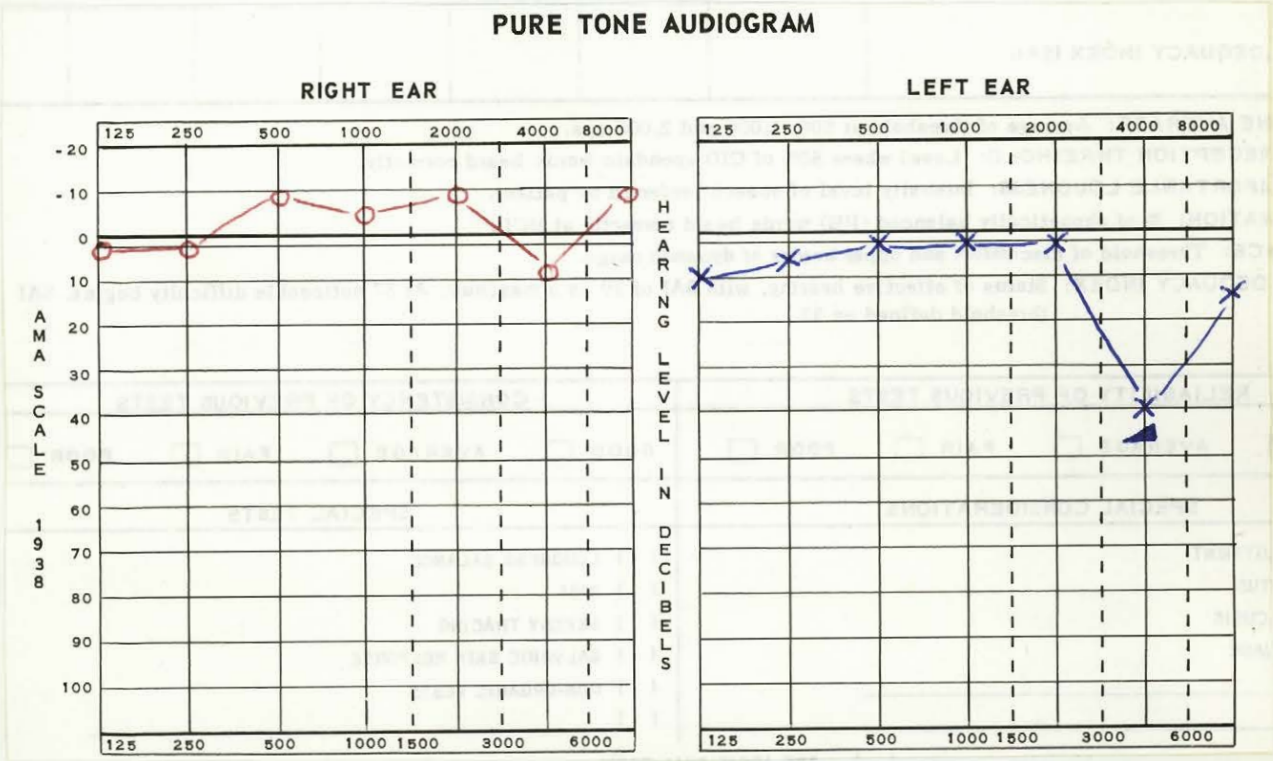


Fig. 24.

Case No. 8. C. R., 20 years old, single, white, male, seaway laborer. On the 2nd of February, 1963, this 20 year-old male, after drinking two bottles of beer in a night club, was struck on the jaw, fell and hit his head on the pavement and was kicked twice in the head. He was unconscious for 5 minutes. On admission to the Royal Victoria Hospital Emergency Department, he was completely amnesic and disoriented as to time and place. He complained of right frontal headache. He had a laceration over the right occipital region which

was sutured. He was admitted to the Montreal Neurological Institute. On admission his vital signs were stable but he was still confused and amnesic. Physical examination was not remarkable. Neurological findings were negative. Skull x-ray was negative. An impression of cerebral concussion was made. Routine hematologic examination, urinalysis and blood serology were normal. Conservative management for head injury was given. ENT examinations were not remarkable. Audiogram revealed normal hearing in both ears. Electronystagmography done on the 4th and 5th of February, 1963 revealed transitory, position-fixed, positional nystagmus to the left in the head-hanging position; alternate cold and hot caloric stimulations showed bilateral hypoactive labyrinths.

Case No. 9. G. R., 23 years old, single, white, male, machine operator. This 23 year-old male was involved in a fight after drinking beer on the 31th of January, 1963. He was hit in the face and fell to the pavement where he was kicked in the head. He was unconscious for full 10 minutes. When he regained consciousness, his sensorium was clouded; he had nausea, vomiting and headache. He was brought to the Royal Victoria Hospital Emergency Department. Physical examination revealed normal and stable vital signs; contusion and abrasion over the right occipital area; blood clots in both nostrils; swollen lips; and a 6 x 6 cms. tender, discolored swelling over the right costo-vertebral angle. Other physical findings were negative. He was admitted to the Montreal Neurological Institute. Complete neurological examination was not remarkable. Skull x-ray revealed a hairline fracture running across the mid-portion of the right parietal bone laterally; there was no evidence of any expanding

intracranial lesion. Chest x-ray and x-ray of the neck were not remarkable. Routine hematologic examination, urinalysis and blood serology were normal. An impression of cerebral concussion with right parietal bone fracture was made. A conservative management for closed head injury was followed. He complained of postural vertigo on sitting up on the following day. ENT examination on the 1st of February, 1963 was not remarkable. Audiogram revealed bilateral normal hearing. Electronystagmography revealed unsustained, position-fixed, positional nystagmus to the right in the sitting up position and in the head-hanging position; alternate cold and hot caloric stimulations showed profound bilateral canal paresis (see Tables IV - X).

Case No. 10. H.K., 36 years old, single, white, male, cleaner. He fell from a steep flight of stairs on the 29th of December, 1962. He was unconscious for approximately one hour. He had been drinking prior to the accident. He was admitted to the Montreal Neurological Institute. When he was seen, he was conscious but confused. His vital signs were normal and stable. He had a right fronto-paristo-temporal scalp hematoma and right periorbital hematoma. He had epistaxis and right hemotympanum. Asymmetry of the face was noted which was attributed to swelling of the right side of his face. Other neurological findings were negative. X-ray of the skull revealed right fronto-temporal comminuted fractures; anteriorly the fracture entered the right frontal sinus and the ethmoid air cells, while posteriorly it entered the right mastoid; the right mastoid air cells, the right frontal, ethmoid, sphenoid and maxillary sinuses were totally filled with blood and/or cerebro-spinal fluid, while the

left paranasal sinuses were partially filled; the left mastoid was clear. X-ray of the cervical spine was normal. X-ray of the right wrist revealed a Colle's fracture. He also had a comminuted fracture of the left 2nd metacarpal bone. Chest x-ray and x-ray of the thoracic spine were normal. An impression of head injury with multiple comminuted skull fracture, a right Colle's fracture and comminuted fracture of left 2nd metacarpal bones was made. Routine hematologic examination, urinalysis and blood serology were normal. Electroencephalogram showed some depression of cortical activity over the left hemisphere posteriorly. Conservative management was given for his head injury. Right peripheral type of facial palsy was noted on the 2nd hospital day. His hearing in the right ear was noted to be down on the 10th hospital day. Otolaryngology was consulted. ENT examination revealed : right hemotympanum; Weber's test was lateralized to the right ear; minimal residual right facial palsy; no evidence of spontaneous nystagmus; nasal findings were not remarkable; other ENT findings were negative. An impression of longitudinal fracture of the right petrous temporal bone with involvement of the right facial nerve was made; the facial palsy has improved considerably. A repeat x-ray of the paranasal sinuses and the mastoids on the 17th of January, 1963 revealed residual mucosal thickening in the paranasal sinuses and in the right mastoid air cells; there was no air-fluid level. Audiogram on the 17th of January, 1963 revealed slight to moderate conductive hearing loss in both ears, worse in the right ear, and a sudden drop of hearing at 4000 cps and 8000 cps of neuro-sensory type in both ears (see Fig. 25). Electronystagmography on the 15th of January, 1963 revealed spontaneous nystagmus to the

left; alternate cold and hot caloric stimulations showed bilateral fairly well functioning labyrinths and marked directional preponderance to the left (see Tables IV - X).

