SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES &TECHNOLOGY (SCTIMST)

(An Institute of National Importance - Govt. of India)
Thiruvananthapuram, Kerala, India- 695011

DIPLOMA IN NEURO TECHNOLOGY

LOG BOOK

Submitted by,

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January 2014- December 2015

CERTIFICATE

I Ms. Shilpa Sebastian hereby declare that I have performed all the procedures listed carried out the project, under report.

Signature :.....

Name :.....

Place: Thiruvananthapuram
Date :

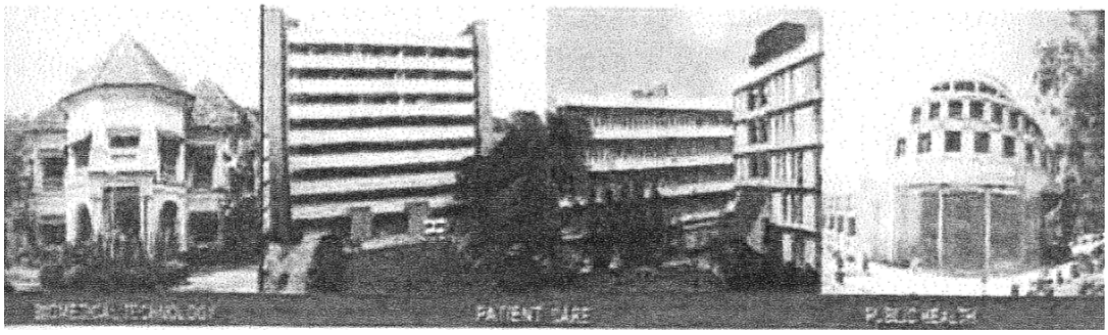
Forwarded. He has carried out the minimum requirement of procedures.

Signature :.....

Head of the department
SCTIMST, Thiruvananthapuram

CONTENTS

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES &TECHNOLOGY (SCTIMST); THIRUV ANANTHAPURAM, KERALA, INDIA.



The Sree Chitra Tirunal Institute for Medical Sciences &Technology(SCTIMST), Thiruvananthapuram is an Institute of National Importance established by an Act of the Indian Parliament. It is an autonomous Institute under the administrative control of the Department of Science and Technology, Government of India.

The Institute signifies the convergence of medical sciences and Technology and its mission is to enable the indigenous growth of biomedical technology, besides demonstrating high standards of patient care in medical specialties and evolving postgraduate training programs in advanced medical ties, biomedical engineering and technology, as well as in public health.

It has a 253-bedded hospital for tertiary care of cardiovascular and neurological diseases, a biomedical technology wing with facilities for developing medical devices from a conceptual stage to commercialization, center of excellence for training and research in public health.

The Institute has the status of a University and offers postdoctoral, and postgraduate courses in medical specialties, public health, nursing, basic sciences and health care technology. It is a member of the Association of Indian Universities and the Association of Indian Universities and the Association of Commonwealth Universities.

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Diploma in Neuro Technology

Diploma in Neurotechnology (DNT) is a two-year academic course under the department of Neurology, Sree Chitra Tirunal Institute for Medical Sciences and Technology. During the course of study in the institute the student is imparted with both theoretical and practical knowledge with regard to the subject. The student in the Institute becomes exposed to very expensive machines of latest technology, which he/she may find difficult to find in ordinary places. Since the Institute is a tertiary care Hospital, the student gets the opportunity to study patients with varied neurological diseases.

The practical grounds where the student learn the work stands to be unmatched elsewhere in our country.

During the two - year period the student is given EEG, EMG and Video EEG posting to get trained in the specific diagnostics. In the EEG lab he gets to know about the connections; recording of EEG and activation procedures of international standards. The student also gets training in acuity testing and field charting since the Optometry Lab forms a part of the Department of Neurology. In the EMG posting the student learns the techniques for nerve conduction, eliciting evoked potentials and machine operation for assisting the consultants to perform the needle EMG and SFEMG study. The student is also trained in Video EEG lab for monitoring prolonged EEG studies like VEEG monitoring, SPECT studies, MSLT, Polysomnography etc. In the Institute the students are allowed for observing Intra operative monitoring and Electrocorticography. The students also get opportunity to assist the programming of VNS therapy. With all this, the presence of many doctors with most excellence brings in the confidence for the student to work under strict conditions, where patient care is given the utmost importance.

ELECTROENCEPHALOGRAPHY

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Itroduction

The electroencephalogram (EEG) is a recording of the electrical activity of the brain. Hans Berger made the first recording in 1929 although similar studies has been carried out in animals as early as 1870. The wave forms recorded are thought to reflect the activity of the surface of the brain, the cortex. The activity is influenced by the electrical activity form the brain structures underneath the cortex.

The electroencephalogram (EEG) is a unique and valuable measure of the brain's electrical function. It is a graphic display of a difference in voltages from two sites of brain function recorded over time.

For the successful interpretation of an abnormal EEG, one must first understand the criteria necessary to define normal patterns. While a normal EEG does not exclude a clinical diagnosis (i.e., epilepsy), an abnormal finding on EEG may be supportive of a diagnosis (i.e., in epilepsy), be indicative of cerebral dysfunction (i.e., focal or generalized slowing), or have nothing to do with the reason that the study was performed (i.e., in headache). It is the clinical application of the EEG findings that imparts the utility of EEG.

