TUTORIAL ON ELECTRONYSTAGMOGRAPHY

(REGISTER NO. M2K11)

An Independent Project submitted in part fulfillment of the first year M.Sc (Speech and Hearing), University of Mysore, Mysore

ALL INDIA INSTITUTE OF SPEECH AND HEARING
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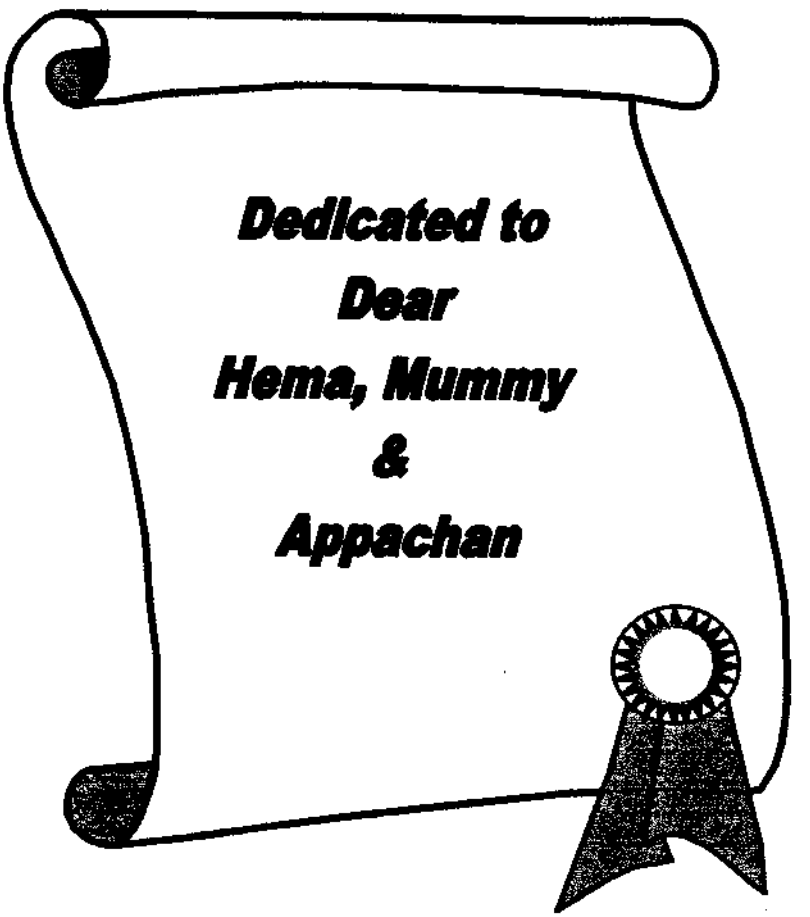
Dedicated to
Dear
Hema, Mummy
&
Appachan

Certificate

This is to certify that the Independent project entitled *"Tutorial On Electronystagmography* " is the bonafide work done in part fulfillment for the degree of Master of Science (Speech and Hearing) of the student (Register No. M2K11).

Director

Mysore

May 2001

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Certificate

This is to certify that the Independent project entitled *"Tutorial On Electronystagmography"* has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any Diploma or Degree.

Guide

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Declaration

I hereby declare that this Independent project entitled *"Tutorial On Electronystagmography"* is the result of my own study under the guidance of Dr. K. Rajalakshmi, Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier in any other University for the award of any Diploma or Degree.

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INTRODUCTION

Equilibrium is the capacity of the body to maintain posture and spatial orientation at rest and during movement. The three systems concerned in the function of equilibrium are

1. The vestibulo - ocular system
2. The vestibulo - spinal system
3. The retino - ocular system

Vestibular system assessment has increased its efficiency especially under the influence of Electronystagmography (abbreviated as ENG). Electronystagmography is a clinical tool that has an objective and quantitative test procedure that assess the vestibulo-ocular function. The other modern equilibriometric tests are Craniocorpography and Posturography, which along with Electronystagmography provides a complete picture about various equilibrium related sensory pathways.

Du. Bois-Reymond in 1849 discovered that a potential difference exists between cornea and retina in man i.e., Corneoretinal potentials. Eye movement away from midline changes this corneoretinal potential. This change in corneoretinal potential enables an acquisition of permanent quantifiable records of eye movement on an Electronystagmography instrument. So Electronystagmography is the electrical recording of a particular type of rhythmic, involuntary, back-and-forth eye movement called nystagmas. Electronystagmography combines traditional vestibulometric methods with electronic graphic recording techniques. The Electronystagmography

examination is essentially a battery of tests drawn from the otologic, neurologic and neurophysical examination. Electronystagmographic and Audiometric examination have the same basic diagnostic significance. Both examinations anatomically localize pathology but do not provide clinical diagnosis. Electronystagmography can only differentiate central nervous system lesion from peripheral vestibular ones (the term "peripheral vestibular" both vestibular nerve and organ). Electronystagmography is an important tool in a neurotological diagnosis which makes it possible to decide about the patients problems related to vertigo, dizziness, balance disorders etc. It helps to arrive at a conclusion that the patient has a normal vestibular mechanism, or a peripheral end organ lesion or a central nervous - retro labyrinthine lesion.

The present tutorial aim's at providing intensive training in the area of "Electronystagmography testing". The word tutorial as defined by scientific and English dictionaries refers to an "instruction book" or "intensive instruction in some area". It aims at providing supplementary instruction in order to present better opportunities to students and concerned professionals to actively participate in the learning process and receive immediate feedback. The information is carefully selected and delivered in an organized and structured manner.

There is an increasing demand on professionals in the field of Audiology to perform Electronystagmography. It is important for students and professionals in this field to know about Electronystagmography testing procedures and interpretation in depth. This independent project deals with

Electronystagmography testing under which a great amount of information has been presented in a comprised form. This information has been collected from books, journals, manuals and other sources.

Thus, this particular independent project has been developed to serve the following purposes:

1. To provide a clear picture of the vestibular anatomy and physiology
2. Give intensive information about Electronystagmography testing.
3. To test one's knowledge of the topic.
4. To serve as a guide for students and other concerned professionals.
5. To train and evaluate trainees during training program
6. An effort will be made to provide a test protocol for Electronystagmography testing.

ANATOMY AND PHYSIOLOGY OF VESTIBULAR SYSTEM

Did you grow up thinking that there are only five senses: sight, hearing, taste, smell and touch?. This common idea ignores several other senses whose receptors are diffuse and less obvious such as pain and one that has a prominent receptor organ, the vestibular sense. Although we constantly use our vestibular sense, we are consciously aware of it on those unhappy occasions when it malfunctions or is over stimulated. Under normal circumstances, however, the vestibular sense constantly sends information to the brain about the head's position and its position changes, about the speed and direction of change. This information is vital in maintaining balance through postural adjustments, maintaining the proper relationship of head and body movements. Before we learn the assessment of vestibular system using electronystagmography. It is imperative that one understands the anatomy and physiology of the system responsible for one's equilibrium.

Vestibular Anatomy

To understand the physiology of vestibular apparatus an appreciation of the anatomy and ultra structure of the peripheral vestibular system and its central pathways is required. The inner ear or labyrinth lies in the temporal bone and is divided into bony and membraneous portion. The membraneous labyrinth contains the sensory epithelium of the cochlea and vestibular structures, lies within cavities surrounded by bony labyrinth. The peripheral vestibular system is an integral part of the labyrinth, which lies in the otic capsule in the petrous portion of the temporal bone. This otic capsule houses

both the cochlea and vestibular end organ. The cochlea and vestibular end organ being structurally interconnected are functionally different. The otic capsule is composed of three chambers: the cochlea interiorly, the peripheral vestibular system in the vestibule and the posterior vestibular chambers. The membraneous labyrinth is surrounded by perilymph and is suspended by fine connective tissue strands from bony labyrinth. The membraneous labyrinth consists of the saccule, utricle and semicircular canals and is filled with endolymph. The three bony semicircular canals open into the posterior vestibular chambers by means of five round apertures. The two vertical canals (the superior and posterior canals) join posteriorly to form a single cris commune. The membraneous labyrinth consists of five areas of sensory epithelium; the vestibular receptor organs: two maculae of otolith organ (utricle and saccule) and three cristae ampullares of the semi circular canals.

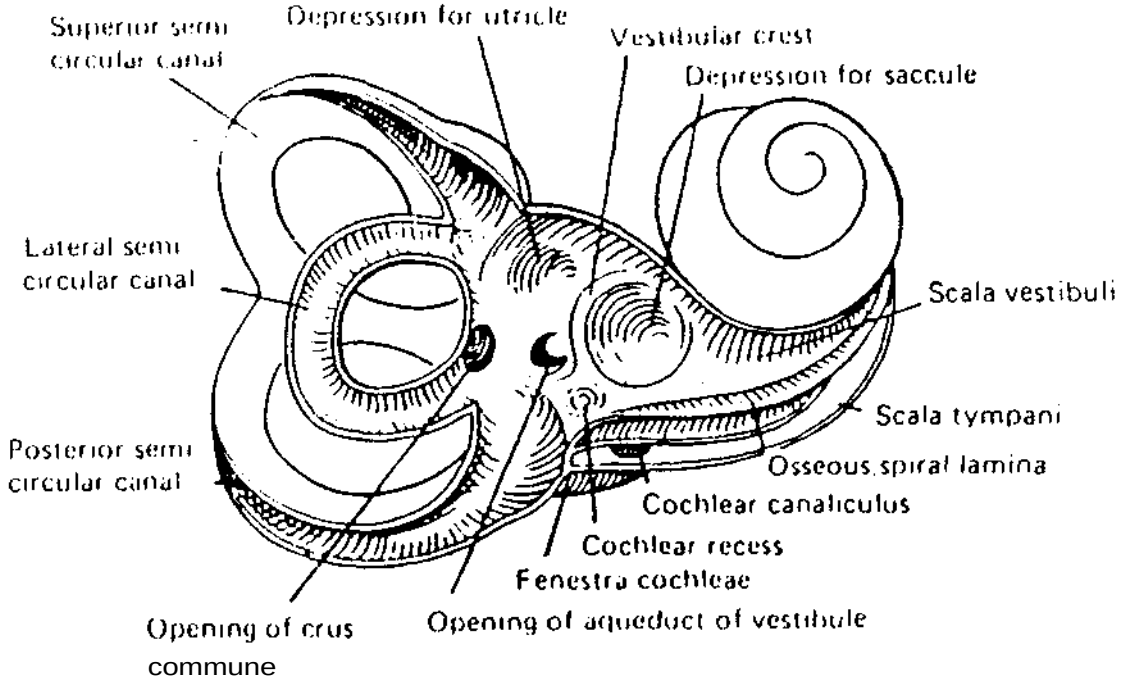


Fig 1: Bony Labyrinth View from Lateral aspect

Saccule, Utricle and Semicircular Canals

The Saccule lies in a recess near the opening of the scale vestibuli of the cochlea and is almost globular in shape. It is connected anteriorly by the ductus reunions to the cochlea and posteriori via the utriculo saccular duct to the endolympatic duct. On its anteroventral wall is the elliptical saccular macula - a thickened area of sensory epithelium. The utricle is larger than the saccule and lies posterosuperiorly to it. It is irregularly oblong in shape and is connected anteriorly via the utrculo-saccular duct to the endolymphatic duct. The three - semicircular canals open into it by five openings, the posterior and superior semicircular canals sharing one opening at the cris commune. On the floor of the utricle lies the sensory epithelium of the comma-shaped macula. The three semicircular canals are small ring like structures, each forming two-third of a circle. Each semicircular canal is a continuous ring of fluid, which is blocked by the cupula, a gelatinous plug that completely fills the lumen of the canal in its ampullated portion. One end of each canal is dilated to form the ampulla, which contains a saddle shaped ridge, the crista ampullaris, on which lies the sensory epithelium, vestibular sensory cells are embedded in the cristae ampullaris of the lateral, anterior vertical and posterior vertical semicircular canals.

Vestibular Receptor Cells

There are a total of 23,000 hair cells in the three human cristae (Rosenhall, 1972) and between 45,000 to 60,000 in the two maculae (Rosenhall, 1972). The hair cells are surrounded by supporting cells, which are attached to

the basement membrane, in which is found the neural and vascular tissue. The vestibular hair cells can be classified into two types: type I and type II. Type I cells are flask-shaped, while type II cells are cylindrical, although it is likely that the classification of the vestibular hair cell is more complicated than this. The sensory cells are neuroepithilial hair cells. Each bearing 50-100 thin steriocilea and a single thick and long kinocilium on the apical surface. The steriocilia vary in height, but are graded with reference to the kinocilium, the tallest about 100 micrometer, being closest to the kinocilium. The kinocilium projects from the cell cytoplasm through a segment of the cell lacking a cuticular plate.

Blood Supply

The blood supply of the labyrinth is derived principally from the internal auditory artery, which arises usually from the anterior interior cerebellar artery, sometimes directly from the basilar artery.

Applied physiology of the peripheral vestibular system

The functions of the vestibular system are

1 The detection of body motion (linear and angular acceleration) as monitored by head motion.

2. The detection of the head in space relative to the gravitational vector (tilt).

The five vestibular receptors in each labyrinth can be classified according to the specific stimulus to which they respond. The macula utricle and macula sacculi, collectively known as the otolith organs, form one type of

receptor; they respond to linear accelerations of the head. Linear in this case means straight line. The upper portion of both the steriocilia and kinocilium are embedded in an otolith membrane - a gelatinous structure that contains many small crystals of calcium carbonate called otoliths or otoconia. These crystals add weight to the otolithic membrane and enables it to respond to two type of stimulus. They are:

1. Tilting of the head

Gravity pulls on the heavy membrane and bends the ciliary tufts, causing excitation or inhibition depending on the direction of tilt.

2. Rapid linear acceleration or deceleration of the head

When the head moves, inertia delays the movement of the heavy otolith membrane and that causes it to pull on the cilia. This is similar to what happens to an accelerating car. When the car moves forward, you are pushed backward because your movement drags behind that of the car. If the brake is suddenly applied the car stops faster than you do and your inertia thrusts you forward.

As stated earlier, the hair cells are polarized by the placement of the kinocilium. Moreover, they are arranged in such a way that the kinocilia of half the hair cells of each macula 'Point' in one direction; those of the other half, in the opposite direction. In other words, half of the nerve fibers innervating maculae are excited by bending the head in one direction and half by sending the head in the opposite direction. Thus the maculae of the utricles and sacculus

provide the brain with constant updated information about the head's testing position and rapid linear movements.

The semicircular canals form the other type of receptor; they respond to angular accelerations of the head. The receptor hair cells of the crista ampullaris are present in each semicircular canal. The steriocilia and a kinocilium are embedded in a gelatinous membrane - in this case, a cupula - that totally block the semicircular duct. Unlike the otolith membrane, the cupula does not contain otoliths and therefore has the same approximate weight and specific gravity as the fluid around it; therefore it does not respond to gravity. It does respond to angular acceleration and deceleration and gives the brain precise information about head movements.

(When head and body moves together, it is a linear movement, detected by the maculae. When the head moves relative to the rest of the body, the movement is angular, due to the way the head articulates with the vertebral column. The cristae ampullares of semicircular canals are exquistely sensitive to these angular movements, partly because of the curved column of endolymph in each semicircular duct. When the head is rotated in the plane of a semicircular duct, inertia causes the endolymph to lag slightly: this pushes on the cupula in the direction opposite to the rotation and bends the ciliary tufts of the hair cell that are embedded in it. The bending of the ciliary tufts opens the potassium channels of the steriocilia, allowing potassium into the hair cells and activating them to release neurotransmitters; the neurotransmitters cause nerve

impulses in the vestibular division of the vestibulocochlear nerve. As in the maculae, the hair cells are direction sensitive: they are excited when the cilia bend towards the kinocilium and inhibited when they bend away from the kinocilium.

Furthermore, the ducts on one side of the head are paired with those on the other side for complementary coding. To illustrate, the right superior and left posterior ducts are in the same plane and respond maximally to movements that are down to the right and up to the left. They respond to lesser degrees to movements that deviate from this plane but such movements stimulate other canals to greater or lesser extent.

Response to Caloric Stimulation

The semicircular canal receptors are also sensitive to thermal gradient in the temporal bone. Audiologists and Neurotologists make use of this phenomenon to produce semicircular canal responses by irrigating the external auditory canal with warm or cool water (or air) in the caloric test. The caloric stimulation is more clinically useful than rotation, because the two labyrinths can be stimulated separately and their response can be compared. The caloric stimulus is usually administered while the patient is in the caloric test position, i.e., supine with his head ventroflexed by 30°. When the head is in this position, the lateral semicircular canals are vertical and are thus maximally sensitive to thermal stimulation. The mechanism of stimulation for a warm temperature irrigation is that the warm temperature wave transverses the temporal bone, the first site it reaches in the labyrinth is the most lateral portion of the lateral

Canal; thus it first warms the endolymph in this region. As the endolymph becomes warm, it becomes less dense and tends to rise, increasing the pressure on the lateral side of the cupula and making it deflect medially. Medial deflection on the lateral canal cupula excites its hair cells. For this reason, warm temperature irrigation excites the irrigated ear. Cool temperature irrigation produces a response in the opposite direction. The fluid in the most lateral portion is cooled, becomes more dense, sinks and deflects the cupula laterally, thus inhibiting the hair cells.

The combination of inputs from all six semicircular ducts gives very precise information about two things.

1. The direction of movement
2. The change in movement velocity because it is when the head starts, stops or changes velocity that the endolymph's inertia has its mechanical effect on the cupula.

Centra) Vestibular System

The vestibular ganglion (Scarpa's ganglion) of the vestibulocochlear nerve is made up of cell bodies of bipolar neurons that are the first order neurons of the vestibular system and lie in the internal auditory meatus. Their peripheral processes synapse with the hair cell of the maculae and the cristae ampullaris.

Their central process forms the vestibular division of the vestibulocochlear nerve. The cell bodies, peripheral and central processes are

myelinated. The vestibular division of the vestibulocochlear nerve enters the brainstem at the cerebellopontine. Most of its fibers synapse on second order neurons in the vestibular nuclei, which lie partially in the medulla and partially in the pontine tegmentum. There are four nuclei on each side; the medial, inferior, superior and lateral vestibular nuclei as shown in figure 2. The lateral vestibular nucleus also called Deiters nucleus is characterized by large, multi-polar neurons with heavy nissl substance. The other three vestibular nuclei, like most sensory nuclei contain smaller neurons with less nissl substance. Some vestibular nuclei receive only primary vestibular afferents but with the exception of the neurons of the interstitial nucleus. The majority receive afferents from cerebellum, reticular formation, spinal cord and conrralateral vestibular nuclei. The vestibular system also receives pathways from the visual system. The largest afferent supply to the vestibular nuclei crisis in the cerebellur. The exact afferent connections to and efferent projections from the vestibular nuclei have not been totally identified but the following generalization may be made about the four groupings of vestibular nuclei.

Superior vestibular nucleus

The afferent input is from cristae of semi circular canals and cerebellum. The efferent output runs in the median longitudinal bundle to innervate the motor nuclei of the extensive eye muscle. This nucleus is therefore particularly important in the control of the semicircular canal - ocular reflexes.

Lateral Vestibular Nucleus

The afferent input is from primarily from the cerebellum and utricular macule. The efferent are primarily involved in vestibular spinal pathways and interior part of the median longitudinal bundle.

The superior and lateral vestibular nuclei project to the thalamus in an anterior projection that run lateral to the red nucleus and dorsal to the sub thalamic nucleus to the ventral posterolateral nucleus of the thalamus. A small projection runs in the lateral lamniscus to end near the medial geniculate.

Medial Vestibular Nucleus

The afferent input is primarily from the cristae and cerebellum with a few fibres from the reticular formation and utricular maculle. The efferent output projects in the median longitudinal bundle to both the oculomotor nuclei and the cervical spinal cord. It is important in coordinating eye, head and neck movements. Other efferents from this nucleus are to vestibulocerebellum, the reticular formation and contralateral vestibular nuclei.

Descending Vestibular Nucleus

The afferent input is from the utricular and secular maculae. The efferent output is mainly to the cerebellum and reticular formation. In addition numerous commissioral fibers supply the contralateral ascending medial and lateral vestibular nuclei. The vestibular nuclei connects with the occulomotor nuclei, the spinal cord, the cerebellum, the autonomic nervous system, the thalamus and the contralateral vestibular nuclei (Furuya, Leawono and Shimazu, 1976). It is at the level of the vestibular nuclei, labyrinthine

information is integrated from other somatosensory systems. Electro-Physiological studies have identified two groups of secondary vestibular neurons - type I and type II. (Shimazu and Percht, 1966). Type I connections tend to be ipsilateral and excitatory. Whereas type II neurons are activated by contralateral type I or by neurons in the reticular substance. The result of head rotation is to stimulate the ipsilateral labyrinth and inhibit the contralateral labyrinth.