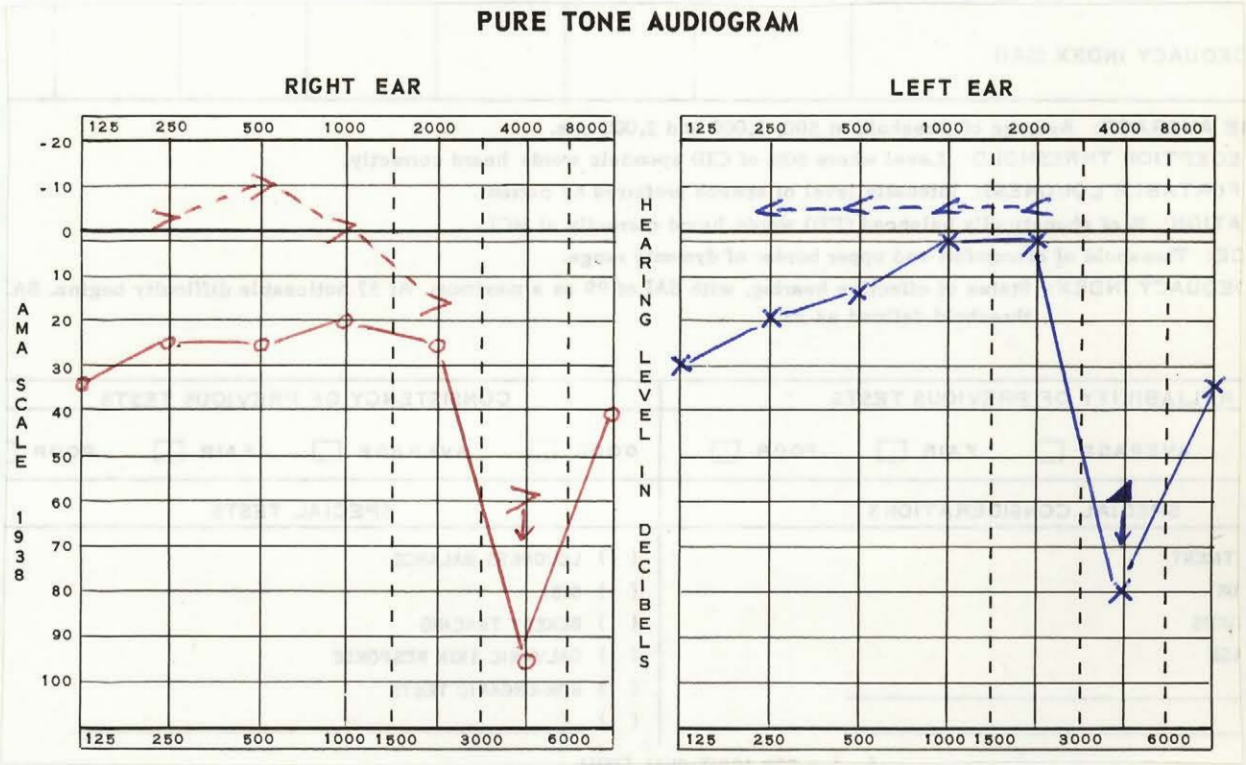


Fig. 25.

Case No. 11. P. A., 22 years old, white, female, nurse. She was involved in a car accident on the 9th of February, 1963. She struck her head, right side of her jaw and right shoulder. She had only momentary loss of consciousness but complete amnesia about the accident. She complained of frontal headache, pain in the right side of her mandible and severe pain in the right shoulder with limitation of movements due to the pain. She was brought to the Royal Victoria Hospital Emergency Department. On examination her vital signs were normal and stable. She had an abrasion over the right frontal region, bruises and tenderness over the right side of the mandible,

and pain on movement of the right shoulder; there was no deformity of the shoulder. She was admitted to the Montreal Neurological Institute. Complete neurological findings were negative. An impression of mild cerebral concussion was made. X-ray of the skull, shoulder and chest were normal; there was no evidence of fracture. Routine hematologic examination, urinalysis and blood serology were normal. Conservative management for head injury was administered. She had always been in good health. She never had any ear, nose and throat problem. There were no symptoms referrable to the ear after the accident. ENT findings were not remarkable. Audiogram done on the 13th of February, 1963 showed bilateral normal hearing. Electronystagmography on the 11th of February, 1963 revealed unsustained, position-fixed, positional nystagmus to the left in the head-hanging position, associated with marked dizziness; repeated electronystagmography on the 13th of February, 1963 (2 days later) failed to elicit any positional nystagmus and vertigo; alternate cold and hot caloric stimulations revealed bilateral fairly well functioning labyrinths with marked directional preponderance to the left (see Tables IV - X).

Case No. 12. R.C., 16 years old, single, white, male, student. He was hit by one of his mates with a coca cola bottle in the right temporo-occipital region on the 16th of February, 1963. He lost his consciousness immediately, and when he regained his consciousness, he lapsed into unconsciousness. This lasted for one hour, and he had profuse vomiting several times. He was admitted to the Montreal Neurological Institute. On admission, he was conscious and fully alert. The vital signs were stable and normal. Physical examination revealed : a hematoma in the right temporo-occipital region which was quite tender and fluctuant; multiple small lacerations of the right

pinna; other physical findings were negative. Complete neurological examination was negative. X-ray of the skull including the temporal bones and of the chest was normal; there was no evidence of fracture or expanding intracranial lesion. An impression of cerebral concussion was made. Conservative management for closed head injury was followed. He had no complaints in the ear since the accident. ENT examinations were negative. Audiogram showed bilateral normal hearing. Electronystagmography on the 18th of February, 1963 revealed unsustained, position-fixed, positional nystagmus to the left in the head-hanging position; alternate cold and hot caloric stimulations showed fairly good responses from both labyrinths with slight directional preponderance to the left (see Tables IV - X).

Case No. 13. A.S., 39 years old, married, white, male. This 39 year-old male was involved in an automobile accident on the 14th of December, 1962. He was thrown out of the car after the cars crashed. However, he was completely amnesic of the accident. He was brought home, and the next day he felt fine except for dizziness which he described as if he would fall and had to grab on to something. On the 16th of December, 1962, he developed severe mid-frontal headache. There was no nausea or vomiting. The headache was persistent and aggravated by light. His dizziness would recur everytime he tried to lift things or when he lay on his right side with the eyes looking upwards. He was admitted to the Montreal Neurological Institute on the 19th of December, 1962 upon recommendation by a doctor. On admission, physical examination revealed : B.P. of 110/70; pulse, 64 per minute; respiration, 20 per minute; temperature was normal; inability to sit up in bed due to limitation of flexion of the lumbo-

sacral spine; other physical findings were negative. Neurological examination was negative. A lumbar puncture showed the pressure of the cerebro-spinal fluid to be normal but the fluid was xanthochromic; microscopic examination revealed the presence of 4000 rbc/cu mm. and 250 wbc/cu mm; protein and sugar were normal; Pandy was negative. Skull x-ray did not show any evidence of fracture or any evidence of intracranial expanding lesion. X-rays of the chest and lumbosacral spine were not remarkable. Electroencephalogram revealed minimal depression of electrical activity over the right hemisphere which could be related to right sided sub-dural collection of fluid or cortical contusion; however, the significance was uncertain and could be within normal limits. An impression of cranio-cerebral trauma with cerebral concussion and mild contusion with subarachnoid hemorrhage was made. His subsequent course in the hospital was uneventful. He was discharged from the hospital on the 24th of December, 1962 slightly improved. He was seen again on the 14th of February, 1963. At this time he had no neurological or other complaints. The dizzy spells had disappeared about two weeks. He never had any ear, nose and throat troubles. There was no history of exposure to intense noise. ENT examinations were negative. Audiogram on the 14th of February, 1963 revealed bilateral sudden drop of hearing, neuro-sensory type, at 3000 cps, 4000 cps and 8000 cps (see Fig. 26). Speech audiometry was normal. Electronystagmography on the 14th of February, 1963 revealed sustained, position-fixed, positional nystagmus to the left associated with severe dizziness in the head-hanging position; alternate cold and hot caloric stimulations showed marked canal paresis of the right labyrinth and

normal response from the left labyrinth (see Tables IV - X).

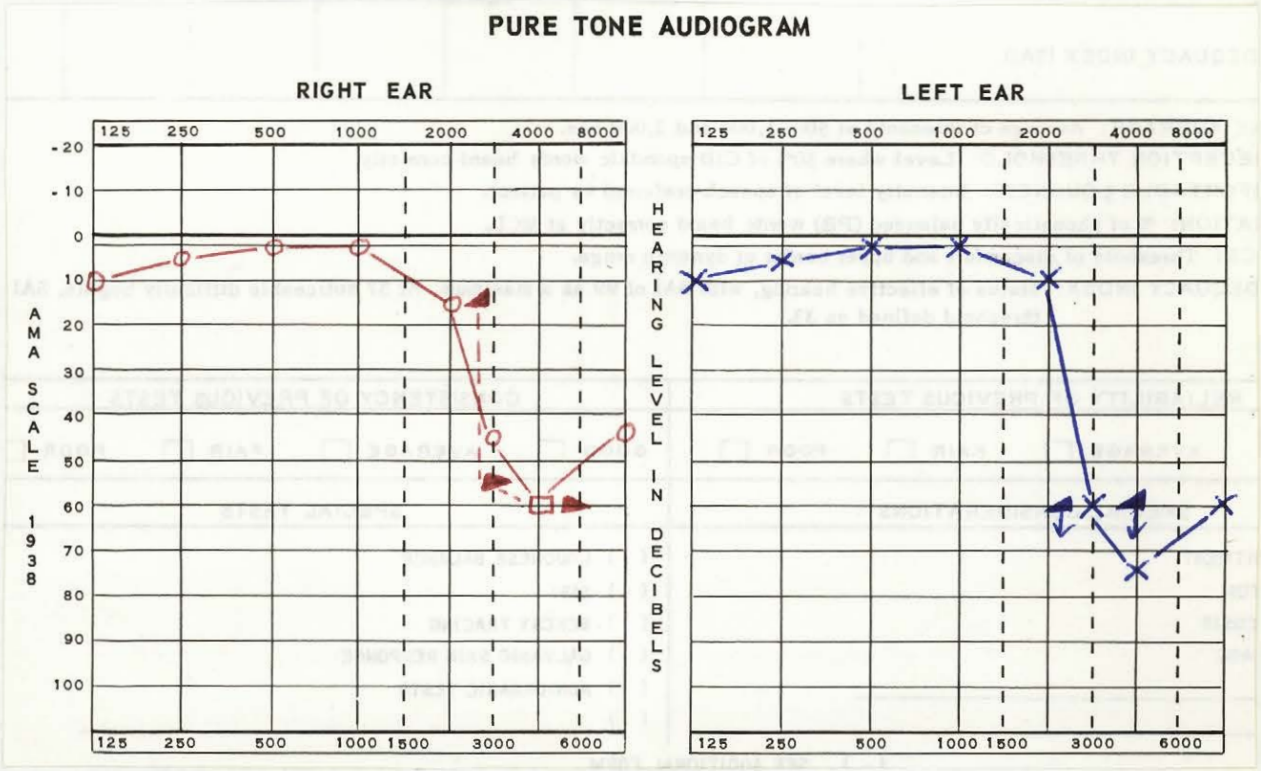


Fig. 26.