Basic physiology of cerebral potentials

The origin of cerebral potentials is based upon the intrinsic electrophysiological properties of the nervous system. Identifying the generator source(s) and electrical field(s) of propagation are the basis for recognizing electrographic patterns that underly the expression of the "brain waves" as normal or abnormal. Most routine EEGs recorded at the surface of the scalp represent pooled electrical activity generated by large numbers of neurons. Electrical signals are created when electrical charges move within the central nervous system. Neural function is normally maintained by ionic gradients established by neuronal membranes. Sufficient duration and length of small amounts (in microvolts) of electrical currents of cerebral activity are required to be amplified and displayed for interpretation. A resting (diffusion) membrane potential normally exists through the efflux of positive-charged (potassium) ions maintaining an electrochemical equilibrium of –75 mV. With depolarization, an influx of positive-charged (sodium) ions that exceeds the normal electrochemical resting state occurs. Channel opening within the lipid bilayer is via a voltage-dependent mechanism, and closure is time dependent. Conduction to adjacent portions of the nerve cell membranes results in an action potential when the depolarization threshold is exceeded. However, it is the synaptic potentials that are the most important source of the extracellular current flow that produces potentials in the EEG. Excitatory postsynaptic potentials (EPPs) flow inwardly (extracellular to intracellular) to other parts of the cell (sinks) via sodium or calcium ions. Inhibitory post-synaptic potentials (IPPs) flow outwardly (intracellular to extracellular) in the

opposite direction (source), and involve chloride or potassium ions. These summed potentials are longer in duration than action potentials and are responsible for most of the EEG waveforms. The brainstem and thalamus serve as subcortical generators to synchronize populations of neocortical neurons in both normal (i.e., sleep elements) and in abnormal situations (i.e., generalized spike-and-wave complexes).

Volume conduction characterizes the process of current flow from the brain generator and recording electrode. Layers of cortical neurons are the main source of the EEG. Pyramidal cells are the major contributor of the synaptic potentials that make up EEG (Figure 1.1A). These neurons are arranged in a perpendicular orientation to the cortical surface from layers III, IV, and VI. Volumes large enough to allow measurement at the surface of the scalp require areas that are >6 cm2 , although probably >10 cm2 are required for most IEDs to appear on the scalp EEG because of the attenuating properties incurred by the skull. All generators have both a positive and negative pole that function as a dipole (Figure 1.1B). The EEG displays the continuous and changing voltage fields varying with different locations on the scalp.

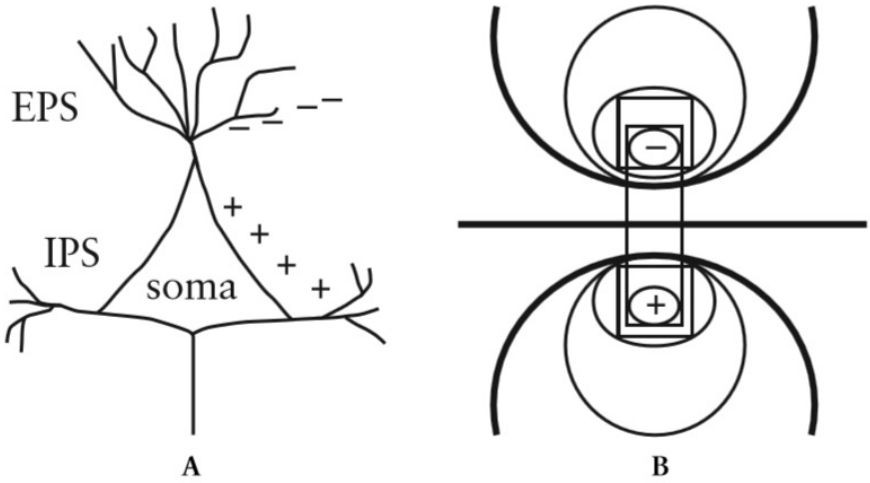
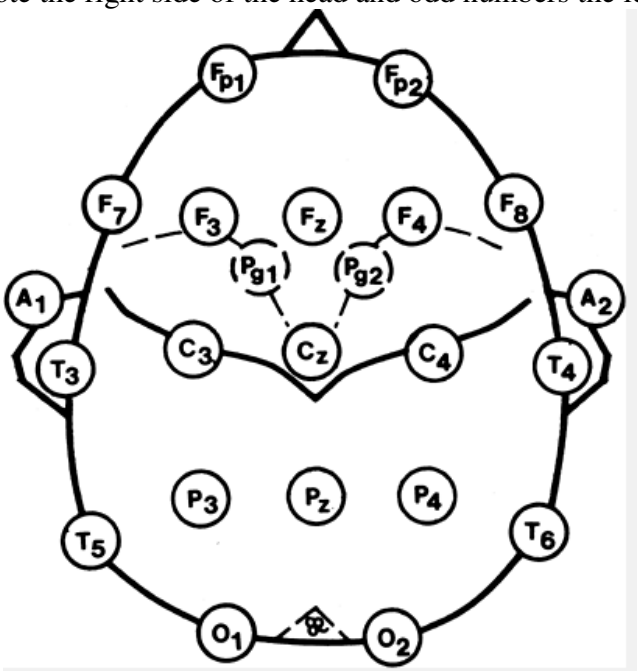


FIGURE 1.1. (A) A pyramidal cell with excitatory postsynaptic potentials and inhibitory postsynaptic potentials. (B) Dipole depicting a field of charge separation.

The nerve cells in the brain produce signals that are called action potentials. These action potentials move from one cell to another across a gap called the synapse. Special chemical called neurotransmitters, one will help the action potential to move to the next cell, the other will stop it moving to another nerve cell. The brain normally works hard to keep an equal amount of each of these neurotransmitters in the brain. EEG activity is quite small, measured in micro volts (m V) with the main frequencies of interest up to approximately 30 Hertz (Hz)

Electrodes

Small metal discs called electrodes are placed on the scalp in special positions. The technologist who measures the head using the International 10-20 System identifies these positions. This relies on taking measurements between certain fixed points on the head. The electrodes are then placed at points that are 10% and 20 % of these distance. Each electrode site is labeled with a letter and a number. The letter refers to the area o£ the brain underlying the electrode. e.g. F - frontal lobe and T- temporal lobe. Even numbers denote the right side of the head and odd numbers the left side of the head.