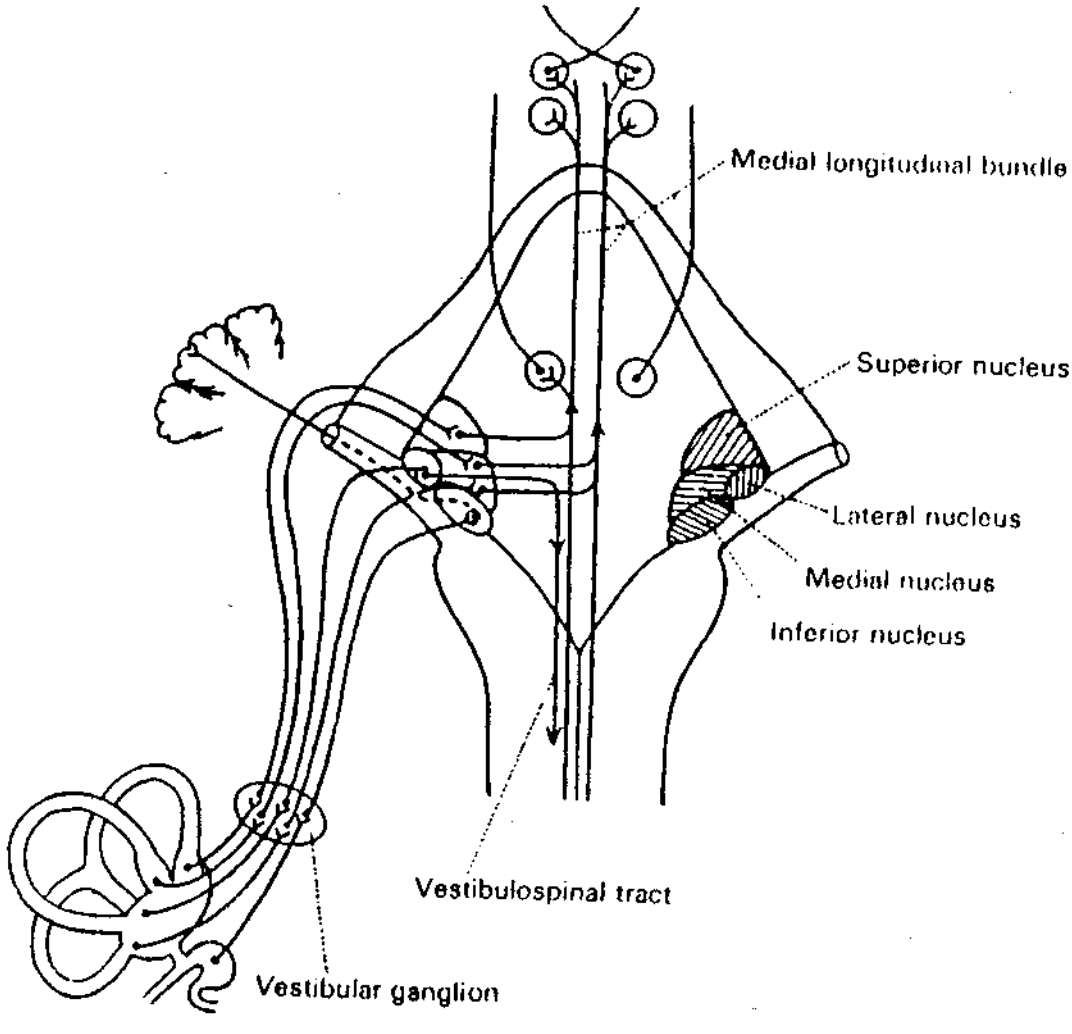


Fig 2: Central connections of vestibular nerve

Ascending Vestibular Projection

Following electrical stimulation of the vestibular nerve, monophasic potentials can be recorded in the contralateral and suprasylvian gyrus just an interior to auditory area (Watzl and Mountcastle, 1949). The thalamo-cortical projection and at the lower end of the interior parietal sulcus near the post - central gyrus (Fredrickson, Kornhuber and Schwatz, 1974). These thalamic and cortical areas also receive proprioceptive and visual projections.

Functionally there are three types of response following peripheral vestibular stimulation

1. The Vestibulo-occular reflexes
2. The vestibulo-spinal reflexes
3. The vestibulo-collic reflexes.

1. Vestibulo-occular reflexes

The vestibular system is extremely important in controlling conjugate eye movements reflexively in reponse to head movement and to the position of head in space. The vestbulo-ocular reflexes provide a simple example of a reflex arc, comprising the vestibular receptor, primary, secondary and tertiary neurons and the effector organ, the oculomotor muscles. An asymmetry resulting from action of the semicircular canals causes a compensatory reflex eye movement in the plane of the canals being stimulated (Baloh and Honrubia, 1979). This compensatory reflex movement of the eye is called vestibulo-ocular reflex. Vestibulo-ocular reflex includes the semicircular canal-occular reflexes and the otolith-ocular reflex.

A. Semicircular Canal-Ocular Reflexes

This is dominant of the vestibulo-ocular reflexes. The measurement of the lateral semicircular canal-ocular reflex, by examination of the oculomotor response to precise vestibular stimuli, is of immense clinical value. Under physiological circumstances, an angular acceleration produces an exact mirror image of events which take place simultaneously in opposite labyrinths. Each utriculopetal stimulus in one labyrinth is matched by an equal, but opposite, utriculofugal displacement in the functionally paired canal of the other ear. In this push pull arrangement, the lateral canals form a pair, while the posterior canal, because of its anatomical disposition, is parallel to, and therefore paired with the opposite superior canal. The difference in input to the vestibular nuclei from the left and right labyrinths is the basis of the vestibular response and mediates all labyrinthine reflexes.

B. Otolith - Ocular Reflexes

Stimulation of the uticular nerve induces eye movements (Suzuki, Tokumasu and Cohan, 1969), but the otolith control of extraocular muscles has proved more difficult to delineate than semicircular relationship. The sensory receptors of the otolith organs are oriented towards many different planes. Nevertheless rotational and torsional compensatory eye movements, produced by head tilt, are well documented. In humans, counter torsional movements are produced by lateral tilt, while vertical rotation results in forward / backward tilt.

2. Vestibulo Spinal Reflexes

The labyrinth influences posture and orientation through neck, axial and limb motoneurons. The vestibular apparatus exerts an influence on the control by way of the myotatic reflex (the deep tendon reflex), which is the elementary unit for the control of tone in the trunk and extremity skeletal muscles. This reflex is under the influence not only of the vestibular system but also of the multiple supraspinal centers including basal gangalia, the cerebellum and the reticular formation.

3. Vestibulo Collic Reflexes

The role of these reflexes in man is uncertain (Outerbridge and Jones, 1971), however, persons lacking vestibular function have impaired head stabilization in response to unpredictable oscillations (Bronstein, 1988).

Gaze Fixation

An important role of the vestibular system is the maintenance of the fovea on a fixation point during changes of head and body position. The interaction between visual, vestibular and cervical information enables a more precise eye movement to be achieved and thus better occular stability then would be possible if only one system alone were functioning. The vestibulo-ocular and cervico-ocular reflexes fixate the fovea upon its target with compensatory eye movements that rotate the eye on the opposite direction to the head movement. These reflexes are modulated by the action of central vestibular structures and depend on the integrity of the pathways mediating eye

movements. A disturbance of this function can cause vertigo and oscillopsia. The visual, vestibular and proprioceptic system can work in conjunction to maintain the fovea on newly appearing visual targets. Many areas of cerebral cortex are involved in eye movement control, including the frontal, occipital and occipitoparietal lobes. In area 8 of each frontal lobe lies the "frontal eye field". Stimulation of which causes contralateral conjugate deviation of the eyes and pathology of which causes ipsilateral conjugate deviation with the inhibition of ipsilateral pursuit eye movements. Area 18 and 19 of the occipitoparietal lobes are involved with slow pursuit eye movements.

While the response to stimulation of the peripheral vestibular apparatus or cervical proprioceptors is a compensatory eye movement, there are many other causes of eye movement: both voluntary and involuntary. Among the involuntary eye movements are nystagmus, saccadic intrusions, ocular flutter, opsoclonus, ocular spasms, ocular myoclonus and ocular bobbing. Voluntary eye movements are saccades. The volitional or involuntary following of a . visual target is affected by a pursuit eye movement or optokinetic nystagmus.

There are three visually controlled occulomotor system, which are of clinical importance in terms of their relationship with the vestibulo ocular reflexes. They are

1. The Saccadic system
2. The pursuit system
3. The Optokinetic system

The Saccadic System

A saccade is a fast eye movement around 350-600 degrees per second increasing with increasing amplitude of eye movement (Robinson, 1964). They can be voluntary or involuntary. Voluntary saccades are used to move the eye between targets in the shortest possible time. The rapid eye movements of the fast phase of nystagmus and the rapid eye movement phase of sleep are involuntary saccades. Thus saccades can be generated as part of a reflex, volitionally towards a target or in the absence of a visual target as for example towards a remembered target. The purpose of the involuntary saccadic system is to maintain the target on the fovea. The direction of the saccade is such as to move the orbit rapidly towards target already moving towards the fovea i.e., the saccade resets the fovea on a target. In a rotation to the right, the new target is coming from the right. So the saccade is to the right.

The saccade is generated by groups of 'burst' neurons in the paramedian pontine reticular formation and pretectal region. The burst neurons in the paramedian pontine reticular formation fire in short burst just before the onset of involuntary horizontal fast saccades or involuntary saccades. The burst neurons in the pretectal region generate vertical saccades (Hoyt and Daroff,1971). The pretectal areas for downward saccades are distinct from those for upward saccade. The eye position is maintained by tonic cells, which continue to fire after the saccade is complete (Cohen and Henn, 1972). The ablity to make saccades may depend on the integrity of projections between the

frontal eye field, caudate nucleus, substantia nigra reticulata and the deep and intermediate layers of superior colliculus.

Ocular Stabilizing Systems

The visual system itself acts to stabilize the fovea on visual targets during head and environmental change using smooth pursuit eye movements and optokinetic nystagmus.

The Smooth Pursuit System

In humans, the smooth pursuit is responsible for maintaining gaze on moving target, so that the target is stabilized on the fovea. The pursuit system monitors the rate of slippage on the retina and sends this information to the premotor system, a control system designed to reduce the slippage to zero. The smooth pursuit system is of particular clinical importance as it is considered to be intimately related to the mechanisms by which the vestibular ocular reflex is suppressed by optic fixation and this is of utmost importance in differentiating peripheral from central vestibular pathology.

The smooth pursuit system has its origin in the sensory cells of fovea. The afferent limb passes in the optic nerve to the ipsilateral and contralateral lateral geniculate bodies and calcarine cortex.

Pursuit movements are for the most part, involuntary. Although it is true that one can disregard a moving object if it is small and the visual background is stationary, one is almost compelled to follow movements of the whole visual fields. Pursuit movements, unlike saccade, usually cannot be made in the absence of a moving visual target. If the pursuit movement carries the eye

beyond the limits of gaze, it is periodically interrupted by saccades in the opposite direction and the result is optokinetic nystagmus.

Optokinetic nystagmus

Optokinetic nystagmus is a reflex oscillation of the eyes, induced by movement of large areas in the visual field. The most common example of this phenomenon can be observed in the jerking eye movements of a train passenger as he views a landscape whose features traverse the field of vision with the motion of the train. In everyday life, the optokinetic response rarely act independently, but interacts with the vestibulo-ocular reflex during the execution of spontaneous head movements and with the smooth pursuit system during the visual following of a moving target.

Ter Broak (1936) identified two types of optokinetic nystagmus : the first was "active" and was elicited by attempting to follow a series of small moving target, the second was "passive" and was elicited by the movement of the entire surroundings. The first causes a "look" nystagmus, the second stare nystagmus. Different pathways mediate the "look" and "stare" nystagmus. The "look" response involves the fovea and calcarine cortex (Zee et al., 1982) and may share the same pathways as the smooth pursuit system. The 'stare' response involves the peripheral retina and crosses the optic chiasm via the accessory optic tract, the afferent pathways reaches the contralateral mid brain nuclei and interior olive and the ipsilateral flocculus (Maekawe and Simpson, 1973) from the midbrain a further pathway reaches the ipsilateral paramedian

pontine reticular formation before crossing to the contralateral vestibular nuclei, thus bypassing the focculus (Baloh, Yee and Honrubia, 1982; Honrubia et al., 1982 ; Yee et al., 1982).

This chapter being an introductory chapter in understanding anatomy and physiology of the vestibular system with its central connection is essential for understanding the subsequent chapters.

BASIC PRINCIPLES AND DEFINITIONS

Electronystagmography (abbreviated as ENG) is the electrical recording of a particular type of rhythmic, involuntary, back and forth eye movement called nystagmus. Although on rare occasions nystagmus may be produced in other ways, it usually originates in either the visual or vestibular system. Electronystagmography is a process that provides means of tracking eye movements behind closed eyelids or in darkened environment. Electronystagmography owes its existence to the fact that the eye is a battery. The cornea is the positive pole, the retina is the negative pole and the potential difference between the two poles is normally at least 1 microvolt. This electrical potential, called the corneoretinal potential, creates in the head an electrical field that changes in orientation as the eyeballs rotate. The corneoretinal potential is a direct current electrical potential of approximately 1 microvolt. Because of this electrical potential difference between the positive cornea and the negative retina, the eye acts as a dipole. Although the corneoretinal potential is quite small, it may be detected at some distance from the eye. These electrical changes can be detected by electrodes placed on the skin, when the changes are amplified and used to drive a writing instrument, a tracing of eye position is obtained. When the eyes are at midposition, there is a certain voltage between the electrodes that serve as a baseline. The recording system is arranged so that as the eyeballs move to the right, the change in voltage between electrodes cause an upward pen deflection and when the eye balls move to the left, the change in voltage causes a downward deflection.

This horizontal eye movement is monitored by two electrodes placed bitemporally, i.e., one on the right temple and the other on the left temple. A second pair of electrodes, one above and the other below one of the eyes, is used to monitor vertical position of the eye on another channel of the recording instrument. Upward movement produces on upward pen deflection and downward movement produces a downward pen deflection. The way in which the corneoretinal potential permits recording of eye movements is shown in the figure. 3

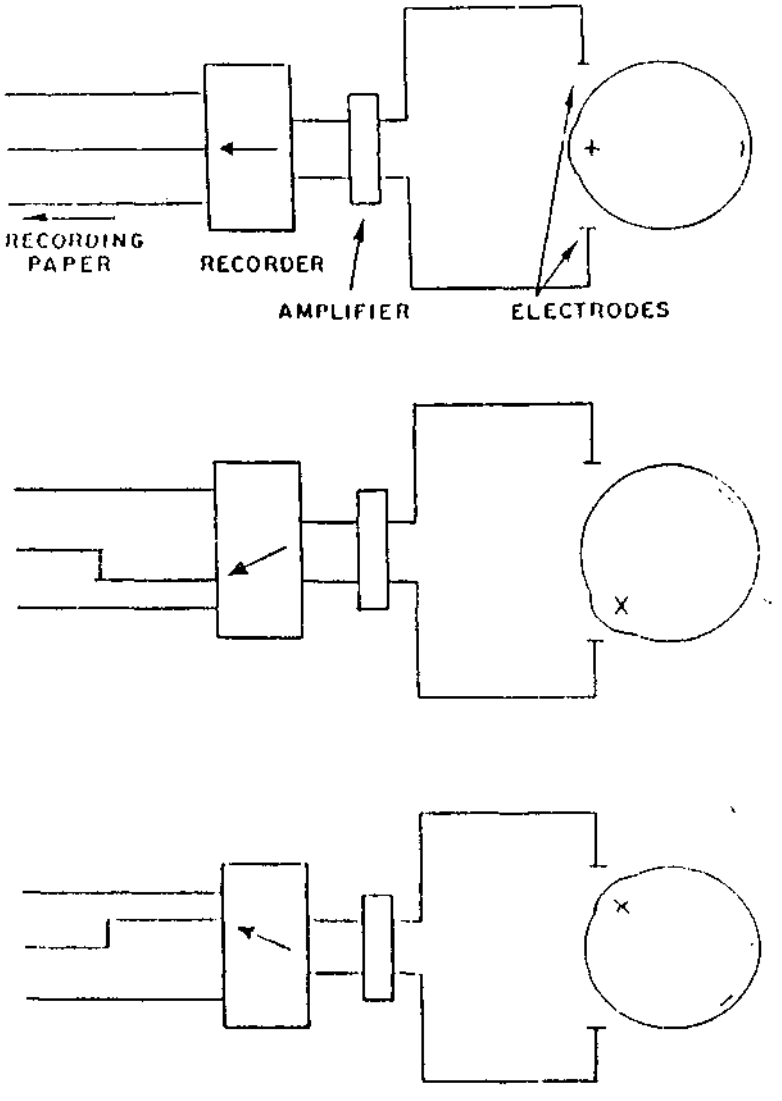


Figure 3: The principle of electrical recording of eye movements. Eye movement to the left creates a downward deflection of the pen and eye movement to the right creates an upward deflection of the pen.

When the eye deviates to the left the positive voltage in the front of the eyes is more nearer to the left electrode, and the negative voltage at the back of the eyes moves nearer the right electrode. Because the tissue of the body conducts electricity, this eye movement will create a voltage difference between the electrodes, with the left electrode positive and the right negative. This difference is amplified and made to drive a pen writing recorder to produce the Electrony stagmogram.

Attaching the Electrodes

A. General considerations

The electrode usually used in the Electronystagmography examination is a silver or gold disc about 4 millimeter in diameter, which is often slightly cupped to hold the electrode paste. It is very easy to put Electronystagmography electrodes on, but it is not easy to put them on appropriately. Care and attention to detail are very important and can make the difference between very good and very bad records. When attaching the electrodes one must try to achieve

1. The lowest possible electrical resistance between the electrode and the skin.
2. Placement of electrode as close as possible to the eyes consistent with stability of the electrodes when the patient blinks or move his eyes.

Low electrode resistance is achieved by thoroughly cleaning the skin as the electrode site and by being certain that the electrode is taped as firmly as possible to the skin.

Procedure

1. Preparation: Seat the patient in a chair of sufficient height so that you can comfortably reach his eyes. Place the electrodes and the previously cut squares of adhesive tape within easy reach
2. Choosing application sites: Standard bitemporal electrode placement for routine Electronystagmography examination is shown in the figure 4 given below.

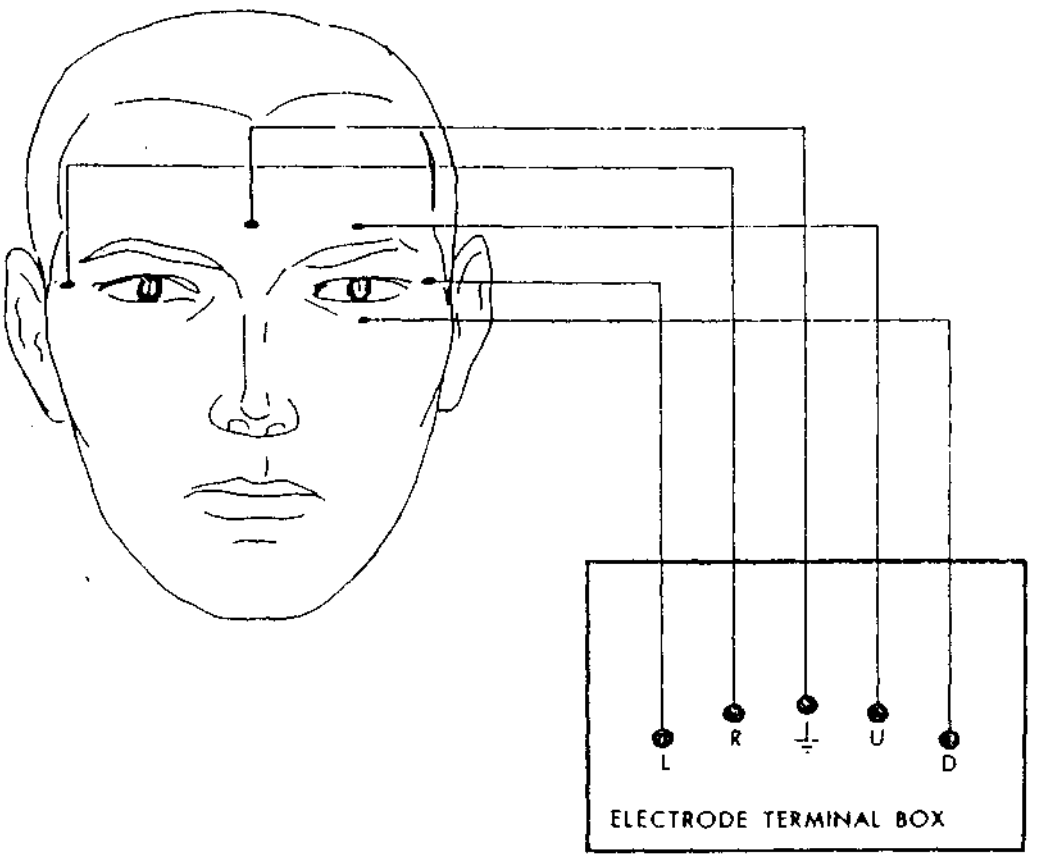


Figure 4 : Electrode placement for recording horizontal and vertical eye movements

Ground electrode should be placed in the middle of the patients fore head. The horizontal electrode records horizontal eye movements and should be placed just lateral to the outer canthi. They should be as close to the eyes as possible but far enough away that they do not move when the patient blinks his eyes. On older patient's with folds of loose skin about the eyes, it will be necessary to place the electrodes some what farther away from the eyes than on younger patient's. The vertical electrodes will be placed above the eyebrow and on the ridge of bone just beneath the eye. They must be in line through the patient's pupils when he is looking straight ahead. As with the horizontal electrodes, the vertical electrode sites should be chosen to minimize eye-blink artifacts. However, with the vertical electrodes being used frequently it will be impossible to eliminate blink artifacts.

Recording the movements of each eye individually

The standard bitemporal electrode placement adds together the movements of both eyes. When a patient with one blind eye is encountered, it may considerably improve the Electronystagmography record if the electrodes are placed so that movement of only the good eye is recorded. To do this, one electrode is placed on the good eye side of the bridge of the nose and other electrode is put in the standard position (i.e., lateral to the outer canthus) on the good-eye side of the head. Single-eye records do not always improve the results obtained from a patient with one blind eye. Therefore, at the beginning of the examination, obtain two calibrations, one with the electrodes across both

eyes and the other with the electrodes across only the good eye and on the basis of these calibrations select the best method of recording.

Cleaning the electrode sites

Electrode placement sites must be cleaned with cotton pledget saturated with ether-acetone and then squeezed out. One must be careful to keep the ether-acetone out of the patient's eyes. All make- up and surface skin oil must be completely removed.

Applying electrode paste and electrodes

It is easier to place the electrode symmetrically if they are applied in pairs (i.e., attach both horizantal electrodes before starting with the vertical electrodes). Later, the electrode and the electrode site with a thin layer of paste. Slightly rub the paste over the electrode site. The paste should cover a circular area of the skin only slightly larger than the electrode. It is important to apply just the right amount of paste. If too much is applied, it will squeeze out when the electrode is taped down. It gets between the tape and the skin and prevents secure placement of the electrode. If too little paste is applied, the electrode will not make good contact.

The electrode must be taped in place and there must be at least 4mm of tape on all sides of the electrode. After all electrodes are in place, tape the wires to the patients shoulder, leaving enough slack so that the patient can turn his head freely in all directions. This is done so that if the wires should be inadvertently pulled on the stress would be applied where they are taped to the shoulder and not to the electrodes.

Electrode Impedance

After the electrodes are placed appropriately, the electrode impedance test needs to be done. In an electrode impedance test, one can see the impedance of each electrode, but the quality of the electrooculography signal is related to the difference between the electrode pair for any channel. In order to check the impedance of an electrode in a particular channel one must have the common electrode attached to the skin as well as both electrodes for other channels. A valve of 5-kilo ohm for the difference between electrode pair is considered acceptable. If the impedance difference is higher than 10-kilo ohm a noisy signal is possible. In the event of higher impedance then it is necessary to re-clean the patient's skin to set a better signal especially if the patient uses a lot of make-up. Then reattach the corresponding electrodes.

Calibration

The next step is to calibrate the recording system. First the tester must determine whether the electrode polarities are correct before asking the patient to look alternately to right and left, then up and down. The pen of the channel to which the bitemporal leads are connected should move upward when the patient looks rightward, and the pen of the channel to which the vertical leads are connected should move upward, when the patient looks upward. If the pen of either channel moves in the wrong direction the appropriate leads should be reversed. This electrode polarity is arbitrary but conventional in clinical Electrony stagmography.