Case No. 14. G.M., 56 years old, single, white, female, secretary. She was struck by a car on the 8th of January, 1963. She was knocked down and struck her left hip and her head. She was unconscious for a few seconds. Except for pain in the lower back and left hip, she had no other neurological complaints. She was admitted to the Royal Victoria Hospital. On admission, physical examination revealed : fully conscious; oriented; B.P. of 100/68; pulse, 84 per minute; respiration, 18 per minute; a 3 x 3 cms. fluctuant hematoma on the occiput with a small laceration; tender hematoma over left lateral hip with limitation of movement of the left leg. There were no abnormal neurological findings. Other physical findings were negative. X-ray of the skull

showed a soft tissue swelling over the vertex of the skull; there was no evidence of fracture. X-ray of the pelvis showed fractures of the superior and inferior rami of the pubic bone on the left side with minimal displacement; possible fracture through the acetabulum. Conservative management for the fracture was given by the orthopedic surgeon.

On the 3rd hospital day she developed mild frontal headache and vertigo. She had dizziness when she turned her head quickly. There was no dizziness on lying down quietly. She had no nausea or vomiting. Neurosurgery and Otolaryngology were consulted. Right Bottle's sign and peri-orbital ecchymosis were described. There were no other neurological deficits. According to the opinion of the neurosurgeon, the Bottle's sign and the orbital ecchymosis were suggestive of basal skull fracture; however, there was no evidence of intracranial lesion. She never had any ear, nose and throat problems prior to the accident. She now had intermittent momentary attacks of dizziness which usually disappeared when she lay still; she had no hearing impairment or tinnitus; ENT findings were not remarkable. Audiogram revealed no significant loss of hearing in both ears. Electronystagmography revealed spontaneous nystagmus to the left; alternate cold and hot caloric stimulations showed slightly to moderately hypoactive right labyrinth and normally functioning left labyrinth and slight directional preponderance to the left (see Tables IV - X).

Case No. 15. G.D., 10 years old, single, white, male, student. This 10 year-old boy fell while he was skating on the 14th of Feb-

ruary, 1963 and hit his head. He was unconscious for 10-15 minutes. After regaining consciousness, he had complete amnesia for the accident. He was brought home, and he started to complain of left temporal headache, photophobia, dizziness and nausea. He vomited several times. He was then brought to the Montreal Neurological Institute. On admission, his vital signs were normal and remained stable. Physical examination revealed a tender hematoma over left parietal area; other physical findings were negative. Complete neurological examination failed to elicit any neurological deficit. He complained much of dizziness, especially on change of position. He never had any ear, nose and throat problem. There was no tinnitus nor any hearing impairment. Ear, nose and throat findings were not remarkable. Skull x-ray did not show any evidence of fracture or any expanding intracranial lesion. Chest x-ray was negative. Routine hematologic examination, urinalysis and blood serology were normal. Electroencephalogram revealed mild disturbance in the occipital region which was stated as a common occurrence in children following mild head injury. Audiogram revealed perfectly normal hearing in both ears. Electronystagmography showed unsustained, position-fixed positional nystagmus to the left in the head-hanging position, associated with vertigo; alternate cold and hot caloric stimulations revealed both labyrinths to be normal.

Case No. 16. C.P., 49 years old, single, white, female, nurse. She was involved in a car accident on the 9th of February, 1963. She struck her head, and she was unconscious for 4-5 minutes. On regaining her consciousness she was confused and amnesic with respect to the accident and other past events prior to the accident. She was

also disoriented as to place and person. She complained of slight dizziness and headache. She had no nausea or vomiting. She was brought to the Royal Victoria Hospital Emergency Department. On examination, the vital signs were normal and remained stable. There was some tenderness over the right zygoma and over the left occipital region. Other physical findings were not remarkable. She was admitted to the Montreal Neurological Institute. Neurological examination was negative. An impression of moderately severe cerebral concussion was made. X-ray of the skull and the chest was negative. Routine hematologic examination, urinalysis and blood serology were normal. She never had any ear, nose and throat trouble. ENT findings were not revealing. Audiogram revealed a sudden drop of hearing at 4000 cps and 8000 cps in both ears (see Fig. 27). SISI test was positive for recruitment. Electronystagmography on the 11th of February, 1963

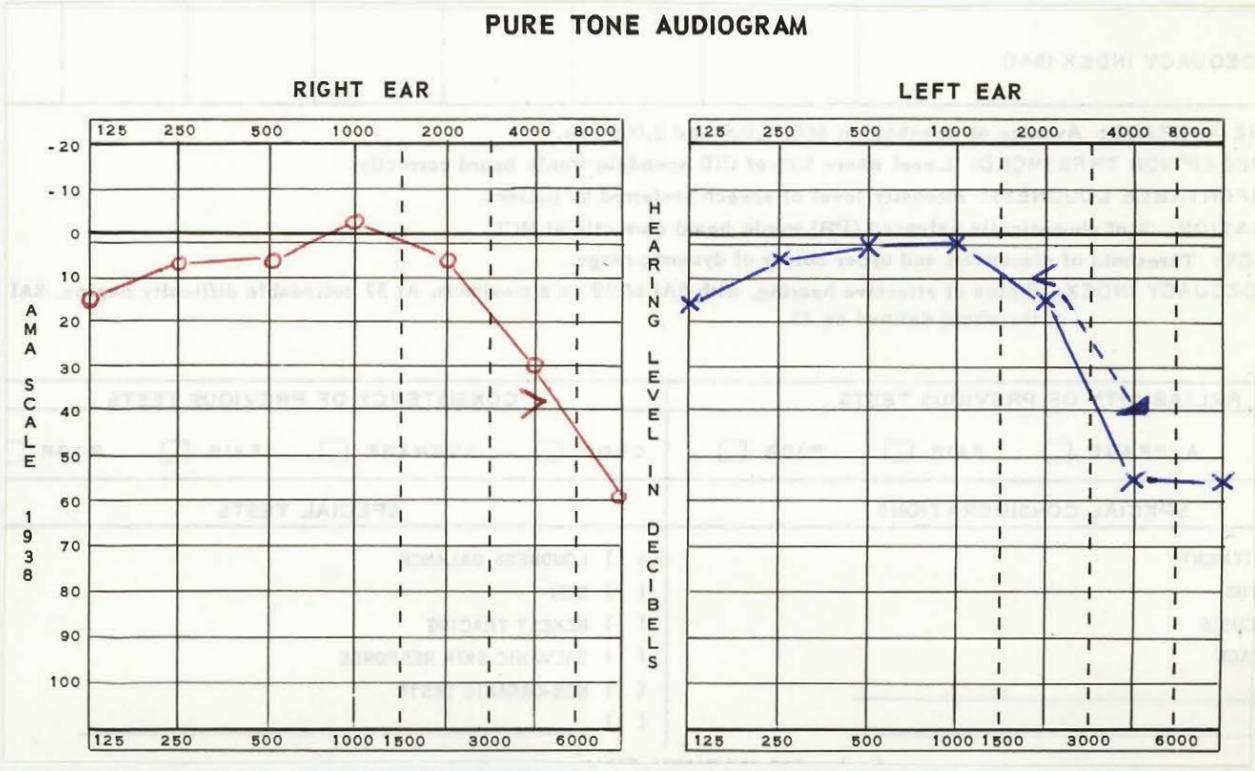


Fig. 27.

revealed marked unsustained, position-fixed, positional nystagmus to the right associated with marked vertigo in the sitting up position with the head turning to the right and in all the head-hanging positions. Electronystagmography repeated 2 days later failed to elicit any more positional nystagmus and vertigo; alternate cold and hot caloric stimulations showed a normally functioning right labyrinth and a hyperactive left labyrinth; the record (nystagmograph) of the left hot stimulation was not completed because of severe nausea and vomiting which occurred about 80 seconds after stimulation (see Tables IV - X).

Case No. 17. R.B., 19 years old, single, white, male, warehouseman. This 19 year-old male was involved in an automobile collision on the 11th of February, 1963. He was found unconscious. On the trip to the hospital he regained consciousness. He was brought to the Royal Victoria Hospital Emergency Department. He had no neurological complaints. On examination, the vital signs were normal. There was marked swelling of the right eye with subconjunctival ecchymosis; some bruises on the face were noted. Other physical findings were not remarkable. He was admitted to the Montreal Neurological Institute. Crepitation and tenderness of the left frontal area were found. Complete neurological examination was negative. Skull x-ray revealed a simple left frontal vertical fracture which extended across the posterior wall of the left frontal sinus and probably also into the right ethmoid region; the left frontal sinus and the right ethmoid cells were cloudy; fluid level in the left frontal sinus was demonstrated. Fracture of the right fronto-zygomatic suture was suspected. Chest

x-ray was negative. Routine blood and urine examinations were normal. An impression of left frontal bone fracture with cerebral concussion was made. Conservative treatment for closed head injury was given. Audiogram revealed bilateral normal hearing. Electronystagmography revealed occasional spontaneous nystagmus to the left; alternate cold and hot caloric stimulations showed slightly hypoactive left labyrinth and normally functioning right labyrinth, and directional preponderance to the left (see Tables IV - X).