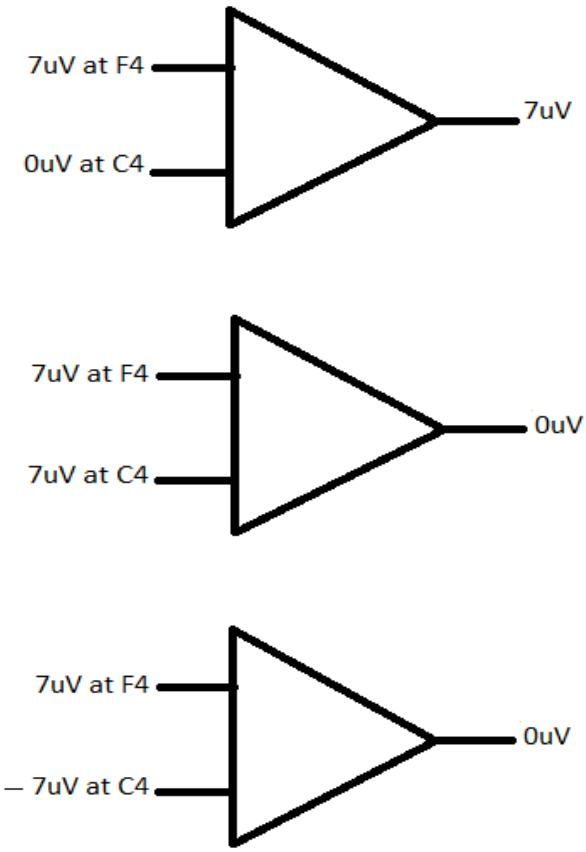
There are a great variety of electrodes that can be used. the majority are small discs of stainless steel, tin, gold or silver covered with a silver chloride coating. These normally have a lead attached. Alternative methods consist of a cap in which the electrodes are already embedded.

Montages

EEG machines use a differential amplifier to produce each channel or trace of activity. Each amplifier has two inputs. An electrode is connected to each of the inputs.

Differential amplifier

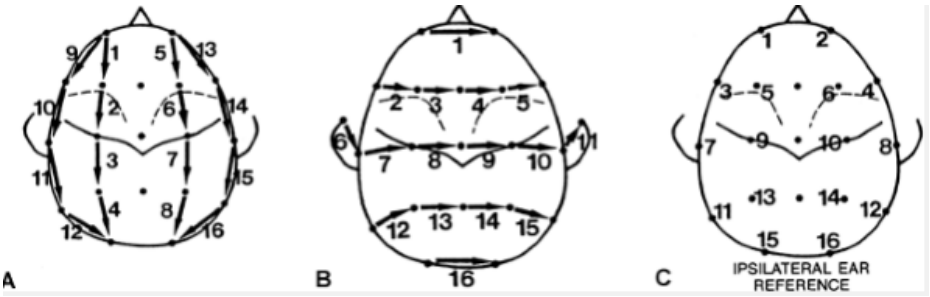
Differential amplifiers measure the voltage difference between the two signals at each of the inputs. The resulting signal is then amplified and then displayed as a channel of EEG activity.



The manner in which the pair of electrodes are connected to each amplifier of the EEG machine is called a montage.

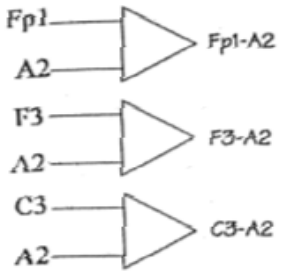
If one is mainly interested in a striking pattern, such as a spike-and-wave complex, then the display of such a pattern in a way that makes it stand out at a fairly large amplitude is desirable. For such a pattern, which is generally predominantly parasagittal, the use of the earlobe, whether ipsilateral or joined, is generally an excellent montage. If, on the other hand, the abnormality looked for is a fairly low-voltage limited pattern, perhaps involving only the midtemporal electrode and the reference electrode itself, there

is great danger that the pattern can cancel out between the involved electrodes and not show up well. The use of reference electrodes also can lead to some rather complex situations in which there appears to be out-of-phase waveforms occurring both anteriorally and posteriorally (Garvin and Gibbs, 1971). In at least some instances, this phenomenon does not represent a physiological reversal of activity but the contaminated reference electrode effect on uninvolved electrodes (Reilly and Seward, 1980).

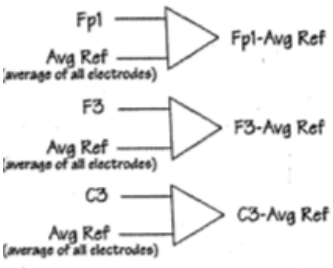


A: A representation of bipolar (scalp-to-scalp) montage in a longitudinal or anteroposterior (AP) direction. This montage is designed for easy comparison of left-right differences in the parasagittal or temporal area, but a montage with strict left-to-right sequence of sets of four could provide the same information. B: This is a typical bipolar (scalp-to-scalp) montage in the transverse or coronal direction. C: This is a reference (monopolar) montage. This particular sequence is designed to allow a front-to-back sequencing of channels to provide anatomic continuity

Common reference derivation: Each amplifier records the difference between a scalp electrode and a reference electrode. The same reference electrode is used for all channels. Electrodes frequently used as the reference electrode are Al, A2, the ear electrodes, or Al and A2 linked together.