Calibration of the horizontal channel is performed by asking the patient to fixate alternatively on two dots or lights placed on the wall or digital light bar, so that they are separated by a known horizontal distance (usually 20 visual angle). As the patient performs this task, the tester adjusts the gain control of the horizontal channel so that the pen moves 1 millimeter per degree of eye displacement. Calibration of the vertical channel is done in the same manner while the patient alternately fixates on two other dots or light placed known vertical distance apart.

Once the calibration has been performed, the patient is ready for testing. The tester cannot assume, however, that the initial calibration will remain valid for the entire testing period. The calibration changes during testing in an unpredictable manner because of fluctuation in the magnitude of the corneoretinal potential, therefore, recalibrations must be performed at regular intervals, preferably before each caloric irrigation and before each of the other major test. Major subtests in Electronytagmography are discussed in the subsequent chapters. Before understanding the testing procedure it is essential to understand some basic concepts like what is a nystagmus, how to identify vestibular nystagmus, identify the direction of the nystagmus, measure the intensity of the nystagmus and to know the various types of nystagmus.

Nystagmus

The term nystagmus is derived from the Greek word nystagmos "to be sleepy" and nystazein "to nod". Medically it refers to a repetitive involuntary,

oscillatory movements of the eyes. Nystagmus may be undulating (Pendular nystagmus - sinusoidal movements of the eyes) or rhythmic (Jerk nystagmus - eye movement which has a slow velocity in one direction and a fast velocity in the opposite direction). The most common way of describing a nystagmus is in terms of the plane of eye movement (i.e., horizontal, vertical and rotatory) and the direction of eye movement (right, left, up, down, clockwise or counter clockwise). By definition, the direction of the nystagmus is named after the direction of the quick component. Although the slow phase is the vestibular phase of vestibular nystagmus, it is the convention to designate the direction of the vestibular nystagmus by the direction of the fast phase, which is mediated by the reticular formation. This convention originated because the fast phase is easier to see than the slow phase (figure 5).

Thus according to convention a rotational nystagmus beats in the direction of the rotation, and the nystagmus which is induced by a cold caloric irrigation of a subject in the supine position beats away from the irrigated ear.

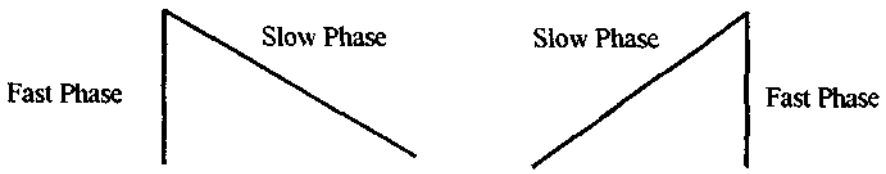


Figure 5 : A right and left beating nystagmus

A multitude of classification systems have been proposed for nystagmus. This has resulted in confusion and overlap in terminology.

Nystagmus can be classified into the following categories:

1. Ocular (visual) Nystagmus
2. Peripheral Nystagmus
3. Central Vestibular Nystagmus

1. Ocular (visual) Nystagmus

The term ocular nystagmus can be defined as nystagmus that is produced by reflex as affecting the retina, eye muscles, optic nerve and their central projections. Ocular nystagmus may be congenital, gaze, central and optokinetic (OKN) nystagmus.

I. Ocular-congenital Ocular Nystagmus

This form of nystagmus is characterized by an undulatory (sinusoidal movement of the eyes with an equal velocity in all direction) or rhythmic nystagmus (which has a slower velocity in one direction than the other). A characteristic feature of this nystagmus is its variability especially with changes in direction of size. Vertical nystagmus is not seen in congenital ocular nystagmus, and upward gaze usually produces a horizontal or undulatory nystagmus. It is also characterized by the presence of a null paint located off central gaze where the nystagmus is markedly diminished or disappear and the marked reduction or absence of nystagmus with convergence.

(i) Ocular-Gaze nystagmus

Gaze nystagmus is referred to nystagmus produced by changes in eye position. The presence of nystagmus in extreme lateral and medial gaze is normal. The fast phase is towards the direction of gaze, the slow phase is away.

The presence of nystagmus at a gaze of less than 30° is considered pathologic. If a gaze nystagmus is present bilaterally. It will beat in opposite direction with right and left gaze. Gaze nystagmus is generally not present in the midline position.

(ii) Ocular - Central Ocular Nystagmus

Rebound nystagmus is a type of central gaze nystagmus, that is opposite beating and occur in the position of primary gaze after a gaze nystagmus is elected. It is felt to be due to cerebellar dysfunction. Central ocular nystagmus can also be produced by dysfunction of the medial longitudinal fasciculus. This nystamus is characterized by a gaze nystagmus when looking towards the lesion (from damage to the ipsilateral pons) and a vestibular nystagmus when looking away (from damage to the ipsilateral vestibular nerve and nuclei).

(iii) Ocular - Optokinetic Nystagmus

It is an induced rhythmic nystagmus resulting from attempting to follow repetitive visual targets moving across the visual field. A rotating drum with contrasting stripes parallel to the axis of rotation is often used to elicit an optokinetic nystagmus.

II. Vestibular Nystagmus

Peripheral vestibular nystagmus is caused by an imbalance in the resting discharge rate of the paired peripheral end organs, either the semicircular canals or otolith organs. The nystagmus may be spontaneous or induced by a myraid of stimuli. In certain pathologic conditions, nystagmus may be induced

by a static change in head position due to stimulation of the spinovestibular tract or gravitational effects on the semicircular canals.

(i) Vestibular - Spontaneous nystagmus

According to Coats (1971), nystagmus that is present with eyes open or closed and fixed in the midline is called spontaneous nystagmus. Spontaneous nystagmus is persistant and does not require an eliciting stimulus. Spontaneous nystagmus can be classified as 1° when present only in lateral gaze in the direction of the fast component (for example away from labyrinthine Lesion); 2° when present in midline gaze and in the direction of fast component; 3 when present in lateral, midline and medial gaze. This classification of spontaneous nystagmus was given by Alexander et al., as early as 1924. It should be noted that according to Coats (1971), Alexander's 1° spontaneous nystagmus would be classified as a gaze nystagmus. Disorders which produce spontaneous nystagmus include labyrithitis, meniere's disease, brain tumors and trauma. Spontaneous nystagmus resulting from permanent non-progressive unilateral decrease in end organ output should only last 2 to 3 weeks, after that central compensation will occur and the nystagmus will cease.

(ii) Vestibular - Positional Nystagmus

Positional nysatgmus was defined by Nylen (1931) to indicate nystagmus produced by changes in head position. Positional nystagmus is described interms of its latency (time period from change in head position to initiation of the nystagmus), duration (total length of time that the nystagmus

beats), fatigability (shorter duration of nystagmus on repeated stimulation), and direction. The direction of nystagmus can be defined and anatomically (right or left) or in relationship to gravity (geotrophic, towards earth; ageotrophic away from earth).

Nylen proposed a classification of nystagmus in 1931 and according to him, positional nystagmus can be classified as

1. Nylen type I: Direction changing positional nystagmus. The nystagmus beats in only one direction in each head position but the direction changes with head position.
2. Nylen type II: Direction fixed nystagmus. Nystagmus beats in the same direction in all positions. It is present in only one position or shows marked attenuation in intensity in the other positions.
3. Nylen type III: Irregular positional nystagmus. Nystagmus that may change direction in a given position. This group also includes nystagmus not included in type I and II.

Aschan et al., (1956) modified Nylen's classification by including the terms persistent and transitory nystagmus. Persistent nystagmus is defined as nystagmus with an infinite duration. Transitory nystagmus is defined as nystagmus with a short duration (usually less than 60 sec)

Aschan Type I: Direction - changing persistent nystagmus
Aschan Type II: Direction - fixed, persistent nystagmus
Aschan Type III: Transitory nystagmus, direction changing or direction fixed

The other most common type of positional nystagmus is rotatory or elliptical. Rotatory nystagmus is described as clockwise or counterclockwise or as right or left. Right and left refers to the direction of the quick components

vector along the globe at 12 'o' clock. Thus a clockwise nystagmus can also be described as to the left. Horizontal direction fixed positional nystagmus of upto 6° / sec (slow phase velocity) is normal in adult subjects evaluated with their eyes closed provided it is present in only two of five head positions tested.

(iii) Vestibular-Positioning Nystagmus

Barber and Stock well, (1976) refers to positioning nystagmus as that elicited by quick movements of the head, i.e., Hallpike maneuver. Uemura et al., (1976), defines positional nystagmus as persistent nystagmus which is induced by different head positions and positioning nystagmus as a transitory nystagmus which is induced by different head position.

III. Central Vestibular Nystagmus

Central nystagmus refers to nystagmus that is not characteristically elicited or found in normal subjects or subjects with end organ disease. Central vestibular nystagmus is produced by dysfunction of the central vestibular system. The following are some of the central vestibular nystagmus:

(i) Central vestibular - Inverted Nystagmus

This refers to nystagmus, which beats in the opposite direction than expected in the caloric stimulation.

(ii) Central Vestibular - Periodic Alternating Nystagmus

This nystagmus is characterized by a rhythmic nystagmus which builds to maximum velocity in one direction then slowly diminishes and starts beating in the opposite direction. This cycle is repeated indefinitely. Periodic

alternating nystagmus is distinguished from direction changing, positional nystagmus in that the former is recorded during testing for latent and spontaneous nystagmus and the latter is elicited during positional testing.

(Hi) Central Vestibular - Vertical Nystagmus

Vertical nystagmus strongly indicate central lesions. Vertical nysagmus may be upbeating or downbeating. Up beating may suggest lesions in the posterior fossa or drug intoxication, down beating may suggest a lesion in the medulla or upper cervical spinal cord.

(iv) Central vestibular - cervical nystagmus

This refers to nystagmus produced by changes of the labyrinth in relationship to the subject's body. This nystagmus is often pathologic and felt to be from the input of the spino vestibular tracts on the vestibular nuclei in the brain stem.

Measurement of strength of nystagmus

Whether one induces nystagmus by rotation, changes in position or caloric's, a method of quantifying the nystagmus response is needed. There are three ways of measuring nystagmus i.e., Duration of nystagmus, frequency (i.e., number of beats per unit time) and velocity of slow phase component. The velocity (slope) of the slow component is the parameter most widely used in clinical practice.

The following chapters will describe the various subtests of Electronystagmography which gives a bird's eye view of the procedure and test interpratation.

GAZE TEST

The function of the gaze system is to maintain visual fixation of an object on the fovea of the eye (area on the retina of greatest visual acuity) during fixed visual gaze.

Procedure

During the gaze test, the patient is asked to fixate visually on a stationary target placed directly in front, 20° or 30° to the either side of the center i.e., 20° or 30° to the right or to the left, and 20° or 30° above and below the central fixation point. Eye movements are recorded for at least 20 sec with eyes open and at least 20 sec with eye closed in each gaze position. The clinician must alert the patient to maintain the gaze position and not to let his eyes wander.

The primary purpose of the gaze test is to detect nystagmus. Nystagmus usually becomes weaker and may disappear entirely when the patient becomes relaxed. Here the patient needs to be alerted by giving certain alerting task like, counting forward or backward or giving a string of mental arithmetic problems, asking him to supply answer after each problem.

Normal Variations

Many normal individuals are able to maintain a steady eye position with eyes open or closed in all direction of gaze as shown in figure 6. Others permit their eyes to wander about and these deviations can became even longer with their eyes are closed. Some normal but highly aroused tensed individually

display square ware movements when their eyes are closed. Normal persons may also exhibit sinusoidal oscillations of the eye at a frequency of approximately 0.3-Hertz. This type of eye movement indicate that the person is drowsy and must be alerted if nystagmus is being sought.

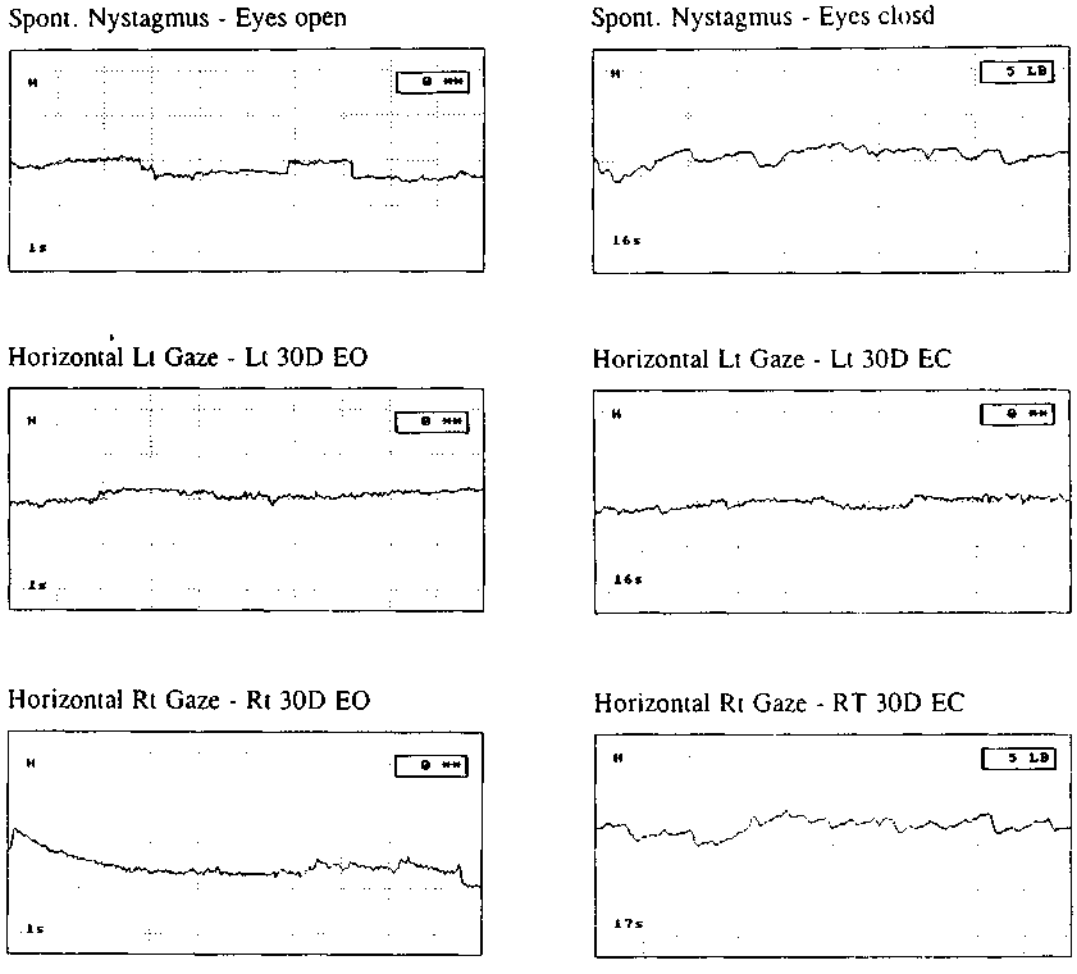


Figure 6 : Normal gaze response and Gaze test, in eye center, left and right gaze positions

Diagnostic Significance

Nystagmus may not be present with the eye's centered, but may appear when the eyes are deviated from the center. Gaze nystagmus is any nystagmus that appears when the eyes are moved away from the center.

1. Direction changing gaze nystagmus

If gaze nystagmus is present and its direction changes when the patient changes the direction of gaze (eg. Right-beating nystagmus with right lateral gaze, left-beating nystagmus with left lateral gaze) is indication of central lesion with lesion is isolated in the brain stem/cerebellum and not in the peripheral vestibular system. In addition, the right beating nystagmus will be most intense, the further the eye is deviated to the right. The left-beating nystagmus will be most intense the further the eye is deviated to the left. This phenomenon is a function of Alexender's law that states that the nystagmus beats most intensely when the eyes are deviated towards the side of the fast phase of the nystagmus.

2. Direction fixed gaze nystagmus

If the gaze nystagmus beats in only one direction (right-beating or left-beating) and horizontally, irrespective of the patient's direction of gaze (figure 7). The cause is due to an acute, unilateral peripheral vestibular lesion. This is referred to as a direction-fixed gaze nystagmus and is caused by the fact that one peripheral vestibular system is weaker in its neural output than the opposite side. This asymmetry creates a neural imbalance in the vestibulo-ocular reflex that causes the eyes to be pulled (slow phase of the nystagmus) towards the weaker ear, followed by a rapid saccade (fast phase of the nystagmus) directed towards the stronger ear. In other words, a peripherally based vestibular nystagmus will beat away from the weaker ear in most cases. In addition, a gaze nystagmus of peripheral origin should increase in intensity

and amplitude when visual fixation is eliminated by eye closure or in darkness. It is strongest when the eye gazes in the direction of the fast phase and it declines in intensity as time passes, because of central compensation.

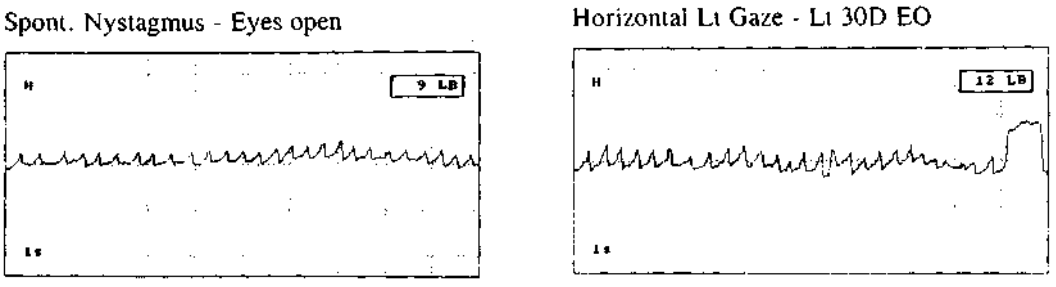


Figure 7 : Direction fixed Gaze nystagmus in center and left eye open positions

3. Periodic Alternating Nystagmus (PAN)

(Periodic alternating nystagmus is a form of gaze nystagmus that usually is present in the primary (center) gaze position. This nystagmus changes direction every 2 to 6 minutes and includes a null period between each half cycle. This is, without the patient, changing his or her direction of gaze, the nystagmus beats in one direction for a couple of minutes, stops and beats in the opposite direction before stopping only to repeat the cycle. This condition often is secondary to a cerebellar lesion, although it also can be seen in patient's with space occupying or vascular lesions of the brain stem and mid brain, and in some cases no evident pathology is found.

4. Rebound Nystagmus

Rebound nystagmus is a type of central gaze nystagmus, that is opposite beating and occur in the position of primary gaze after a gaze nystagmus is elicited. It is felt to be due to cerebellar dysfunction.

5. Spontaneous Ocular Square Waves

Spontaneous ocular square wave observed during the gaze test often are caused by lesion of the brain stem / cerebellum, although this type of eye movement sometimes is characteristic of a tense or nervous patient.

6. Vertical Nystagmus

Vertical nystagmus may be found on gaze upward or downward and less frequently in the primary position. Its recognition is of great clinical importance, and is caused almost invariably by central nervous system disease usually in the brainstem.

6.1 Up beating Nystagmus

Up beating nystagmus is fairly common in brainstem lesions and usually present when the eyes are deviated upwards.

6.2 Down beating Nystagmus

Down beating gaze nystagmus has been reported in patient's with lesions of the vestibular nuclei (Cogan, 1977) and with lesions in the flocculus of the cerebellum. This conditions usually causes a down beating gaze nystagmus when the eyes are in the primary gaze position as opposed to the lateral gaze position. Down beating nystagmus, especially with lateral gaze is

observed frequently in lesions of the cervicomedullary junction (Barber and Stockwell, 1976).

7. Rotatory Gaze Nystagmus

Rotatory nystagmus is rotation of the eye around an axis, and is not recordable using an electronystagmography instrument. Clinician must visually observe the presence of rotatory nystagmus. Rotatory nystagmus usually is consistent with a brain stem lesion, often involving the vestibular nuclei. It is observed in such disease process as multiplesclerosis, fourth ventricle cysts, and space occupying lesions that distort the fourth ventricle where the vestibular nuclei are housed. Rotatory nystagmus has been reported in cerebellar disease as well (Zee, 1987). In very early stages of an acute, unilateral peripheral vestibular lesion, a rotatory component to predominantly horizontal nystagmus may be present, and this should be considered when a patient is in an acute stage of vertigo.

Pitfalls

There are several possibilities for serious errors when one is performing or interpreting the gaze test.

Effects of Drug

Eye movement abnormalities provoked by drugs like wandering pendular eye movements or jerk (often oblique or vertical) nystagmus may be seen. Some drugs especially barbiturates, antihistamines, and tranquilizers, suppress nystagmus by lowering alertness, therefore nystagmus may not be

observed. All patients referred for Elecrronystagmography examination should be instructed to avoid any but life-supporting drugs for 48 hours before testing, the exception being epileptic patients, who should not discontinue medication taken to control seizures.

Congenital Nystagmus

The term congenital nystagmus is used to describe nystagmus that appears at birth or soon after in other wise healthy individual. From an Elecrronystagmography point of view, it is extremely important to identify this special form of nystagmus because it is non-lesional. Congenital nystagmus may be pendular or jerky type in character. In congenital nystagmus there is a particular direction of gaze called null point, at which the nystagmus declines mostly or stops. Congenital nystagmus is nearly always horizontal or rotatory and rarely vertical. An important features of congenital nystagmus is that nystagmus on gaze upward is virtually always horizontal, not vertical. Another important feature of congenital nystagmus is reduction or abolition of the nystagmus on convergence.

For cases in which the differentiation of congenital from acquired nystagmus is difficult, exploration of these three features - null point, upward gaze, and convergence effect - may provide decisive information.

Alertness

Nystagmus may be abolished or reduced in intensity or amplitude if the patient is not mentally alert or if he is drowsy. So patients need to be constantly

alerted, especially during parts of the test in which their eyes are closed. For this the patient may be instructed to perform some arthematic problems.

Excessive Eye Deviation

The clinician searching for nystagmus on eccentric gaze should ask the patient to deviate his eye no more than 30° from the primary position. If the patient is asked to deviate his eye too far (for example say 40° from primary position) nystagmus will occur in perhaps 75 % of normal individuals. This is called physiologic end point nystagmus. The punctum of the lacrimal sac on the lower lid may be used as a reference point for horizontal gazes, when eyes are open.