Case No. 18. D.C., 56 years old, male, married, white, elevator man. This 56 year-old male slipped and fell on the 28th of February, 1963, hitting his left occipital region. He was unconscious for approximately 3 hours. He was brought to the Royal Victoria Hospital Emergency Department and was admitted for observation. On regaining consciousness he complained of bifrontal headache. His vital signs remained stable, and slight bleeding was noticed in the left ear. Physical examinations and neurological examinations were not remarkable. ENT examinations revealed blood in the left external auditory canal, otherwise the other findings were negative. Skull x-ray was negative for fracture. He was discharged on the next day. Since the accident he has been troubled with some hearing loss in the left ear, postural vertigo, especially on lying down on his left side, on sitting or standing up from the supine position, and on bending his head backwards, and occasional episodes of headache. There was no amnesia or other mental changes.

He never had any vertigo or any ENT troubles prior to the accident. He came to the Neurology Clinic on the 14th of March, 1963 for the

above complaints. Repeat examination did not reveal any neurological deficit. A repeat skull x-ray showed a transverse fracture of the left petrous temporal bone extending backwards to the occipital bone. Electroencephalogram was normal. He was referred to the Otolaryngology Clinic. ENT examinations revealed slightly dull tympanic membranes without any evidence of perforation; other findings were normal. There was no gross evidence of spontaneous nystagmus. There was no staggering or incoordination. Audiogram revealed bilateral neuro-sensory hearing loss with an average of 17 db in the right ear and 25 db in the left ear in the speech range, and worse in the higher frequencies, particularly the 4000 cps and 8000 cps frequencies (see Fig. 28). SISI test was positive for recruitment in both ears. Electronystagmography revealed transitory form of position-fixed positional nystagmus to the left in the sitting up position and in the head-hanging position; alternate cold and hot caloric stimulations showed marked directional preponderance to the left, otherwise both labyrinths were fairly active (see Tables IV -X).

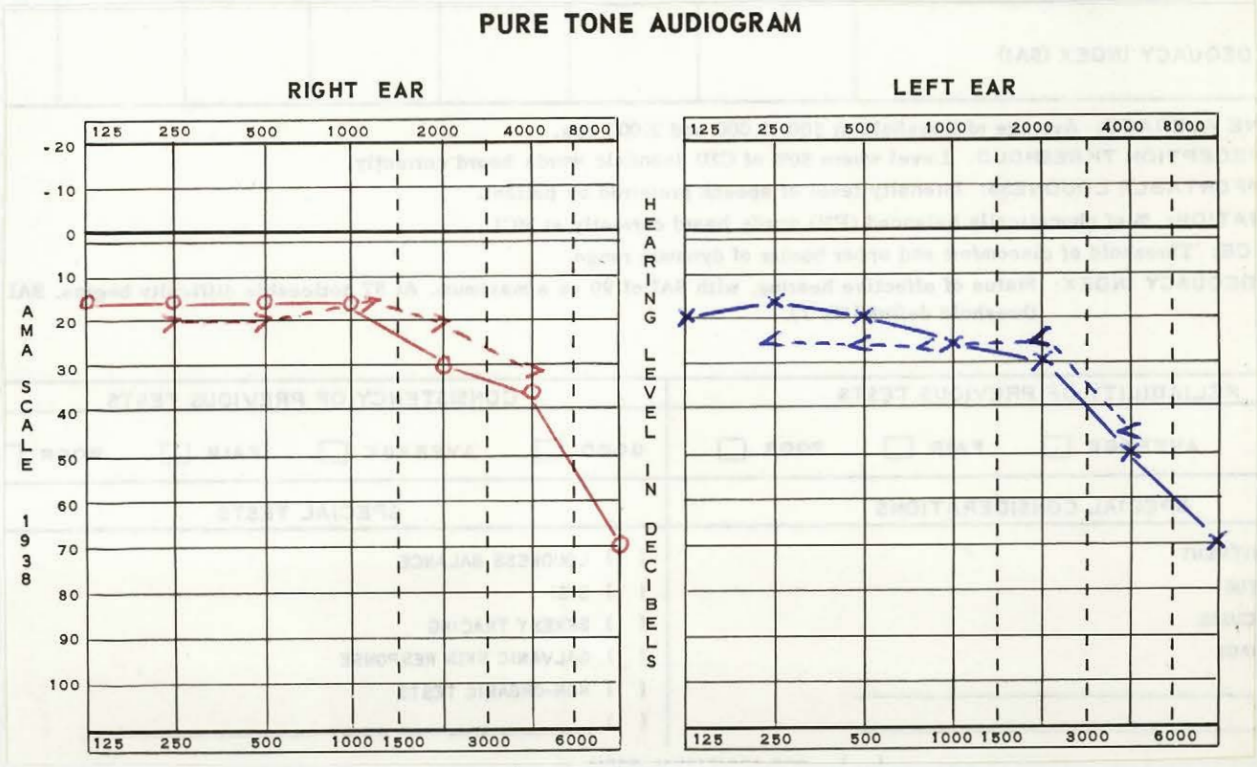


Fig. 28.

IV. Results.

A. Control.

Table I

	Spontaneous Nystagmus		Positional Nystagmus									Directional Preponderance		Pure Tone Audiometry	
			Supine			Sitting up			Head-hanging						
	to Right	to Left	For.	Rt.	Lt.	For.	Rt.	Lt.	For.	Rt.	Lt.	to Right	to Left	Nor.	Abn.
No. of cases	3	3	0	0	0	0	0	0	0	0	0	4	4	19	0

Legend : For. = head forwards.
Rt. = head turning to the right.
Lt. = head turning to the left.
Nor. = normal hearing.
Abn. = abnormal hearing.

Tabulation of the Occurrence of Spontaneous Nystagmus, Positional Nystagmus, Directional Preponderance and Hearing Tests of the Controls.

Table II

		Right Cold : 30°C	Left Cold : 30°C	Right Hot : 44°C	Left Hot : 44°C
Duration in Seconds	Mean	176	168	163	165
	Std. Deviation	22.6	23.2	27.6	27.0
Latent Period in Seconds	Mean	22	27.8	31.1	24.7
	Std. Deviation	11.4	16.0	13.3	15.3
Total Number of Nystagmus Beats	Mean	191	178	183	207
	Std. Deviation	76.2	67.0	64.7	83.9
Total Amplitude in Degrees	Mean	2332	2080	2089	2377
	Std. Deviation	896	778	981	1158
Maximal Intensity in Degrees/Second	Mean	36.8	31.2	38.3	49.3
	Std. Deviation	16.1	13.7	20.7	27.5

Tabulation of the Mean and Standard Deviation of the Controls.

Table III

		Vestibular Sensitivity		Unidirectional Sensitivity	
		Right	Left	Right-beating	Left-beating
Duration in Seconds	Mean	340	334	332	341
	Std. Deviation	29.9	28.2	46.3	43
Latent Period in Seconds	Mean	53	52	59	47
	Std. Deviation	19.3	27.5	27.9	22.4
Total Number of Nystagmus Beats	Mean	374	385	361	398
	Std. Deviation	126	134	125	151
Total Amplitude in Degrees	Mean	4417	4457	4169	4709
	Std. Deviation	1643	1669	1558	1850
Maximal Intensity in Degrees/Second	Mean	75.1	80.6	69.5	86.2
	Std. Deviation	33.5	39.1	33.6	41.1

Tabulation of the Right/Left Vestibular Sensitivity and the Unidirectional Sensitivity (Right-beating Nystagmus and Left-beating Nystagmus) of the Controls.

B. Head Injury Patients.

Table IV a

Cases	Subjective Dizziness	Spontaneous Nystagmus to Right	Spontaneous Nystagmus to Left	Positional Nystagmus: Supine Position For.	Supine Position Right	Supine Position Left	Sitting-up Position For.	Sitting-up Position Right	Sitting-up Position Left	Head-hanging Position For.	Head-hanging Position Right	Head-hanging Position Left
1	≠	-	≠	-	-	-	-	-	-	≠IIaL	-	-
2	≠	-	≠	-	-	-	-	-	-	-	-	-
3	≠	-	≠	-	-	-	-	-	-	-	-	-
4	-	-	-	-	-	-	-	-	-	-	-	-
5	≠	-	-	≠Ib	≠Ib	≠Ib	≠Ib	-	≠Ib	-	-	-
6	-	-	-	-	-	≠IIbL	-	-	≠IIbL	≠IIbL	-	≠IIbL
7	-	-	≠	-	-	-	-	-	-	-	-	-
8	-	-	-	-	-	-	-	-	-	≠IIbL	≠IIbL	-
9	≠	-	-	-	-	-	≠IIbR	≠IIbR	-	≠IIbR	≠IIbR	-
10	-	-	≠	-	-	-	-	-	-	-	-	-
11	≠	-	-	-	-	-	-	-	-	≠IIbL	-	-
12	-	-	-	-	-	-	-	-	-	≠IIbL	≠IIbL	-
13	≠	-	-	-	-	-	-	-	-	≠IIaL	≠IIaL	-
14	≠	-	≠	-	-	-	-	-	-	-	-	-
15	≠	-	-	-	-	-	-	-	-	≠IIbL	-	≠IIbL
16	≠	-	-	-	-	-	≠IIbR	≠IIbR	-	≠IIbR	≠IIbR	≠IIbR
17	-	-	≠	-	-	-	-	-	-	-	-	-
18	≠	-	-	-	-	-	≠IIbL	-	≠IIbL	≠IIbL	-	≠IIbL

Legend : ≠ = positive or present ; I = position-changing PN
- = negative or absent ; II = position-fixed PN
R = to the right ; a = sustained
L = to the left ; b = unsustained
PN = positional nystagmus

Note : All positional nystagmus elicited were associated with vertigo.

Tabulation to Show the Occurrence of Subjective Dizziness, Spontaneous Nystagmus and Positional Nystagmus and Its Characteristics in the Head Injured Patients.