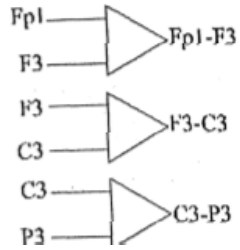


Average reference derivation: Activity from all the electrodes are measured summed together and averaged before being passed through a high value resistor. The resulting signal is then used as a reference electrode and connected to input 2 of each amplifier and is essentially in.active. All EEG systems will allow the user to choose which electrodes are to be included in this calculation.



Average reference derivation

Bipolar derivation: These sequentially link electrodes together usually in straight lines from the front to the back of the head or transversely across the head. For example the first amplifier may have electrodes Fpl and F3 connected to it and the second amplifier F3 and C3 connected to it



Analogue EEG instruments

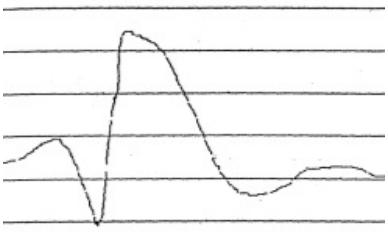
Conventional analogue instruments consist of an amplifier, a galvanometer and a writing device. A galvanometer is a coil of wire inside a magnetic field. The output signal from the amplifier passes through the wire causing the coil to oscillate. A pen mounted on the galvanometer moves up and down each time the coil moves. The pen draws the trace onto paper moving below it

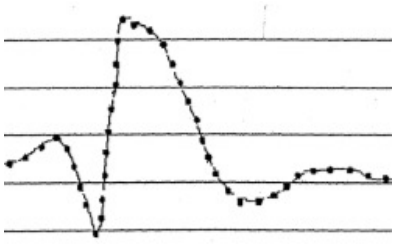
High and low frequency filters and sensitivity controls control the amplifier output. The high and low frequency filter values will set the window within which the EEG activity is recorded. This is known as the bandwidth. The sensitivity controls the size of the activity displayed. For example a sensitivity of 10 microV/mm means that a signal with amplitude of 100 microV will produce a 1 cm vertical deflection.

The speed at which the paper moves on will also affect the appearance of the waveforms.

Digital EEG instruments

A digital EEG system converts the waveform into a series of numerical values. This process is known as Analogue-to-Digital conversion (ADC).





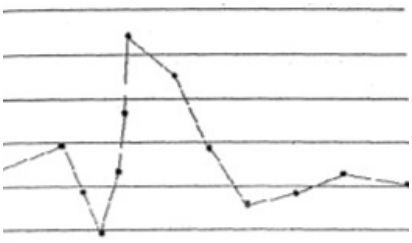
Analogue waveform Sampling rate of 240 Hz

The values can be stored in the computer memory, manipulated and then redisplayed as waveforms on a computer screen. The rate at which the waveform data is sampled in order to convert it into a numerical format is known as the sampling rate.

The sampling rate is usually expressed in Hz, for example 240 Hz is 240 times per second. The minimum acceptable sampling rate is 2.5 times greater than the highest frequency of interest but most digital EEG systems will sample at 240 Hz.

Some recordings which involve recording activity directly from the brain surface, may have activity of a higher frequency for example 200Hz. Therefore some digital EEG systems will have optional sampling rates of 480Hz available.

Sampling at rates lower them this will mean that when the signal is converted back to analogue form, it will not resemble the original waveform



Sampling rate 50 Hz

A second factor that affects the accuracy of the waveform is *sampling skew*. Sampling skew occurs when all channels are not sampled simultaneously. Many digital EEG systems sample channel first. then sample channel2, then channel3 etc. The time lag between sampling of each channel is known as sampling skew. To reduce the sampling skew, some digital systems use *burst mode sampling*. This increases the speed between successive channels sampling in order to reduce the amount of sampling skew.

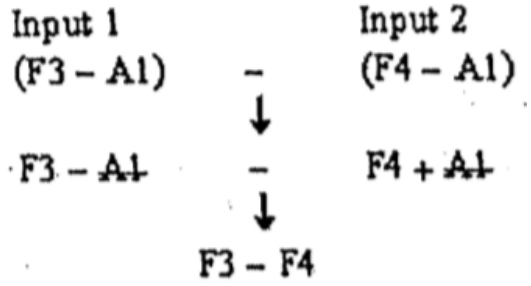
A third factor that affects the accuracy of digital EEG waveforms is the display. The accuracy of a monitor display depends on the number of points or pixels that are available. The number of pixels available is referred to as the screen resolution. Screen resolution is

described in numbers that represent the pixels available in the horizontal and vertical axis.

A VGA display has a resolution of 640 x 4$0 pixels while a monitor with a Super VGA display will have a screen resolution of around 1024 x 768 pixels. A typical page of BEG contains 10 seconds of data. A digital EEG system, sampling at rates of 240 I-tz will need to display 2400 samples horizontally for each recording channel. The highest screen resolutions available today do not have enough pixels to match the number of data samples. Systems that draw every other sample or every third sample in order to match the screen resolution Will have the effect of reducing the sampling rate and displaying incomplete data. An accurate digital system will draw two data samples per screen pixel. This means that all data points can be displayed and sampling rates will not be decreased.

EEG signals that have been digitized can be manipulated to change the montage ‘on-line' at the time of recording or 'off-line' after the recording is completed. This 'remontaging’ is accomplished by recording all EEG channels with a common reference e1ectrode. Regardless of the montage used to display the data while it is being recorded, data is stored into the computer memory in common reference mode. This allows the data to be displayed using different montages at a later time. Since digital systems store the analogue signal as numerical values, remontaging is a simple subtraction process, which results.in cancellation of the common reference.

An example is shown in the next figure. The reference electrode Al is common to both channels on input 2. It has the identical value in each channel. Rernontaging these two channels together into one new channel is by subtraction, which mathematically will cancel the value at the reference electrode. The resulting channel will therefore display the potential difference between F3 (input) 1 and F4 (input 2).



