E L E C T R O N Y S T A G M O G R A P H Y

- A Review of Literature.

REGISTER No.9

An Independent Project Work submitted as
Part fulfillment for First Year M.Sc.
(Speech & Hearing)
to the
University of Mysore .

ALL INDIA INSTITUTE OF SPEECH & HEARING
M Y S O R E - 570 006

to dearest

babi and daddy

who continue to inspire.

C E R T I F I C A T E

This is to Certify that

the Independent Project entitled:

" ELECTRONYSTAGMOGRAPHY- A REVIEW OF LITERATURE "

is the bonafide work done in part fulfilment

for First Year M.Sc(Speech and Hearing),

of the student with Register Number:9

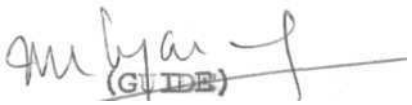
DIRECTOR,
ALL INDIA INSTITUTE OF SPEECH AND HEARING, MYSORE- 570 006.

C E R T I F I C A T E

This is to Certify that the Independent Project Entitled:

" ELECTRONYSTAGMOGRAPHY- A REVIEW OF LITERATURE "

has been prepared under my guidance and Supervision.



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DECLARATRION

This Independent Project Entitled-

" ELECTRONYSTAGMOGRAPHY- A REVIEW OF LITERATURE "

is the result of my own work undertaken under the guidance of Mr.M.N.Vyasmurthy, Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore,-6 and has not been submitted earlier at any University or Institution for any other Diploma or Degree.

Mysore:

Dated :30th April,1982 REGISTER NO. 9

I am grateful to. - -

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ELECTRONYSTAGMOGRAPHY- A Review of Literature:

Chapter-1

INTRODUCTION.

Electronystagmography is a clinical tool which involves the acquisition of a permanent quantifiable record of eye-motion. Du Bois-Reymond(1849) discovered that a potential difference exists between the cornea and the retina in man (Corneoretinal potential). The Cornea was found to be positively charged and the retina was found to be negatively charged. The eye therefore functions as a'rotating dipole' and this characteristic is utilized in the 'skin electrode technique' known as "electronystagmography" ENG records may be interpreted with consideration of a growing body of information regarding normal and abnormal patterns of eye motion associated with the function of the oculomotor and vestibular systems.

ENG has become an important element in the complete nero-otoaudiologic diagnostic procedure particularly for individuals with unilateral hearing loss tinnitus dizziness and vertigo. ENG makes it infinitely easier to decide that the patient has:

(a) A normal vestibular mechanism?

(b) Disease of one or other labyrinthine end organs

(c) Retrolabyrinthine or central nervous systerr disease.

ENG makes it possible to discover nystagmas which is otherwise suppressed with the eyes open. It makes possible to measure the velocity of mystgrmus. ENG provides us with an objective repertable, permanent record that will be of significant

advantage in medicolegal problem involving the vestibular and CNS system.

The correlation of ENG recordings with the total patient evaluation by the Physician will be the best way to arrive at the diagnoses.

Chapter-II

Vestibular Apparatus

Anatomy

The sensory structures of the vestibular system known collectively as the vestibular apparatus, are contained within a membranceaslabyrinth which is supported by fibroas strands inside a contoured cavity, the bony labyrinth, in the petrous portion of the temporal bone. The membranous labyrinth an interconnected series of tubes and sacs, forms a closed container filled with a fluid, endolymph. The space outside the membranous labyrinth, between it and the bony labyrinth, is filled ē perilymph. The membranous labyinth contains two types of sensory organs: the semi-circular ducts, whose sensory receptors are primarily sensitive to angular accleration, and the otolith organs, the utricle and saccale, whose sensory receptors are primarily sensitive to linear acceleration and change in linear acceleration.

Three semicircular ducts, the anterior, the posterior and the lateral, form the part of the membranous labyrinth in each inner ear. In man, each duct is roughly in a single plane and approximately at Rtrs to the other two, the lateral duct is in a plane inclined about 30° from the horizontal head plane, while the anterior and posterior ducts are approximately in vertical head planes. The anterior semicircular duct of the labyrinth in one ear is coplanar with the posterior semicircular

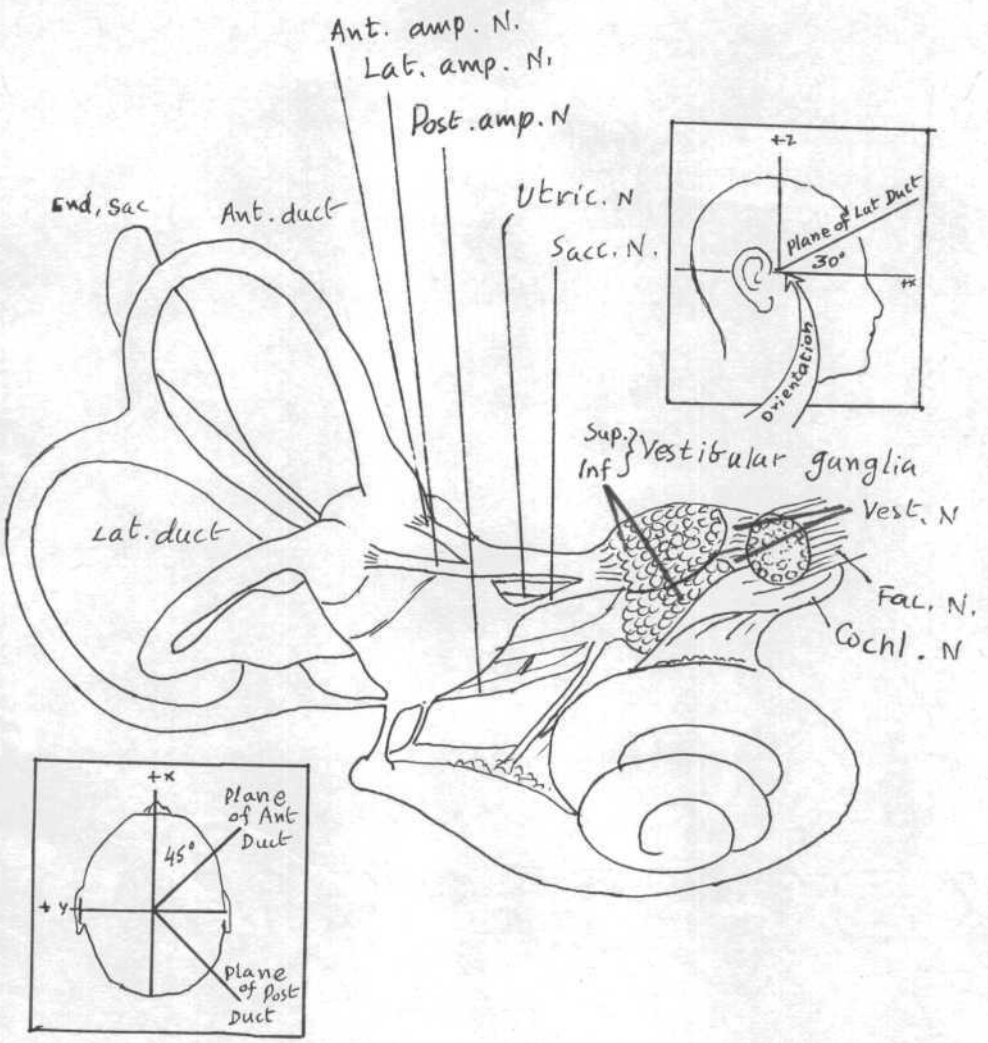


Fig A: Orientation and gross morphology of the semicircular ducts and otolith organs as well as their innervations.

Inset: The major planes of the SC Ducts relative to their cardinal head axes (Hixson et al., 1966)

duct of the labyrinth in the contralateral ear. This plane is approximately 45° from the sagittal head plane.

Each semicircular duct widens at one point before it joins the utricle. This dilation is the ampullar. It contains the neuroepithelium of the semicircular duct. This neuro-epithelium as well as supporting cells, connective tissue, blood vessels, and nerve fibres form the crista ampullaris. The crista ampullar's is a saddle-shaped ridge which extends across the floor of the ampulla at rt Ls to the long axis of the ampulla.

Chapter-III

Physiology.

Vestibulo-ocular interaction:

The peripheral vestibular system consists of 5 sensory receptors that are contained within each membranous labytrinth These receptors are (1) the macula of the saccule; (2) the macula of the utricle and(3-5) the crista -cupula apparatus in each of the three semicircular canals. Each of, these receptors contains sensory cells having cilia that are in intimate contact with an overlying membrane; in a fashion similar to that of the cochlear hair cells and tectorial membrane. The spiral cochlea is phylogenetically newer than the vestibular apparatus and represents a refinement of the vestibular sensory system.

The sensory nerve fibres from each of the 5 vestibular receptors join together to form the vestibular branch of the auditory nerv and they project with the brain stem to form synaptic connections in the vestibular necleus.

The vescibular nucleus consists of four discrete divisions from which neurons emerge to supply information to the visual and somatosensory systems. The medial division of the nucleus receives a portion of its input, from the semicircular canals and send projections to the oculomotor, trochleay and abducen nuclei. The abducens nerve (VI nerve) emerges from its nucleus and innervates the lateral Rectal muscle, which pulls the eye outward. The trochlear(IV) innervates the superior

oblique muscle which pulls the eye downwards. The oculomotor(III) supply the medial rectus, which rotates the eye inward. The oculomotor nerve also supplies the inferior oblique and inferior rectus muscles.

While the overall organization of the vestibulo-ocular system is very complex, the innervation pattern just describes allows each of the semicircular canal receptors to influence eye motion in the plane of the canal in which the receptor resides .
Of particular importanceto the ENG evaluation is the fact that the horizontal canal receptors have a substantial effect upon the motion of the eye in the horizontal plane, or "to and fro" from the individual's right to left side. In the normal case this influence vestibulo labrynth contains endolymph which lags behind the motion of the head itself whenever the head is acclerated or declerated. The fluid lag is due to inertia, and it results in a brief relative motion within the membranous labyrinth. The fluid motion in turn results in a displacement of the cristacupula apparatus housed within each canal in a manner that would occur with any pliable object placed in the path of the flowing liquid.

The diagram in Fig.2 illustrates these effects. Part A of the figure is a schematic of the horizontal semicircular canal in the resting position The crista-cupula apparatus is shown to be contained within an enlargement of the canal C is called the ampulla. The ampulla is located in the rostral portion of the

canal(the portion closest to the front of the head) when the head is rotated towards the left side, as shown in Part-B, the endolymph which is present in the canal legs briefly and undergoes a relative motion towards the ampulla.. The direction of this motion is called ampullo-petal. The crista-cupula apparatus will be displaced in the direction of the momentary flow of the endolymph. Wher experiencing rotation of this type the normal individual will produce compensatory eye motion towards the Right side, which is opposite to the direction of rotation. The eyes will move slowly to the Right until a maximum deviation is reached and they will return quickly before repeating the slow compensatory motion to the Bight . It is the left horizontal canal which has promoted this compensatory^ ** ' - - "... ^ * -'. t* * ** ^ *' J$eye motion, acting, of course in concert with the canal on the Right side of the head.