Table IV b

Cases	Directional Preponderance to Right	Directional Preponderance to Left	Closed Skull Fractures: Temporal Bone	Closed Skull Fractures: Other Cranial Bone	Pure Tone Audiometry	SISI Test	EEG
1	-	///	negative	negative	slight P-L	not done	not done
2	-	//	negative	negative	profound P-R	not done	not done
3	-	//	/ R trans.	/ multiple	T-R	not done	not done
4	-	-	negative	negative	severe P-R;T-L	not done	not done
5	/	-	negative	negative	slight P-L	/ recruit	not done
6	-	-	negative	negative	bil. severe P	not done	not done
7	-	//	negative	negative	slight P-L	not done	abnor. R
8	-	-	negative	negative	N	not done	not done
9	-	-	negative	/ R-parietal	N	not done	not done
10	-	//	/ R longitu.	/ multiple	bil. C & P (4000cps)	not done	abnor. L
11	-	//	negative	negative	N	not done	not done
12	-	/	negative	negative	N	not done	not done
13	-	-	negative	negative	bil. high tone P	not done	not done
14	-	/	negative	negative	N	not done	not done
15	-	-	negative	negative	N	not done	Min. abn.
16	-	-	negative	negative	bil. high tone P	/ recruit	not done
17	-	/	negative	negative	N	not done	not done
18	-	//	/ L trans.	/ L-occipital	bil. high tone P	/ recruit	not done

Legend : R = right side ; L = left side ; Trans. = transverse fracture ; Longi. = longitudinal fracture ; Recruit = recruitment ; N = normal hearing ; P = perceptive (neuro-sensory) hearing loss ; C = conductive hearing loss ; T = total hearing loss

Tabulation to Show the Occurrence of Directional Preponderance, Skull Fractures, Cochlear Function, Recruitment Phenomenon and Electroencephalographic Findings in the Head Injured Patients.

Table V

Cases	Right Cold:30°C	Left Cold:30°C	Right Hot:44°C	Left Hot:44°C	Vest. Sensitivity Right	Vest. Sensitivity Left	Unidirect. Sensitivity Right-beating	Unidirect. Sensitivity Left-beating
1	360	158	192	294	552	452	350	654
2	230	176	190	170	420	346	366	400
3	no resp.	150	no resp.	134	no resp.	284	-	-
4	no resp.	no resp.	100	no resp.	100	no resp	-	-
5	138	328	172	240	310	568	500	378
6	182	100	no resp.	no resp.	182	100	-	-
7	240	200	185	210	425	410	385	450
8	125	200	170	180	295	380	370	305
9	no resp.	83	84	58	84	141	167	58
10	170	154	140	174	310	328	294	344
11	200	180	185	190	385	370	365	390
12	200	160	170	180	370	340	330	380
13	188	208	121	170	309	378	329	358
14	130	144	116	124	246	268	260	254
15	210	160	220	240	430	400	380	450
16	166	240	210	vomiting	376	vomit.	450	vomiting
17	160	92	112	166	272	258	204	326
18	193	95	108	172	301	267	203	365

Legend : Vest. = vestibular
Unidirect. = unidirectional
resp. = response

Tabulation of the Duration of Caloric Reaction (Nystagmus) in Seconds of the Patients.

Table VI

cases	Right Cold:30°C	Left Cold:30°C	Right Hot:44°C	Left Hot:44°C	Vest. Sensitivity Right	Vest. Sensitivity Left	Unidirect. Sensitivity Right-beating	Unidirect. Sensitivity Left-beating
1	602	89	156	542	758	651	245	1144
2	204	168	186	276	390	444	354	480
3	no resp.	148	no resp.	250	no resp.	398	-	-
4	no resp.	no resp.	48	no resp.	48	no resp	-	-
5	93	623	351	345	444	968	974	438
6	38	29	no resp.	no resp.	38	29	29	38
7	384	330	322	481	706	811	652	865
8	95	110	40	39	135	149	150	134
9	no resp.	25	17	22	17	47	42	22
10	365	337	275	476	640	813	612	841
11	292	223	148	302	440	525	371	594
12	181	130	137	189	318	319	267	370
13	73	172	12	107	85	279	184	180
14	251	240	160	235	411	475	400	486
15	210	200	273	260	483	460	473	470
16	113	136	259	vomiting	372	vomit.	395	vomiting
17	216	83	149	166	365	249	232	382
18	194	88	102	186	296	274	190	380

Legend : Vest. = vestibular
Unidirect. = unidirectional
resp. = response

Tabulation of the Total Number of Nystagmus Beats of Patients.

Table VII

Cases	Right Cold:30°C	Left Cold:30°C	Right Hot:44°C	Left Hot:44°C	Vest. Sensitivity Right	Vest. Sensitivity Left	Unidirect. Sensitivity Right-beating	Unidirect. Sensitivity Left-beating
1	8984	1364	2109	9550	11093	10914	3473	18534
2	3796	2630	3167	4734	6963	7564	5797	8530
3	no resp.	1714	no resp.	3629	no resp.	5343	-	-
4	no resp.	no resp.	460	no resp.	460	no resp	-	-
5	900	8175	5675	4324	6575	12499	13850	5224
6	358	222	no resp.	no resp.	358	222	222	358
7	6000	4068	3544	6228	9544	10296	7612	12228
8	520	1023	475	260	995	1283	1498	780
9	no resp.	91	84	108	84	199	175	108
10	4435	4116	2134	4780	6569	8896	6250	9215
11	3386	2090	1045	2931	4431	5021	3135	6317
12	3107	2243	2114	2862	5221	5105	4357	5969
13	1416	3972	166	2000	1582	5972	4138	3416
14	2592	3072	1152	2592	3744	5664	4224	5184
15	3310	3360	3000	3200	6310	6560	6360	6510
16	1468	1715	3350	vomiting	4818	vomit.	5065	vomiting
17	4153	1390	2770	2870	6923	4260	4160	7023
18	3083	1180	1260	3142	4343	4322	2440	6235

Legend : Vest. = vestibular
Unidirect. = unidirectional
resp. = response

Tabulation of the Total Amplitude of Nystagmus in Degrees of the Patients.

Table VIII

Cases	Right Cold;30°C	Left Cold;30°C	Right Hot;44°C	Left Hot;44°C	Vest. Sensitivity Right	Vest. Sensitivity Left	Unidirect. Sensitivity Right-beating	Unidirect. Sensitivity Left-beating
1	50.1	14.9	24.1	84.5	74.2	99.4	39.0	134.6
2	39.3	35.3	36.3	63.8	75.6	99.1	71.6	103.1
3	no resp.	24.2	no resp.	45.3	no resp.	69.5	-	-
4	no resp.	no resp.	18.3	no resp.	18.3	no resp	-	-
5	12.9	39.8	65.6	55.8	78.5	95.6	105.4	68.7
6	insuff.	insuff.	no resp.	no resp.	-	-	-	-
7	60.2	30.6	33.5	50.8	93.7	81.4	64.1	111.0
8	16.1	27.0	16.4	36.9	32.5	63.9	43.4	53.0
9	no resp.	no resp.	insuff.	insuff.	-	-	-	-
10	64.4	51.4	32.2	71.7	96.6	123.1	83.6	136.1
11	34.3	17.6	16.6	34.1	50.9	51.7	34.2	68.4
12	29.3	26.6	29.8	21.2	59.1	47.8	56.4	50.5
13	61.3	69.7	insuff.	55.5	-	125.2	-	-
14	39.8	28.2	23.0	28.1	62.8	56.3	51.2	67.9
15	38.0	30.4	30.6	41.6	68.6	72.0	61.0	79.6
16	45.6	48.2	49.4	82.0	95.0	130.2	97.6	127.6
17	48.1	36.7	72.2	60.3	120.3	97.0	108.9	108.4
18	33.1	30.8	26.5	34.8	59.6	65.6	57.3	67.9

Legend : Vest. = vestibular
Unidirect. = unidirectional
resp. = response

Tabulation of the Maximal Intensity of Nystagmus in Degrees/Second of the Patients.

Table IX

		Right Cold:30°C	Left Cold:30°C	Right Hot : 44°C	Left Hot : 44°C
Duration in Seconds	Mean	160	157	137	158
	Std. Deviation	57.5	58.9	45.8	43
Latent Period in Seconds	Mean	27.3	32.8	45.6	26.8
	Std. Deviation	-	-	-	-
Total Number of Nystagmus Beats	Mean	183	173	146	228
	Std. Deviation	155	139	102	134
Total Amplitude in Degrees	Mean	2639	2356	1085	3130
	Std. Deviation	2155	1874	1447	2191
Maximal Intensity in Degrees/Second	Mean	31.8	28.4	26.3	42.5
	Std. Deviation	16.4	14.6	17.6	19.6

Tabulation of the Mean and Standard Deviation of Caloric Reactions of the Patients.

Table X

		Vestibular Sensitivity		Unidirectional Sensitivity	
		Right	Left	Right-beating Nystagmus	Left-beating Nystagmus
Duration in Seconds	Mean	298	305	294	319
	Std. Deviation	109	132	89	118
Latent Period in Seconds	Mean	67.4	57.2	71.6	48.5
	Std. Deviation	-	-	-	-
Total Number of Nystagmus Beats	Mean	330	405	320	416
	Std. Deviation	223	256	247	277
Total Amplitude in Degrees	Mean	4445	5524	4162	5838
	Std. Deviation	3144	3493	3082	4457
Maximal Intensity in Degrees/Second	Mean	58.1	70.9	54.8	74.3
	Std. Deviation	28.4	29.8	26.8	32.0

Tabulation of the Mean and Standard Deviation of the Right/Left Vestibular Sensitivity and the Unidirectional Sensitivity of the Patients.