SACCADIC TEST

Saccades are fast eye movements created by the saccadic system. The velocity of these movements varies between 200° /sec and 600° /sec in humans. The velocity increases with increased range of eye movement (Robinson, 1964). Saccadic eye movements are used for moving the eyes between visual targets and to achieve the best visual image of the desired object in the shortest possible time. Therefore they provide a stabilizing function of the eye. Saccadic eye movements can be voluntary or involuntary such as the fast phase of vestibular nystagmus and any jerky nystagmus. The rapid eye movement of the rapid eye movement phase of sleep are also considered to be saccades.

In clinical practice, the saccadic test is always done before any of the other tests. Its purpose is two-fold : (1) To calibrate the recording system, and (2) to examine the patient's ability to perform saccades. The saccade test is repeated at intervals during the Electronystagmography examination to recalibrate the recording system.

Testing Procedure

The patient is in the sitting position and is instructed not to move his head. On a non computerized instrument, the patients eye movements are recorded as he looks back and forth between two spots located on a wall directly in front of him at a distance of 5 feet. The spots are positioned so that the patient's eye sweeps 20° of visual angle in the horizontal plane as he looks from one spot to the other. As the patient performs this task, the clinician adjusts the gain of the horizontal channel of the Electronystagmography so that

the recording pen moves 20 millimeter each time, the patient refixates. Thus to calibrate the horizontal channel, the patient looks 10° left, center, 10° right, center, 10° left, and so on. The vertical channel is calibrated the same manner. This method works well for calibration, but saccadic defects seem easier to detect, when the patient performs a 20° saccades/

In clinical practice, the tracing yielded may be inspected for disorders of saccadic eye movement, the accuracy and the velocity of the saccades are commonly assessed. The evaluation of the reaction time for the initiation of saccades (latency period) may also give valuable information.

Normal Variations

When a normal individual makes saccades, his eye more rapidly and usually stop precisely on each target (figure 8). However, some normal individuals consistently undershoot or overshoot the target by a small amount and then must reach it by making one or two small corrective saccades.

Abnormalities

1. Accuracy of the saccades

In certain localized lesions within the central nervous system the saccades are performed with gross imprecision. One function of the cerebellar hemisphere is to control smooth integration of the body muscles that function in an agonist-antagonist relationship. Disease of the cerebellum or its neural connections in the brain stem (collectively known as the cerebellar system) cause defects of limb movements, such as dysdiodochokinesis. The occular counterpart of dysdiadochokinesis is occular dysmetria. Occular dysmetria is

characterized by overshooting or undershooting of the target when visual fixation is transferred from one point to another (figure 9). Overshooting of the target is called hypermetric and undershooting is called hypometria.

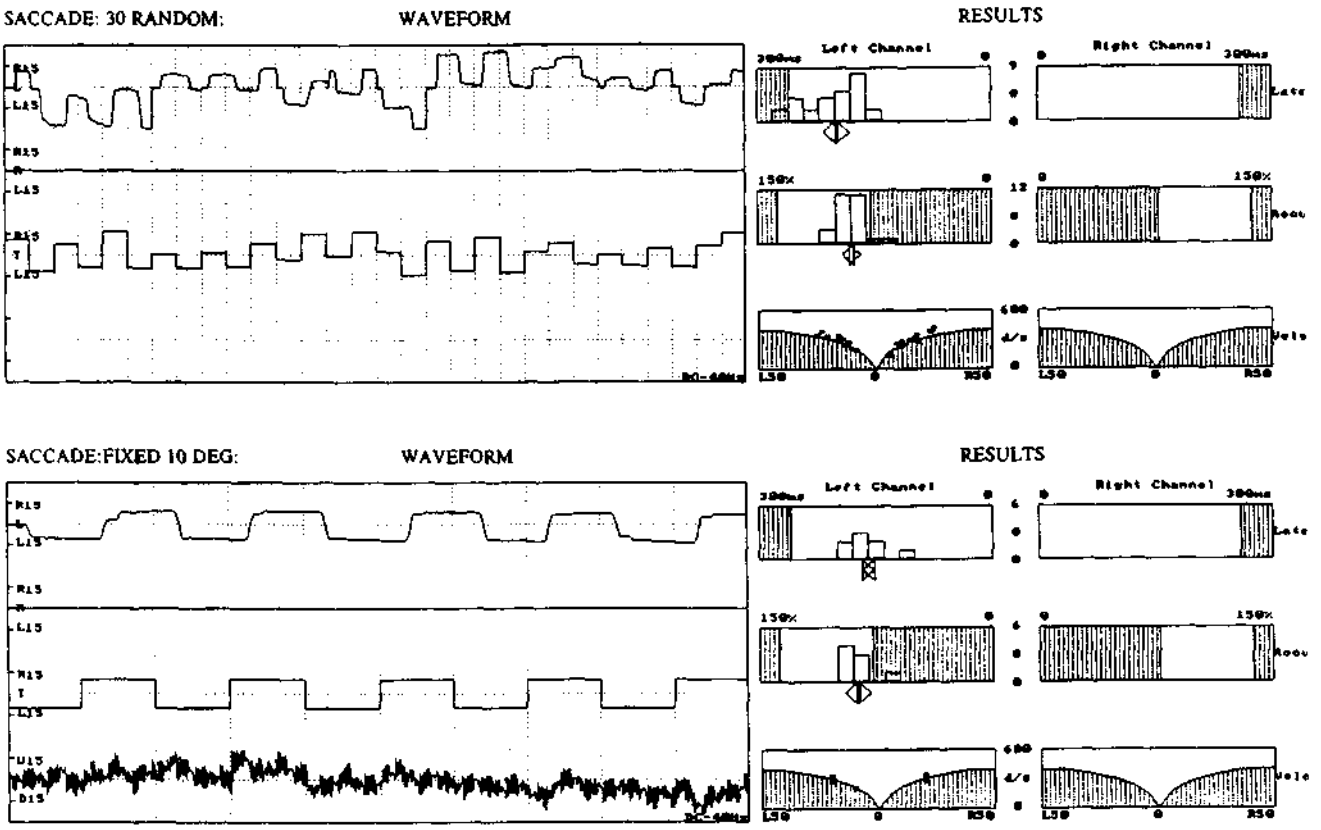


Figure 8 : A normal saccadic waveform : Random saccades and fixed saccades

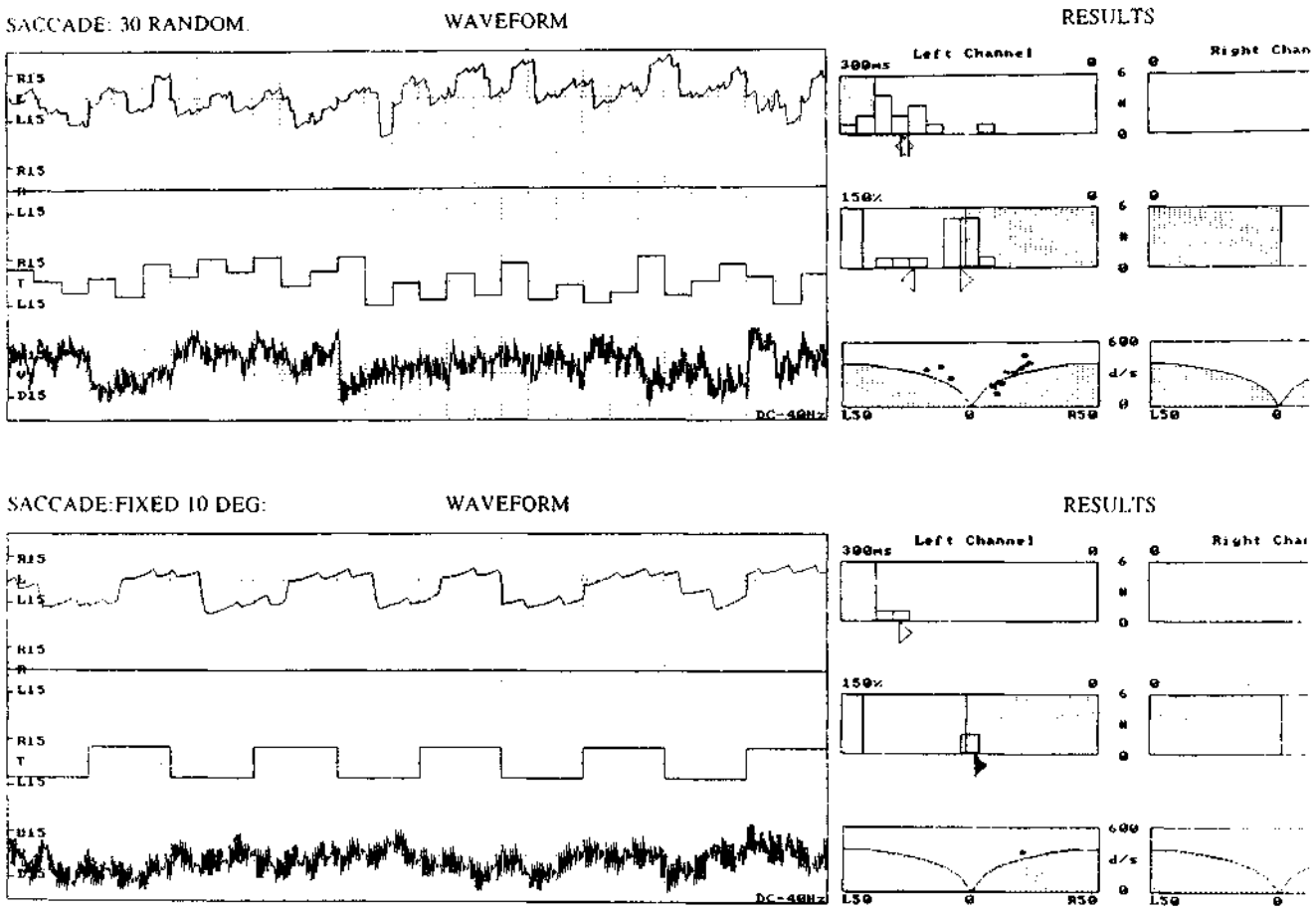


Figure 9 : Hypometric saccades, or undershoots, a form of ocular dysmetria.

Clinical Significance

Saccadic accuracy is not altered in peripheral vestibular disease. Both hypometria and hypermetria can be seen in cerebellar disease. In unilateral cerebellar lesions, dysmetria occurs when the saccadic movement is towards the side of the lesion. In intrinsic brain stem lesions, hypometric saccades are more common than hypermetric saccades. Intrinsic brain stem lesions include vertebrobasiler insufficiency, multiplesclerosis, infiltrating tumours and degenerative disease (Baloh, Honrubia and Sills, 1977; Henriksson, et a1, 1981).

2. Saccadic Velocity

In normal subjects saccade peak velocities vary among 180° /sec to 600° /sec for 5-60° of eye movement (Pyykhko and Schalen, 1984). The longer the movement the higher the velocity.

Clinical Significance

Saccade velocity is not affected by peripheral vestibular disorders. Slowing of the horizontal saccades is usually caused by pontine disease, affecting the pontine paremedian reticular formation, and vertical saccade slowing is a result of pretectal lesions. So saccadic slowing is seen in central vestibular pathology (Bolah, Honrubia and Sills, 1977).

3. Saccadic Initiation : Saccadic reaction time

The saccadic reaction time is the latent period between the command given to the subject for a voluntary eye movement and the onset of a saccade. In normal subjects the average reaction time is about 250 milliseconds, the upper limit being 350 milliseconds.

Clinical Significance

Saccadic reaction time is normal in patient's with peripheral vestibular and pure brain stem and cerebellar lesion. However, in multiple sclerosis or wide spread degenerative disease (That is progressive supra nuclear opthalmoplegia) of CNS, delayed reaction time are found (Soligen et al., 1977; Mastaglia, Black and Collins, 1979). Saccadic reaction time is consistently delayed in basal ganglia disorders (Bolah, Honrubia and Sills, 1977). In cortical

lesions abnormal saccadic reaction time delay has been reported in bilateral frontal lobe lesions (Bolah, Honrubia and Sills, 1977).

Pitfalls

Several possibilities for serious error are present when one interprets the saccade test.

Superimposed Gaze Nystagmus

The clinician must carefully examine the tops and bottoms of the tracing of saccadic movements. Most normal individuals show a smooth regular tracing here, through a minor degree of "Quiver" perhaps from muscle potential or microsaccade is within normal limits. Occasionally bilateral gaze nystagmus may be superimposed on the saccadic tracing which must be identified, and not confused with hypometric saccades.

Superimposed Congenital Nystagmus

It is possible to have congenital nystagmus superimposed on the saccadic tracing, but congenital nystagmus is easiest to recognize in the gaze test.

Drugs

Drugs mentioned in the earlier chapter in sufficient dosage can produce deterioration of saccadic eye movements. The abnormality most commonly seen is occular dysmetria. If one detects an abnormality in saccadic test, one must rule out drug as the cause before accepting the findings, as evidence as of organic lesion.

Inattentive patient

An inattentive patient performs saccades poorly and requires several reflations before he manages to hit the target. Such a tracing would probably be judged abnormal ; thus the clinician must coax the patient until he is convinced that he has elicited the patients best effort. In addition to making sure that the patient is performing at his best, the clinician must be sure that the patient can see the targets. If the patient has removed his glasses for testing and his saccades are poor, the patient should perform the task again using his eyeglasses on.

Eye **Blinks**

The patient who blinks his eye whenever he performs a saccade can produce a misleading tracing. Sometimes these eye blinks, produce a tracing that appears similar to one produced when the eye overshoot the visual target. However, the clinician can make a discrimination by examining the tracing of vertical movements ; eye blinks produce prominent spikes in lower tracing, whereas true overshoots do not. The blinks can be easily identified in the tracing of vertical movements. They produce sharp-pointed spikes in the horizontal tracing. In this case, the true overshoots can be distinguished from eye blinks i.e., the overshoots have flattened tops.

OCULAR PURSUIT TEST

When a visual target moves slowly and smoothly across the visual field, the eyes are able to track it smoothly up to a velocity of 60-70° /s with minimal saccadic intrusion. In frequency terms the smooth tracking limits are upto 1 to 1.5 Hertz. Above these velocity and frequencies, saccades come into play to catch up with the movements and smoothness is lost. Smooth pursuit is considered to be an eye movement that monitor constantly a moving target, partly by anticipating the movement and partly by using visual feedback arising from the retina. Thus the image of the moving object is stabilized on the fovea.

Clinically, the patient is asked to follow the examiner's finger moving across the visual field to and fro, for the horizontal eye movement, or is asked to follow a pendulum while the eye movements are observed. Electro-nystagmographic recording can be made at the same time. On a computerized Electronystagmography an array of light emitting diodes that are lit up in sequence is used and in this way the stimulus is more precisely controlled. The target moves to and fro in a certain visual angles, with a constant velocity or sinusoidally. Quantitative analysis of this eye movement can be done using a computerized Electronystagmography. The best quantitative analysis of eye movement is the gain of the pursuit. To calculate the gain of the pursuit, the velocity of the measurable pursuit components, is measured, ignoring the saccades, and this is compared with target velocity by taking the ratio of eye velocity / target velocity. This is the gain of the pursuit and in normal subjects is expected to be 1. This is virtually up to 40° / sec. Normal pursuit test is done

for frequencies ranging from 0.2 through 0.7 Hz. The normal / abnormal range is determined based on sex and age, and values below or above 2 Standard Deviation is considered abnormal.

Testing Procedure

The patient is in sitting position and is instructed not to move his head. He is asked to track the movement of the pendulum or the light spot that moves in front of the patient on the digital light bar. Pursuit is tested at different frequencies 0.1, 0.2, 0.4 Hz and the gain of the pursuit is compared with a normative data.

Normal Variations

The normal individual is sometimes able to follow the target with negligible error, producing a tracing that is nearly the perfect image of the target motion (figure 10). However most persons-particularly elderly ones-follow the target somewhat imprecisely, occasionally allowing it to slip off the fovea and then performing corrective saccades.

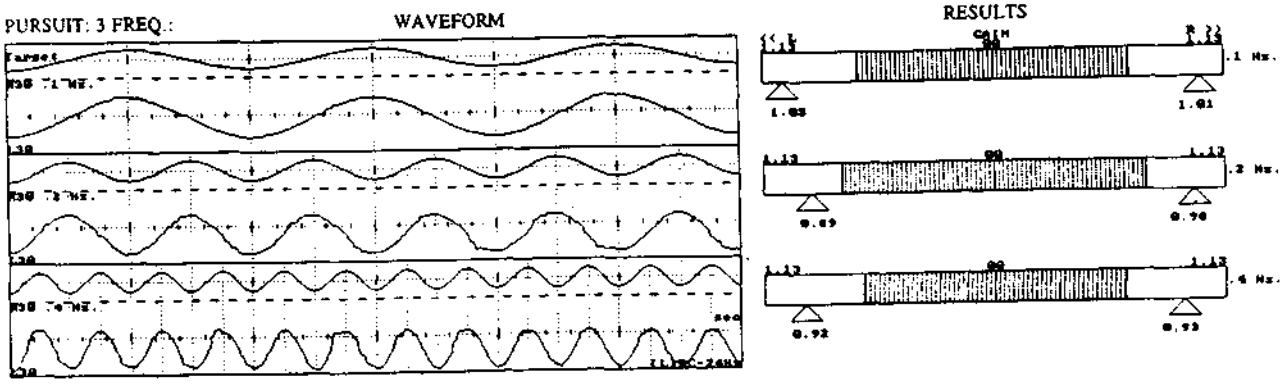


Figure 10 : Normal pursuit tracking

Abnormalities

Saccade **Pursuit**

When a patient has brain stem disease involving the pursuit system, he substitutes saccadic movements in varying degree for the smooth tracing capacity. The eye falls behind the moving target briefly, then retrieves it with a saccade. This is the main diagnostic abnormality one looks for in the tracing. The term "cog wheeling" is used to describe the appearance of the tracking record when there is marked saccadic pursuit.

Pursuit abnormalities are characterized by decrease in the gain and the appearance of superimposed saccades at low velocities, in which the movement is normally achieved smoothly. The number of saccades is considered to be proportional to the degree of severity of pursuit abnormality (figure 11).

In chronic peripheral vestibular disorder smooth pursuit is normal. In acoustic neuromata (vestibulocochlear schwannomate) pursuit is not impaired unless the rumour is situated in the cerebellopontine angle and compresses the brain stem (Bolah, Kumley and Sills, 1976). In cerebellar lesions the gain of the pursuit system is reduced, but this is compensated by increasing the saccadic component of eye tracking. Therefore the movement is broken up by many saccades (Jung and Kornhuber, 1964), and the frequency of saccades is highest in cerebellar disorder (Pyykoko and Sehalen, 1984). In brain stem lesions the eye tend to remain stationary at relatively high velocities and the compensating saccades are randomly executed and are small in number. In multiplesclerosis, pursuit abnormalities are common. In basal ganglia disorders

(Parkinson's disease and Huntington's chroea) smooth pursuit is bilaterally affected (Baloh, Honrubia and Sills, 1977). Saccadic abnormality are also seen in degenerative central nervous system disease (Progressive supranuclear opthalmoplegia, Organic brain syndrome, Alzheimers disease etc.). The abnormality seen in this condition is usually bilateral.

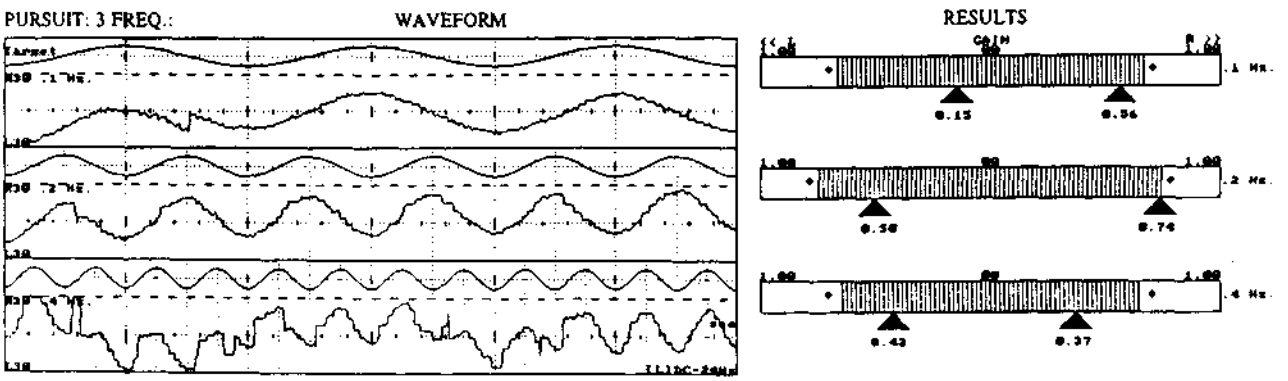


Figure 11 : Saccadic pursuit, an abnormal tracing

Benitez (1970) has proposed a clinical classification of the qualitative features of tracking records.

Pattern 1 is a sine wave record that is perfectly smooth.

Pattern 2 is an irregular record with periodic 'bites' that occur in some normal individual.

Pattern 3 is a record features by saccadic substitution of pursuit movements and Pattern 4 is featured by disorganization of pursuit eye movement.

Pitfalls

The pursuit test, or pendular tracking test as it is also known, can be affected by a variety of conditions including patient's age and alertness, examiner's instruction and various medications. At times, an overriding gaze nystagmus can make it difficult to determine what portion of the abnormal smooth pursuit tracing is secondary to abnormal pursuit function per se, and what is due to the overriding nystagmus. Superimposed congenital nystagmus may also be present on the pursuit tracking. The pursuit system may be normal in these cases, although the pursuit tracing is certainly not smooth. Breakup of the pursuit tracing may be due to the spontaneous nystagmic activity rather than to dysfunction within the actual pursuit system.

OPTOKINETIC TEST

Optokinetic nystagmus is elicited by passing before the patient's eyes, a repetitive visual pattern that fills at least 30 % of the patient's visual field. The function of the optokinetic system is to maintain visual fixation when the head is in motion. This system compliments the vestibular system in this regard but functions primarily at frequencies lower than those of the vestibular system. The optokinetic nystagmus in the laboratory is usually elicited by instructing the patient to look at a rotating drum or moving belt which has on its surface black stripes on a white back ground. On a computerized Electronystagmography instrument, the patient is instructed to focus on the light that is centered on the light bar. As the light moves, the patient should follow it until it drops off. Once the light is off the patient should pick up the next light following it until it drops off and so on. This produce a nystagmus, with its slow (following) phase in the direction of movement of the stripes or light and its fast (corrective) phase in the opposite direction. When properly elicited and interpreted, optokinetic nystagmus can be of considerable help in diagnosing disorder of the central nervous system.

Optokinetic responses to drum rotation/light moving in clockwise and counterclockwise directions are elicited. For this reason, the clinician must be very careful to make the speed of drum rotation as equal as possible in the two directions. This is particularly important if a hand - operated drum is used.