ARTIFACTS IN EEG

Although EEG is designed to record cerebral activity, it also records electrical activities arising from sites other than the brain. The recorded activity that is not o£ cerebral origin is termed artifact and can be divided into physiologic and extra physiologic artifacts. While physiological artifacts are generated from the patient, they arise from sources other than the brain (i.e., body). Extra physiologic artifacts arise from outside the body (i.e., equipment ; environment)

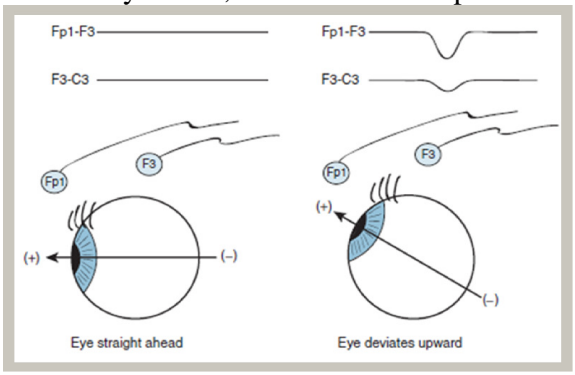
Artifacts originate from a variety of sources, and their recognition, identification, and possible elimination are a primary responsibility of the electroencephalographic (EEG) technologist. If unrecognized, can lead to misinterpretation of the recordings.

Most recorded physiological activity will have a logical topographic field of distribution with an expected falloff of voltage potentials. Artifacts have an illogical distribution that defies the principles of localization

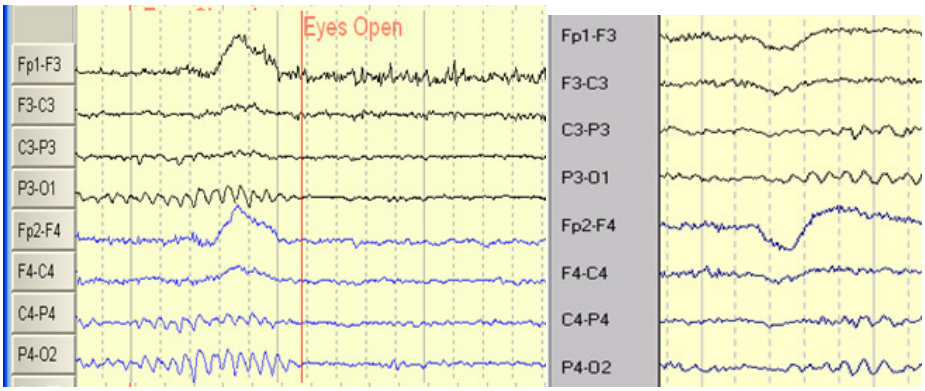
Physiological artifacts

Eye Movements

The eye movements are generated by the corneoretinal potential. This generator produces a direct current (DC) potential of approximately 50-100 mV. The electrodes involved are the ones closest to the eyeball: Fp1, Fp2, F7, and F8. This potential is best regarded as a dipole with the positive pole localized to the cornea and the negative pole to the retina. With an eye blink, the cornea rolls up due to normal Bell's phenomenon.



Upward movement is detected by a positive potential recorded at the supraorbital electrodes placed at Fp1 and Fp2. The activity recorded at F3 and F4 will be smaller in amplitude.When the eyeball moves in a downward direction, the inverse occurs. In EEG it appears as a bifrontal, diphasic, synchronous slow wave with field does not extend beyond frontal .

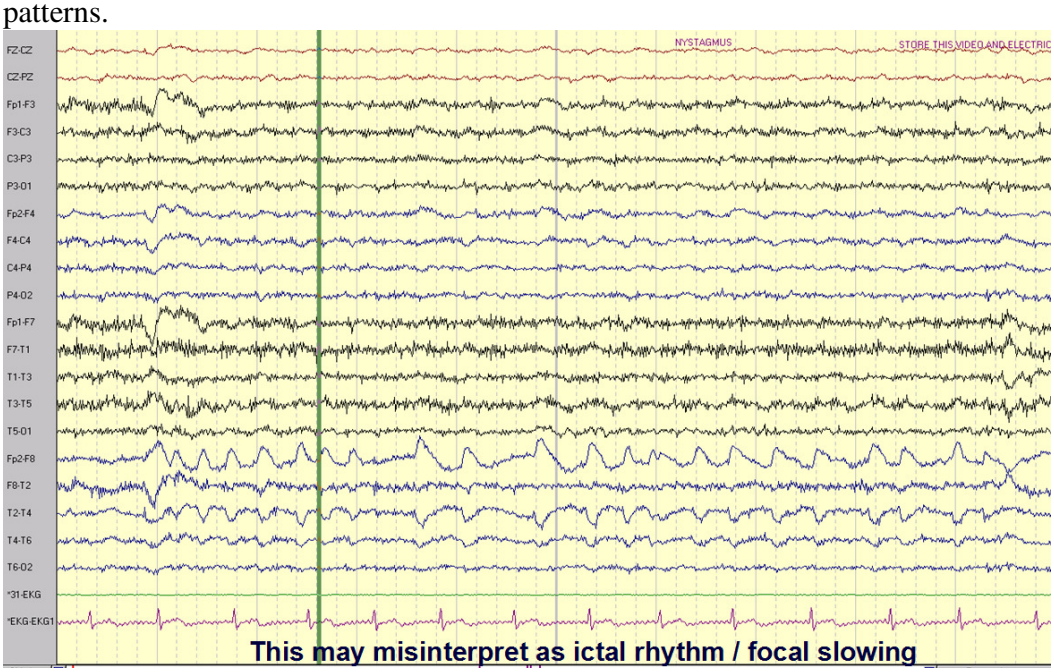


Eyelid flutter

Less easy to evaluate. Rhythmic activity, at a frequency of 5-10 Hz, that will be intermittent but occurs for many seconds at a time. May mimic intermittent rhythmic theta/delta activity. It usually low voltage slowing. Detected at only the Fp1 and Fp2 electrodes Lightly place the fingers over the eyelids. (for the identification) For the typical monitoring – place electrodes on the left/right side above outer canthi and left/right below outer canthi. Ocular artifact produces a phase reversal between infra orbital electrode and supra orbital electrode (ie, LOG and ROG)