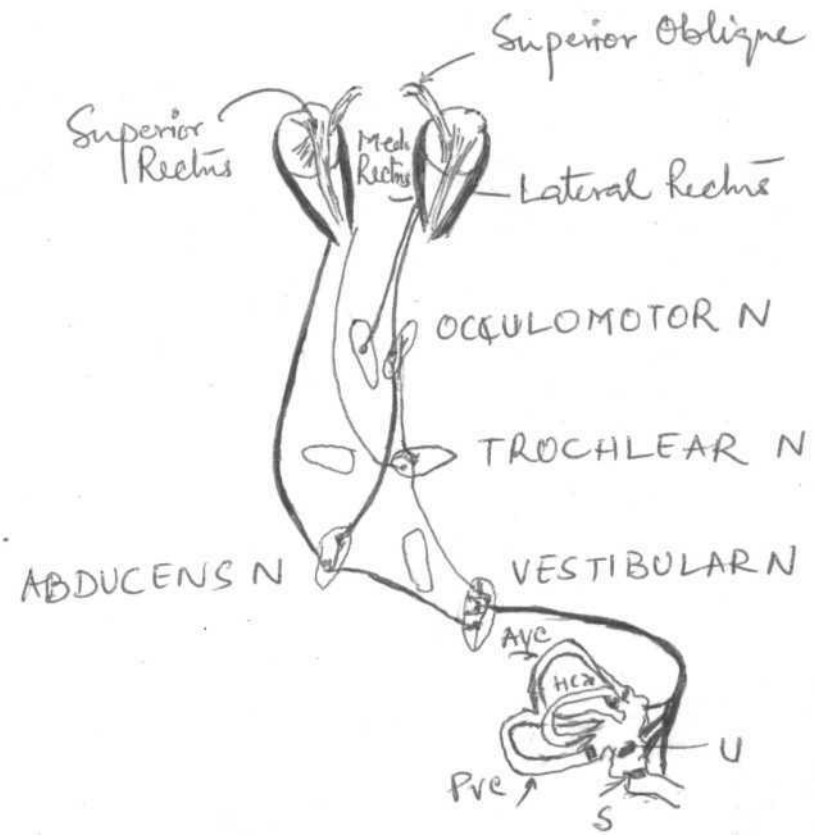
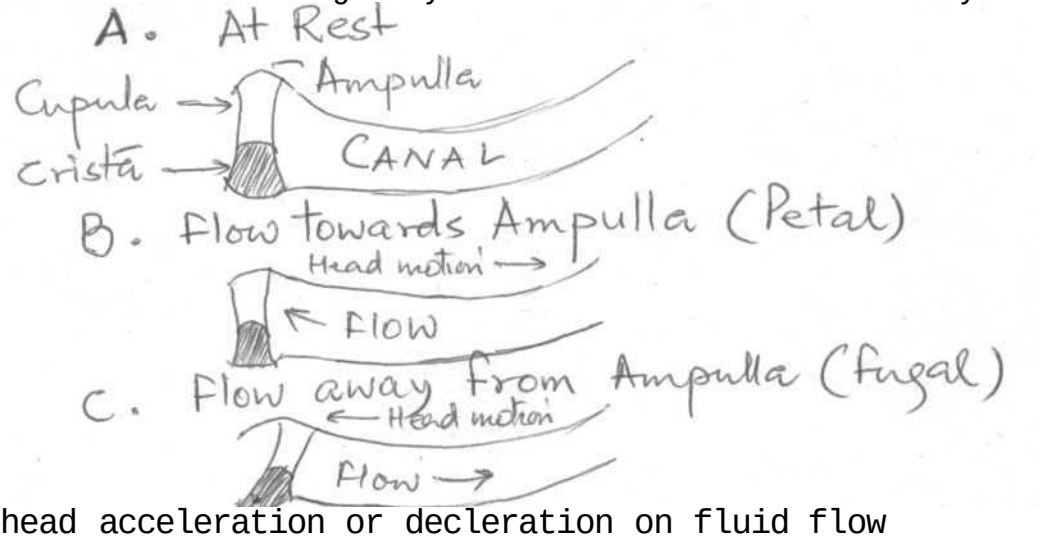


Fig-I

Figure-1: Simplified innervation scheme for a horizontal semicircular canal. The Brain stem nuclei involved in control of the extrinsic eye muscle are shown in schematic form C their possible connections via the vestibular nucleus to the sensory receptors vestibular nerve in the HC. In case of Right labyinth note that principle connections are made to the mediolrectus of the right eye and the Lato rectuss of Lt eye

Figure-II



Effect of head acceleration or decleration on fluid flow in the horizontal semicircular canal.

Part-C: of the figure illustrates the effects of head rotation to the right. Rotation to the right results in a relative motion of the endolymph away from the ampulla or in an ampulla-fugal direction. The criste-cupula displacement corresponds to this fluid motion and the resulting slow compensatory eye motion is towards the left side, with rapid recovery to the Right.

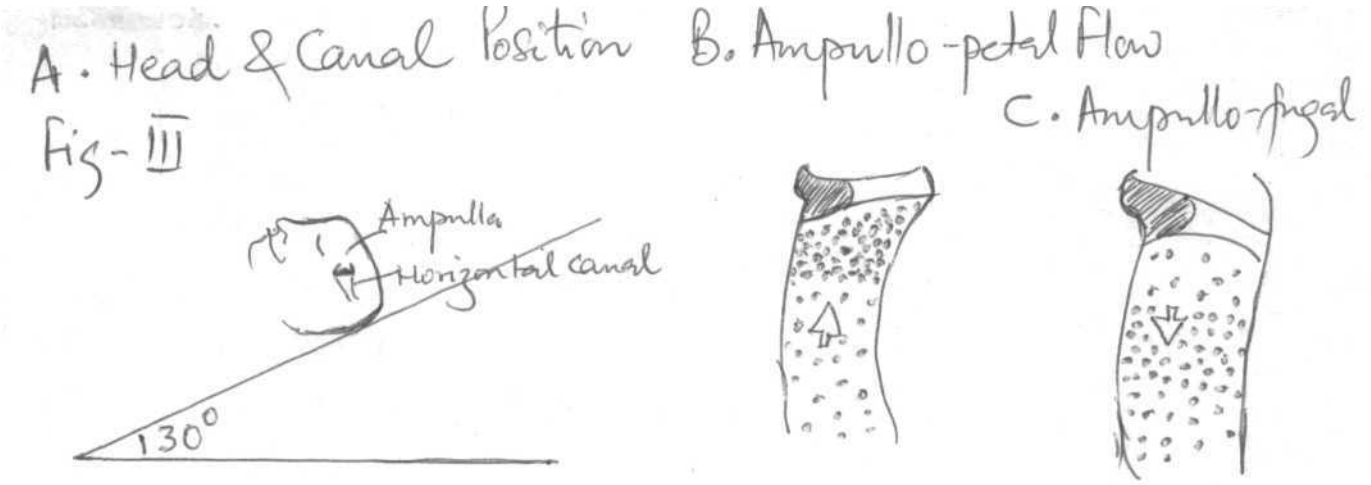
The "to and fro" motion of the eyes, with slow travel in one direction and rapid travel in the other, is '"true vestibular nystagemus". When true vestibular nystagemus is present, its direction is named by the direction of the fast phase of the eye motion. The magnitude of the nystagimus is measured in terms of rate of eye motion in degrees of rotation per second, during the slow phase of the motion. The fast-phase direction and the slow phase velocity(SPV) of nystaginus are two of its features that are used in clinical descriptions.

CALORIC STIMULATION:

In the norrral case, both the Left and Right vestibular systems function to influence eye motion. A. major (systems f-unction to influence-eye me portion of the complete ENG examination consists of observing and measuring eye motion under normal conditions of stimulation eg.- under conditions of changes in head or body position. A portion of the examination requires that each vestibular labyninth be stimulated singly, without known stimulation of the opposite labyninth. This cannot be accomplished by spinning or otherwise moving the individual, since both labyrinths would receive stimulation when motion was introduced. The usual method of single labyrinth stimulation involves the use of a "Calorie Stimulus'.

When caloric stimuli are used, the individual reclines, and the head is brought to an elevation of 30° above a true horizontal plane. The head position appropriate for Calorie stimuli is illustrated in Part-A of Fig-III. As suggested

by the figure the 30° elevation results in a nearly vertical orientation of the horizontal canals. The ampulla of the canal is placed in a position above the canal itself, when the appropriate position is assumed. The caloric stimulus consists of liquid (water or air) which is different than normal body temparature and which is introduced into the external auditory meatus



Effect of Caloric irrigation on fluid flow in the H.C.- The meotus, tympanic membrane and nuclear all undergo a temperature change. Since the horizontal canal passes very close to the middle ear space, the fluid in the horizontal canal also undergoes a change in tewparature, when a warm stimulus is used as in Part-B of Fig-III, the fluid nearest the middle ear space becomes warmer and less dense than normal. Because of the change in density, the warmed fluid effectively"rises" towards the ampulla of the horizontal canal. This ampullo-petal motion corresponds to that which would result from head rotation to the left, which was shown in left rotation and warm irrigation of the left ear both produces a nystagenus with a slow compensatory motion to the Right side and a fast phase to the Left side.

When a cool irrigation is used, the fluid nearest the middle ear becomes move dense than normal Part C of Fig-III The fluid "Falls" toward the dorsal part of the canal due to its density and the effects of gravity. The resulting ampullo-fugal flow of endolymph corresponds to head rotation to the Right. The nystagmns C is evoked by this form of stimulation will have a slow phase towards the left side and a fast phase towards the Right side.

In summary Calorie stimulation provided to either labyrinth may produce nystagnus effects which corresponds to rotation, warm stimulation if a labyrinth results in nystagmus effects which would follow from rotation towards the irrigated side. Cool stimulation produces effects which would normally result from rotation away from the irrigated side. The slow and the fast phases of the nystagmus complement this form of stimulat-ion Cool irrigation produces nystagmus with a fast phase and from the irrigated side. Warm irrigation results in a fast phase towards the irrigated side. The specific application of this form of stimulation in clinical testing will be described later.

Chapter-IV.

Definitions.

NYSTAGMUS: is an involuntary rhythmical movement of the eyes. Two successive movements occur at regular intervals, one to and one fro. If the movements are equal, nystagmus is oscillating or undulating. If unequal, we have jerking diphasic or directed nystagms, Nystagms of the unequal type is a non-voluntary movement.

NYSTAGMOGRAPHY: is the technique of recording nystagomus. A number of methods of obtaining the recordings have been utilized:

(1) Mechanical method.

(2) Photographic method.

(3) Photoelectric method and

(4) Skin electrode technique known as Electronystagmography.

NYSTAGMOGRAM: Is the actual tracing or recording produced by the technique of electronystagography

I. NYSTAGMUS:

(A) Components: (1) Slow (2) Fast;

(B)Direction: (1) Horizontal, (2)Vertical (3) Oblique or Rotatory

(C)Intensity or Degree(Alexander's Classification)

(l)Ist Degree (2)2nd Degree (3) 3rd Degree

(D)Types: (l)Optokinetic (2)Vestibular (3)0cular

(E)Noninduced:

(1) Spontaneous

(2) Positional: (a) Nylen's classification:

(i) Nylen I

(ii) Nylen II

(b)Cawthrone's Classification:

(i) Fatigable

(ii) Constant

F) Induced:

(1) Galvanic ;

(2) Rotational

(3) Mechanical

(4) Calorie.

2. NYSTAGMOGRAPHY:

Techniques:

1. Mechanical devices.
2. Photographic
3. Photoelectric
4. ENG or Skin Electrode

(a) Corneoretinal Potential

(b) Rotating Dipole

(c) Positive or Negative direction.

(d) Electrodes: (i)Horizontal;

(ii)Vertical; and iii)Ground.

(e) Preamplifier: AC and DC

(f) Galvanic Potentials

(g) Channels: (i) Raw or Conventional

(ii) Differentiated

3. NYSTAGMOGRAM :

(A) Types: (1) Rt. Beating;

(2) Lt. Beating.

(B) Parameters: (1) Duration;

(2) Intensity

(3) Latency

(4) Response Decline.

(a) Velocity of Fast and Slow component

(b) Total number of Beats

(c) Frequency per Unit/Time

(d) Culmination Value

(e) amplitude.

(C) Recording Types: (1) Hypoactive

(2) Paretic or Non-reactive

(3) Increased response Intensity or Hyper excitability

(4) Directional preponderance

(5) Secondary Phase nystagmus

(6) Intermittent or Sporadic nystagmus

(D) Eye movements other than Nystagmus:

(1) Table Mountain- To and Fro or Square waves

(2) Sinus Rhythm

(3) Blinking

(4) Eyelid Tremor.

Chapter-V

The Electronystagmograph in Clinic.

An Electronystagmograph is simply a standard biological pen-writing recorder. Table below lists the specifications that tailor the recorder to record nystagmus. Three of the more critical of these specifications are discussed.

Specifications of an ENG suitable for routine Clinical use:

I - Gain (Sensitivity)

25-500 mV/cm per deflection continuously variable with calibrated setting.

II. Frequency response (3 dB down points)

High 15 Hz

Low 0.1 Hz (Time constant = 3 sec)

III. Common-mode rejection ratio

Greater than 80 dB at 60 Hz

IV. Input impedance

Greater than 1 megohm with differential input

V Paper speed

10 mm/sec.

VI. Number of recording channels

one/two.

I. Frequency Response: Bioelectric signals never occur as isolated phenomena. The particular event one wishesto record is always immersed in a milieu of competing electrical events of diverse origins. One important way of selectively recording the desired event is to tailor the frequency response of the recorder to reject unwanted signals. Care must be taken,

however not to restrict the recording frequency response to the point that the desired count is not recorded with fidelity.