Instrumentation

The optokinetic test is usually done with a hand-operated drum. Desirable characteristics of this instrument are

1. Free rotation (a bearing mounted drum is preferable)
2. At least 6" in diameter to minimize inaccuracy produced by drum curvature.
3. At least 10" high to stimulate a sufficient percentage of the visual field.
4. Can be held either vertically or horizontally to elicit horizontal or vertical nystagmus.

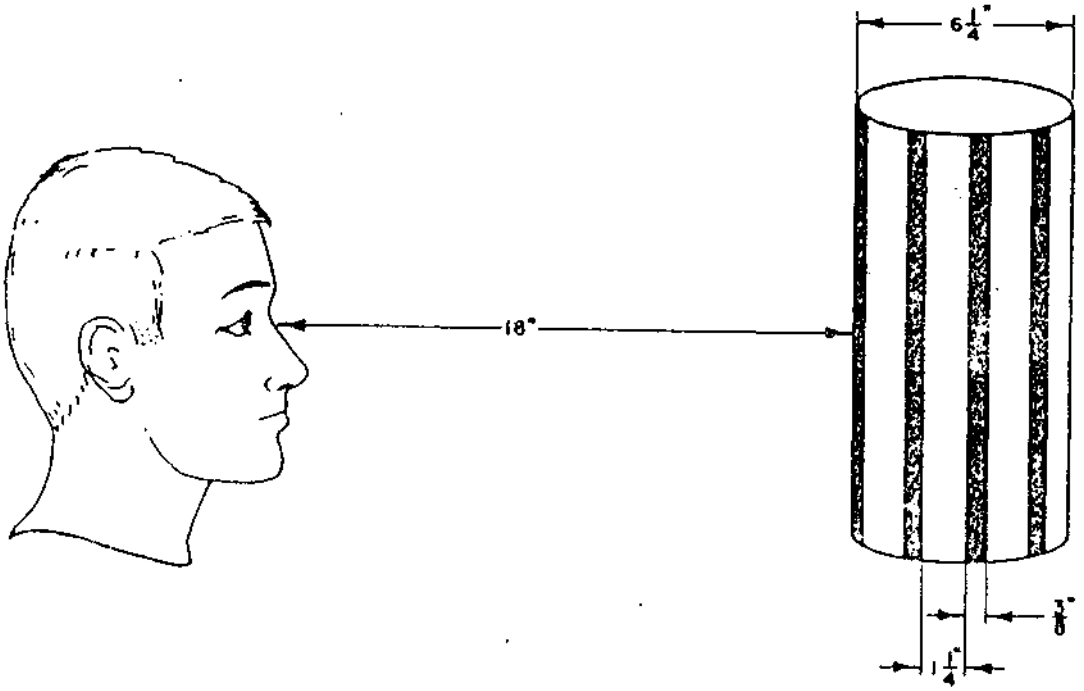


Figure 12 : Optokinetic test drum with suggested dimensions

Large Rotation Cylinder

Some laboratories use a large cylinder that is lowered over the patient's head for optokinetic testing. The advantage of this instrument is that it stimulates the entire visual field, thus producing a more regular nystagmus,

which is less readily controlled by the patient. Also, if motor operated, this instrument has the advantage of providing a quantitatively controlled stimulus. Disadvantage of the large cylinder are 1. It occupies a relatively large amount of laboratory space and 2. It cannot provide a vertical stimulus.

Optokinetic Projector

It is an instrument, which projects an optokinetic stimulus pattern onto the examining-room wall. When the lights are off, the pattern projected by this instrument fills the patient's visual field almost as effectively as the large drum. However, unlike the drum, the projecting stimulator is compact and can be rotated to provide a vertical stimulus. Also, it allows a quick change of stimulus.

Computerized Optokinetic Test

This involves the patient to follow the lights on a light bar controlled by specific software. Sub test involves, the sinusoidal optokinetic velocity step test, and optokinetic after nystagmus in which patient's nystagmus eye movements are recorded as she / he watches a series of stimuli that simulate a spinning environment.

Test Procedure

Optokinetic nystagmus provides an excellent check of the recording system and also of the ability of the patients oculomotor system to generate nystagmus. Therefore it is advantageous to do the optokinetic test before doing the positional and caloric tests.

The patient is seated comfortably in front of the digital light bar or in front of the drum at eye level, at a predetermined distance, which varies according to the instrument used. If the patient wears eyeglasses, they should be on.

In eliciting optokinetic nystagmus, it is very important that the patient not be over instructed, it is rarely necessary to tell the patient to "pick up each stripe or light as it goes by" or "count the stripe or light". The tester should simply tell the patient to "watch the stripe or light". If this produces, a well formed optokinetic nystagmus, no further instruction is necessary. If it does not, admonishing the patient, "you are not watching them", or "Focus on the stripes or light", will usually produce the desired response. On a computerized Electronystagmography test, there are two variations of testing that can be done. They are

(1) sinusoidal optokinetic nystagmus test, in which, the stimulus shifts from clock wise to counter clockwise direction alternatively,

(2) optokinetic step velocity test where optokinetic test, at various velocities from 20° /sec to 60° /sec is tested, in steps of 20°, 30°, 40°, 50° and 60° can be tested for both clockwise and counterclockwise rotation.

The optokinetic after nystagmus is tested in complete darkness or with eyes covered, after a sufficient period for inducing well developed optokinetic nystagmus (ideally maximal optokinetic nystagmus is established by accelerating stimulus), a nystagmus, following stimulation is recorded.

At the faster velocity, some patient's are incapable of generating a well-formed nystagmus and in such patient's the examiner need not persist in his attempts to obtain with response. If desired, vertical optokinetic responses can be monitored while the patient watches vertically moving stripes or lights. Ordinarily, it will be sufficient to elicit up and down beating optokinetic nystagmus at low velocity.

Normal variations

When a normal individual watches an optokinetic stimulus, the speed of his eyes during the nystagmus slow phase matches the speed of the stimulus, up to a stimulus speed of approximately 30° /sec (figure 13). As the stimulus speed is increased further, eye speed continues to increase up to 40° to 50 /sec for stimulus speed but it tends to fall progressively below target speed. As the stimulus speed is increased still further, eye speed declines until the fusion limit is reached.

A normal individuals optokinetic responses are symmetrical, which means that for a given stimulus speed, the intensity of his left-beating optokinetic nystagmus (provoked by a rightward moving stimulus) is approximately the same as his right-beating nystagmus (provoked by a leftward moving stimulus). On a computerized instrument it is possible to measure "gain". The average ratio of the slow phase velocity to the target velocity in each direction is called the gain. A gain between 0.60 and 1.1 is considered normal for both sinusoidal and velocity step optokinetic.

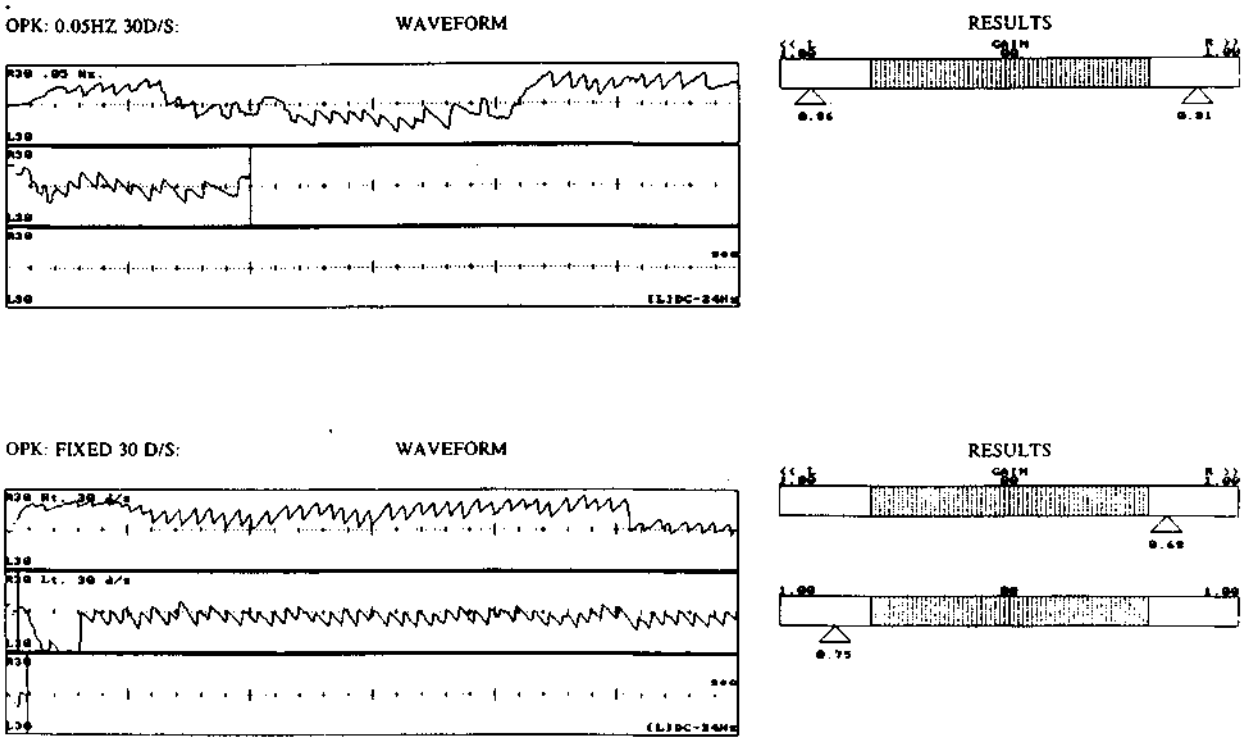


Figure 13 : Normal optokinetic recording, both sinusoidal and fixed optokinetic test

Abnormalities

In chronic peripheral vestibular disorders, optokinetic nystagmus is unaffected and symmetrical (Zee, Yee and Robinson, 1976). In acute unilateral peripheral lesions it may be transiently affected causing an asymmetry and reflex decline. In the presence of a lesion affecting the optokinetic system at various levels, the following abnormalities can be seen.

1. Asymmetry : Gross irregularities in its rhythm and amplitude

In cerebellar atrophy, the impairment of optokinetic system is usually bilateral and characterized by reflex decline and irregularities in amplitude and frequency of the response. An asymmetry may be found in unilateral cerebellar lesions and the derangement is detected, when the stimulation of the optokinetic nystagmus is towards the affected side (Baloh and Honrubia, 1979). Brain stem lesions usually cause bilateral optokinetic nystagmus reflex depression (Cogan, 1956), although limited unilateral intrinsic lesions may be associated with reflex depression when the optokinetic stimulus movement is towards the side of the lesion. An asymmetry in which there is a reduced slow phase velocity in one direction with difference of 10° /sec to 30° /sec is considered abnormal. Lesions of the perietal, occipital and frontal lobes are known to produce optokinetic abnormalities (Coats, 1971).

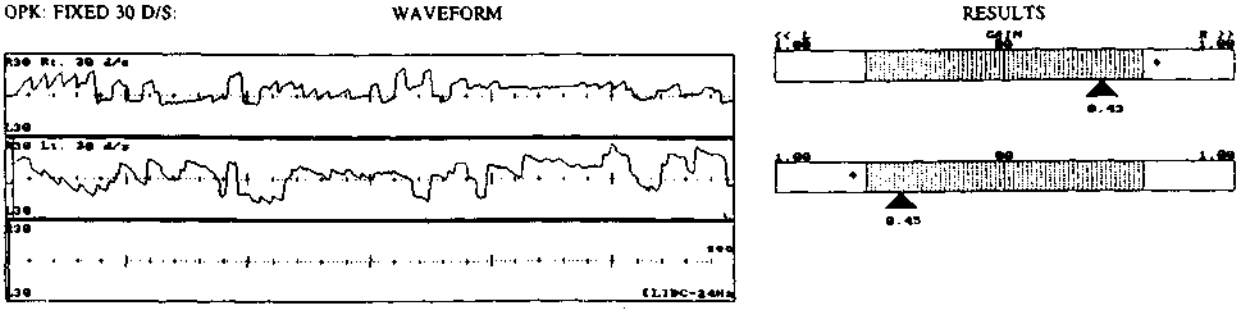


Figure 14 : Optokinetic recording showing reduced gain

2. Unilateral or bilateral absence of optokinetic nystagmus

In brain stem lesions the fast component of optokinetic nystagmus may also be affected as well as the slow component, thus resulting in complete absence of the response ; this may be unilateral or bilateral.

3. Inversion or Reversal of optokinetic nystagmus

The presence of congenital nystagmus may cause marked alteration of the optokinetic record. In this the nystagmus beats appropriately to the left when the light / stripes are moving to the right. However, when the target reverses direction and moves to left, the nystagmus also beats to the left, that is, in a direction inappropriate to the stimulus. At the increased target velocity the same optokinetic pattern is seen i.e., again the nystagmus beats inappropriate direction when the stripes move to the left. This phenomenon is called inversion or reversal of optokinetic nystagmus.

4. Optokinetic after- nystagmus

Optokinetic after-nystagmus is a nystagmus beating in the same direction of the fast phase as the preceding optokinetic nystagmus, when tested in darkness, after a sufficient period for inducing well developed optokinetic nystagmus. This optokinetic after nystagmus is transitory, much smaller in amplitude and of lower frequency than perstimulatory optokinetic nystagmus. Optokinetic after-nystagmus gradually fatigues and disappears. It may be followed by a low grade, transitory phase with reversed direction which is considered to be a secondary phase of optokinetic nystagmus.

Optokinetic after-nystagmus is evaluated by measuring its duration, slow phase velocity and frequency and comparing those values obtained from optokinetic nystagmus stimulus to the right and left. Normally these values are expected to be more or less the same. A significant difference or a directional preponderance is indicative of a central vestibular imbalance.

Pitfalls

Drugs and Inattentiveness

As with the saccade and tracking tests, the patient who is taking drugs can produce an abnormal tracing in the optokinetic test. The clinician should also be aware that the patient can suppress his optokinetic nystagmus at will by allowing the stimulus to blur before his eyes. Therefore, if nystagmus is of poor quality or is absent in either or both directions, the clinician must constantly encourage, the patient to watch the stripes and accept the result as valid only when he is sure that the patient is performing at his best.

The Dix-Hallpike Test

Or

Dynamic Positioning Test

The Dix-Hallpike test for benign paroxysmal vertigo is a dynamic positioning maneuver, and is an important part of the Electronystagmography test battery. The positioning test corresponds to the classical Dix and Hallpike maneuver in which the patients head is briskly brought into the critical position and then kept there for an appropriate period of time. This is a test for a specific response to a specific maneuver. This maneuver exposes the patient not only to a particular position but also to rather violent movement. The response often consists of a burst of intense nystagmus and vertigo that dies out within 1 to 2 minutes after the patient has been brought into the test position. The response may not begin immediately after the patient arrives at the test position it may have a latency period of 5 to 10 seconds. Because of the latent period and the violence of the response, a positive Dix-Hallpike test may take the inexperienced clinician by surprise. The first impulse of the patient will be to sit up and if not prevented, he will do so before a recording of the nystagmus can be obtained. Another common characteristic of a positive Dix-Hallpike response is fatigability, i.e., the intensity of the response is markedly reduced on subsequent attempts of elicitation. Because of this it is particularly important to obtain a good recording the first time.

The phenomenon of benign Paroxysmal positioning vertigo (BPP V) was described by Barany (1921) and popularized several years later by Dix and Hallpike(1952).

Test Procedure

In the Dix-Hallpike maneuver, the examiner instructs the patient to fixate on a point when in an erect position. Then as the examiner rapidly moves the patient from that position to another (i.e., Head hanging left), the examiner will provide with another fixation point for up to 30 seconds. If the Dix-Hallpike maneuver is conducted with the patient eyes closed, the examiner need only to instruct to what position he/she will be moved and then help the patient assume that position at the correct time during the test. Explain to the patient that you will want him to lie back rapidly at the same time turn his head all the way to the right. Also tell him that you will guide him as he goes down. With one hand grasping the patient's head and the other placed against his back, guide him as rapidly as possible into the down and right position. Keep the patient in this position for at least 30 seconds, and throughout this time remain at his side. Recording should begin immediately after the patient assumes a head hanging position. Then lift the patient back up and leave him in the sitting position for 15 seconds. Repeat this procedure with the down-and-left position.

While the patient is in each test position, ask him if he is dizzy. If the response is "yes" determine whether or not the dizziness reproduces his symptomotology. Write this information on the Electronystagmography record.

If nystagmus is produced by either or both of the Dix-Hallpike maneuvers repeat which ever maneuver produced the nystagmus. This is very important, since a vital characteristic of the Hallpike response is its fatigability with repeated elicitation.

A pure rotatory nystagmus without a horizontal or vertical component can not be detected on an Electronystagmography recording even with electrodes surrounding the eye. Hence it is imperative that the patients eyes be monitered visually during the Dix-Hallpike maneuver. The optional method is to observe the eyes behind lighted Frenzel lenses, but direct observation may also suffice. Thus the least favourable method is to test with the patients eye closed and rely on the recorder to determine the nytagmic response, given that this method will fail to identify those patients with pure rotatory nystagmus. Conversely if the nystagmus contains horizontal or vertical components the abnormality can be recorded behind closed eyes.

Normal Variations

In normal individuals, the Dix-Hallpike maneuver provokes no vertigo and no nystagmus with the eye open, although a few beats are sometimes seen in Electronystagmography tracing when one is recording with the patients closed eyes (figure 15).

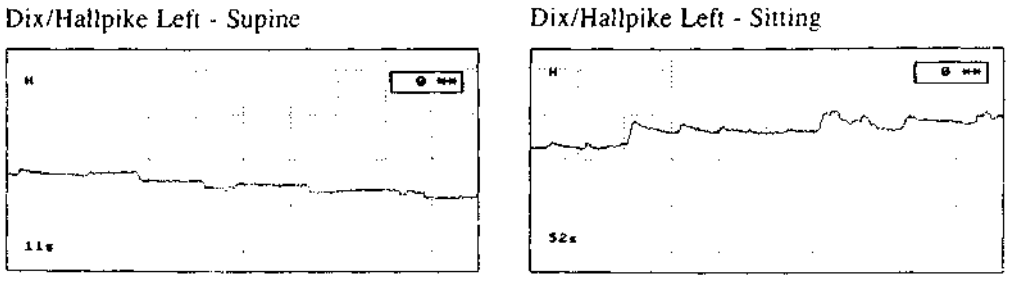


Figure 15 : Normal Dix and Hallpike record : left supine and left sitting positions

Abnormalities

The most common form of nystagmus provoked by the Dix-Hallpike maneuver is the positional nystagmus that can be classified into two categories.

1. Benign Paroxysmal Positional Nystagmus (peripheral)
2. Central positional nystagmus.

1. Benign Paroxysmal Vertigo / Positional nystagmus

This type of nystagmus appears after the patient has been placed in either the head hanging right or head hanging left position, rarely both. Often it briefly reappears, beating in the opposite direction when the patient resumes the sitting position (figure 16). Positional nystagmus of the classic benign paroxysmal type always possess four features

1. It is delayed in onset, appearing no sooner than several seconds after the patient's head is placed in the critical position.
2. It is accompanied by vertigo, lasting approximately as long as the nystagmus.
3. It is transient, never lasting longer than 30 seconds.

4. It is fatigable, becoming progressively weaker each time the maneuver is repeated.

Identification of this rather specific type of positioning nystagmus is important because it is the most common form in clinical practice. If the response is determined to be classic, a benign peripheral vestibular lesion in the under most ear is suspected.

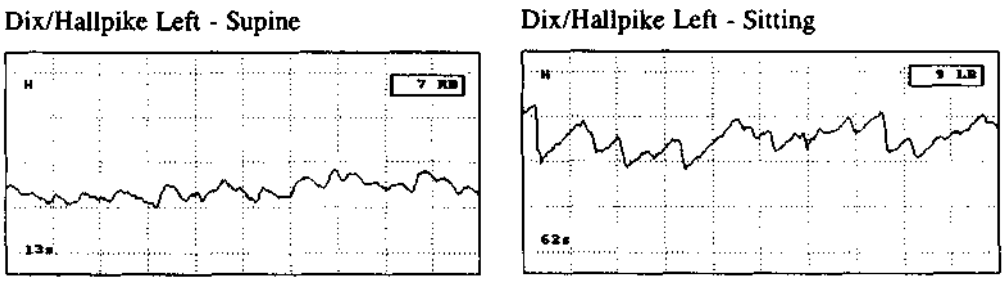


Figure 16 : Recording of abnormal Dix and Hallpike response

2. Central Positional Nystagmus

Some times the Dix-Hallpike maneuver also identifies positional nystagmus. This type of nystagmus differ markedly from nystagmus of the benign paroxysmal type. It appears without a latency, is horizontal, oblique, or vertical (not rotatory), last for a longer time (usually as long as the head position is held), is accompanied by little or no vertigo, and fatigues little or not at all upon repeated testing. This type of positional nystagmus is of particular importance because it usually denotes significant central vestibular lesion.

However, unidirectional nystagmus caused by acute unilateral peripheral vestibular lesions also may be transiently intensified by the Hallpike maneuver.

Pitfalls

Positioning nystagmus of the benign paroxysmal type is fatigable, therefore the Dix-Hallpike maneuver must be done correctly on the first attempt. The response may be absent for the second time. If the clinician intends to perform the Hallpike maneuver, he should do it before other manipulations of the patient that could trigger the response. The examiner should prepare the patient before hand, telling him what he will do and what he wants to see. The patient who has benign paroxysmal vertigo is usually reluctant to perform the Hallpike maneuver. Even when convinced of its necessity, he is opt to become agitated and tightly close his eyes once the vertigo starts, making visual observation or recording of nystagmus impossible. He must be told to keep his eyes open at all costs. So it is essentially important for clinicians to instruct the patient appropriately before beginning the test.

POSITIONAL TEST

The static positional tests are conducted to determine if changes in head position causes or modify nystagmus. This procedure is designed to test the effect of body position on spontaneous nystagmus (if present) or to uncover a nystagmus that might be present in some position other than supine. The test is conducted with the patients head and body placed in various positions and with eyes closed. The purpose of eye closure is to eliminate the effect of visual suppression on potential nystagmus. Positional test provokes spontaneous or positional nystagmus, and hence should be done before the caloric test.

Test Procedure

Records of at least 30 sec with eyes closed should be obtained with the patient in each of the following test positions.

1. Sitting-patient seated comfortably, facing straight ahead.
2. Supine-patient lying on his back, head level with chest.
3. Right lateral-patient lying on right side with pillow under his neck so his neck is straight.
4. Left lateral- as with right lateral expect lying on left side.
5. Head right-patient lying on his back with his head turned as far to the right as possible.
6. Head left - as with head right expect head turned to the left.
7. Head hanging - patient lying on his back with his head as far below the horizontal as possible.