Nystagmus

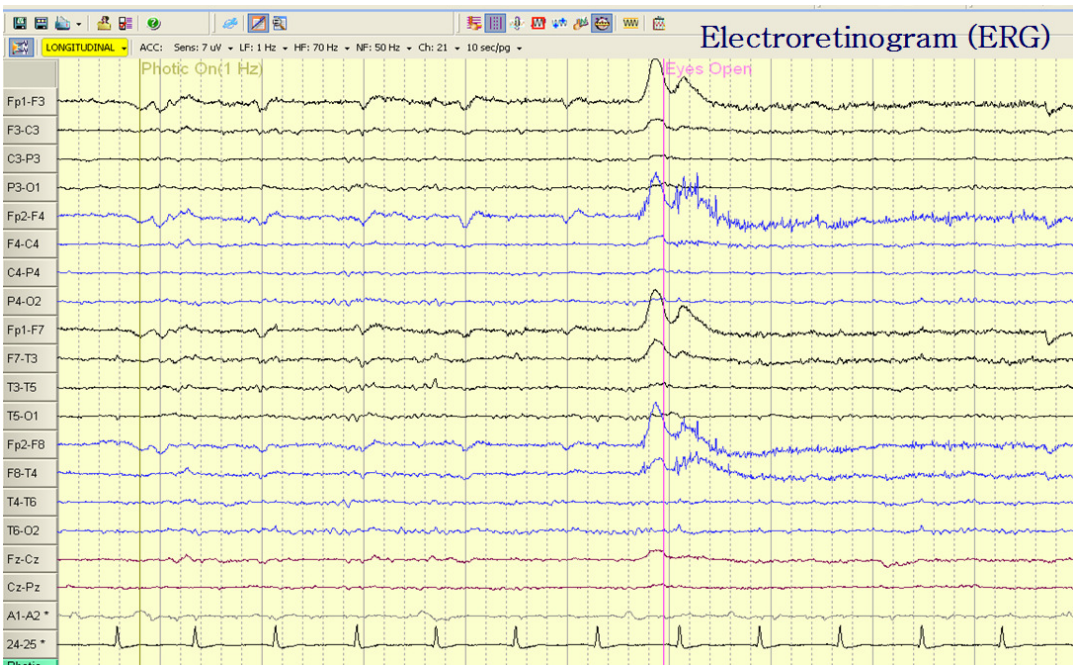
Horizontal nystagmus normally occurs bilaterally, but it is often detected unilaterally. Electrode detected – either F7 or F8. Movement may be recorded only by the electrode on the side of the fast direction of the nystagmus. Mimic – seizures or other physiological patterns.



Electroretinogram (ERG)

Low-voltage(<50microV) response to light stimulus of the retina. Appears in the anterior head regions. Especially during recording of electrocerebral inactivity, retinal response can be seen during photic stimulation. Confused with an electrode artifact generated by a silver electrode reacting to the light source. Confused with abnormal frontal sharp waves.For the faster rates of flashes, cannot be respond. If the response is constant, with no delay to the stimuli, and no decrease in amplitude, then the artifact is most likely due to the chip in the plating of the electrode. This artifact can be identified by shielding the frontal electrodes with an opaque covering that prohibits the light source

from being recorded by a faulty electrode.



Roving eye movement

It occurs during drowsiness with the frequency of less than 1 Hz. Direction of eyes is towards the side where the deflections are out of phase or where the pens separate (positivity). Slow roving movement artifact does not have abrupt changes.

Lateral eye movement

Which has a more abrupt transition. With bipolar montages, positive and negative phase reversals are seen at the F7 and F8 electrodes.

Asymmetric eye movement artifact

When ever there is decreased movement of 1 eye, Asymmetrical placement of electrodes, Unilateral enucleation and prosthetic eye replacement, External ophthalmoplegia , Bell's palsy, Skull defect(eye movement artifact will be lower in amplitude ipsilateral to skull defect.) we can see asymmetric eye movement artifact.

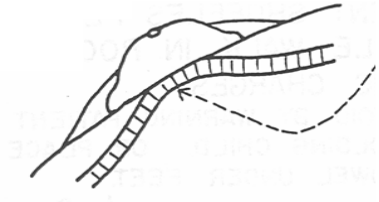
Electrocardiographic Artifacts

EKG artifact may appear simultaneously with prominent QRS complexes seen in several channels. Seen in obese patients, short necks and infants, in whom the head is close to the thorax.Most often detected by referential montages, especially those using ear electrodes as reference. Maximally over posterior head region. Extra systoles and cardiac arrhythmias are frequently detected in the temporal chain of bipolar montages but not in the parasagittal derivations, because the temporal electrodes are closer to the electrical

field of the heart. These cardiac beats often mimic cerebral sharp waves, spikes, or even temporal theta activity and may be misinterpreted because the field of electrical activity. The artifact is especially prominent in electrocerebral inactivity(ECI) recordings .