In ENG, 60-Hz hum and muscle potentials are probably the most serions of the rapidly changing interfering signals. These signals can be minimised, however, by restricting the high side of the frequency-response curve. Also, such slowly changing electrical signals as the electrodermal respcnse(due to sweating) and electrolytic potentials at the electrode-skin interface can be eliminated by restricting the high side of the frequency response curve. Also, such slowly changirg electrical signals as the electrolytic potentials at the electrode- skin interface can be elimated by restricting the low side of the ENG's frequency response.

2. Common-mode Rejection Ratio: Biomedical recorders almost always use " differential amplifiers " to amplify the recorded signals. The main advantage of the differential amplifier is that it tends to reject interfering electrical " noise " radiated from nearby sources eg.- 60 -Hz power lines. In short, a differential amplifier is two amplifiers connected so that their outputs subtract. Thus, signals that are the sare at both inputs (common-mode signal) cancel out, whereas signals that differ at the two inputs pass through and are amplified.

Slight differences in the electronic components of the two sides of the differential amplifier will create slight differences in common-mode signals. Since the electronic components are not

perfectly uniform, real-life differential amplifiers never completely reject common-mode signals. However, some are better than others. The ability of the differential amplifier to reject common mode signals is thus an important indication of its quality. This ability is measured by applying a signal eg.- a 60-HZ sine wave, so that it is first in phase at the two inputs and then 180° out of phse The ratio of in-phase to out-of-phase voltages required to produce the same output is termed the " common-mode rejection ratio " This the 90 - dB common-mode rejection ratio specified above means that an in-phase signal must be about 13,000 times as strong as an out-of-phase signal to produce the same output.

3. <u>Single-versus Dual-channel Recorder:</u> The advantages of a two-channel recorder over a one-channel recorder are :

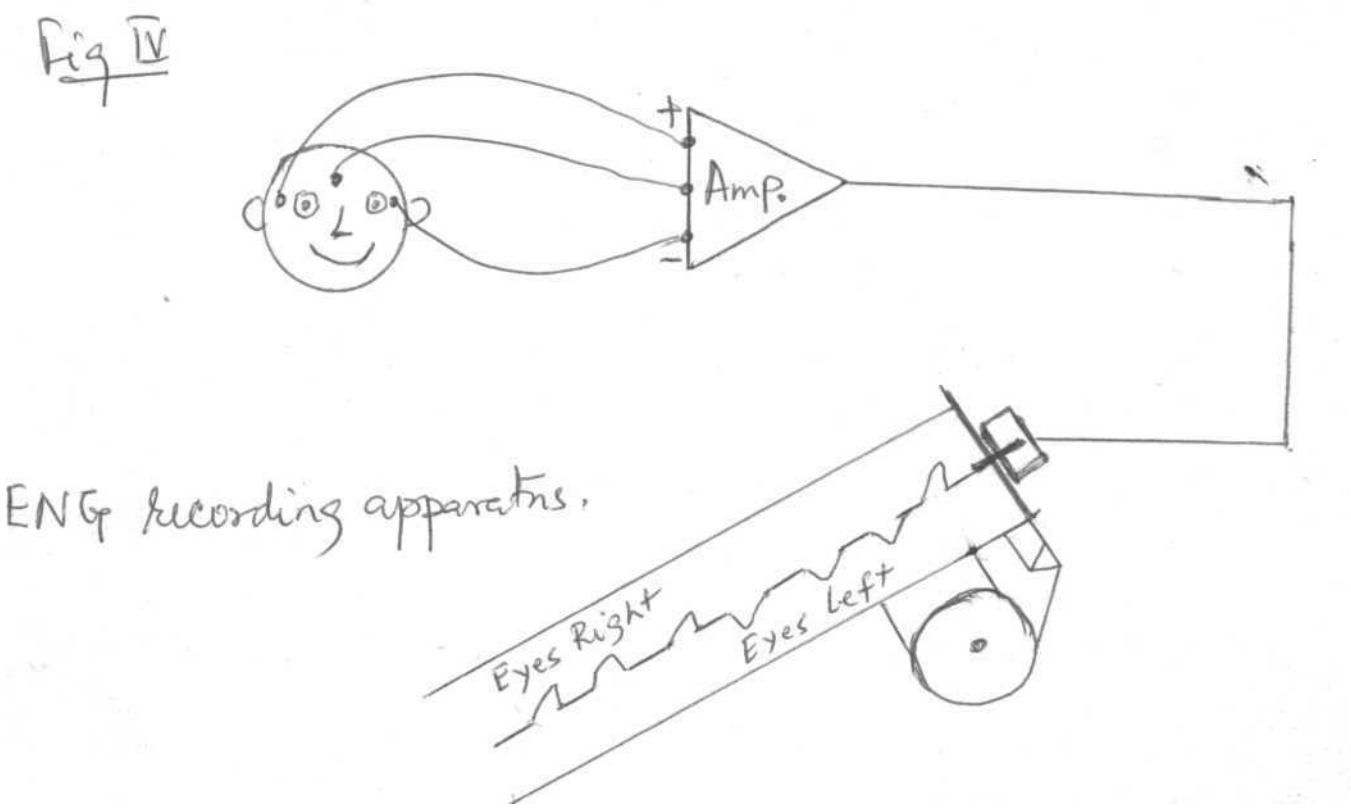
1. Vertical eye movemen s can be recorded. A vertical eye-movement channel occasionally reveals paraoxysmal nystagmus that otherwise would be missed and also, vertical ocular abnormalities may be important signs of central nervous system pathology.
2. The vertical channel helps to detect eye blinks in the horizontal channel.
3. The second channel provides ox back up channel so that the ENG need not shut down if one channel develops difficulties .
4. If the recorder has modular preamplifiers the second channel can be used as a " velocity " channel which, in theory provides a semiauomatic computation of nystagmus intensity.

The mayer disadvantage of the dual-channel recorder is that it costs about twice as much as a single-channel recorder + An additional minor disadvantage is that the dual channel recorder is somewhat larger than the single-channel recorder (17" v/s 10").

Physical Basis for ENG:

The ENG recording is made possible because the eyeball behaves as an electrical'dipole' In this sense, the eye wight be thought of asa battery C is constantly maintained and renewed by the metabolic processes associated C it. The dipole Ch/: of the eye results in a corneal retinal potential(CRP), or voltage with a relative posi ive(+) value at the cornea and a negative(-) value at the retina. Although the CRP is quite small it may be detected at some distance from the eye. A typical ENG recording situation is shown below Disc-shaped electrodes are placed over-each outer canthus as close to the eye as is possible without interference with eye or eyelid motion. A 3rd electrode is placed between the eyes on the lower forehead. The lead from the the electrode nearest the right eye is connected to the active or "+ " input of a " differential " amplier.

Fig IV



ENG recording apparatus.

The lead from the Left side is attached to the reference or " - " input of the amp., and the 3rd lead is attached to the grounded terminal of the amp. The amplifier is called " differential" because it functions to detect the difference in the voltage appearing at the active and reference inputs relative to the grounded point. In amplifying only the differences, the amplifier excludes noise which is common to the active and reference electrodes, such as 60 Hz line-frequency interference. Eye rotation towards the Right side of the skull results in the development of a positive voltage at the active input of the amplifier and rotation towards the left side of the skull results in a negative voltage at the active input. When the amplifier is attached to a stripchart recorder by rotation towards either direction will result in a pen deflection having a magnitude and direction proportional to the magnitude and direction of the eye motion. In Fig.4 rotation to the right results in a pen-motion in the opposite direction. If recording of vertical motion of the eye are desired the active and reference electrodes are placed above and below the eye and the same recording principles obtain.

Calibration and measurement of Nystagmus:

In order to obtain an accurate input of the magnitude of eye motion the electrodes are applied and the individual is instructed to look at a spot or a light source which has been placed at some distance to the right or left of the center point

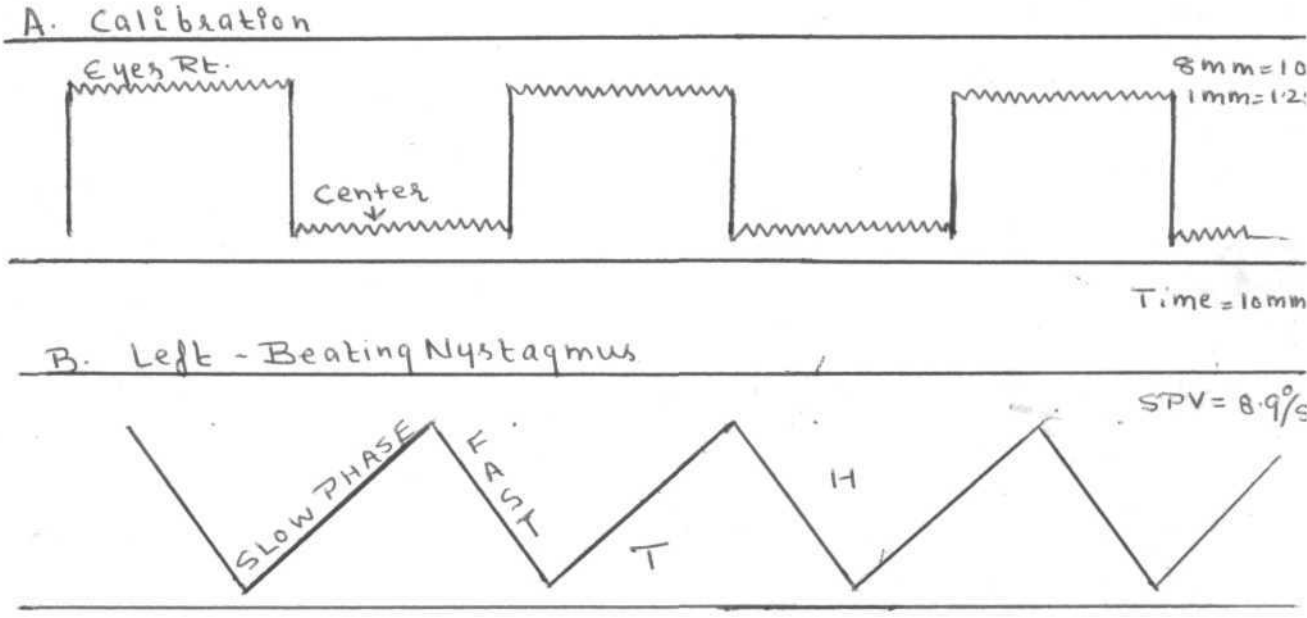
of his field of vision The distance should be sufficient to cause eye rotation in the amount of 10° to the right of center and 10 degree to the left of canter. The recorder is activated and the amount of pen deflections in mm, corresponding to the 10° X eye rotation is noted. The tracing in part A of Fig.5 shows an ^ ; example of a calibration tracing where the individual was instructed to look at a center line target and then one to the right of center. Examination of the tracing reveals that 10° of eye rotation corresponds to 8 mm of pen deflection. This means that 1 mm of.pen deflection is equivalent to 1.25° of eye rotation.

Part B of Fig.5 shows how the calibration information would be used to measure the SPV of a nystagmus beat. The nystagmus illustrated in Part B has a slow component reflection in gradual pen motion toward the top of the tracing and a fast phase toward the bottom. According to our convention the slow phase corresponds to right rotation of the eye, and the fast phase corresponds to left rotation of the eye. The paper speed has been set to 10 mm/sec. If the distance between the start of one nystagm beat and the next is measured then the time course of a beat may be computed by dividing the distan e by lOmm/sec. In the exarrple in Fig.5 the distance covered by the slow phase portion of each beat is 14mm. Therefore the duration of the slow phase component is 1.4 sec. This is symbolized

by in set in the graph. The height of the nystagmus beat sympolized by H in the figure is 10 mm corresponding to a total eye rotation of 12.5° If total eye rotation(H) is divided by the time course(T) of the slow phase component, the resulting number will be the rate or velocity of eye motion. Eg.- in Fig 5 the computation would be:

$$\frac{\text{Magnitude of slow phase}}{\text{Duration of slow phase}} = \frac{12.5^\circ\ (10\ \text{m}\hat{})}{1.4\ \text{sec}(14\text{mm})} = 8.9^0/\text{sec.}$$

In the Part B- it is a left-beating nystagmus C an SPV of 8.9° /sec.



Another approach to the calibration and input of nystagmus is to require that the individual look at points which are 1CP off center and then to adjust the recorder sensitivity until the 10 of eye rotation results in exactly 10mm of pen deflection. Then 1 mm of pen deflection will correspond to 1° of eye notion, and the examiner need only measure the height of the pen deflection(in mm) to arrive at eye rotation directly. The CRP may be too small in some individuals to permit this, however.