Since the purpose of the positional test is to separate the effect of position from the effect of movement, the patient should be moved slowly into each test position. During all positional test it is extremely important to keep

the patient mentally alert. It may be also important to use various levels of mental alerting with each patient. Although most adult subjects produce the strongest nystagmus during active alerting, such as performing a mathematical task, some subjects respond better to questions, some better to tactile stimulation, and some better to no overt task. In other words, using only one specific mental alerting task (e.g. mathematics) for all patients may be inappropriate. Mental alerting task that are too difficult can cause facial tension, eye blinking, and random excessive eye motion that can effect the purity of the nystagmus response thus making it difficult to identify and quantify. Therefore with each patient, the tester must select a concentration task of the appropriate level of difficulty.

The Electronystagmography recording is usually made after abolition of optic fixation with eye closure, it can also be done in eyes open if required. Rotatory component will not appear in the recording. The clinician who wishes to observe the rotatory component, he must use the Frenzel glasses.

Normal Variations

No normal individual has positional nystagmus with eyes open, but many have it with eyes closed (figure 17). Since many normal individuals have horizontal positional nystagmus with eyes closed, one needs definite criteria for determining whether it is pathologic in a given patient. Barber and Wright (1973) established the following criteria: Horizontal positional nystagmus with eyes closed is considered abnormal

1. If it changes directions in any head position.
2. If it is persistent in three or more of the head positions tested.
3. If it is intermittent in four or more head positions.
4. If the slow phase eye speed of the three strongest consecutive beats exceeds 6° /sec in any head position.

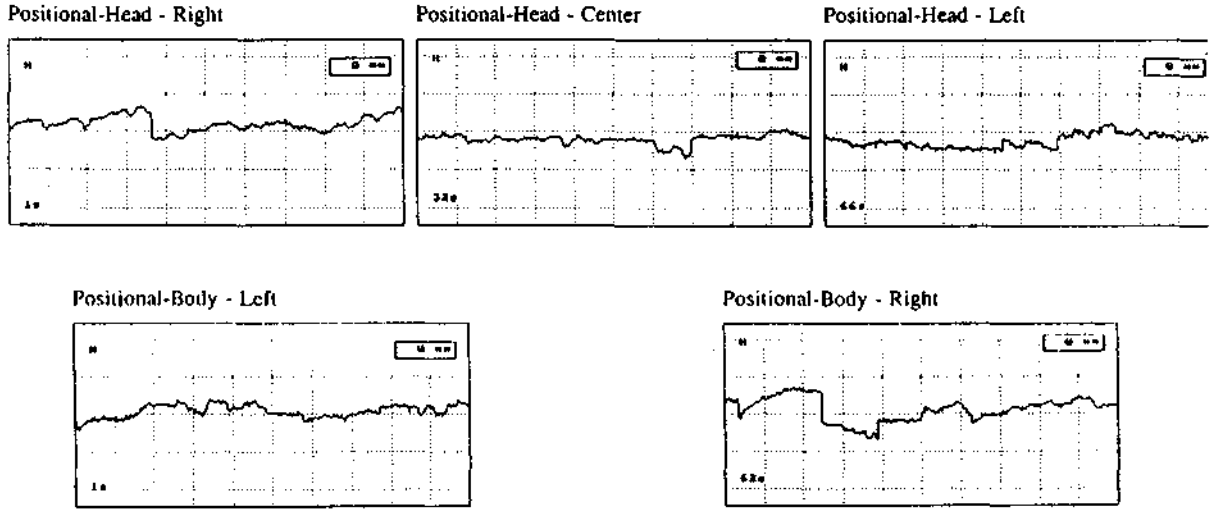


Figure 17 : Normal positional test record in head right, center and left positions and body left and right positions

Abnormalities

Before one considers the various abnormalities in the positional test. It is important that one learns to differentiate positional nystagmus with spontaneous nystagmus. The term spontaneous nystagmus has different meanings to different people. It is preferable to define spontaneous nystagmus as nystagmus that is direction fixed and beating with about the same intensity in all head positions, when the eyes are closed. So it is clear from this definition that spontaneous nystagmus is direction fixed, whereas positional

nystagmus may be direction fixed or direction changing. Direction fixed positional nystagmus is differentiated form spontaneous nystagmus by a certain variability of intensity in different head positions or by absence of the nystagmus in one or two positions whether the eyes are closed or open.

Some common abnormalities seen in the positional test are

1. Positional nystagmus with eyes open

Positional nystagmus with eyes open is always abnormal, hence is of great importance. It is usually accompanied by little or no vertigo and persists for as long as the head position is maintained. Whether direction fixed or direction changing, it is a good evidence of CNS disease or lesion (Barber and Stockwell, 1976).

2. Direction-fixed positional nystagmus with eye closed

As mentioned earlier direction fixed positional nystagmus (with eyes closed) is rather common in normal population. If the positional nystagmus fits into the criteria that was mentioned earlier it is considered abnormal. Although direction - fixed positional nystagmus with eye closed is usually indicative of a peripheral disorder, it may sometimes occur in CNS disease. When the intensity of nystagmus is beyond normal limits it implies only the non physiological (pathological) nature of the nystagmus, but it is not of any definite localizing or lateralizing value.

3. Direction — changing positional nystagmus

Here the direction of the nystagmus beat is not fixed in various positions, but will change in different position (figure 18). Again the direction

changing positional nystagmus is non-localizing, may be present due to either peripheral or central vestibular lesion.

Positional nystagmus may be classified as geotropic or ageotropic. (Geotropic meaning nystagmus beating towards gravity, i.e. fast phase of nystagmus beats towards the undermost ear. Ageotropic means that the nystagmus beats away from gravity, i.e., fast phase of nystagmus beats towards the uppermost ear.) The direction-changing, ageotropic positional nystagmus (a nystagmus that changes direction when head position changes and beats away from the down ear in the supine-head-left and supine-head-right positions) has been observed more commonly in central lesions and in bilateral, peripheral lesions (Barber, 1975). The above findings are generalizations, however, and either type can be seen in central or peripheral vestibular pathology.

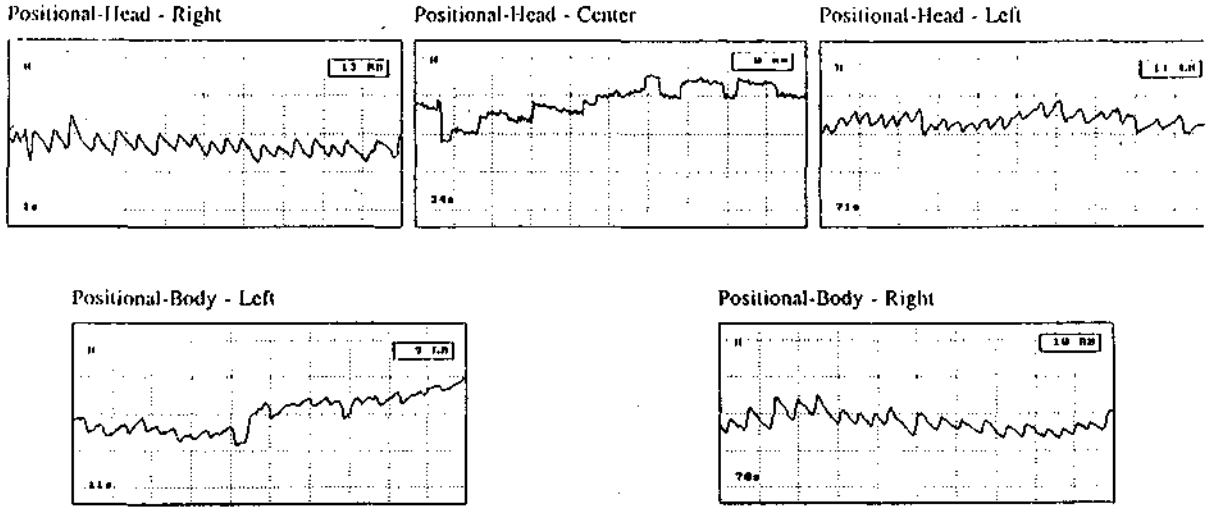


Figure 18: Direction changing positional nystagmus

4. Direction changing nystagmus in a single head position

Here the direction of nystagmus beats changes from left to right or right to left in a single head position. Initially the nystagmus will be beating in one direction, and then disappears, after a few seconds, nystagmus reappears this time beating in the opposite direction. This uncommon positional nystagmus that changes direction in a single head position is probably confined to central vestibular lesion (Barber and Stockwell, 1976).

5. Positional Alcohol nystagmus (PAN)

Direction-changing positional nystagmus is produced by large doses (Blood alcohol level about 40 mg / 100 ml or more) of alcohol. The nystagmus appears approximately half an hour (1/2 hr) after the alcohol ingestion and is most prominent when head is in right lateral and left lateral positions. This nystagmus is called PAN I and it is geotropic. It may be present with eyes open but is much stronger when they are closed. After 3 or 4 hours PAN I disappears. Later, at least 5 hours after ingestion of alcohol, nystagmus appear again. This nystagmus is called PAN II, it is a geotropic and persists for as long as 24 hours. It is important to identify this type of nystagmus as it can interfere with test findings.

Pitfalls

Since direction changing positional nystagmus can be provoked by alcohol ingestion, one must not attribute this abnormality to an organic lesion until alcohol intake has been ruled out as a cause.

Several other variables can influence the result of the positional test. One variable is alertness. As mentioned previously, it is important for the clinician to keep the patient alert by some task, such as mental arithmetic, when looking for nystagmus when the patient's eyes are closed, because lack of alertness causes nystagmus suppression. The problem is increased when the patient is taking central nervous system - depression medication.

Another variable is direction of gaze when placed in the right lateral or left lateral positions, with eyes open patient tend to look either at the ceiling or at the floor. If the patient has gaze nystagmus is may appear on the tracing and may be erroneously interpreted as positional nystagmus.

Positional test is designed to evaluate the effect of head position, not movement, on nystagmus. Therefore, position changes should be performed slowly to minimize the effect of movement. Finally if nystagmus appears when the patient assumes the right lateral or left lateral positions by turning only his head, the clinician must ask him to assume the same position again. This time the patient must be turned on to his side without neck rotation, so that the clinician can determine weather the neck rotation or the head position caused the nystagmus (i.e., to rule out the neck involvement).

CALORIC TEST

Caloric test as the name suggest involves using varied temperature in the external auditory meatus to induce nystagmus and to assess the vestibular system. Of all the Electronystagmography tests, the caloric test is the most difficult and time consuming for both the tester and the patient. Most clinical Electronystagmography laboratory utilizes a caloric test produce that is based on the bithermal test procedure described by Fitzgerald and Hallpike in 1942. The technique consists essentially of obtaining cold and warm caloric response from each ear.

Instrumentation

Three types of caloric irrigation apparatus are commercially available

1. Open water irrigators
2. Closed loop irrigators
3. Air caloric irrigators

The open water irrigator contains two temperature controlled water reservoirs that hold water at the warm and cold irrigating temperature. Upon activation of a foot switch or other controls, an automatic timer releases water from one reservoir (Pre-selected via a warm/cold control switch) for a preset period of time. The water flows into the patient's ear canal via a hand held irrigating tip and is caught in a catch basin held underneath the patient's ear; hence the term open water system.

The closed loop water irrigator operates in essentially the same manner as the open water irrigator, except that the water from the reservoir is circulated

through a silastic bag which expands to fill the patient's ear canal while the temperature controlled water is circulating through it.

The air caloric irrigator delivers a temperature controlled air stream than water. Although the closed loop and air irrigator systems have the theoritical advantages of being less 'messy' and avoiding the problems associated with water irrigation of an ear with a perforated tympanic membrane or external otitis, lack of any consensus regarding stimulus parameters for these irrigation systems and also minimal availability of normative data makes presentation of any form of "consensus" technique utilizing these irrigators impossible. Therefore, this chapter will continue discussing the caloric test using open caloric irrigation apparatus.

Desirable basic characteristics of a caloric irrigator are

1. Warm and cold irrigating temperatures maintained in separate reservoirs with ± 0.1 ° c accuracy.
2. Automatic, timed delivery, preferably activated by a foot switch, leaving both hands free to position the irrigating tip.

Positioning the Patient

During the caloric test, the patient lies in supine position with his head elevated 30° above the horizontal. In theory, this aligns the horizontal canal vertically, which is the optimum position for stimulation. A stretcher, flat examining table or electronystagmography chair that can be put into the reclining position may be used. A headrest, to elevate the patient's head and allow convenient positioning of the catch basin is desirable.

Calibration Points

Fixation points should be placed on the ceiling above the patient at center gaze and at 10° on either side of the center. These are used to calibrate prior to every irrigation and also for the patient to fixate during failure of fixation suppression test.

Procedure

The caloric test is performed by irrigating each external auditory canal twice - once with warm water (or air) and once with cool water (or air) and recording each of the four provoked nystagmus responses. The irrigation temperatures advocated by Fitzgerald and Hallpike was 30° c and 44 c. These are 7° below and above body temperature (37°), hence theoretically produces an equal and opposite stimuli. The following schedule for performing the caloric test is suggested:

1. Calibrate (Patient in test position ; spots on ceiling)
2. Perform the Right ear cold irrigation
3. Provide 5 minutes rest period
4. Calibrate
5. Perform Left ear cold irrigation
6. Provide 10 minute rest period
7. Calibrate
8. Perform Left ear warm irrigation
9. Provide 5 minute rest period
10. Calibrate
11. Perform Right ear warm irrigation

The rest periods are measured from the time the preceding caloric responses stop. They are necessary because if successive caloric responses are too close together, the first response will influence the second. The order of irrigations is chosen so that successive responses will always be in opposite directions. Now it should be understood here than on irrigation of the ear canal with cold water, the induced caloric nystagmus will beat away from the ear irrigated, and on irrigation of the ear canal with warm water, the induced caloric nystagmus beats towards the irrigated ear.

The first schedule includes a calibration prior to each irrigation because the calibration often varies as the caloric test progresses. To minimize this source of error, the calibration preceding each response is used to calculate the intensity of that response.

Outline of the procedure for performing a single irrigation

Following is a general outline of the technique of eliciting a caloric response. The more critical aspects of this procedure will be discussed in more detail in the following section.

1. If the patient wears contact lenses, have him/her remove them. This is because contact lenses may produce a false-positive failure of fixation-suppression test.
2. Tell the patient very briefly what is going to be done and that the procedure may be a little uncomfortable; but will not hurt.
3. Position the patient. Tell him that he will be required to start the concentration task on command shortly after irrigation stops, and that he requires to keep his eyes closed after the irrigation has stopped.
4. All necessary preparation for irrigation should be made, like

a) Preparing the irrigator, switching to the proper reservoir (warm or cold), and checking the reservoir temperature.
b) Positioning yourself comfortably, and placing the basin beneath the patient's ear.
c) To turn on the recorder.
d) Insert the tube and irrigate
e) After irrigation stops, tell the patient to begin the concentration task
f) After the caloric nystagmus has receded from its maximum (usually about 40-60 seconds after cessation of the irrigation), have the patient open his eyes, fixate on the center fixation point on the ceiling, and continue the concentration task. The patient should maintain fixation for about 20 seconds. This part of the test procedure in which the patient's fixation to caloric induced response is tested is called "Failure of fixation suppression test".
g) Wait until the caloric nystagmus has completely subsided before turning off the recorder.

Control of the Caloric-Test Variable

The caloric test is analyzed by comparing the response from one ear with the response from the other ear. It is therefore important to irrigate both ears in exactly the same way. Following are the variables over which one must achieve control:

a. Position of the Subjects Head

It is much more important to be certain that head position remains constant from irrigation to irrigation than to achieve a head elevation of exactly 30 . Same patients have a tendency to rotate their heads during caloric response. This is a reflex "neck-torsion" response that is part of the effect of

the caloric irrigation. The tester should prevent it by placing his hand gently but firmly on the patient's forehead.

b) Positioning of irrigating tube within the canal

It is important to insert the tube exactly the same distance and direct it at the same part of the tympanic membrane every time the irrigation is performed.

c) Control of the irrigating temperature

Of the variables in the caloric test procedure, irrigating temperature is by far the most important. When irrigating water is taken from a constant-temperature reservoir, much of the problem of controlling irrigating temperature is solved. The chief remaining source of error is cooling of the water in the tube leading from the bath to the irrigating tip (both irrigating temperatures are above the room temperature). One way to minimize this source of error is to insulate the tube, leaving about 1 foot at the end uninsulated to provide the flexibility needed for proper insertion of the tube. With this procedure, some cooling of the irrigating water still occurs. This can be controlled by setting the thermostatic control on the reservoirs slightly above the desired irrigating temperature to compensate for cooling in the tube during the irrigation. The difference in temperature between the reservoir and irrigating tip is estimated using a calibrated thermometer.

d) Control of the quantity and duration of the irrigation

The quantity and duration of the irrigation are less important sources of error than the control of irrigating temperature. However, some control over

them must still be maintained. If automatic timing of the irrigation is available, control of this variable is provided. The orifice diameter of the irrigating tip is the most important determinant of the rate of flow of the irrigating water. The simplest way to keep this constant is the use of same tip for all irrigation's.

e) Control of "alertness" of the patient

In general, any maneuvers that increases the patient's alertness or reduces their ability to think about the sensation of vertigo produced by the irrigation tend to increase the regularity and intensity of the caloric nystagmus. Therefore, having the patient do a "concentration task" during the caloric response will greatly improve the record. This task should be done aloud. Its difficulty must be tailored to meet the abilities of the patient. A too-difficult task will create excessive voluntary eye movements and muscle artifact, while a too easy task will not accomplish its intended purpose. It is best to change the task with each irrigations and to progressively increase its difficulty with successive irrigations.

Following is a list of suggested concentration tasks in the order of progressively increasing difficulty:

1. Count by 1 's
2. Count by 2's
3. Count by 3's
4. Subtract l's serially, beginning with some high number
5. Subtract 2's
6. Subtract 3's
7. Recite the alphabet backward

Measurements of Caloric Response

Parameters of response: After all your irrigations have been completed, the tester needs to obtain a quantitative estimate of the strength of each of the four responses. In the past, three indexes of response strength have been used. The first is duration of the nystagmus response, which is usually defined as the interval between the beginning of the irrigation and the last beat of the nystagmus. The second index is peak nystagmus frequency, which is usually defined as the average frequency of nystagmus beats during the 10 sec interval in which nystagmus is most intense. The third index of response strength, which is most widely used, is maximum slow phase eye speed, which is defined as the average slow-phase eye speed during the 10 seconds interval in which the response is most intense. Perhaps the simplest way to obtain an estimate of maximum slow phase eye speed is to measure eye speed of the three strongest beats of nystagmus within the chosen 10 seconds interval and then to average these values.

Unilateral Weakness: After the maximum slow phase eye speed for each of the four caloric responses is calculated, we can calculate unilateral weakness, or the amount by which the two responses provoked by right ear irrigation's differ in intensity from those provoked by left ear irrigation's. Unilateral weakness is calculated by the following formula:

$$\text{Unilateral Weakness} = \frac{(RW+RC) - (LW+LC)}{RW+RC+LW+LC} \times 100$$

Where RW is peak slow phase eye speed of the response following the right-ear, warm temperature irrigation, LW is the peak response for the left ear-warm temperature irrigation RC is the response for the right ear-cool temperature irrigation, and LC is the peak response for the left ear-cool temperature irrigation. Unilateral weakness is thus the amount by which the responses to irrigation of the two ears differ, expressed as a percentage of the sum of all four responses.

Directional Preponderance: The same four elements are used to calculate directional preponderence, which represents the difference in intensity between the two right-beating nystagmus response (provoked by right ear-warm temperature and left ear-cool temperature irrigation's) and the two left beating response (provoked by left ear-warm temperature and right ear-cool temperature irrigation). Directional preponderence is calculated by the following formula:

$$\text{Directional Preponderence} = \frac{(RW+LC)\ (LW+RC)}{RW+LC+LW+RC} \times 100$$

Where the abbreviations are same as those used in formula for calculating unilateral weakness.

Fixation Index: The fixation index (FI) is a measure of the effectiveness of visual fixation in suppressing caloric nystagmus (Baloh et al.,1977; Barber, 1981). It is calculated by the following formula.

$$FI = \frac{SPES\ (EO)}{SPES\ (EC)}$$

Where SPES (EO) is the slow-phase eye speed of two or three representative beats occurring while the eyes are open and fixating, and SPES (EC) is the slow-phase eye speed of two or three representative beats, occurring just before the eyes are opened. The FI should be calculated for at least one right-beating and one left beating caloric response.

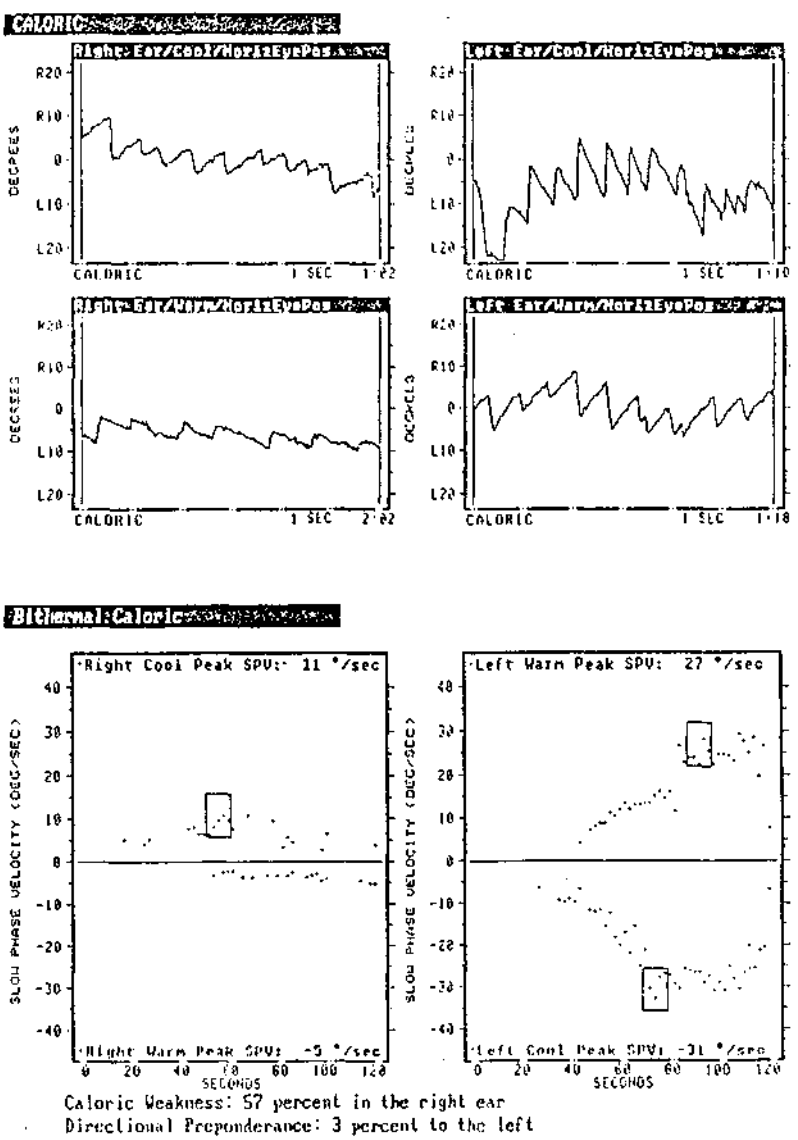


Figure 19: Abnormal bithermal caloric test report

Clinical Implication Of Unilateral Weakness, Directional Prepronderance And Fixation Index

Unilateral weakness calculation using caloric response indicate weakness of response to one ear. It is customary to denote a unilateral weakness according to the side of the weaker response, e.g. a unilateral weakness on the right side. A negative value of unilateral weakness indicate a weakness on the right and a positive value indicate a weakness on the left, although the weakness is usually apparent from inspection of the tracings. Various studies have indicated that a unilateral weakness of more than 15-25 percent are pathological (Barber and Stockwell, 1980). For standard clinical practice a unilateral weakness of more than 20 % is considered pathological. A significant unilateral weakness is usually considered as evidence of a lesion involving the labyrinth or vestibular nerve on the side of the weakness response (Uemuria and Cohen, 1973 ; Baloh et al., 1977).