Pulse artifact

Usually confined to a single electrode. Appears as a slow-wave potential. It occurs when an electrode is placed over surface arteries. It is easily monitored by using an electrocardiogram (ECG) lead. The ECG signal will be time locked with lag (200-300msec) to the slow wave and always occurs at the same location in respect to the slow wave. Confirms the movement of the electrode with the pulse and alters its appearance on the EEG as pressure is applied. Slight change (1mm) in the position of electrode or head could rectify the artifact



by the movement of electrode with each pulsation cause more negativity to that particular electrode

Ballistocardiograph

Ballistocardiographic potentials are movement artifact that is time locked to the EKG. Microscopic movements of electrodes & wires from the recoil effect of beating heart produce rhythmic activity of low amplitude. Cardiac monitoring shows relationship of the cardiac signal to these pulsations, but not always time locked. Often mimic low-voltage theta, alpha, and beta frequencies, and mixtures of these low-voltage frequencies often resemble cerebral activity. Usually seen in brain death EEGs.

Pacemaker-generated artifact

High-voltage. Short-duration spike activity easily monitored with electrodes placed on the chest. This artifact may be continuous or intermittent, depending on the type of pacemaker.

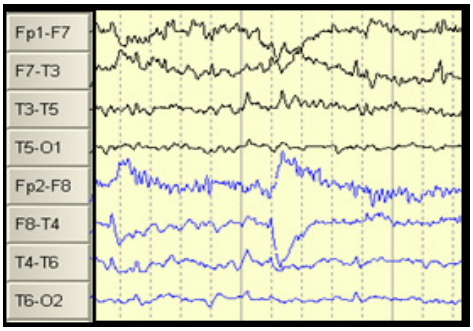
Cardiac arrhythmias

Cardiac arrhythmias can be misinterpreted as ictal events because – its evolving pattern. These patterns are easily monitored by recording from electrodes attached to the chest

Electromyographic Artifacts

Lateral rectus artifacts

Generated by low-voltage motor unit potentials localized to the lateral rectus muscles. They are typically recorded from the F7 and F8 surface electrodes, and the positive component is most commonly seen.appears with a sharp positive deflection of very short duration with a slow falloff as the muscle relaxes.The activity is usually not seen from the corresponding contralateral electrode. (ref :- Pedly). It mimics the appearance of a calibration signal.



A sharp transient is seen at the onset of each positivity in F8. Although this transient does resemble an epileptic spike, the fact that it occurs at the onset of each lateral eye movement helps to indentify it as a lateral rectus spike artifact on gaze to right.

Single motor units

May be recorded from any electrode placed over one of the scalp muscles. They appear as repetitive negative or positive deflections that have a comb-like appearance and are typically recorded from a single electrode.

Frontalis electromyogram (EMG)

Recorded over frontal electrodes when patients are tightly closing eyes The frontalis muscles are often activated by photic stimulation, and a photomyoclonic response is recorded. It may obscure EEG activity and even be confused with a photoconvulsive response

Temporalis EMG

Recorded from temporal electrodes when patients tightly close their jaws or make chewing movements. Frequent in patients with oral automatisms prior to or during an ictal event or who have orofacial dyskinesias

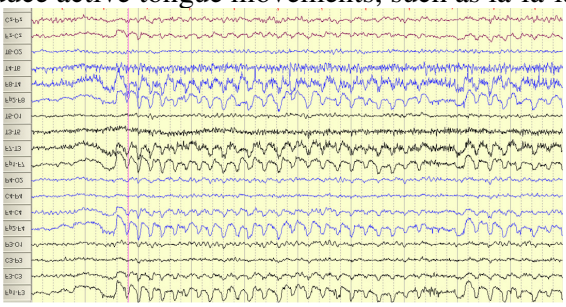
Note: EMG actiivity recorded over the frontal and occipital areas are common in tense individuals.

Most EMG artifacts can be reduced or eliminated with the use of relaxation techniques, such as reassuring the patient, comforting the patient, or simply massaging the muscle groups

The use of high-frequency filters to eliminate the artifact should be avoided, because these filters rarely eliminate the high frequency; rather, they alter its appearance from a sharp or spike wave to a more sinusoidal frequency that may look more like cerebral beta activity.

Glossokinetic Artifact

Movement of the tongue produces a DC potential. The tip of the tongue is negative in polarity with respect to its base. Potentials generated are frequently recorded as slow movements from the temporal electrodes and frontal. The activity may be unilateral or bilateral depending on the direction of the tongue movements. Monitoring the activity is done by placing an electrode on the cheek and another on the submental muscle of the lower jaw – activity with greater amplitude than at scalp. Frequency will be time locked. The artifact may be reproduced by having the patient repeat words or phrases that produce active tongue movements, such as la-la-la or ta-ta-ta.



Galvanic skin response

Perspiration artifacts are recorded as high-amplitude, very slow (0.25 – 1 Hz) potentials.Patients resting on perspiration-soaked pillows may display a voltage asymmetry created by a salt bridge. (This can be eliminated by cooling the patient and taking his or her head off the pillow by using a neck roll). When electrodes react with this (NaCl + Lactic acid) sweat and paste changing electrode impedence.
Perspiration artifact can be reduced by drying the scalp with fan or alcohol, reducing the room temperature (by use of a cooling fan or air conditioning). The use of standard low-frequency filters generally reduces the amount of this artifact.

Physiological Movements

The artifact is caused by movement of the head electrodes in contact with the bed or pillow. Tremor, Myoclonic limb movements, nocturnal leg movements, hypnic jerks, causes movement of the head. Monitored by placing two electrodes in close proximity on the moving limb

NON-PHYSIOLOGICAL ARTIFACTS

Generated by recording electrode, equipment, environmental sources. Electrode artifacts related to poorly attached electrodes, high resistance, broken wire, changes in the lead-scalp interface, movement of electrode leads.

Instrumental and Environmental

Electronic noise : Generated by moving electrons within the recording amplifiers can be evident at high-gain settings.