CALIBRATION VARIABILITY:

The CRF is highly variable overtime. It varies with the dark adaptation of the eye, in particular. Since dark adaptation will be affected by room illumination, calibration procedures should be accomplished with C is approximately equal to that which will be used during the various phaseset of 3NG examination. Many portions of the examination are accomplished with the individuals eye s closed, and therefore subdued illumination is the best condition to use in the examining room.

The CRP will vary over time, even with constant illumination in the test facility. Since the magnitude of the CRP will influence the amount of pen deflection, the true calibration of the recording will vary over time, as well. It is imperative to recalibrate the recorder sensitivity for eye displacement several times during the course of a complete evaluation. Failure to do so may invalidate recordings taken as few is 5 min subsequent to a calibration procedure.

Electrode application:

Misapplication of the electrodes is a common problem encountered in ENG evaluation, proper application requires the skin at the electrode sites be cleared thoroughly C alcohol or some other solvent. A small amount of electrode paste or jelly is rubbed onto the recording sites. Additional amounts are applied to the electrodes and the electrodes and the electrodes are placed on the sites & fixed there C suitable adehesive tapes

The electrode paste application and the cleansing of the skin help to improve the electrical conductivity of the skin. Several problems may result from a failure to apply the electrodes properly. The most serious of these is that unwanted electrical signals which should appear to be identical at the active and reference electrode sites will actually appear to be different. The unwanted signal will be amplified by the differential amplifier and obscure the desired CRP recording loose electrodes may produce small voltage changes due to their motion at the electrode recording site. The small voltages may result in an compression of erratic eye motion, even though eye motion may be normal. Finally, failure to clean the recording site or apply the electrode significant reduction in the arparant amplitude of CRP C a subsequent failure to detect occular motion. Practically difficulties encountered C electrodes may result from a combination of three factors and reapplication of all electrodes is the most prudent course of action if erratic, recordings are encountered.

Chapter-VI.

THE ENG EXAMINATION.

The complete ENG Examination includesat least the 11 steps shown in the outline below. The steps numbered 3 thr ugh 11 require between 60 and 90 min., for completion. Each of the steps and the specific tests associated with them will be described in the Sections which follow:

ENG EXAMINATION SEQUENCE:

1. Pre-evaluation instruction.
2. Hlstory(dizziness questionnaire)
3. Nondiagnostic dtoscopy
4. Electrode application,
5. Calibration.
6. Gaze nystagmus,
7. Optokinetic nystagmus
8. Visual tracking
9. Spontaneous nystagmus.
10. Positional nystagmns.
11. Caloric testing.

Pre-evaluation Instruction:

Several forms of medication and alcohol, may affect the vestibular system. In order to obtain valid ENG recordings, it is imperative that the patient who is to be evaluated be instructed to abstain from sedatives tranquilizers, anti-histamines and pain medication for 12 hours prior to the evaluation. Additionally, no alcbholic beverages should be

consumed by the patient within 24 hours prior to the evaluation. It is advisable that upon making the appointment for the evaluation instructions regarding mediations be provided to the patient in written form to minimize the possibility of mis-understanding. Occasionally a patient who will not or cannot abstain from medications will be encountered . Therefore, even though instructions have been provided to them, the examiner should ask all patients about their medications and should report the results of that enquiry along with the examination findings.

History: The ENG evaluation, like a hearing evaluation, is only a portion of a complete oto-nemologic examination. The patient's history with respect to vertigo, dizziness medications and hearing may all be of value to the physician who must ultimately arrive at a medical diagnosis. In most instances the history may be gathered by using a questionnaire which is sent to the patient prior to his arrival at the clinic. Among the iterms which might be included on such a questionnaire, the following topics may be most helpful:

Tinnitus: Whether unilateral or bilateral, episodic or constant long standing or of recent onset.

Vertigo:(True spinning sensation) Whether long standing or recent episodic or constant associated with trinnitus sudden motion, hearing loss etc.

Dizziness: Whether long standing or recent, episodic or constant motion induced etc.-

Hearing Loss: extent of less, duration of loss, medical or surgical history related to the loss, unilateral or Bilateral

General Physical Condition: Including cardiovascular neurologic, and surgical history.

Medications and Drugs: Use of sedatives, stimulators tobacco, alcohol, antihistamins, pain relatives

Nondiagnostic Otoscopy: Referrals for ENG evaluation will usually originate with a physician who has determined that the patient's ears are normal otoscopically. However, it is prudent to examine the patient's ears at the tine of evaluation to be certain that there are no conditions which would pre-elude the successful or safe administration of caloric stimulation. If such a condition exists, the non-caloric portion of the examination may be completed and the basis for refusal to obtain caloric induced responses may be noted in the report.

Abnormal ENG Findings & their Significance.

(A) GENERAL CLINICAL SIGNIFICANCE:

The ENG examination may help differentiate between Pathology involving the peripheral vestibular system and that involving the central nervous system. The division between " peripheral " and " central " as the point at which the VIIIth nerve enters the brainstem'Reger-1972).ENG is thus analogous to audiometry. The basic significance of both tests is their ability to localize pathology anatomically. Audiometry, however, provides more refined localization because it can separate nerve from end-organ lesions, whereas the ENG examination described herein cannot. Several ENG findings indicate pathology but do not distinguish between a peripheral and central location. This category of ENG abnormalities is referred to as "non-localizing".

(B) Ocular-dysmetria Test:

Ocular-dysmetrea is an over-or undershoot of the ocular rotation that occurs when visual fixation is transferred from one place to another (a "refixation movement" or "saccade"). Ocular dysmetra is thought to Originate in the cerebellum(Cogan,l954) Higgins and Daroff,l966; Orzechowski,1927), but in practical clinical application it should probably be considered indicative of either cerebellar or brainstem pathology. Lesions involving the cerebellum(whether vascular or neoplastic) So frequently involve brainstem structures as well, that, when localizing

central lesions, several authors (Coats, 1970, Daroff and Hoyt, 1971) group the cerebellum and brainstem together.

Because of the fleeting nature and small amplitude of the dysmetric ocular movement, obtaining a graphic record of it has obvious advantages. Noorden and Preziosi(1966), Ellenberger etal(1972), and Haring and Siemens(1973) have published records of ocular dysmetrid- Haring and Simmens point out that ocular dysmetric may be recorded in the course of the routine ENG calibration("Calibration overshoot") Thus a valuable central sign may be detected during the ENG examination with virtually no investment of additional examination time. Most normal subjects demonstrate an occasional overshoot during the ENG calibration. Therefore, when inspecting the record for ocular dysmetria one must have in mind some criterion for "limit of normal" Haring and Simmens(1973) require that 50% of the calibrations be overshot before ocular dysmetria is diagnosed. However this criterion is based on an intuitive finical impression. A systematic normative study would greatly enhance the clinical usefulness of the Ocular-dysmetria test.

When inspecting the calibration record for Ocular-dysmetria, one must also keep in mind that eye-blink artifacts, which are often synchronized with the refixation movement, may produce a very good initation of an overshoot. Since the vertical channel will register the eye blink but not the overshoot, it helps to distinguish between

true and artifactual over-shoots.

If a vertical channel is not available, the examiner must observe the Patient's eyes during the calibration and record the occurrence of the eye-blinks.

C- GAZE TEST:

Nystagmus may not be present with the eye centered but it may appear when they are deviated from center. The gaze test examines for such a ""gaze nystagmus"? it also examines for paresis of ocular deviation, when present, gaze nystagmus almost always demonstrates the following characteristics:

(1) It is divided into slow and fast phases with the fast phase in the direction of eye deviation.

(2) its intensity(amplitude and possibly slow-phase speed) increases with increasing eye-deviation. Congenital nystagmus presents the most not-able exception to these characteristics.

(**a) Technique:** Compared to the routine physical examination, the gaze test done in the ENG laboratory usually incorporates the following refinements: (i) the amount of eye rotation is quantitat-vely controlled; and (ii) the eye movements are recorded. The gaze test is done immediately after the calibration, while the patient is still in front of the fixation points. The patient gazes steadily at the 20° and 30o fixation points to right and left and above and below center. Gaze is maintained for 30 sec in each of the right eye positions.

Gaze nystagms may be present but not recorded, either because its amplitude is too low or because it is rotatory. Also, paretic eye movements may be easier to observe visually thaw to record. Therefore during the gaze test, it is very important to observe the patient's eyes visually as well as to record their movements.

(b) Diagnostic significance of Gaze abnormalities: With certain exceptions gaze nystagmus and gaze paresis indicate the presence of CNS pathology. Particularly if persistent for a month or more these findings further suggest brainstem involvement(Daroff and Hoyt, 1971; Dow and Manni,l964)

(a) End point nystagmus:

(Aschan et al, 1957 a; Kestenbaum; 1961; Walsh and Hoyt 1969X There has been no systematic quantitative study of end-point nystagmus. According to Ascher et al (1957 a)" Gaze nystagmus (at) 20° "30 or less . . . . . should probably be regarded as pathological ". In Bloombergs(1955) series of 100 normal subjects nystagmus was never present at "about 45°" from Centre. So Gaze deviations are set at 20° and 30° from center.

Classification of Gaze Nystagmus Kastenbaum(1961)

I. Normal

" End Point " or "end-position" nystagmus: a nystagmus C appears on extreme gaze.

(40 or more, rarely at 30°)in a large % of normals.

II Abnormal:

A - Vertical: present on up and/or down gaze

B- Horizontal:

1. Bilateral, equal: nystagmus of approximately equal amplitude and frequency in both directions.
2. Bilateral unequal: nystagmus of clearly unequal amplitudes in the two directions.
3. Unilateral; nystagmus present only in one direction of gaze.

(b) Vertical Gaze Nystagmus:

Vertical gaze nystagmus indicates CNS pathology, probably involving the brainster. Upward vertical gaze nystagmus, is much more common than downward vertical gaze nystagmus. When vertical gaze nystagmus appears without associated horizontal gaze nystagmus it suggests either a midline or bilateral lesions in the upper pons or midbrain(Daroff and Hoyt 1971; Kesterbaum-61)

(c) Bilateral, equal horizontal gaze nystagmus:

As with other forms of gaze nystagmus, bilateral equal horizontal gaze nystagmus indicates CNS pathology, probably involving the brainstem. However, when this form of gaze nystagmus appears as an isolated sign, one must rule out drug effects, particularly barbiturates(Bergman et al 1951) diphenylhydanton(Dilantin) (kitt et al 1964) and alcohol (Aschan, 1958)

(d) Bilateral unequal horizontal gaze nystagmus:

Since drug effects rarely produce asymmetrical abnormalities' bilateral, unequal gaze nystagmus argues against A drug toxicity. Hence this type of gaze nystagmus strongly suggests organic CNS pathology.

(e) Unilateral horizontal gaze nystagmus: A unilateral horizontal gaze nystagmus cannot be considered a central sign until one has ruled out the possibility that it is a manifestation of of an intense vestibular spontaneous nystagmus. Visual fixation may suppress a spontaneous nystagmus so that it is not present with the eyes centered. However, deviating the eyes towards the fast phase may sufficiently enhance the nystagmus that it " breaks through " the visual fixat on suppression and becomes visible.

The most straight forward way to determine if a unilateral horizontal gaze nystagmus is due to a vestibular spontaneous nystagmus is to have the patient close his eyes. If, on eye closure, there appears a spontaneous nystagmus in the same direction as the gaze nystagmus with slow phase speed greater than 8 per sec, then the gaze nystagmuss is not a central sign (coats 1970)

D. Sinusoidal Tracking Test:

1. Technique: The sinusoidal tracking test is done by having

the patient fixate on a spot which is moving in a sinusoidal pattern (Benitez,1970; Tung and Komhuber 1964; Ohm 1940) Tested with frequences of 0.3 and 0.6 Hz, and the pattern subtends an angle of ± 20° from center gaze. Tracking movements are obtained in both the vertical and horizontal directions.

2. Clinical Significance: A normal individual should be able to track the pattern smoothly, although brief fixations on other objects may occasionally interrupt the smooth sinusoidal pattern. In the generally recognized abnormal sinusoidal tracking pattern saucadic eye jerks in the direction of spot movement repeatedly '* break up " the smooth sinusoidal pattern.

Findings of Benitez(1970) and Jung and Kornliber(l964) that (1) abnormal sinusoidal tracking occurs in central oculomotor lesionS, usually involving the brainstem (2) it is usually associated with gaze nystagmus and bilateral optokinetic diminution and (3) barbiturate sedation may cause it.