C. Comparison of the Graphic Representations of the Frequency Distributions of the Magnitudes of Caloric Reactions of the Controls and the Patients (Head Injury Cases).

1. Duration of Caloric Reaction (Nystagmus).

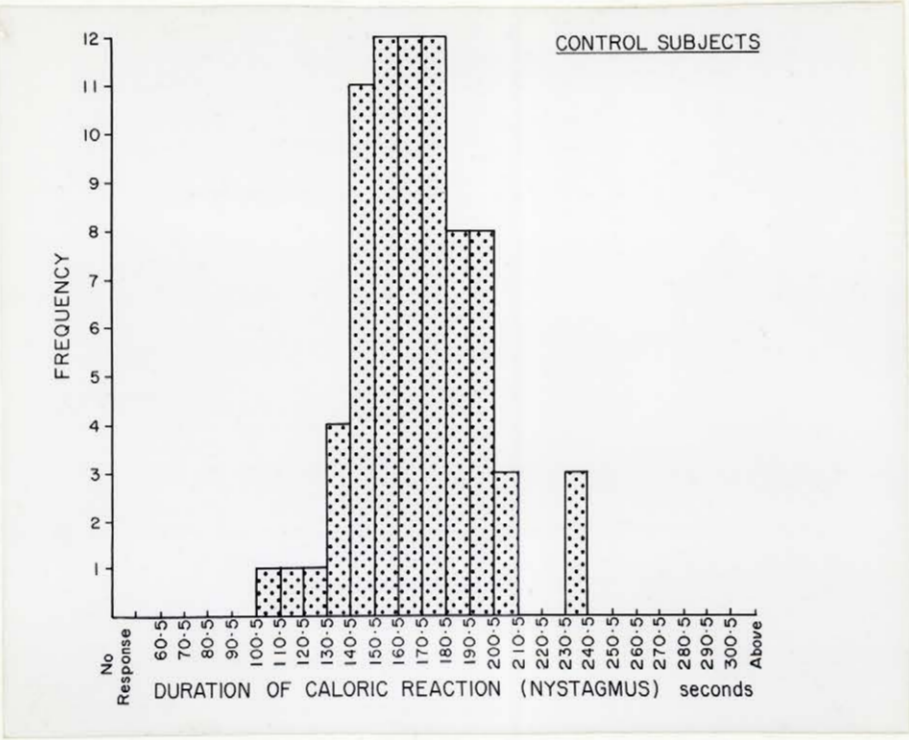


Fig. 29.

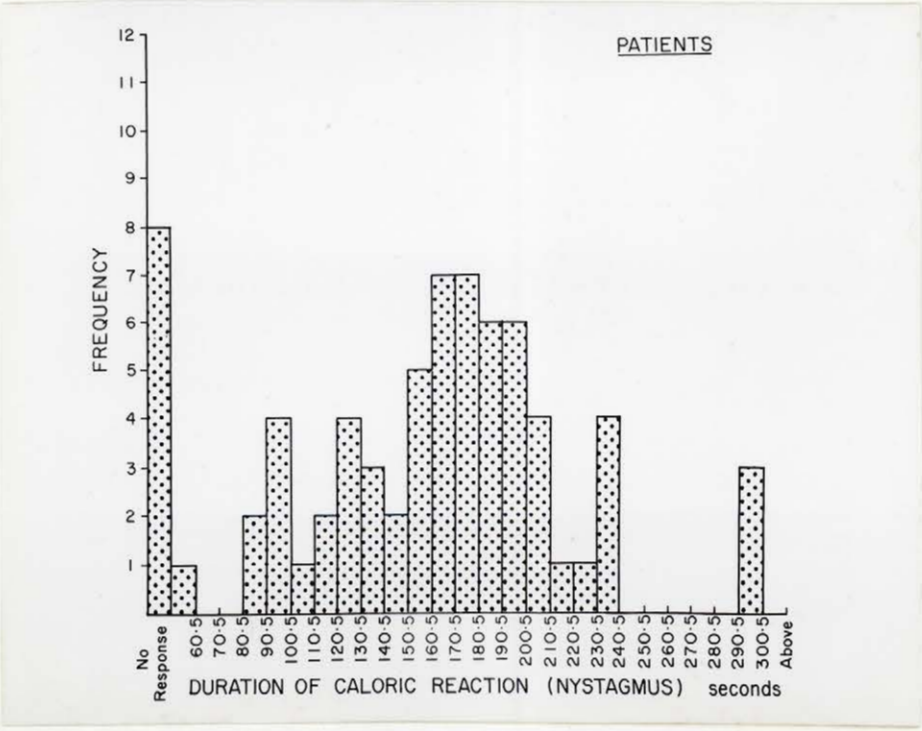


Fig. 30.