Directional Preprondarance calculations identifies the right beating or left beating response are stronger. It is customary to denote a directional preprondarance according to the direction of the stronger responses, e.g. "a directional preprondarance to the right". A positive valve indicates a preprondarance to the right, and a negative valve indicates with preprondarance to the left, but it is usually easier to determine the direction of any significant preprondarance by inspecting the tracings. A preprondarance greater than approximately 20-30 percent is usually considered pathological (Barber and Stockwell, 1980). In standard clinical practice a value of greater than 25 % is

considered pathological. A significant directional preprondarance is usually considered as evidence of a lesion involving the vestibular pathways, either peripheral or central (Baloh et al., 1977).

Fixation index as mentioned earlier is the ability to suppress caloric nystagmus by fixating on a visual target. Alpert, (1974) has reported that on FI of 0.60 or greater is pathological, and Barber (1981) considers patients FI of 0.70 or greater to be pathological.

Abnormalities in Caloric test

Unilateral Weakness and Directional Preproderance

A significant unilateral weakness is a most important Electronystagmography finding. It commonly signifies a peripheral vestibular lesion on the weak side. With the exception of the identification of positional nystagmus of benign paroxysmal type with the Hallpike maneuver, no other Electronystagmography finding so positively identifies and lateralizes a peripheral lesion. A significant directional prepondarance, on the other hand, is of little clinical value. It is a sign that something is probably wrong, but it offers no localizing value, because is accompanies a wide variety of both peripheral vestibular and central nervous system disorders.

Bilateral Weakness

In some patients, the caloric response of both ears are very weak or absent when a response on both sides is less than 11° /sec to the warm irrigation's and less than 6° /sec to the cool irrigation's, bilateral weakness is

present; because it falls outside the 95 % lower limit of normal variation. Bilateral caloric reduction occurs with both peripheral vestibular lesions and central nervous system lesions.

Hyperactive Response

Caloric response may exceed the upper limit of normal variation, 50° /sec - slow phase eye speed for cool irrigation's and 80 /sec for warm irrigation's. Hyperactive response may occur if the caloric transfer qualities of the stimulated ear are greatly enhanced, such as when a mastoidectomy cavity is present or when the tympanic membrane is perforated, or retracted. Otherwise, these responses are generally explained as resulting from reduction of normal central nervous system (mainly cerebellar) inhibition of caloric activity.

Failure of Fixation Suppression

In all normal individuals, all patient's with peripheral vestibular disorders, and in some patients with central nervous system disorders, caloric nystagmus is suppressed by visual fixation. However, in some patient's with central nervous system disorders, nystagmus intensity with eye open nearly equals, matches, or exceeds that with eye closed. This effect is known as failure of fixation suppression and is good evidence of central nervous system localization.

Premature Caloric Reversal

The caloric response generally reaches peak intensity between 45 and 90 sec after the start of irrigation, and the nystagmus then slowly declines until it

stops after 200 sec. If the recorder is allowed to run, weak secondary nystagmus sometimes reappears, beating in the opposite direction, than it too declines to nil. This phenomenon is called caloric reversal. When the caloric reversal occur too early and is particularly strong, it denotes a disorder of central nervous system (probably cerebellar system) mechanisms that modulate the caloric response, and it may be a feature of posterior fossa lesions. We consider a true premature caloric reversal to be pathologic and indicative of central nervous system disease if the ear drums are intact after the onset of irrigation, and if slow phase eye speed is more than 60 to 70 /sec (Barber and Stockwell 1976).

Caloric Inversion and Perversion

The term caloric inversion refers to an entire caloric response that beats in the direction opposite to that expected. The term caloric perversion refers to the occurrence of vertical or oblique nystagmus as a response to a caloric irrigation. Both caloric inversion and perversion are taken as evidence of brain stem disease, but these abnormalities are rarely observed clinically (Barber and Stockwell, 1976).

Pitfalls

Weak or absent response due to inadequate irrigation, lack of temperature control, duration of flow, lack of alertness should be controlled to obtain accurate caloric response nystagmus. Similarly superimposed congenital nystagmus or gaze nystagmus must be identified that may result in miscalculation of the caloric response.

Extended Caloric Tests

The alternate binaural bithermal caloric test in which warm and cold water is sequentially injected into each ear canal is the standard form of caloric test used in most clinical set up. Various authors have proposed various types of caloric tests that can be used in electronystagmography. The electronystagmographic caloric test can be varied in several ways. Both the physical parameters and the temperature of the stimulus can be changed. Bithermal stimulation can be accomplished normally by the use of warm and cold water, but also by use of either warm and cool air or closed loop water system in which the water it self does not touch the tympanic membrane. In addition to these modification of the standard alternate binaural bithermal caloric test, other type of caloric testing can be done, such as a monothermal test using ice water, cool water, or warm water. The amount and duration of the stimulus can also be varied, as can the patient's position during the test. Another modification is the presentation of identical stimuli simultaneously to both ears. Described below are some of the other types of caloric test that can be used in electronystagmography.

Air Caloric Test

The air caloric test is performed by alternately injecting warm and cool air into the external auditory canal. The air caloric test has not become as standardized as the water alternate binaural bithermal test and various authors suggest different temperatures, rates of administration, and duration of the stimuli. In general most authors have tried to make the air stimulus produce the

same magnitude of response as the water stimulus. On the basis of a comprehensive study by coats et al., (1976) suggested the following air caloric parameters: temperature of 27.5 and 45.5 degree centigrade for 100 seconds at 13 liters/minute.

Advantages

Caloric stimulation using air has several advantages over the more commonly used water stimulation. It can be used in patient's with tympanic membrane perforation in whom the standard water irrigation's would be contraindicated and in patients with mastoid cavities, for whom the risk of a cavity infection with water irrigation's would be fairly high. In addition, the initial warm up of air irrigators is faster than the warm up of water irrigators.

Disadvantages

Disadvantages of the air irrigation system include patient discomfort from the noise stimulus produced by high airflow, burning sensation of the external auditory canal, and earache.

Simultaneous Binaural Bithermal Test

The simultaneous binaural bithermal caloric test has been advocated in recent years primarily by Brookler (1976). This test is performed by irrigating both ears simultaneously with water at 30° C for 60 sec while the patient's head is in the usual 30° upright position. After a five-minute pause the test is repeated using water at 44 C. The maximum slow phase velocity is recorded for each irrigation. Brookler categorized the results as follows

Type 1: No nystagmus to either warm or cool simultaneous stimulation.

Type 2: Nystagmus beats in one direction after stimulation with one temperature and in the opposite direction after stimulation with the other temperature.

Type 3: The direction of nystagmus remains the same regardless of the water temperature.

Type 4: Nystagmus with one temperature but not with the other.

Mono Thermal Caloric Tests

Mono thermal caloric tests have been recommended both as screening tests and as alternative to the standard alternate binaural bithermal test.

Warm Caloric Test

Warm caloric test is used as a screening tool in which caloric irrigation is done only for warm water as stimuli. The difference in slow phase velocity between two ears are considered for screening purpose.

Kobrak Minimal Caloric Test

The Kobrak minimal caloric test uses 5 ml of ice water to irrigate the ear canal. The duration of the nystagmus can be measured without the use of electronystagmographic recording but is more accurate when the electronystagmographic machine is used and slow phase velocity is calculated (figure 20). The main advantage of this test is its simplicity. There are, however, several disadvantages with ice water caloric stimulation. This test is not as accurate as alternate binaural bithermal stimulation in detecting vestibular abnormalities. The cold stimulus appears to be a stronger stimulus

than either of the standard bithermal stimulus and provokes more patient discomfort.

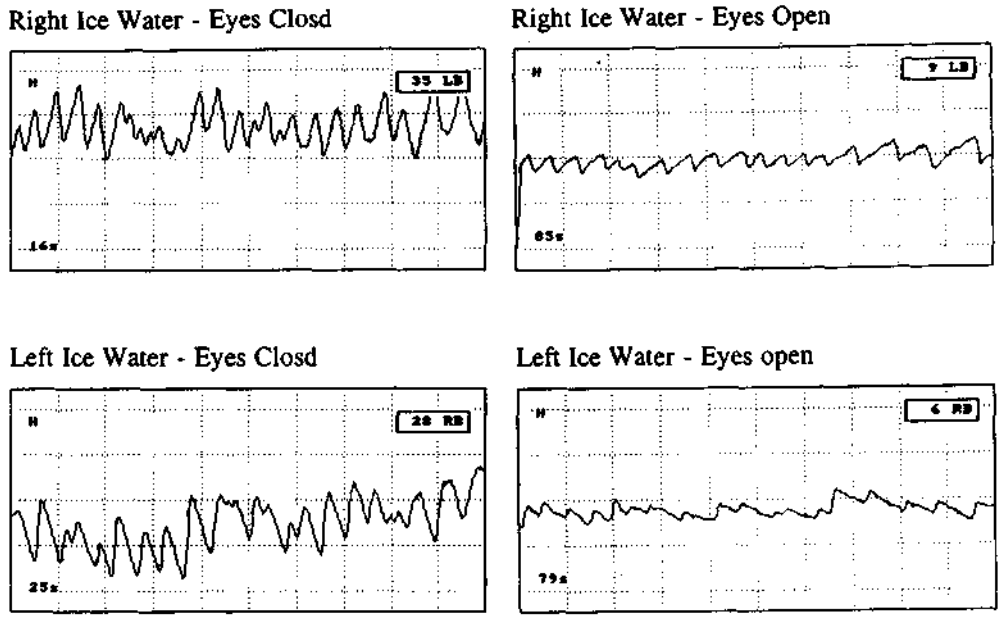


Figure 20 : Kobraks ice cold caloric test report

Monothermal Upright Inverted Caloric Test

Becker and Clemis (1979) examined a method of producing bidirectional nystagmus with a monothermal stimulus as a possible alternative to the alternative binaural bithermal test. The patient was seated with his head extended 60 degree. Eighty seconds after the onset of the irrigation, his eyes were opened for 5 sec to note fixation suppression, and then his eyes were closed and his head was placed forward to invert the lateral canal. The electronystagmographic tracing was continued for another minute while in this position. The average slow phase velocity was used to determine unilateral weakness and directional preprondarance using 20 % as the limits of normal.

Torok Monothermal Differential Caloric Test

In the Torok monothermal differential caloric test, each ear is irrigated with 10 ml and 100 ml of water at 20° centigrade for 5 and 20 seconds, respectively. The maximum frequency of nystagmus over 10 sec period is calculated for each irrigation and the ratio of the strong stimulus over the weak stimulus is determined. The normal range for this ratio is 1.2 to 3.5. Response greater than 3.5 are called recruitment and less than 1.2 is termed decruitment. In vestibular recruitment, the disproportionately greater response is seen with the strong stimulus. This response is seen in most patients with end organ pathology. In vestibular decruitment the response to the strong stimulus is equal or less than the response to the weak stimulus. The majority of patients who exhibits this phenomenon have evidence of central nervous system disease.

The ratio of the two ears is also compared and caloric response is considered asymmetric if the difference between the two ears exceed 25 %. A hypoactive response is one in which the maximum frequency of nystagmus for both irrigations in one ear are below normal. The main difference between the Torok monothermal differential test and the alternate binaural bithermal test are the monothermal irrigations at two intensities.

Summary

The standard alternate binaural bithermal irrigation remains the most popular test of vestibular function. The use of air caloric irrigators or closed

loop water system seen to be scientifically sound alternatives to use of open water irrigation. Although controversy still surrounds the use of monothermal warm calorics it can be used as a screening test. Some of the other test mentioned may be helpful as ancillary tests for use in patients in whom the standard test produce inconsistent findings.

TORSION SWING TEST

Rotational stimulation of the vestibular apparatus has been performed for over 150 years. It was Barany who introduced rotatory stimulation as a clinical test for vestibular disorder. With rotatory testing, the patient is seated in a chair that rotates about its vertical axis. The head is fixed so that angular rotation occurs in the plane of one of the semicircular canal pairs (usually with head tilted 30 degrees forward so that the lateral canal will be horizontal). Rotatory tests of the vestibulo-ocular reflexes have not been widely accepted as a part of routine vestibular examination for two reasons.

1. Expensive, bulky equipment is usually required in order to generate precise rotatory stimuli
2. Rotatory stimuli affect both labyrinths simultaneously compared to the selective stimulation of one labyrinth, which is possible with caloric tests.

4 Three types of angular acceleration have been used to clinically evaluated the vestibulo-ocular reflex: (1) impulsive (2) constant (3) sinusoidal. With the development of electronystagmography came the ability to record nystagmus during rotation. Soon rotatory tests using constant and sinusoidal acceleration became popular in several clinical laboratories.

Torsion Swing Chair

Several investigators have suggested that the sinusoidal rotatory stimulus is more efficient and quantitative stimulus than impulsive or constant acceleration. A sinusoidal stimulus is defined by two simple variables

1. The period of oscillation and
2. The amplitude of oscillation

Both of these variables can be controlled with relatively simple mechanical devices.

Torsion swings are in expensive, uncomplicated and with electronystagmography can very easily record per-rotatory test nystagmus. A per-rotatory test investigates the nystagmus evolved during rotation, in contrast to the rotatory tasks where post rotatory nystagmus is studied.

During torsion swing test the patient is seated in a chair whose rotation is mechanically controlled by action of a calibrated spring, when the chair is moved from its neutral position it slowly returns to that position with a damped sinusoidal oscillation. This stimulus alternatively deviates the capula in ampullopetal and ampullofugal directions producing nystagmus that alternates direction with each half cycle of rotation. Most commonly, the maximum or the average slow component velocity and maximum nystagmus frequency are used to quantify the response during each half cycle. Responses to clockwise and counter clockwise rotation can be compared in a given subject; however, the stimulus intensity is dependent on the weight and distribution of mass in the chair and so varies from subject to subject.

Besides nystagmus, compensatory eye movements can also be recorded on the torsion swing, when the excursions of the swing are very small, we can observe varying eye deviations, which are essentially the expression of the

slow nystagmus phase. When the stimulus of the swing is small, the slow phase does not reach the critical value to prevoke the quick phase.

Interpretation of Torsion Swing Results

Although the torsion swing chair has not enjoyed tremendous popularity, there are situations where its use may be desirable, certainly the stimulus is more physiologic than the water in the ear canal and is much more pleasant. In the context of vestibular testing, the torsion swing chair serves an adjunctive relationship to caloric testing. Interpretation of rotatory testing results encompasses the following treatment: directional preprondarance, maximum slow component velocity and the rotatory ocular fixation index.

Direction Preponderance

Directional preponderance of nystagmus is calculated by simply counting the number of nystagmus beats in one direction compared to the number of beats in the opposite direction. When the beats in one direction outnumber the beats in the other direction by more than 23 %, a directional preponderance is said to exist.

Baloh et al., (1979) found that the variance associated with difference measurements comparing clockwise (CW) and counterclockwise (CCW) responses in the same subject is much less than the variance in response between subjects.

$$\text{Directional Preponderance} = \frac{CW - CCW}{CW + CCW} \times 100$$

This formula is analogous to the directional preponderance formula used with caloric testing. The mean value for the normalized difference between clockwise and counter clockwise response is approximately zero at each stimulus intensity, demonstrating that clockwise and counter clockwise responses are symmetrical in normal subjects. In abnormal subjects these difference measurement were consistently more sensitive indicator of impaired function than absolute magnitude measurements. When slow component velocity was used in the equation, it proved even more effective in identifying unilateral disease.

Maximum Slow Component Velocity

As in the case of caloric testing maximum slow component velocity is more useful for distinguishing normal from abnormal reactions. For rotatory stimuli the log of maximum slow component velocity increases linearly with the log of stimulus intensity. Even with precisely controlled rotatory stimuli, however, there is a large variation in the vestibulo-ocular reflex function measured in normal subjects. This variability in response is not related to the type of rotatory stimulus since it is identical for the impulsive and sinusoidal stimuli. Factors such as stress, fatigue, level of mental alertness, and habituation all contribute to the variability.

Abnormalities

Unilateral Disease

Although rotatory testing can consistently identify complete unilateral peripheral vestibular paralysis, it infrequently identifies partial peripheral

vestibular lesions. Patients with unilateral loss of vestibular function should have asymmetric responses to rotatory stimuli because of the difference in excitation and inhibition with ampullopetal and ampullofugal stimulation of the intact labyrinth.

Bilateral Disease

Rotatory stimuli are ideally suited for testing patients with bilateral symmetrical peripheral vestibular lesions since both labyrinths are stimulated simultaneously. Frequently, patients with absent response to caloric stimulation may have recordable rotatory induced nystagmus particularly at higher stimulus intensities. Nystagmus frequency and maximum slow component velocity can be compared on a serial basis using torsion swing. This rotatory test is more precise than calorics in this situation since bilateral testing is being done and the stimulus intensity is easily quantified and reproducible over time.

Central Lesions

As with lesions of the peripheral vestibular structures, lesions of the central vestibulo-ocular reflex pathway can lead to a decrease or asymmetry in the velocity of slow component of nystagmus. The spectrum of abnormality associated with central lesions, however, is more diverse than a simple decrease in the slow component velocity of induced nystagmus. The highly organized pattern of nystagmus usually produced by rotatory stimuli in normal subjects may become very disorganized in patient's with central lesions, especially when the prepontine reticular formation causes abnormal fast

components. In the patient with cerebellar atrophy, the nystagmus pattern is disorganized with fast components occurring in random fashion causing marked beat-to-beat variability in amplitude. This type of abnormality has been termed nystagmus dysrthythmia and is commonly found in patient's with all varieties if cerebellar lesions.

The role of torsion swing test is clearly adjunctive to caloric testing. The question is how can the torsion swing test augment caloric testing, not whether caloric testing can be replaced by torsion swing. The swing test is definitely useful in recording minimal residual labyrinthine function. The pattern of nystagmus induced by torsion swing chair can be faithfully analyzed in patient's with central disease. Furthermore, there is little known about the contribution of the otolithic organs to this form of testing.

STUDY QUESTIONS

I. Fill up the blanks

1. The internal ear is embedded within the petrous portion of the————
2. The fluid present in the bony labyrinth is————and in the membraneous labyrinth is————
3. The component parts of the vestibulae are————and————
4. The arterial blood supply of the labyrinth is derived from————artery
5. The artery that supplies the vestibular nerve is————
6. ————;————and————are the three semicircular canals present in the inner ear.
7. Actual tracing or recording produced by using electronystagmography is called————
8. The two component of vestibular nystagmus are————and————
9. The direction of nystagmus is named after the direction of———— component.
10. ————potential is the principle underlying the recording of electronystagmography
11. The electrical potential in the cornea is————charged and that of retina is————charged.
12. The two vertical semicircular canals-the posterior and superior join posteriorly to form a————
13. The macule utricle and macule sacculi are collectively known as the ————organ

14. The utricle and saccule communicates posteriorly by————————duct to the endlymphatic duct.
15. The semicircular canal responds to the————————acceleration of the head.
16. In caloric test————————semicircular canals are maximally sensitive to thermal stimulation.
17————————system is responsible for maintaining or stabilizing the gaze of a moving target on the fovea in the eye.
18. In a vestibular nystagmus————————phase of the nystagmus is as a result of weak labyrinth pulling the eye towards it.
19. In persons with conginetal nystagmus, optokinetic test show————————
20.————————,————————and————————are the major characteristics of benign paroxmysal positional vertigo.
21. The caloric irrigation temperature for warm water is————————and cool water is————————
22.————————caloric test is preferred in case the patient has a tympanic membrane perforation
23.————————caloric test uses ice water to irrigate the ear canal.
24.————————,————————and————————.~ are the three types of angular acceleration that can be used to evaluate vestibulo-occular reflex.
25. In Torok monothermal differential caloric test a ratio of greater than 3.5 is called————————.

II. Choose the appropriate answer

1. A nystagmus can be classified as

   a) occular nystagmus
   b) peripheral nystagmus
   c) central nystagmus
   d) all of the above.

2. Widely used parameter for measurement of nystagmus strength is

   a) duration of nystagmus
   b) frequency of nystagmus
   c) velocity of slow phase component
   d) all of the above.

3. Direction changing gaze nystagmus seen in gaze test is indicative of

   a) central vestibular lesion
   b) peripheral vestibular lesion
   c) both central and peripheral lesion
   d) None of the above.

4. Vertical nystagmus is caused by

   a) peripheral vestibular lesion
   b) central vestibular lesion
   c) both central and peripheral lesion
   d) None of the above.

5. The plane of the eye movement in nystagmus are

   a) horizontal
   b) vertical
   c) rotatory
   d) all of the above

6. Nystagmus present for an infinite duration is called————Nystagmus

   a) transitory
   b) rotatory
   c). persistent
   d) positional

7. The nystagmus that is referred to as clockwise or anticlockwise is———— nystagmus

   a)> rotatoiy
   b) positional
   c) tansitory
   d) all of the above.

8. Which of the following type of simulation can elicit and induce nystagmus

   a) rotation
   b) caloric
   c) cervical
   d) all of the above.