E. Spontaneous N-stagmus:

1. Definition and Test procedure: Spontaneous nystagmus is a nystagmus that is present in the absence of any known nystagmographic stimulus. Assumption of a neoneutral position(positional nystagmus) and deviating the eyes from center(gaze nystagmus) may be considered nystagmogenic stimuli. Spontaneous nystagmus is defined as a nystagmus present in the Read upright position(the

best approximation of the "neutral" head position in the human) and with eyes centered. Recording of vertical and horizontal eye movements is done with the eyes centered for 30 sec sec. to 1 min first open and then closed. Since the patient's head is upright during the gaze test, this eye-centered recording fulfills both criteria of spontaneous nystagmus.

2. Clinical Significance: It is useful to classify pathological spontaneous nystagmus according to its probable system of origin, i.e., vestibular, ocular or central. This classification is analogous to cogan's (1956) "otologic " "ocular" and "neurologic" divisions Others have proposed similar classifications (Toghia and Moreno 1971).

(a) Normal Spontaneous nystagmus with eyes closed:

(1) Vertical: Behind closed eye lids a vertical jerk type nystagmus, usually up-beating is present in about 80% of normal subjects (Flurr and Eriksson,1961) and may be rather intense i.e., slow phase speed greater than 10° per sec.

(**2)Horizontal:** Although it is generally agreed that vertical spontaneous nystagmus behind closed eyelids occur in normal persons, similar agreement about the presence of normal horizontal spontaneous nystagmus has not been reacted. Although there were dissenters, most early investigators of clinical ENG reported(or implicity assumed) that horizontal

nystagmus present without visual fixation has the same clinical significance as spontaneous nystagmus present with visual fixation i.e., it is always abnormal(see, for example, Aschan et al 1956-a) however considerable the evidence has accumulated recently which supports the existance of a low-intensity horizontal spontaneous(or positional) nystagmus in 15-30° of otologically normal subjects(Barber and Wright 1973, Bos et al 1963, Coats 1969; collins et al 1973; Flur & Eriksson 1961; Lansberg, 1962; Spector 1971; Visser, 1962) The slow-component speed of this nystagmus is usually less than 7° per sec(Coats-69)

Classification of Spontaneous Nystagmus:

I. Normal:

Vertical, behind closed eyelids Horizontal, behind closed eyelids 7-8° / sec. Voluntary.

II. Vestibular(otologic)

Fast and slow phases

Horizontal

Conjugate, Suppressed by visual fixation.

III. Ocular(Opthalmologic)

Congenital

Occupational

IV. Central(neurologic)

Diagnosis made by excluding other types.

(b) Normal Spontaneous nystagmus with eyes open:

"Voluntary nystagmus '" is the only known normal "spontaneous " nystagmus present with eyes open and fixed. It is pendular (no divisions into fast and slow phases) conjugate, extremely rapid (3-15 oscillations per sec) small-amplitude nystagmus which is initiated and maintained by voluntary effort(Blai-et al 1967: Lipman 1972, XanxscKKJc Rosenblum and Shafer 1966) several reports indicate that voluntary nystagmus is rare(Rosenblum and Shafer 1966? Walsh and Hoyt 1969). Voluntary nystagmus is relatively common (Blair et al 1967).

The main clinical importance of voluntary nystagmus is that it could be mistaken for convergence nystagmus which is a patho-logical nystagmus of dorsal midbrain origin. However convergence nystagmos is a coarse, jerk type, disconjug atotynystagmus.

(c) Vestibular(otologic)Spontane us nystagmus:

To be considered vestibular, a Spontaneous nystagmus must have the following characteristics:
(Jung and Karnhuber,1964):

1. divided into fast and slow phases(jerk type");
2. horizontal or primarily horizontal(rotary component);
3. Conjugate(both eyes move together)

(This criterion of course does not apply if there is a peri-pheral ocular paresis or paralysis)

4. Suppressed by visual fixation.

Vestibular spontaneous nystagmus, particularly if it is intense, is usually caused by a peripheral vestibular lesion(Coats 1970) However, occasionally it may be caused by a central lesion which is localized to the vicinity of the vestibular nuclear complex on one side(Jung and Komhuber 1964, Scala and Spiegel 1938). Because vestibular spontaneous nystagmus can be done to either peripheral vestibular or CNS pathology it is a nonlocalizing abnormality;

(d) Ocular(opthalmologic) Spontaneous nystagmus: A synonymous term for this type of spontaneous nystagmus is "fixation nystagmus " (Jung and Komhuber 1964) Congenital nystagmus is by for the commonest type of ocular spontaneous nystagmus. Congenital nystagmus is present from birth or early infancy. It may have a pendular or "spikelike" wave form(Aschan and Bergstedt 1955, Jung and Komhuber 1964) The wave form is extremely variable from patient to patient and often changes when fixation distance or direction of gaze is changed. Eye closure usually either abolishes or changes the direction of congenital nystagmus. It rarely enhances congenital nystagmus as it does vestibular nystagmus.

A visual abnormality may or may not accompany congenital nystagmus. Many nero-opthalmologists separate congenital nystagmus into different clinical types, depending on the presence or absence of a visual abnormality eg.— Cogan's(1967) Sensory defect" and '"motor defect'" types.

Congenital nystagmus may be mistaken for central spontaneous particularly in the patient, with normal vision who does not discover his nystagmus until relatively late in life. (Cogan 1967) characteristics are:

(1) There is no oscillopsia in spite of what is often a very large amplitude nystagmus.

(2) Congenital nystagmus is almost always normal.

(3) On vertical gaze a vertical component rarely develops and a horizontal pendular component may frequently appear.

4. Convergence usually suppresses congenital nystagmus. In the presence of congenital nystagmus, both optokinetic and vestibular nystagmus, hath are notoriously difficult to elicit and even when elicited, they may be so grossly distorted as to preclude quantitation. The distortion of caloric responses makes quantitative vestibular-function assessment impossible in many congenital nystagmus patients.

(e) Central (nerologic)Spontaneous nystagmus: A Spontaneous nystagmus is of central origin by a process of exclusion. If the the spontaneous nystagmus fails to meet one or more of the criteria of a vestibular type and an ocular nystagmus has been ruled out as outlined above , then the nystagmus must be central . A central spontaneous nystagmus usually indicates a brainstem or cerebellar lesion(Jung and Koruhuber,1964)

(F) Optokinetic Test:

Optokinetic text nystagmus(OKN) occurs when one looks at a moving repetitive pattern(black stripes on a white background are usually used clinically) which fills most or all of the visual field. OKN has slow(following) phases in the direction of pattern movement and fast(rafixation) phases opposite to the direction of pattern movement. Optokinetic nystagmus is probably involuntary if fixation on the moving pattern is maintained(Cogan 1956, Smith 1963) The larger the stimulus pattern, the less the variability in OKN due to voluntary visual fixation changes.

An abnormal OKN test may be manifested as an asymmetry (difference in oppositely directed OKN in spite of equal stimulus speeds) or as a bilateral dimension. The OKN test is one of the more sensitive tests for central oculomotor pathology(Coats 1970; Jung and Komhuber 1964).

1. Instrumentation and Test Technique: The OKN Test is usually done with a hand-operated drum. In order to fill the patient's visual field, some use a large internally lighted cylinder which is lowered over the patient's head.

Basically the clinical OKN test consists of eliciting nystagmus in opposite directions at the same stimulus speed. In the normal routine neurological examination OKN is elicited only by two brief oppositely directed horizontal stimuli.

(a) Vertical OKN: Although early OKN studies involved only the horizontal response, the importance of vertical OKN testing has been emphasized recently (Coats 1970; Jung and Koruhuber 1964; Rosborg et al 1972; Smith 1962). One reas n for this emphasis is that if vertical OKN is excluded a significant number of abnormalities be missed.

An isolated or predominant vertical OKN abnormality has localizing value, since it is usually due to a high midbrain lesion(Jung and Koruhuber 1964, Roseborg 1972,Smith 1962)

(b) Eliciting OKN at more than one stimulus speed: OKN asymetries are usually enhanced when stimulus speed is increased (Jung and Koruhubar 1964); Suguti & Komatsugki 1962) Therefore eliciting OKN at more than one stimulus speed often clarifies the presence of an asymmetry.

2. Recognizing OKN Abnormalities:

(a) Bilateral dimension: Although OKN is involuntary if fixation is maintained, in practice it is under considerable voluntary control because of the patients' ability to influence it by varying fixation.(Jung and Koruhuber 1964; Smith 1963) Hence lack of cooperation must always be suspected when bilateral OKN diminition or absence is present.

(b) Asymmetry: There are two recognizable patterns of OKN asymmetry: (1) poorly formed, and (2) slow-phase speed. Both patterns involve the Slow phase. Usually an OKN asymmetry presents as a combination of the two patterns and a slow-phase - speed asymmetry can often be converted into a poorly formed asymmetry by increasing stimulus speed.

(**c) Importance of conservatism in OKN abnormalities:** It is very important that OKN abnormalities be clearly present before being considered significant(coats 1970 Smith 1963) WRxrBgSBX J&XxSJSMKR&RSX If there is any d ubt about an abnormality we repeat the stimulus to confirm its presence.

3. BBR3E33HHXBgXEgKXKHX Clinical significance of OKN Abnormality

(a) Detection of Central Pathology: If the patient has been cooperative, OKN asymmetry and bilateral diminition usually indicate the presence of CNS pathology. Peripheral ocular pathology eg.-Strabisms severe extraocular muscle paresis and long standing unilateral blindness, must also be ruled out as a course of OKN asymmetry(Coats 1970)

(b) Localization of central pathology:

(1) Association with gaze abnornalities(paresis or nystagmus) OKN abnormalities due to cerebral and brainstem lesiops can be distinguished on the basis of associated gaze-test findings. An OKN abnormality is due to brainstem oreerebellar pathology is always accompanied by either a gaze nystagmus or a gaze paresis, In contrast an OKN abnormality with a normal gaze test is usually due to a cerebral hemisphere lesion(Coats,1970; Cogan & Loebl949; Davidoff et al., 1966; Jung and Komhuber,1964).

2. Vertical OKN asymmetry: Suggests pathology involving the brainstem. An isolated vertical OKN asymmetry further suggests bilateral or midline lesions in the midbrain or upper pans (Jung & Komhuber 1964; Rosborg et al 1972, Smith,1962).

Precautions: (1) A sight vertical OKN asymmetry(down-beating response usually predominant) may be present in some normal subjects. Therefore, if a vertical OKN asymmetry is present as an isolated finding, it must be very large before it can be considered significant.

2. Vertical electrodes record eyelid movements with extreme sensitivity: The possibility then are apparent abnormally formed vertical OKN is due to superimposed eye blinks or other eyelid movements must be ruled out.

3. Bilaterally absent or deficient OKN: Provided that failure to fixate on the stimulus has been ruled out, bilaterally absent or deficient OKN(either horizontal or vertical or both) has essentially the same localizing significance as vertical OKN asymmetry; i.e., it suggests a high bilateral or midline brain-stem lesion.

4. Lateralizing Significance of horizontal OKN asymmetry in Brainstem lesions:

In animal experiments, lateral lesions of the pons and midbrain below the level of the oculomotor nucleus cause a predominance of the OKN beating away from the side of the lesion(Teng et al 1958). A similar OKN asymmetry has been reported in humans with well-localized upper brainstem lesions(Ios et al, 1972); the laterializing value of OKN asyrmmetry in brainstem lesions in poor in the routine clinical situation, probably because the lesion usually in large relative to the size of the structures responsible for the OKN asymmetry.

5. Lateralizing significance of horizontal OKN asymmetry in cerebral-hemisphere lesions:

In contrast to brainstem-cerebellar lesions, the lateria-lizing significance of OKN asymmetry in cerebral-hemisphere lesions is quite good.

G. Paroxysmal Nystagmus Test:

1. Nomenclature: In 1952, Dix and Hallpike described a maneover for eliciting what they termed " positional hystagmus of the benign paroxysmal type ". In normal subjects, the Dix-Hallpike manoever elicits no nystagmus or possibly only a brief weak nystagmtus behind closed eyelids. However, in patients with certain types of vestibular system disorders the maneuver elicits a usually rather intense, transient nystagmic response.