2. Total Number of Nystagmus Beats.

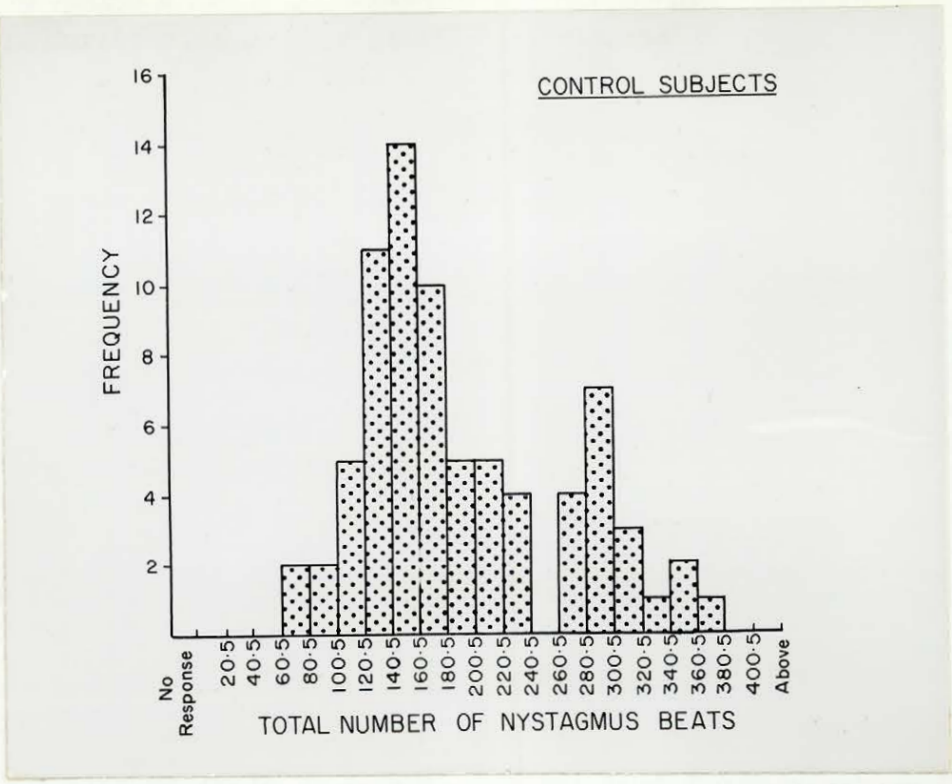


Fig. 31

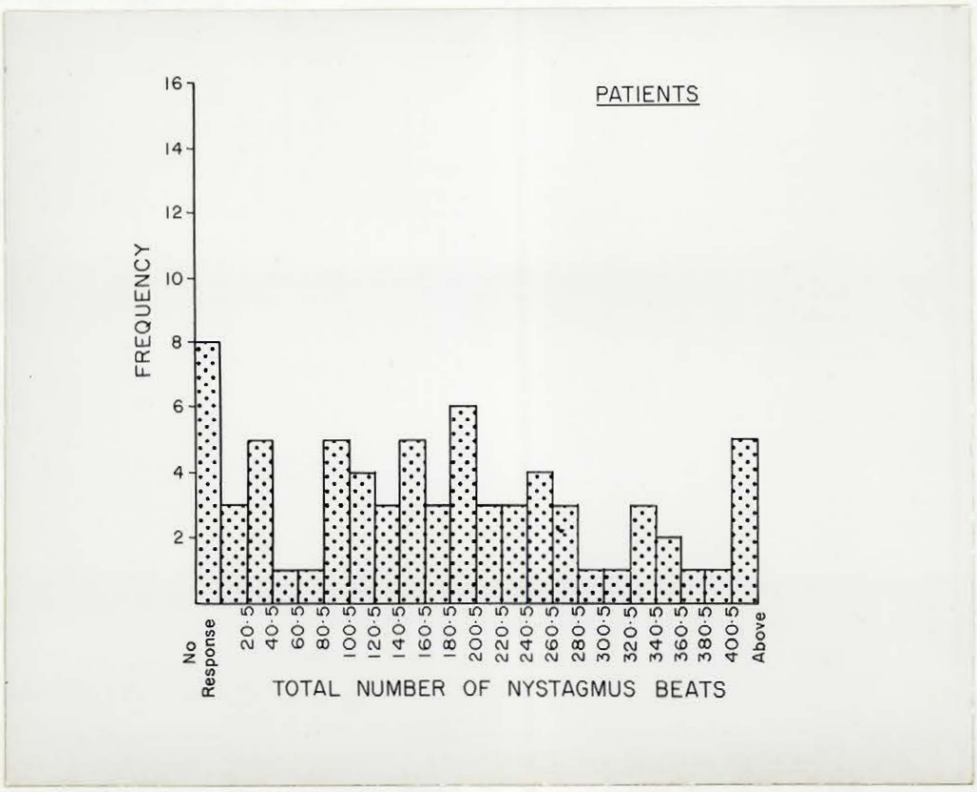


Fig. 32

3. Total Amplitude of Nystagmus.

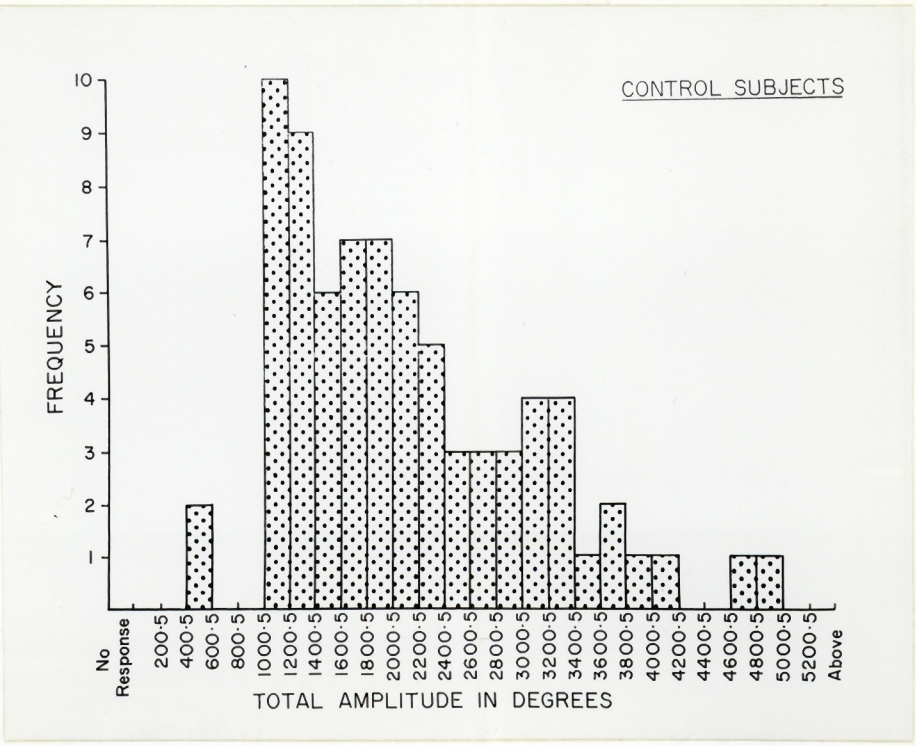


Fig. 33.

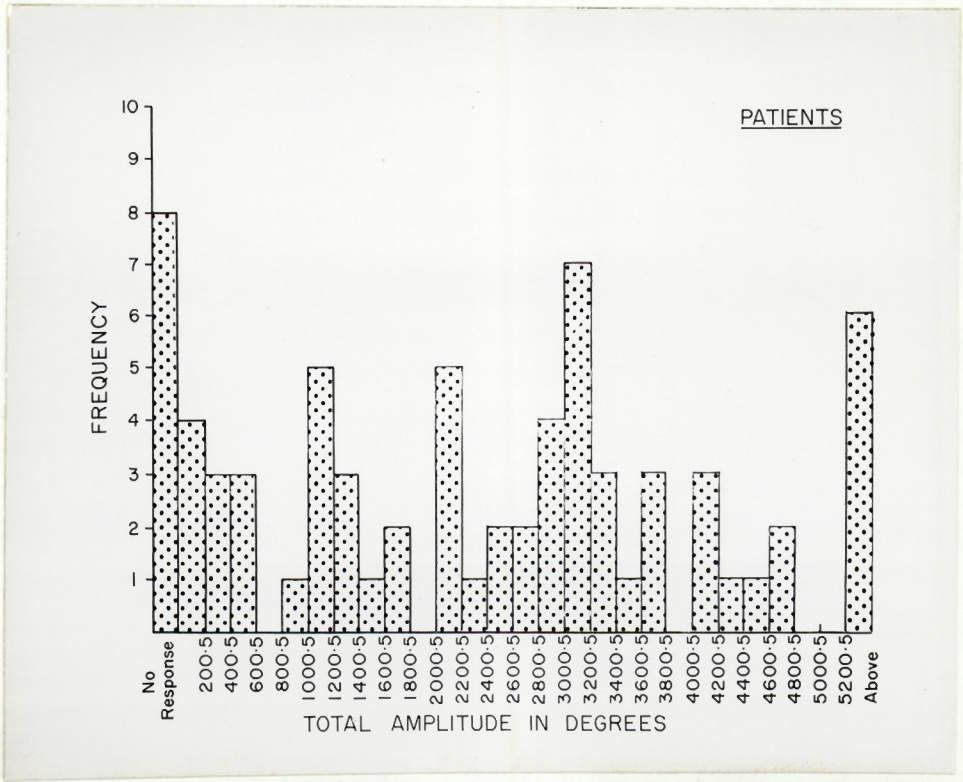


Fig. 34.

4. Maximal Intensity (Maximal Slow Speed) of Nystagmus.

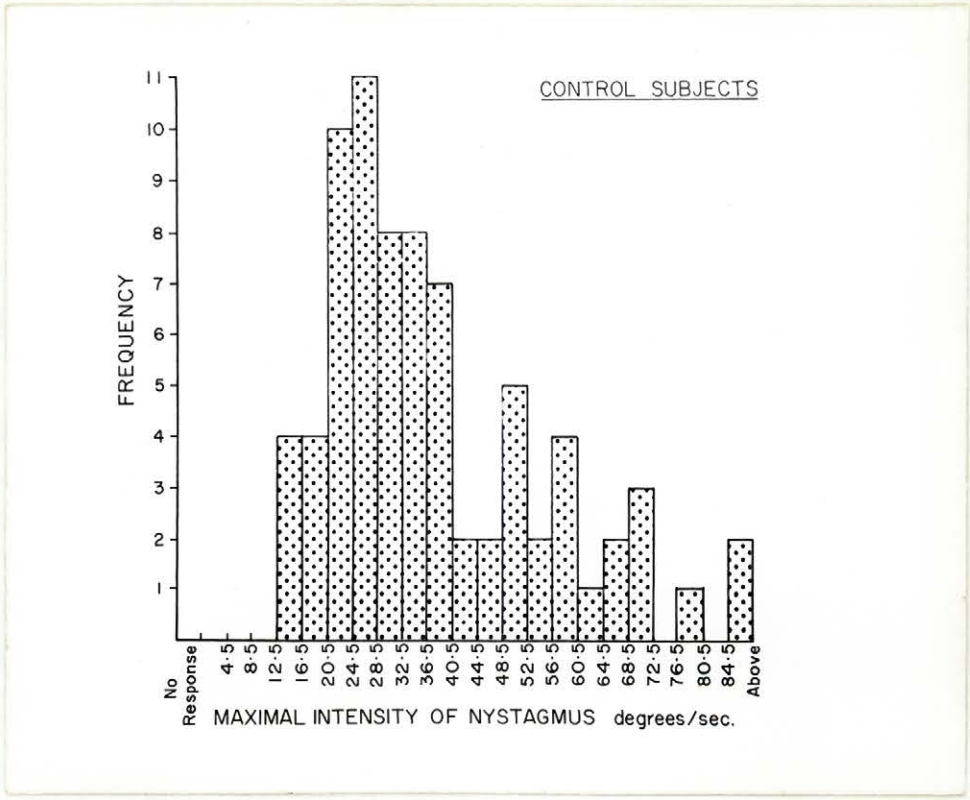


Fig. 35.

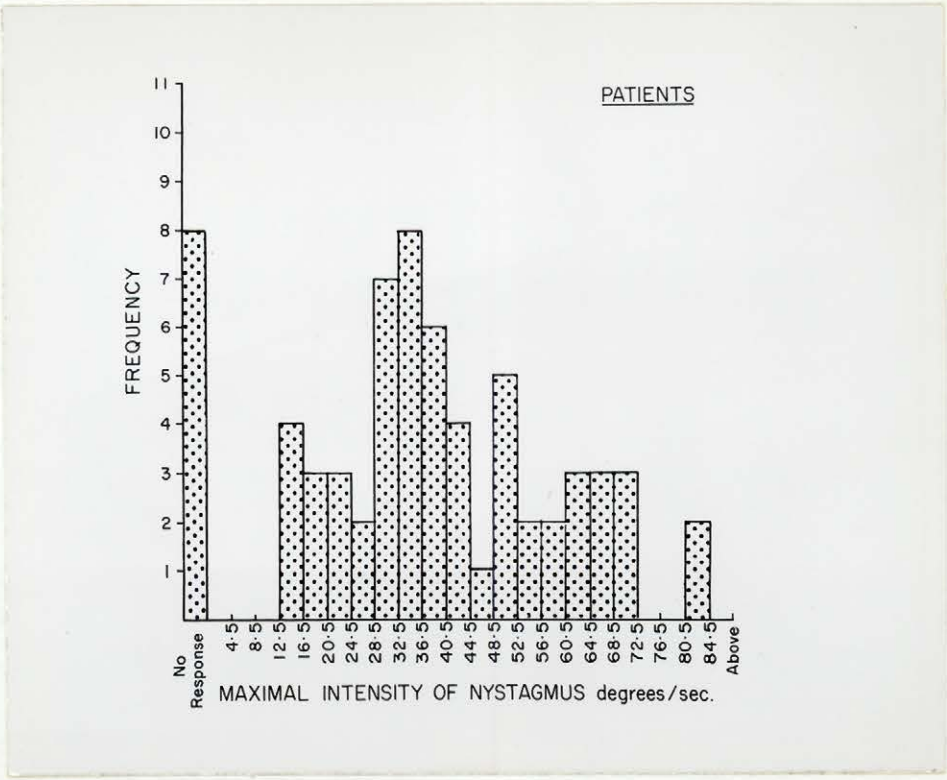


Fig. 36

V. General Discussion.

A. Statistical Aspect.

The statistical data of the magnitudes of the caloric reactions (the duration of the caloric reaction, the total number of nystagmus beats, the total amplitude of nystagmus and the maximal intensity of nystagmus) of the Control Group and the Post-traumatic Group (Head Injury Patients) were presented in Tables II, III, V, VI, VII, VIII, IX and X. Ordinarily, the analysis of the data is done by using the standard mathematical technique. However, in the present investigation it was felt that the standard method of employing the t-test to determine the significance of differences of means and confidence limit techniques to ascertain the probability of the Post-traumatic Group falling into the Control Group classification would have little value because of the small number of samples in both groups. A graphic analysis of the frequency distributions of the Control Group and the Post-traumatic Group (see Figs. 29-36) was employed to show the significance of differences between the two samples. Although the results were not conclusive, a good indication of striking difference of the distributions of the different parameters of the caloric reactions (the duration of the caloric reaction, the total number of nystagmus beats, the total amplitude and the maximal intensity of nystagmus) between these two groups was obtained. Generally, the "spread" of values was very much greater for the Post-traumatic Group (Head Injury Patients) and very few definite maximums could be determined. On the other hand, it was quite obvious that for the Control Group a definite distribution with well-defined maximums existed.