9. The recording obtained in an electronystagmography testing is

   a) Audiogram
   b Calorigram
   c). Nystagmogram
   d) None of the above

10. Direction fixed persistent positional nystagmus was classified by Aschen et al., as

   a) Aschan Type 1
   b) Aschan Type II
   c) Aschan Type III
   d) None of the above

11. Exactly————degree of elevation of head is required for performing the caloric test.

a) 20
b) 25
c) 30
d) 35

12. Which of these caloric irrigator has temperature controlled water circulated through a silastic bag

a) open water irrigator
b) closed loop irrigator
c) air caloric irrigator
d) all of the above.

III. State True or False

1. Saccule is connected anteriorly by the ductus reuniens to the cochlea.

2. A long kinocilum is present on the opical surface of the vestibular hair cells.

3.. The otolith organ responds to angular acceleration

4. A saccade is a slow eye movement

5. The saccades are generated by burst neurons in the paramedian pontine reticular formation and pretectal region

6. In ENG recording system the movement of eyeballs to the right produces downward deflection of the recording pen.

7. The horizontal electrode are placed just lateral to the outer canthi close to the eye

8. The fast phase of nystagmus is mediated by reticular formation

9. The fast phase of nystagmus is as a result of the weak labyrinth pulling the eye towards it
10. Congenital nystagmus can interfere with gaze nystagmus recording
11. Saccadic pursuit is seen in saccadic test is indicative of central vestibular lesion
12. Benign Paraoxysmal positional vertigo is indicative of a central vestibular lesion
13. Bithermal caloric irrigation is done at 7 degrees above and below the body temperature
14. In caloric test the patient is placed in supine with head ventroflexed by 45 C- degrees
15. In alternate bithermal caloric test both ears are irrigated with either hot or cold water simultaneously

IV. Match the following

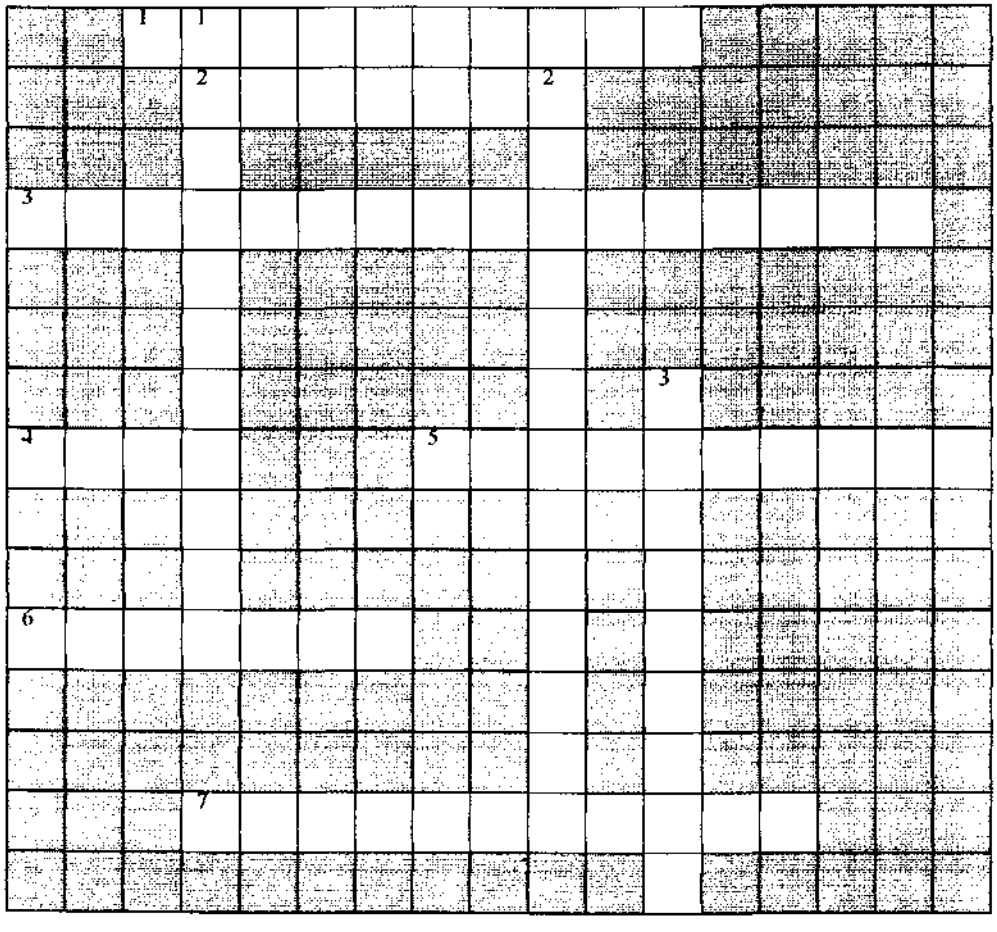
1. Congenital Nystagmus	A. Cog wheeling
2. Occular square waves	B. Alcoholic
3. Saccadic pursuit	C. Brainstem lesion
4. Absent optokinetic nystagmus	D. Tense / nervous patient
5. Benign paroxysmal positional vertigo	E. Undershooting response
6. Hypometric saccades	F. Peripheral lesion
7. Positional alcohol nystagmus	G. Null point / convergence effect

V. Given below are Anagrams of different types of Nystagmus unscramble them

1. NETONCGILA
2. AZGE
3. IICTENOOPTK

4. ANONTPSUSED
5. SITALNIOOP

VI. CROSS WORD PUZZLE : Identify the word from clues given below and the number of letters the word has as given in bracket.



Across :

1. Nystagmus resulting from various body position (10)
2. Slow eye movement (7)
3. Device for pouring hot / cold water to ear canal (16)
4. Type of abnormal nystagmus present on lateral eye position (4)
5. Nystagmus present since birth (10)
6. Refers to fast eye movement (7)
7. This is done before each sub test (11)

Down:

1. Nystagmus that can be seen while watching objects from a running train (11)
2. A type of rotational test (12)
3. Nystagmus that beats towards gravity (9)

VII. Identify as many types of nystagmus as you can in this word grid.

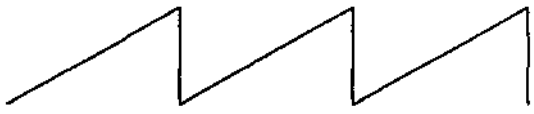
G	**R**	**O**	**P**	**I**	**Z**	**A**	**T**	**0**	**D**	**E**	**F**	**F**	**C**
N	**Y**	**R**	**0**	**T**	**A**	**T**	**0**	**R**	**A**	**Z**	**D**	**N**	**0**
I	**H**	**M**	**M**	**A**	**T**	**P**	**A**	**0**	**L**	**I**	**T**	**U**	**N**
T	**T**	**N**	**E**	**T**	**A**	**L**	**B**	**B**	**X**	**Y**	**0**	**P**	**G**
A	**O**	**T**	**N**	**L**	**U**	**A**	**T**	**N**	**G**	**I**	**L**	**B**	**E**
E	**P**	**T**	**O**	**C**	**A**	**X**	**N**	**T**	**O**	**N**	**T**	**E**	**N**
B	**S**	**P**	**0**	**N**	**T**	**A**	**N**	**E**	**O**	**U**	**S**	**A**	**I**
N	**U**	**S**	**W**	**L**	**M**	**N**	**G**	**B**	**E**	**Z**	**P**	**T**	**T**
W	**T**	**X**	**Y**	**Z**	**A**	**A**	**I**	**0**	**T**	**A**	**Q**	**I**	**A**
0	**B**	**S**	**E**	**N**	**Z**	**T**	**M**	**1**	**E**	**T**	**T**	**N**	**L**
D	**C**	**I**	**T**	**E**	**N**	**I**	**K**	**0**	**T**	**P**	**0**	**G**	**0**

VIII. Answer the following in two or three lines

1. What are the functions of the vestibular system?
2. Name the areas in which sensory epithelium are present in the vestibular receptor organ?
3. What is a nystagmus?
4. Given below is a vestibular nystagmus, identify the direction of the beat?

5. What is a rebound nystagmus?

6. How will you differentiate between a gaze nystagmus and a congenital nystagmus?
7. What is saccule and how does it communicate with the endolympatic duct?
8. Write briefly about the capula?
9. What happens when the endolymph moves in the semi-circular canals?
10. Write the various direction of eye movement in nystagmus?
11. How is the peripheral vestibular nystagmus produced?
12. What do you mean by central vestibular nystagmus?
13. What is electronystagmography?
14. Identify the slow and fast phase of the nystagmus in graph given below?



15. What do you mean by vestibulo-ocular reflex?
16. How can a vestibulo-ocular reflex be elicited?
17. What is a positional test?
18. Name any two mental alerting task?
19. Write the test instruction for gaze test?
20. Write the test instruction for saccadic test?
21. Write how calibration is done in electronystagmography?
22. Is it necessary to calibrate before each sub test. If so why?
23. What are the common sources of artifacts in electronystagmography testing?
24. How would you instruct the patient for pursuit test?

25. What precaution should be taken while carrying out a pursuit test?

26. How will you do a calibration before a caloric test?

27. When would you call a nystagmus seen in positional test as pathological?

28. Mention the three types of caloric irrigator available commercially?

29. What are the basic desirable characteristics of a caloric irrigator?

30. What is meant by unilateral weakness?

31. What is meant by directional preponderance?

32. Given below is the formula to calculate the unilateral weakness. Find the missing component?

$$UW = \frac{(RW+RL) - (LC-LW)}{RC + RW + \text{———} + \text{———}}$$

33. What is fixation index?

34. How is fixation index calculated?

35. Calculate the unilateral weakness and directional preponderance using the following data?

    a) Right warm : 11 degrees /sec
    b) Right cool : 14 degrees /sec
    c) Left cool : 35 degrees /sec
    d) Left warm : 32 degrees /sec

36. Calculate the fixation index from the following data?

Right beating eyes open = 3 degrees/sec
Right beating eyes closed = 12 degrees/sec
Left beating eyes open = 5 degrees/sec
Left beating eyes closed - 12 degrees/sec

ANSWERS TO STUDY QUESTIONS

I. Fill up the blanks

1. Temporal bone
2. Perilymp, endolymp
3. Utricle, Sacuule
4. Labyrinthine
5. Anterior vestibular
6. Posterior, Lateral, Superior
7. Nystagmogram
8. Slow phase, Fast phase
9. Quick/Fast
10. Corneoretinal
11. Positively, Negatively
12. Single criscommune
13. Otolith
14. Utriculo saccular
15. Angular
16. Lateral
17. Pursuit
18. Slow
19. Optokinetic reversal
20. Delayed onset, presence of vertigo, fatigability
21. 44 degree centigrade, 30 degree centigrade
22. Air
23. Kobrak Minimal
24. Impulsive, constant, sinusoidal
25. Recruitment

11. Choose the appropriate answer

1. **D**
2. **C**
3. **A**
4. **B**
5. **D**
6. **C**
7. **A**
8. **D**
9. **C**
10. **B**
11. **C**
12. **B**

III. State true or false

1. True
2. True
3. False
4. False
5. True
6. False
7. True
8. True
9. False
10. True
11. True
12. False
13. True

14. False
15. False

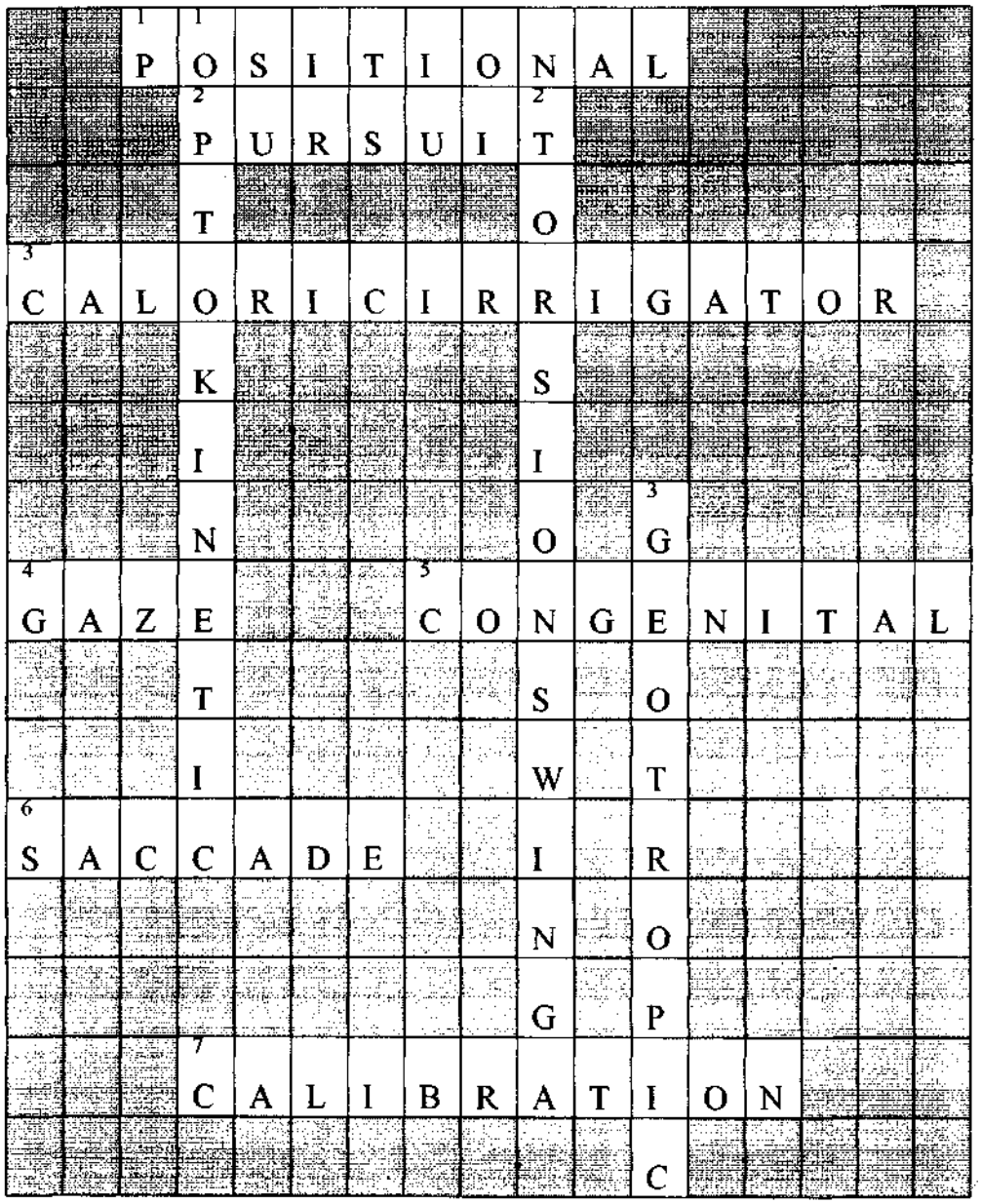
IV. Match the following

1. **- G**
2. **- D**
3. **- A**
4. **- C**
5. **- F**
6. **- E**
7. **- B**

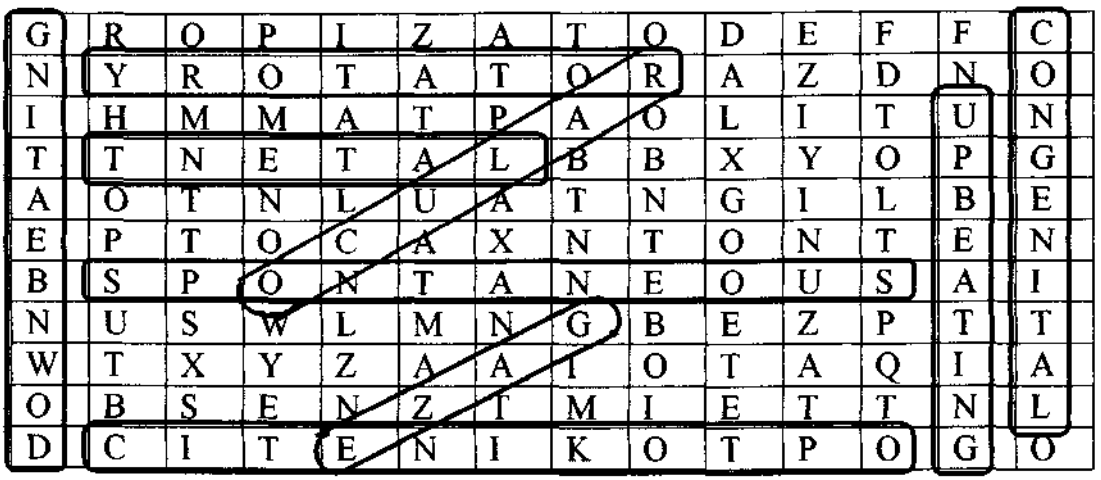
V. ANAGRAMS

1. Congenital
2. Gaze
3. Optokinetic
4. Spontaneous
5. Positional

VI. : Answers to Cross Word Puzzle



VII: Answers to Word Grid



VIII: ANSWERS

1. The functions of the vestibular system are

   a. To detect the body motion (linear and angular acceleration) as monitored by head motion.

   b. The detection of head in space relative to the gravitational tilt.

2. The five areas of sensory epithelium in the vestibular receptor organ are the two maculae of otolith organ (utricle and saccule) and three cristae ampulares of the semicircular canals.

3. A nystagmus is nothing but a repetitive involuntary oscillatory movement of the eye.

4. Right beating nystagmus

5. Rebound nystagmus is a type of central gaze nystagmus that is opposite beating and occurs in the position of primary gaze after a gaze nystagmus is ellicited.

6. Congenital nystagmus can be differentiated from gaze nystagmus in electronystagmographic recording due to the following characteristics.

(i) It has a null point in which nystagmus declines markedly or stops.

(ii) It may be horizontal or rotatory and rarely vertical

(iii) It always horizontal in direction on upward gaze

(iv) Reduction or abolition of the nystagmus on convergence.

7. Saccule is part of the otolith organ, almost globular in shape lying in a recess near the opening of the scala vestibuli of the cochlea. It communicates with endolympatic duct by the utriculo saccular duct.

8. The capula completely scales off the ampulla. The capula of the superior and horizontal semicircular canals are anteriorly placed, while the capula of the posterior semicircular canal is located inferiorly. Movement of the head causes capular displacement in the opposite direction.

9. Movement of the endolymph will either move the capula towards the utricle in the vestibule (utriculopetal) or away from the utricle (utriofugal). When there is no movement of the endolymph in relation to the capule it remains in the mid position in the ampulla.

10. Direction of eye movement in a nystagmus could be towards the right, left, up, down, clockwise or anticlockwise

11. The peripheral vestibular nystagmus is caused by an imbalance in the resting discharge rate of paired peripheral end organs, either the semicircular canals or otolith organs. It could be spontaneous or elicited by a myraid of stimuli.

12. Central vestibular nystagmus refers to nystagmus that is not characteristically elicited or found in normal subjects or in subjects with end organ disease.

13. Electronystagmography is a technique used for recording nystagmus. It is a systamatic test which utilizes the cornea retinal potential to produce graphic records of vestibular functions.

14. Figure

15. The vestibular system is extremely important in controlling congugate eye movements reflexively in response to head movement and to the position of the head in space. The asymmetry resulting from action of semicircular canals cause a compensatory reflexive eye movement in the plane of the canal being

stimulated. This compensatory reflex movement of the eye, is called vestibulo-ocular reflex.

16. Vestibulo-ocular reflex can be elicited by either a caloric test or rotational test like torsion swing test.

17. The positional test is a sub test of electronystagmography in which the patient head and body position are changed to identify the effect of body position on spontaneous nystagmus if present or to uncover a nystagmus which might be present in some position other than supine.

18. Two mental alerting task are

(i) Count 1-100 loudly

(ii) Count subtracting 3's from 50 downwards

19. Test instruction for gaze test is that you will be seeing a light on the digital light frame. Keep your eyes fixed on it without turning your head, and ovoid blinking. When you are asked to close your eye, please close your eye in the some position.

20. Instruction for patient in saccadic test is that a light will be moving on the digital light frame from right to left. Move your eyes quickly with the light i.e. when the light goes to the right, your eye should move immediately to right and similarly to left.

21. Calibration in electronystagmography is done for both horizontal and vertical channels. Patient is asked to alternately look at light fixed at a

precalibrated position usually 20° visual angle. Gain controls needs to be adjusted so that the pen moves lmm per degree of eye displacement.

22. The calibration changes during testing in an unpredictable manner because of fluctuations in the magnitude of corneoretinal, therefore recalibration must be done at regular intervals preferably before each sub test and before each caloric irrigation.

23. Two commonly encountered types of artifacts are

(i) Those caused by electrical interference as caused by broken electrodes, movement of electrode lead wire and

(ii) 60 Hz hum due to high electrode impedance.

24. Patient is instructed to fallow movement of a pendulum or light moving on the digital light bar with his eyes moving back and forth slowly along with the light or pendulum. He should not turn his head with the movement of light.

25. Precaution to be taken are that the patient has understood the instruction for the test, that the patient is not moving his head along with the light and to give verbal feedback on his response pattern.

26 Calibration for caloric test is done in the supine position itself. He is asked to look alternatcly at lights fixed on the ceiling at a predetermined angle.

27. Positional nystagmus is considered abnormal if

(i) It changes directions in any head position

(ii) It is present in three or more of the five head position tested.

(iii) It is intermittent in four or more head positions

(iv) The slow phase eye speed of the three strongest consecutive beats exceeds 6° /sec in any head position.

28. The three type of caloric irrigators available are

(i) Open water irrigator

(ii) Closed loop irrigator

(iii) Air caloric irrigator

29. Caloric irrigator used in electronystagmography needs to have the following basic characteristics

(i) Warm and cold irrigating temperatures maintained in separate reservoirs with ± 0.1 c acuracy.

(ii) Automatic, timed delivery, preferably activated by a foot switch, leaving both hands free to position the irrigating tip.

30. Unilateral weakness is a caloric response that indicate a weakness of response to one side. It is the amount by which the two responses provoked by right ear irrigations differ in intensity from those provoked by left ear irrigations.

31. In caloric test, directional preponderance identifies whether left or right beating caloric response is stronger

32. The missing component is LW + LC

33. Fixation index is an index that represents the ability to supress caloric nystagmus by fixating on a visual target.

34. Fixation index is calculated by the formula

$$FI = \frac{SPES\ (EO)}{SPES\ (EC)}$$

Where SPES(EO) is slow phase eye speed of nystagmus in eyes open position and SPES(EC) is slow phase eye speed of nystagmus in eye's closed position.

35. Formula

$$UW = \frac{(11+14)-(35+32)}{11+14+35+32} \times 100 = -46\%$$

$$DP = \frac{(11+35)-(14+32)}{11+35+14+32} \times 100 = 0\ \%$$

36. For right-beating nystagmus

Fixation Index (FI) = EO/EC = 3/12 = 0.25

For left beating nystagmus

Fixation Index (FI) = E0 /EC = 5/12 = 0.42

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