Barany first observed that some patients exhibited dizziness and nystagmus only, when they assumed a particular "critical position" (Barany 1921; Jongkees,1961). Later Nylen devoted many years to systematic clinical study of this phenomenon which is now termed " positional nystagmus Nylen (1953) emphasized that positional nystagmus caused only by a particular head position. Several other writers advocated moving the patient rapidly into the various test positions under the assumption that the rapid movement wight " provoke" a positional nystagmus which would otherwise be missed (Lindsay-1951); (Williams,1947) The Dix-Hallpike test evolved from this " school of position testing"

2. <u>Test Procedure:</u> The patient is first seated on the examining table with head straight ahead. He is then rapidly brought backward into the head hanging and turned position. He A is left in this position for 30 secs or if a nystagmic response appears for as long as the response persists. If the response persists for more than 1-1/2 min, the test position is terminated and the response considered '" persistant". After this maneuver the patient is brought back to the sitting, eyes-front position, and left there for about 30 sec, and then the maneouver is repeated with the nect twisted in the opposite direction. If either maneuver produces a nystagmic response, the maneuver is repeated to sec, if the response is fatigable. If the response does not decreased significantly by the third elicitation it is declared "non-fatigable ".

In the test, the eyes are closed and the nystagmic response is recorded rather than usually observed. Although paroxysmal nystagmus is usually primarily rotatory, it almost always has sufficient vertical or horizontal components or both, to be recordable(Preber and Silfurskiold 1957? Stable and Terins 1965) However, since the vertical component is often predominant (Cawthome 1954, Preber and Selveskiold 1957), paroxysmal nystagmus frequently will be missed if vertical eye movements are not recorded.

3. <u>Diagnostic Significance:</u>

(a) Classical paroxysmal nystagmus. Following are the salient features of the benign proxysmal type as it was originally described by Dix and Hallpike (1952)

1. Latent period: The nystagmic response does not begin for 0.5-3.0 sec after the patient arrives at the test position.
2. Transient "paroxysmal " response: The nystagmus"increases in a rapid crescendo in a period which may be as short as 2-3 secs or as long as 10 seconds. Thereafter it rapidly declines * Dix and Hallpike(1952) did not fix the total duration of the response but other authors(Ascnam et al., 1956a? Lindsay 1951) mention durations of 20-60 seconds
3. Dizziness: (usually severe) The paroxysmal nystagmus response is accompanied by dizziness which is often so severe that "the patient may close their eyes, cry out in alaram, and make active efforts to sit up again."

4. Fatigability: When the maneuver is repeated, the response either does not reappear or reappears with significantly reduced intensity.

The following additional characteristics of classical paraf-oxysmal nystagmus are often helpful in classifying doubtful responses.

1. The response is usually unilateral

2. The nystagmus usually is directed towards the downward ear, which is also the pathological ear and

3. the calorie and audiometric tests are usually normal.

(b)Pathophysiological types of " classical paroxysmal nystagmus

Patients with classical paroxymal nystagmus tend to cluster into the following clinical types.

1. Elderely patients (Lindsay 1967): A large % of patients with classical, positive Dix-Hallpike tests are older than 55 yrs Although not life-threatening, paroxysmal nystagmus tends to persists in the elderly.

2. <u>Posttraumatic dizziness:</u> Many authors(Barber 196& ,Cawthorne 1954; COpe and Ryan 1959, Dix and Hallpike 1952; Gordon 1954; Harrison 1956; Preber and Silfveskiold 1957; Schuknecht 1969) have noted that paroxysmal nystagmus occurs in a large %(15%) of posttraumatic 'dizziness' following head trauma, and is often the only objective abnormal finding. The prognostid implication of paroxysmal nystagmus after head traumiB: is relatively good, since most of the patients can expect to be symptom free woothen 2-6 months.

3. Middle-ear Pathology: A significant proportion of patients with chronic middle-ear infection who complain of episodic dizziness have classical paroxysmal nystagmus(Barber,1964; Dix Hallpike 1952 ; Schuknecht 1969). As Jongkees(1961) has pointed out, when such a patient is encountered the possibility of a labyinthine fistula must be ruled out.

4. Middle-ear surgery: Classical paroxysmal nystagmus is occasionally encountered after stapes surgery. Barber 1964 S huknecht, 1969; Spector, 1961 Terin's 1963) It is probably related to mechanical manipulation of structures in the vestibule Paroxysmal nystagmus is uncommon following other types of temporal bone surgery such as mastoidectomy labyrintheatomy and tympanoplasy(Schuknut 1969)

5. Rare types: An idiopathic" paroxysmal positional vertigo of childhood " has been described (Chutorain 1972) Also, Falimud et al (1970) reported a case of classical paroxysmal nystagmus in pernicious anemia.

c) Localizing value of paroxysmal nystagmus:

(1) Classical paroxysmal nystagmus: The pathophysiological mechanism of classical paroxysmal nystagmus does not yet been established(Stachle and Ternis 1965). Although some animal experiments suggest that cerebellar lesions may produce a sydiane resembling paroxysmal nystagmus(Femandex and Lindsay-60) the available clinical evidence argues overwhelmingly for a peripheral origin.

(2) Non-classical paroxysmal nystagmus: Cawthrone(l954) and Harison(1968) suggested that non-classical paroxysmal nystagmus indicates CNS pathology.

H. Position Test:

1. Nomenclature: the position test explores a series of standard head positions to determine if the patient has positional nystagmus

To permit reasonably concise communication of position-test results a positional-nystagmus terminology has developed eg (Henriksson et al; 1972.

(a) Spontaneous versus Positional nystagmus.

(1) Spontaneous nystagmus: Any nystgmus present in the head-upright position with eyes centered

(2) Positional nystagmus: A nystagmus that is not present with head upright out is present in one or more other positions.

3) Spontaneous and positional nystagmus:

A nystagmus present in the head-upright position but modified either in intensity or direction by assumption of one or more positions.

(b)Classification of positional nystagmus:

Most authorities(Barber 1964; Fernandez and Lindsay 1960; Jengkees 1961; Lindsay 1967) use the Aschan et al (1957a) modification of Nylen's classification as outlined in Table IV.

Table-IV **Classification of Positional Nystagmus:**

1) Persistent- continues for at least 1 min after assuming the test position.

A. Type Indirection changing): beats in one direction in one (or more) positions and in the opposite direction in other positions (s)

3. Type.II(direction fixed)- beats in the same direction whenever present.

II. Transitory- goes away within 1 min after the test position is assumed. Called "Type III " positional nystagmus also called "positioning" nystagmus.

2) Position Test Technique: We obtain eyes-closed records of at least 30 sec with the patient in each of the following positions:

1) Sitting; 2) supine 3) Rt.lateral; 4) Head Rt. 5)Lt.lateral 6) Head Lt. 7) Head hanging.

The patient is moved into each position as slowly as is practicable Since spontaneous and positional nystagmus are subject to central suppression it may be desirable to have the patient perform a concentration task while in each test position However, this may reduce record quality by introducing voluntary eye movements muscle potentials and eye blinks. Therefore, one must use judgement in applying the concentration task to the positive test.

3. Diagnostic Significance of Position Test Results:

(a) "Idiopathic" positional nystagmus is often seen when the patient is in positions other than sitting and hence? in such instances must be called "positional " rather than "Spontaneous"

(b) Drugs causing positional nystagmus: Alcohol sedatives and salicylates may cause direction-changing positional nystagmus (Nylen 1950) The positional nystagmus produced by alcohol is the only drug-induced nystagmus that has been studied extensively with ENG (Aschan et al 1956b Hill et al; 1973)

(c) Pathologic positional nystagmus(Slow phase speed greater than 7° per sec).

Although some authors report a statistical tendency for direction-changing positional nystagmus to occur in central lesions, and direction fixed positional nystagmus to occur in peripheral lesions(Henriksson et al 1972; Nylen 1950) it is generally agreed that this tendency is not sufficiently strong to be useful clinically(Aschan et al,1956a; Tongkees 1961; Teykees and Phily'szoon 1964; Schiller & Hedbey 1960). Therefore positional nystagmus with slow-phase speed over 7 /sec whether type I & II is a nonlocalizing abnormality

I. Bithermal Calorie Test: Brown-Sequard first described the human caloric response in the latter part of the 19th Centuary However, it was not until Barany's early 20th Century work of

the caloric test found widespread clinical application(Jongkees 1949) In the 1920-1940 period variations of Barany's original technique proliferated(Arslan 1955, Jongkees 1949) but the caloric test achieved acceptance among clinical practitioners only as a rather unreliable qualitative procedure.

Barany Caloric test was subiect to three fundamental weakness:

1. A difference between the cold caloric responses could be due to a difference between Left and Right beating nystagmus(which could be the result of either a brain lesion or a peripheral vestibular deficit) or to a difference in the responsiveness of the peripheral labyrinths. The inability of the cold-water caloric test to distinguish between these two possibilities limited its localizing value.

2. Caloric nystagmus could be observed only while suppressed by visual fixation. As a consequence comfortable degrees of caloric stimulation were required to break through this suppression and even then only a few comparatively difficult to observe nystagmus was available for analysis.

3. The measurement parameters available with naked-eye observation(latency, duration and a subjective assessment of nystagmus " vigor") were not optional. The ENG recorded bithermal caloric test overcomes all of the these weaknesses. The use of warm as well as cold caloric stimuli allows nystagmus-directiondifferences to be distinguished from vestibular-responsiveness differences(Fitzgnald and Hallpike 1942; Kobrack 1943; thornval 1932) The use of ENG to record the nystagmus allows

removal of visual fixation, thus providing a much larger and more accurate assessment of nystagmus intensity than any parameter measurable by direct visual observation.

1. Test Technique: The bithermal calorie test consists essentially of obtaining cold and warm caloric responses from each ear, with the temparatures equally above and below body temparature (Fitzurald and Hallpike, 1942)

The technique as originally described(Fitzurald and Hallpikel942) used temparatures of 30°C and 44°C and was intended for use with eyes open and fixed. With eyes open and fixed 40-secs irrigation at temparatures of 30° C and 44°C were thought to be " minimal stimuli." However when later investigations of the test were done with eyes closed it quickly became apparent th t the stimulus was in fact rather tense. Aschan(1955) recognized the necessity of reducing stimulus intensity and chose to reduce duration rather than the difference between body and irrigation temparatures.

In order to minimize central suppression of the caloric responses(Barber and Wright 1961; Coats 1966), the patient is instructed to perform aloud a "concentration task", eg - substracting serial swens, during each response. The difficulty of the task is adjusted to match the patients' ability.

2. Quantifying the Caloric Test:

(a) Which is the "best" measurement of caloric-response magnitude?

Generally agreed that maximum speed of slow component(SSC) is a better measure of caloric-response magnitude than duration because;

Alternative Measurements of Caloric-Response Magnitude:

I. Responseduration-time elapsed from first beat to last beat

II.Total response magnitude:

A. Total number of beats during response.

B Total amplitude(Sum of the amplitudes of all beats occuring during the response)

III. Maximum response magnitude:

A. Maximum amplitude.

B. Maximum frequency

C. Maximum slow-component speed.

1. Maximum SSC correlates better with stimulus intensity (Aschan,1955; Henriksson,1956), probably because the temparature change time course, rather than the end-organ response, is the primary determinant of caloric response duration(Cawthrone and Cobb 1954; L.Shiyama and Keels,1970)

2. In patients with unilateral SN hearing loss, maximum SSC demonstrates vestibular pathology much more often than does

caloric-response duration.(Fodor,1968, Henriksson,l956;)-(Jongkees and Philipszoon 1964; Koch et al.,1959; Stalile & Bergman, 1967) convenience is the main argument for the various alternatives to maximum SSC. However, this " arguments "force is reduced by the availability of "short cut" hand measurements differentiator or "velocity" channels(Henriksson 1956) and a recently developed instrument which automatically calculates and digitally displays maximum calorie SSC(Coats&Black-73)

(b) Determining Maximum caloric-response SSC: The most direct way of determining SSC is to measure its amplitude and duration and divide duration into amplitude. In terms of records appearance this determines slow-phased steepness or slope. The effect of random slow-phase speed fluctuations is minimized by averaging SSC, across several beats. The approximate measurements may occasionally yield false-negative res lts(Henriksson-1956). Therefore a borderline-significant difference in Calorie responses obtained by an appropriate measurement may prove significant if the responses are measured the "exact" way

(c) Measuring Calorie unilateral weakness and directional Pre-ponderance: Caloric-nystagmus SSC's cannnot be used directly to evaluate the vestibular system, because absolute values in normals very widely(Aschan et al 1956 a; Tongkeas,l948 Tung and Komhuber 1964, Stahle,1958. Therefore comparative measurements are used (Tongkees 1948). Two comparisons are made:

(1) a comparison of the responses from the right versus the left ear(unilateral weakness or canal paresis) &

(2) a comparison of right-versus left beating nystagmus (directional preponderance)

Absolute differences between ears and between right and left beating responses become more variable as the responses became more intense(Fongkees and Philipszoon,1964). Therefore, we express unilateral weakness and directional preponderance in relative terms i.e., as a percentage of the total of all four response intensities. Thus, calorie unilateral weakness (UW) and a directional preponderance(DP) are calculated by egns(l)& (2) resp.(Larts 1965; Tongkees and Philipszoon,1964):

$$(1)\quad UW = \frac{\text{Response from Rt.ear - Response from Lt.ear}}{\text{Total of all four responses.}} \times 100$$

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

$$(2)\quad DP = \frac{\text{Rt.beating response- Lt.beating response}}{\text{Total of all f ur responses}} y$$

$$= \frac{(IC + RW) - (RC + LM)}{RC + LC + RW + LW} \times 100$$

Where:

RC = Maximum SSC of right- cold response
LC = Maximum SSC of left - cold response
RW = Maximum SSC of right- warm response
LW = Maximum SSC of left - warm Response.