B. Diagnostic Aspect.

1. Control Group.

The control group is composed of 19 healthy normal young adults, most of them are student nurses of the Royal Victoria Hospital. Out of the 19 control subjects, 6 had spontaneous nystagmus shown by the electronystagmograph and not by clinical examination; in 3 of these, directed to the left, and in 3 to the right (see Table I). All of these subjects with spontaneous nystagmus had directional preponderance to the direction of the spontaneous nystagmus in at least 3 parameters of the magnitudes of caloric reactions. Only 2 out of the 13 control subjects without any demonstrable spontaneous nystagmus showed directional preponderance (see Table I). Therefore, 8 out of 19 control subjects or 42.1% of the controls had directional preponderance, and 6 out of 19 or 31.5% had spontaneous nystagmus. The direction of the spontaneous nystagmus and the directional preponderance was equally divided. None of the 19 control subjects had positional nystagmus with vertigo during the postural tests.

The Jongkees 20% rule has been used in the present investigation in the evaluation of directional preponderance.

2. Head Injury Patients.

A large proportion of the present series of head injuries complained of vertigo, particularly postural vertigo, either immediately or sometime later after the skull trauma. This is also the observation of many authors as mentioned in chapter V. In the author's small series of head trauma cases, 11 out of 18 or 61.1 % had subjective complaint of postural dizziness.

Positional nystagmus associated with vertigo was a very common occurrence in head trauma cases. It was present in 11 out of 18 cases or 61.1 % in the present investigation (see Table IVa), and in most of them it was brought out in the head-hanging position during the postural tests. The most frequent form of the positional nystagmus elicited in the author's series was the so-called peripheral type of positional nystagmus, i.e., position-fixed, transitory positional nystagmus. Only 1 out of the 11 cases with positional nystagmus demonstrated the central type of positional nystagmus (Nylen's Type I positional nystagmus)m i.e., position-changing positional nystagmus. However, in this particular case the positional nystagmus was not sustained in character. Irregular positional nystagmus was not observed in the present series. The presence of positional nystagmus with vertigo was considered pathological, representing some disturbances of the vestibular system, even though the caloric reactions should fall within the normal limits. The non-occurrence of positional nystagmus among the control subjects gave some support to this assumption. It should be emphasized that postural tests are a "must" in performing the vestibular tests in post-traumatic cases (head injuries) as the positional nystagmus might be the only abnormal finding present in spite of negative neurological findings. This was demonstrated in several cases in this series. The timing of performing the postural tests was likewise important; postural tests for post-traumatic cases should be carried out as soon as possible, preferably the first 24 hours after the injury if the condition of the patient warranted such an examination. More information probably would be obtained in this manner. It is

appropriate to mention here that 2 of the present series of post-traumatic cases demonstrated marked positional nystagmus with marked dizziness one day (within 24 hours) after the injury, and when repeated 2 days later, positional nystagmus and vertigo could no longer be elicited.

Spontaneous nystagmus (other than positional nystagmus) was demonstrated in 7 out of 18 cases or 38.8% of the series. Only one out of 7 cases with spontaneous nystagmus demonstrated both spontaneous nystagmus and positional nystagmus with vertigo in the head-hanging position. In the latter instance, the nystagmus in the head-hanging position was accentuated in relation to the frequency, amplitude and the maximal intensity of the nystagmus, and it was associated with marked dizziness. In the other 6 cases, positional nystagmus with vertigo was not demonstrated. The direction of the spontaneous nystagmus in all these cases was to the left.

Directional preponderance was present in 11 out of the 18 cases or 61.1% in the present investigation; 10 out of the 11 cases, the directional preponderance was to the left, and only one to the right (see Table IVb). Directional preponderance was shown in all the 7 cases with spontaneous nystagmus, and the direction of the preponderance was in the same direction as the spontaneous nystagmus. Directional preponderance was present in 5 out of the 11 cases with positional nystagmus, and the direction of the preponderance was again towards the direction of the positional nystagmus. In case No. 5, the positional nystagmus was of the position-changing type; however, it was most significant to the right, and the directional preponderance was found to be directed to the right. In the present

investigation I was not able to show any correlation between the directional preponderance and the side of the head injury either by the clinical findings or by the electroencephalographic findings. Of course, only a limited number of these cases had electroencephalographic studies.

The results of the alternate cold and hot caloric tests with electronystagmography revealed a wide variation of findings, ranging from no response in several cases to extremely hyperactive responses (see Tables V, VI, VII, VIII, and Figs. 29-36). The caloric reactions of these cases most depended on the severity of the labyrinthine damage.

Temporal bone fractures were demonstrated radiologically in 3 cases out of 18 or 16.6% in the series; one with longitudinal fracture and two with transverse fractures. One of the 2 transverse fractures of the petrous temporal bone was incomplete because the cochlear and the vestibular functions were retained, although there were evidences of disturbance of both divisions. The other transverse fracture was complete; both cochlear and vestibular functions were totally lost. In the case of the longitudinal fracture of the petrous temporal bone, mixed hearing loss and facial palsy were demonstrated (see Table IVb).

The cochlear function after skull trauma varied probably with the severity of the injury and the progressive nature of the damage. 4 out of the 18 cases revealed severe perceptive or neuro-sensory hearing loss to total hearing loss. In one of these 4 cases, the hearing was slightly improved sometime later after the injury, and the vestibular function in this particular case which was found

originally (one week after the skull trauma) to be non-responsive to caloric stimulations showed an almost complete recovery two years later. Many of these cases without any radiological evidence of skull fractures showed bilateral high tone perceptive or neuro-sensory hearing loss with the peak at 4000 cps frequency. SISI test (short increment sensitivity index) was performed in 3 cases with some high tone neuro-sensory hearing loss after skull trauma, and all 3 cases were positive for recruitment, indicating a peripheral lesion (see Table IVb).

The site of injury. The opinions of various authors differed considerably; some ascribed the damage after skull trauma to a central lesion (at the vicinity of the vestibular nuclei) while others attributed the lesion to the peripheral end organs. However, up to now there was no pathological proof to support one or the other contention. From the present investigation, the clinical evidences were in favor of a peripheral labyrinthine lesion for the post-concussional symptoms; Schuknecht and Davison[143] reported the same findings.

VI. Summary and Conclusion.

1. Directional preponderance is frequently seen in normal healthy subjects. It is present in 42.5% of the present series.
2. Spontaneous nystagmus is observed frequently in normal healthy subjects. 31.5% of the control subjects showed spontaneous nystagmus to one or the other direction, and the directional preponderance was always present in these cases. The direction of the preponderance was towards the direction of the spontaneous nystagmus.

3. Directional preponderance is frequently associated with spontaneous nystagmus.
4. Positional nystagmus was not observed in the control subjects.
5. Postural vertigo is the most common to occur in patients suffering from head injury. 61% of the cases in this series manifested or complained of postural dizziness immediately or sometime after the head trauma.
6. Positional nystagmus associated with dizziness is a common finding after head injury. It was present in 61% of cases in the present study. The most frequent form was the transitory, direction-fixed positional nystagmus.
7. The head-hanging position in the postural tests readily activates the positional nystagmus with vertigo in these traumatic cases.
8. Postural tests should always be included in vestibular function tests.
9. Positional nystagmus may be the only abnormality to be found after head trauma.
10. Vestibular disturbances may occur in head injury without associated cochlear disturbance. Many of the cases complain severely of dizziness after head injury when the hearing is perfectly normal.
11. Spontaneous nystagmus (other than positional nystagmus) is quite often observed after head injury. 38% of this series showed the occurrence of spontaneous nystagmus.
12. Directional preponderance was present in 61% of the head injured cases. The direction of preponderance was predominantly to the left (10 out of the 11 cases). It is usually in the direction of the spontaneous nystagmus or of the positional nystagmus.

13. There was no correlation between the direction of the preponderance and the side of the injury in these cases.
14. There was radiological evidence of petrous temporal bone fracture in 16% of these head injured cases.
15. Impairment of cochlear function was found in 61% of this series. The severity of the hearing loss varied. Many of the patients showed bilateral high tone neuro-sensory hearing loss with the peak at 4000 cps frequency. Only 3 cases were tested for recruitment and all three were positive.
16. The responses to the alternate cold and hot caloric stimulations showed a wide range of variation. The histographic representations of the frequency distributions of the control subjects and the post-traumatic cases were strikingly different.
17. Clinical evidence in the present investigation suggests that the lesion producing abnormalities in vestibular function is peripheral rather than central. The definite pathology is not yet determined.

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