3). Normal limits of Calorie-Test Results:

(a) Unilateral weakness(UN) and directional preponderance(DP)

Table summarizes the results of six-independent, normal bithermal calorie test series. In each series UW S DP were calculated according to equations D&L

STANDARD DEVIATIONS OF NORMAL CALORIC UW & DP
(Measured By Maximum Slow-Component Speeds)

Reference & Methods. -	Unilateral Weakness.	Directional Pre-ponderance.
Aschan et al(l956 a) 30-sec irrigations at 30°c & 44°C eyes closed 25 subjects, 25-50 years old.	8.6%	8.8 %
Henriksson(1956), 40-sec irrigations at 30°C and 43.6°C, in dark,25 subjects, 19-45 years old.	9.5%	8.5 %
Tongkees et al(1962)30-sec, irrigations at 30°C and 44°C eyes closed. 47 subjects.	7.5%	8.7 %
Preber(1958)40sec irrigates at 30°c and 44°C eyes closed 50 S's	5.3%	5.8 %
Coats(1965).40 sec irrigations at 30°C and 44°C eye closed 30S's	9.5 %	13.2 % .
3rookler and Pulec(l970)-30sec irrigations at 30oc and 44o C eyes closed .839 "patients without nuro-otologic disease	11.5 %	13.5 %

UW Standard deviations vary from 5.3 % to 11.5%

In all but one of the normal ieries(Henriksson 1956) DP was more variable than UW. Further complicating the determination of HP's normal limit is the possible existence of a " Physiologic" DP in some normals.
(Coats,1966, 1969; Hallpike et al., 1951)
Tongkees et al, 1962)

(b) Bilateral Weakness(SW): Although absolute maximum SSC's vary widely in normals, it is general clinical impression that a very weak(or absent) caloric response from both labyreaths is abnormal. Some difficulties arises, however, when one attempts to define "very weak" because normative data on absolute values of maximum calorie SSC are Scanty. Preber(1958) found a mean normal maximum SSC of about 22^0c per second and a standard deviation(S) of about 6° per sec. Subtracting 25 from the mean SSC would give a lower limit of 10° per sec.
Herriksson(1956) found comparable values of 29° per sec and 11° per sec giving a lower limit of 7° per sec.

(C) Failure of fixation suppression (FPS):

In normal individuals, visual fixation either suppresses or abolishes caloric nystagmus. Several reports suggest that failure to suppress calorie nystagmus with visual fixation is a pathological sign indicating CMS prthology(Hart,1967; Haceario et al,1972; Naito et al; 1963, Preber & Silfverskiold 1960)
Quantifying the amount of fixation suppression may provide a

more sensitive test(Demanez and Ledonx 1970). It is suggested (1) slow phase speed is the best nystagmasparameter to quantify FFS and (2) Suppression of SSC by 20% or less is abnormal(Alpext and Corts)

4. Diagnostic Significance of Caloric Test Results:

(a) Unilateral weakness: A caloric UW can only be caused by a lesion of the vestibular end organ or the primary vestibular nerve fibres. It is therefore a peripheral finding. (Aschan,1955, Aschanet al 1957 b; Tongkees,1948; Tung & Komhuber, 1964; Stalile,l958)

The division between " Central % and peripheral is to some extent arbitrary, since a vestibular-nucleus lesion could involve primary vestibular nerve fibres and therefore produce a UW. Such a lesion always produces central findings alongwith the UW(Coats,l970)

(b) Directional Preponderance:

A Caloric DP can be due to either peripheral or central pathology(Brookler, 1970; Cawthorne et al 1942; Fitzgerald & Hallpike 1942; Koel et al 1959).

It is therefore a non-localizing abnormality.

(c)Bilateral Weakness: Caloric BW may be due to either bilateral peripheral vestibular pmthology eg.-as strepto-mycin toxicity might produced or Central pathology which interferes with the vestibulo-ocular reflex are . Most BWs are due to bilateral peripheral prthology. The patient

almost always has associated central oculomotor signs. In particular the OKN test is usually abnormal.

(d) Failure of fixation suppression:

FFS is a central sign however two benign causes of FFS have been demonstrated and must be ruled out. (1) Sedation particularly by Barbiturates(Coats 1970? Rashbass and Russell-1961) and (2) Contact lenses particularly if they are new or uncomfortable . In additionwith all other central ENG abnormalities peripheral ocular prthology must be ruled out in the patient demonstrating FFS.

DIAGNOSTIC SIGNIFICANCE OF ENG ABNORMALITIES

Test	Normal or of questionable pathological significance	Non localizing	Peripheral	Central
Calibration				Ocular dysmetria ("Calibration over-shoot")
Gaze test				(R/0 eye blinks) Gaze mystagumus Vertical Unilateral (R/0 intense spontaneous NYS) Bilateral equal (3/0 bilateral unequal sedation)
Optokinetic test				Asymmetry (R/0 intense span Bilateral deminution (R/0 lack of voluntary usual fixation and sedation)
Sinusoidal tracking position test	Horizontal & vestibular spontaneous NYS and positional NYS less than 7.0*/Sec. Vertical spontaneous NYS behind/closed eyelids	Vestibular spontaneous NYS & positional NYS greater than 7.0*/Sec.		Central spontaneous NYS.
Paroxysmal Nystagumus test		Non classical	Classical	
Bithermal caloric test	DP between 20 & 30% ,	DP grater than 30%	UW (R/0 BW central interruption of V-O Reflex)	FFS (R/0

"R/0 - Rule out; NYS = Nystagmus; V-0 = Vestibulo-Ocular"

Chapter VIII

ENG FINDINGS IN VARIOUS PATHOLOGIES.

Central Nervous System Lesions:

Coats AC(1970) reported Central Spontaneous nystagmus gaze nystagmus, failure of fixation- Suppression of Caloric nystagmus optokinetic asymmetry and disconjugate oculomotor abnormalities in case of CNS lesions.

Kimm J and Maclean JB(1975) inferred as . . . . however considering the intricate neuroanatomical pathologies within the ocular motor system in addition to the elaborate vesti-bulo-ocular connections, it is reasonable that disconjugate eye movements may result with certain CNS lesions. Technique in which they employ independent eye movement measurement in order to assess the movement of each eye separately during ENG reveal that disconjugate eye movement occurred even with extra axial lesions which spread the medial longitudinal fasciculus- a pattern seen in cases C CWS lesions.

Padoven I et al (1975) listed 60 signs of CNS impairment from there 26 nystagmic irregularities have a small diagnostic value. No sign occurring alone could be taken as pathogno-monic, only a group of signs can be used for the impairment localization.

Besides for the peripheral vestibular and the peripheral vesti bulocochlear impairment, The authors succeeded to make a group containing pathognomonic signs for mixed vestibular impairments as well as for multiplgsclerosis affecting vestivular structures.signs which indicate central impairment are:

1. Spontaneous nystagmus in the direction of the weaker laby-rinth and not in the direction of the healthy are.
2. Spontaneous nystagmus which does not become weaker on fixation.

3. Spontaneous nystagmus which increases on fixation.
4. Vertical nystagmus(multiple Sclerosis)
5. Retraction nystagmus.
6. Oblique nystagmus.
7. Rotatory nystagmus(medulla oblangnta)
8. Square waves(atherosclerosis of the brain)
9. Restlessness of the eyes
10. Vertical gaze nystagmus: Supranystagmus in Supcavasion.
11. Bitemporal gaze nystagmus: dextronystagmus in dextro-versicns and levonystagmus in levoversions.
12. Centrifugal nystagmus: dextronystagmus in dextroversions, levonystagmus In levoversions
    Supranystagmus in supraverssions or in all directions
    (mesencephalic and pontine impairments-inferior cerebellar fossa)
13. Positional nystagmus type Nylen I: persistent unfixed, indefatigable nystagmus(Inferior c rebellar fossa)
14. Positional nystagmus type Nylen II:
    Persistent fixed, indefatigable nystagmus.
15. Nystagmus on torsion of the neck.
16. OPK nystagmus contralaterally weakened
    (Cerebral hemisphere mesencephalon)
17. OPK nystagmus ipsilaterally weakened
    (pons and the cerebellum)
18. OPK nystagmus bilaterally weakened
19. OPK inversion.
20. OPK disorganization.
21. No response to the OPK test(hysteria or blindness)
22. Induced nystagmus of too long a duration or of slow stopping
23. In induced nystagmus the time of the strongest response is shifted.

24. Induced nystagmus with too strong slow component hypernystagmus.

25. Strong Secondary nystagmus.

26. Paradox or perverted nystagmus relative to the expected direction of the Induced nystagmus.

27. Directional preponderance of nystagmus on the affected side.

28. Diminished fixation suppression of nystagmus.

29. Fixation suppression of nystagmus absent.

30. Negative fixational suppression of nystagmus: Stronger nystagmus on fixation than on non fixation.

31. Changes in the direction of nystagmus during fixation.

32. Nystagmic irregularities only in one direction of induced nystagmus

33. Pathologic fixational Suppression in only one direction of the induced nystagmus.

34. Changes in the direction of nystagmus during fixation in one direction of the nystagmus only.

35-60. Nystagmic irregularities.

Cerebral Hemispheric lesions:

Benitez JT(1972) found nystagmographic alternations with eye tracking, octokinetic and calorie tests in patients with temporo parietal legions with extension to the underlying white matter In cases where the lesion was autosuperior in the parietal to be with minimal extension to the white matter, no significant nystagmographic alterations were seen. In temporoparietal lesion that extends deep into the white matter interrupts oculomotor efference which causes a diminition or abolishment

of the ortokinetic nystagmus to the opposite side of the lesion. Thus optokinetic test which is easy to perform and easy to interpret has a greater diagnostic significance that the vestibular directional preponderance in central hemispheric lesions.

Vestibular Imbalance:

A unilateral reduced vestibular response is generally thought to be due to peripheral labyrinthine or VIII nerve pathology. However, a reduced vestibular response noted on a monothermal (either warm or cool) caloric(air or water) examination may be the result of directional preponderance alone, which has no localizing value- Becker GD(1975). The effect of directional preponderance on monothermal caloric examinations may be eliminated when the responses from both the warm and cool caloric stimulations are used in calculating Rt/Lt difference

Only after calculating the results from both the cool and warm (bitherrral) examinations together is it evident that a 100% Rt.directional preponderance is the cause of the monothermal Rt/Lt difference. So here instead of interpreting the results (from the monothermal examination) as being conststant C" peripheral pathology we would include that a vestibular imbalance is present, but is of no localizing value.

Idiopathic Spontaneous nystagmus:

Coats AC(1969) did a study of spontaneous nystagmus in 121

normal subjects and 160 patients. Suggests that an "idiopathic Spontaneous nystagmus" is recorded with the subjects' eyes closed in 20-25% of both the normal and patient population. This z± nystagmus is probably of no significance in the diagnosis of vestigo-producing disorders. It is of low-intensity (usually below 4°/ sec, but occasionally in the 6 - 10°/ sec range) and is more frequently directed to the Left than to the Right spontaneous nystagmus above $l0^0$/sec is of diagnostic significance.

It is probable that such a nystagmus will be of peripheral origin and directed away from the side of the peripheral lesions.

Directional Preponderance-DP

DP is clinical ENG refers to bithermal caloric induced nystagmus the intensity of C is greater in one direction than the other(Brodeler-KH) The bithermal caloric stimulus is becoming widely used in the ENG evaluation of the vestibular system. The finding of DP indicate an abnormality within the vestibular system, including its central connections and has no localizing value.

Perilabyrinthitis: presented a case of perilabyrinthitis with an unusual form of nystagmus altering mechanism. Patients who are f und to leave a decreased or absent response to caloric

or positional testing may on global digital compression; manifest nystagmus as recorded by ENG This form of mental altering is one which has not previously been described and should be kept in mind when evaluating patients with vestibular problem.

ENG in Infants:

Glosowski P et al(1977) tested by Caloric Stimulation (30° c & 44°C) 60 infants 3-6 years old devoid of any ear disease History. The distribution "with sedation" and "without sedation " occurred by chance. According to age and weight of the subject's sedation was obtained by intra-muscular injection of Valiur. Nystagmus registration was done by ENG. The quantitative reductions in the observed nystagmus were as following:

Number of beats:	19% ± 5%
Maximum angular velocity of the slow phase:	24% ± 11%
Total amplitude:	24% ± 11%

For caloric vestibular testing of infants, valium sedation proved to be very satisfactory.

Pilot Candidates & ENG: The Pastrotatong ENG responses of pilot candidates showing no, moderate or strong vestative reactions after Cawioh's stimulations were compared with

those of pilots flying different types of aircraft It could (Scurer M & Frohich(l972)
be illustrated that

(a) the smaller a person's vegetative resistance towards rotatory stimuli, the higher his postrotatory ENG- responses and the higher the difference between Right and Left turns,

(b) Jet pilots during their flying training spot a habituations of their vegetative reflexes and seem to develop a high sensitivity and a good balance of their vestibular system.

(c) This development towards higher sensitivity is less pronounced in fixed-wing pilots

(d) This development was not found in helicopter pilots.

Equilibrium trouble:

Anbry M et al (1968) desired a study to show the static modifications in patients having equilibrium troubles. The tests are performed with or without vestibular stimulation with the aid of

(1) A statokinesimeter which allows the recording of the position of the center of gravity compared to the polygraph of support its shifting in the four cardinal directions and the guide correcting movements during a vertical stand of variable duration (Gen:l min)

(2) An electromyograph which records the simultaneously the muscular activity of some muscular groups.

(3) ENG findings of Pialonx et al demonstrate clearly that every vestibular excitation provokes a functional variation by all the muscle of the body as the vestibular system is strictly connected-with the extrapyramidal system this fact means that the vestibular activity is continually checking the muscular function.

Vector ENS:

Evjatar et al (1968) presented Vector ENG as a method of vestibular system study that may allow differentiation and analysis of responses from both the otolith and the semi-circular canal system. It utilizes a four channel recording system consisting of horizontal vertical and two pairs of diagonal electrodes. Nystagmus, an oscillatory kinetic complex, resulting from the corneo-retinal potential of the eye ball as a dipole and possibly influenced by the electrical fields lends itself to vector analysis. Vector ENG offers the possibility of analysizing the shape and rotatory direction of the nystagmus, factors which are important in localizing the source of nystagmic impulses within the vestibular system. The conclusions are based on multiple channel ENGs performed during measurement of positional nystagmus and during ny labyrinthectony for Menieresdisease. Selective mechanical stimulation of utride, saccule and anterior vertical canal was performed prior to end-organ destruction and the nystagmic responses were recorded on the four ENG channels.

Tumors of the Cerebellopontine Recess:

Benitez JT and Bouchend KR(1974) found a significant nystagmo-graphic alterations in the examination of 10 patients with tumors of the cerebellopontine recess as impaired ocular pursuit movements when the eyes were moving towards the side of

of the lesion as detected by eyes tracking and OPK tests

Unilateral diminition of OPK response was found towards the side of the tumor(moving target towards the unaffected side)

The presence of saccades aiming OPK response provided additional information? even when the responses appeared symmetrical.

Investigation of Spontaneous lateral gaze & positional nystagmus contributed in less extent to the diagnosis of the lesions. A

Caloric test indicated namely the side of the lesion but gave little information as to the size of the tumor.

Multiple Cranial nerve Palsy:

Correa Sojos FA(1975), ENG is an important investigation in

the diagnosis of central or peripheral lesions of the vestibular pathways, whether or not they are associated with other cranial

nerve lesions. Several disorders of CNS whether due to infection degenerative conditions or tumors may present as a disorder of the- vestibulo-acoustic system.

ENG is a method by which in certain cases, the 1st indications

of these conditions may be demonstrated.

Meniere's disease:

Richard WB(1979) examined a case of Menier's disease. Patient

presented with a direction-fixed geotropic positional nystagmus.

Completely consistent with his peripheral end-organ disorder.

Following a glycerol test; despite marked improvement in

cochlear function, he experienced a symptom of vestibular

dysfunction. A ENG shortly thereafter showed the nystagmus had changed to a pattern suggesting the possibility of a central involvement. In this instance, this pattern was not due to the basic disease process or to alcbchol. It was the result of the ingestion of glycerine as a diagnostic and prognostic procedure.

Endolymphatic hydrops or vertigo of Meniere's is a peripheral and global labyrinthine syndrome with cochlear as well as vestibular symptoms. Roquette J and Sartual J(1961) studied quantitatively and qualitatively the result of a functional cochleovestibular examination of 41 patients suffering from unilateral vertigo of Meniere's as well as the meaning of each parameter of the vestibular response, authors ' pay special attention to the possibility of there being constant or typical relations between ENG and audiological findings in illness

Whiplash Injury: Some people who have had "whiplash " injuries complain of positional vertigo precipitated by rotation or extension of the head and this subjective complaint can often be substantiated by nystagmographic examination- Compere WE(1968) Although the sumptom tends to subside with tune reassurance and conservative therapy, the positional vertigo may continue for more than 24 months. The positional vertigo probably results from compression of the 1st portion

of the vertebral artery against the transverse by contracted deep "cervical, fascia and may amenable to surgical treatment if conservative therapy fails.

Dizziness:

Eviator A et al (1970) did a study using ENG Spontaneous and positional nystagmus findings of 70 normal volunteers and 745 patients complaining of dizziness. The results were compared between these two groups. In the group of patients complaining of dizziness the results of the positional tests were compared with the result of caloric examination.

As a group the patients complaining of dizziness were found to have more spontaneous and/or positional persistent nystagmus than the normal volunteers. However the incidence of short duration type of nystagmus was not significantly difficult between the groups.

The majority of patients with spontaneous and/or positional nystagmus were found not to be suffering from CNS lesions thus indicating that such nystagmus in itself does not point to a CNS lesion. Spontaneous and positional nystagmus although an important component of the ENG vestibular examination does not replace the bithermal caloric test and has no significant value without it.

Down's Syndrome:

Zarnoch JM (1980) recorded and described ENG results, particularly caloric responses from a small sample of the Down's syndrome population. The most significant implication of this study is that ENG's can be performed on children, including

those with retardation. It has longbeen the consensus that children under the age of 7-8 years could not be evaluated with ENG largely due to the tasking and calibration problems. However this investigation has demonstrated that through minor SNG modifications a broad picture of existing vestibular functions can emerge.

ENG in Parkinson's disease:

Pialouk(l969) did a study before and after coagulation of thalamus in Parkinson's disease. Confirmed (a) the existence of vestibulo-thalamic fibres.

(b) the unilateral predomenance of vestibular fibres or centres in the thalamus zone,

(c) the existence of the "nystagmogenic centre" in this zone. This centre was discovered by Lachmann and Bergman and Monnier and Montandon.

Cervical Vertigo:

Mccabe B(1975) did ENG examination in a case with cervical vertigo along with many others. The combination of paracity of physical and radiographic findings, betterness of the complaints, together with the pending litigation led them to the conclusion that their patients symptoms were either spurious or emotional. The positive ENG sign was the only sign of organic disease. The physiopathology of cervical vertigo from whiplash injury is not known. It may be due to scanning of either deep cervical musculature vasculative or ligments producing abnormal volleys of discharge along the spino-vestibular tracts onto the vestibular nuclei.

Posterior Fossa Disease:

Parker W(1977) studied ENG findings of 150 patients with confirmed neurologic disease. Reported frequent occurrence of diminished optic fixation inhibition. Literature suggests that this inhibitory reflex is mediated through the cerebellum. Clinical evidence from this group of patients shows a high correlation between diminished fixation inhibition and the presence of other cerebellar findings. A low correlation exists between normal fixation inhibition and the presence of cerebellar signs.

This is considered as strong inferential evidence that optic fixation inhibition of vestibulo-oculomotor activity is mediated through the cerebellum. Conclusive evidence must await specific lesioning experiments, Author described the physiologic importance of the optivestibular inhibitory reflex.

Cerebellar Tumors:

Nagabhyrae (A.R. (1979) concluded that by facilitating the early detection of an intracranial lesion, ENG contributed to a successful outcome in a 38 year old patient who initially presented only with occasional dizzy spells associated with nausea and vometting but C no other CNS cardiovascular otological signs.

Haring Roger D(1973) showed that the presence of very fine ocular dysmetria can be detected as a calibration overshopt during the performance of routine ENG. This overshoot of the standard 10° fixation target are is an excellent sign of a cerebellar defect.

Disseminated Sclerosis:

Collord M and Conranx C(1981) did work on ENG in case of Bisseminated Sclerosis patients. A review of the literature shows the extreme frequency of nystagmus and changes in vestibular reflexes in disseminated sclerosis. During the course of this disease, it is possible to detect nystagmus in at least 2/3rd of cases and in a similar proportion there are changes in vestibular reflexes.

Usually a hyper reflexia, especially in the early phases of DS Subjectively the patients rarely complain of vertigo and such symptoms are in any case past of the early form of the disease. on the other hand disorders of balance are much more frequent but the factors involved are numerous and the role of the vestibular system is often debatable. ENG and Oculography demonstrate the extreme frequency of spontaneous or fixed gaze nystagmus in this disease as well as the extreme frequency of changes in conjugate eye movements changes in reflex, voluntary and automatic conjugated eye movements ENG can help decisively in the diagnosis of DS by revealing changes which reflect damage to the brainstem lesions which are clinically very often silent. In this way the diagnoses of DS may be confirmed. Further more ENG can easily suggest another diagnosis other than the early form of DS of the vestibular type

Multiple Sclerosis: Study done by Aanta,Rickkinen & Frey- (1973) found either positional or spontaneous nystagmus in 60% of the multiple cases tested by them. Multiple Scterosis was

verified using both Schumatchus criteria and CSF findings They suggest ENG and other nerological techniques provide a valuable aid in the early differential diagnosis of Multiple Scterosis because of their sensitivity and the close relationship between dilection sites of MS and those structures which contribute to the maintainance of balance. '

Spooner et al (1972) used phenomenon of hyperthermic electroystymography which sometimes causes new neurological findings in patient with multiple sclerosis.

Bell's Palsy:

Mironenko(1978) examined ENG in patients with facial nerve palsy of Bell's type. 50% cases demonstrated spontaneous nystagmus Vestibular hyporeflexes on the paralysed side were found in 30%

Hyperflex in 25% and normal reflex in 48% .

Authors suggests that vestibular disturbance are of the same etiology on Bell's paralysis.

Sensorineural hearing loss:

Rahko T and Virolainen E(1978) in their study presented two second test tones at frequencies of 500, 1000 and 4000 Hz and varying from 120 dB to 125 dB to 99 hearing impaired ears. In 22 ears (22%) an ENG change was seen after the tone stimulus, 8 cases had 3-10 second nystagmus, 5 cases a nystagmus of longer duration and in seven cases spontaneous nystagmus strengthened. The frequency has not effect on the appearance of nystagmus.

The appearance of the ENG change in all recruitment groups supports the opinion that the effect is not mediated through hearing pathways but by straight stimulation.

ENG after Stapedectomy:

The aim of the study done by Spector M (1973) was to assess the persistent vestibular changes after stepedectomy as shown by ENG. 62 Otosclerotic ears were studied before and after stepedectomy for vertigo, spontaneous and positional nystagmus with closed eyes and intensity of caloric responses, in this sense preoperative vertigo, spontaneous or positional nystagmus poor-boneconduction or caloric hypoactivity did not routinely correlate with post operative vestibular troubles. In most cases vertigo was demonstrated after operation. In 6 cases drilling of the foot plate induced a 66% rate of vestibular dysfunction. A poor hearing result was usually associated with vestibular dysfunction. The vestibular apparatus was more cormronly adversely affected by stapedectomy than the cochlear apparatus.

Skin lesions: (Straham RW(1968)

The application of skin electrodes for ENG has resulted in undesirable skin lesions in two of more than 600 patients tested Though possible etiologies of these skin lesions include electrical burns, sensitivity of the electrode paste and over-zealons preparation of the skin, the lesions were probably caused by a problem specifically related to the i silver electrode and the individuals sensitivity. The presence of calcium in the lesions of these patients is best explained by a soft tissue phenomenon called "dystrophic Calcification ".

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