50-cycle interference recorded in an EEG : Due to high-impedance electrodes. It affect the input circuitry of the amplifier. Also affect the common mode rejection. Use of higher input impedances of the amplifiers can reduce 50 cycle artifact. Reduce the electrode impedance in to < 5kohm May not be from the EEG instrument (also can come from environment) electromagnetic fields produced by equipment like AC, fan, Fluorescent lights. Presence of 50-cycle artifact in all channels - may represent a problem with electrical safety.

Capacitive, inductive, magnetic, and electrostatic artifacts:
Usually related–movement of electrode wires, input cable, AC power cable. Cause of capacitive artifacts is the cable acts as a capacitor (insulator of cables act as dielectric of the capacitor)

Electrostatic artifact: Static electricity stored on a variety of clothing and bedding manufactured of synthetic fibers. This voltage may be discharged by touching a metal bed rail, or even passed from person to person.

Drip artifact : Is due to drop of intravenous fluid falling near the electrode wires. It can reduce by use of micro drips instead of macro.

Ventilator Artifact: Single to multiple low – high voltage transients, Frequency of 2–40 Hz, Can mimic burst suppression pattern.Note each respiratory pump action on the EEG or observe the EEG by stopping the respirator for 20-30 sec.

Muscle artifacts vs 50 Hz artifact

Not regular, Not sinusoidal, Irregular in frequency and amplitude, Increase paper speed and count

Environment for artifact free recording: Quiet atmosphere,Comfortable bed, No synthetic carpeting, Convenient control of lightening, Proper shielding, Comfortable temperature and humidity levels to avoids sweating, tensions, restless. Keeping electrode leads close together, Move away from source (at least 10 feet)(since electrostatic induction is inversely proportional to the distance between the patient and source) Changing the orientation of the patient/source parallel to the electromagnetic lines of force. Locate the EEG lab at least 50 feet away from the AC feeders, transformers etc . One earth point for all equipments. Shielding the source of artifact and machine with earthed metal shield

EEG Activity

EEG activity can be broken down into 4 distinct frequency bands:

- Beta activity > 13 Hz
- Alpha activity 8 Hz-13 Hz
- Theta activity 4 Hz-7 Hz
- Delta activity < 4 Hz

Beta activity: Beta activity is a normal activity present when the eyes are open or closed. It tends to be seen in the channels recorded from the centre or front of the head. Some drugs will increase the amount of beta activity in the EEG.

Alpha activity : is also a normal activity when present in waking adults. It is mainly seen in the channels recorded from the back of the head. It is fairly symmetrical and has amplitude of 40 uV to 100 uV. It is only seen when the eyes are closed and should disappear or reduce in amplitude when the eyes are open.

Theta activity : can be classed as both a normal and abnormal activity depending on the age and state of the patient. In adults it is normal if the patient is drowsy. However it can also indicate brain dy$function if it is seen in a patient who is alert and awake. In younger patients, theta activity may be the main activity seen in channels recorded from the back and central areas of the head.

Delta activity: is only normal in an adult patient if they are in a moderate to deep sleep. If it is seen at any other time it would indicate brain dysfunction.
Abnormal activity may be seen in all or some channels depending on the underlying brain problem.

ATYPICAL BUT NORMAL WAVEFORMS DURING AWAKE

Lambda: Lambda waves have a triangular shape. They occur posteriorly and symmetrically Lambda occurs in the awake patient when the eyes stare at blank surfaces. They are normal waveforms and can occur. singly or in long or short runs.

MU activity : The mu rhythm is a central rhythm of alpha activity frequency (usually 8~ 10 Hz) in which the individual waves have an arch -like shape. The voltage characteristics of the mu rhythm resemble those of alpha rhythm. The mu rhythm does not block with eye opening, but blocks unilaterally with movement of opposite extrernity.

Posterior slow waves of youth : This activity was occurring most commonly in the EEGs of children 8 to 14 years of the age. They are frequently bilaterally syrtchronous and symmetrical. Posterior slow waves of youth attenuating or blocks with eye opening and disappears during drowsiness along with alpha rhy.thm.

THE FEATURES OF EEG DURING DROWSINESS AND SLEEP

The transition from awake to the drowsiness or stage I sleep, is marked by some profound changes in background activity of the BEG. The transition may be gradual or abrupt. The most prominent change is disappearance of the posterior dominant (alpha) rhythm. With the alpha rhythm gone, the background becomes dominated by theta activity, which occurs in generalized distribution, but is com.monly most prominent in central or frontal region.

Vertex waves

Also referred to as V waves or vertex sharp transients, this feature of EEG is most prominent on stage ll sleep. The waves are aptly named, as their focus lies in Cz, the vertex. When the waves are larger amplitude, they also ate picked up in the C3 and C4 electrodes. Their fields frequently spread to fronto central regions and sometimes even extended to parietal regions. V waves usually are biphasic, but occasionally may be triphasic as welL

F waves or frontal waves

Occasionally, a sharp transient like V waves appears on the frontal region without a corresponding wave present at the vertex. Such transient is referred as F waves. V waves and F waves have the same significance. They are normal features of stage II sleep.

K complex

The K complex is slow wave transient, seen in stage II sleep. The amplitude is generally maximum at the vertex. This is large amplitude wave. Sleep spindle immediately follow or associated the K complex. The K complex may last for nearly a second, but at times the duration can be large. The K complex can occur apparently spontaneously. They also can occur in response to sudden sensory stimulation such as unexpected loud noise in EEG laboratory.

Sleep spindles

These are the burst of very rhythmic activity at 11 to 15 Hz that seen in stage II sleep and early phase of stage II sleep. Sleep spindles are generally occur in widespread distribution. Commonly they are of higher amplitude in the central areas, sometimes an anterior dominance is noted. Sleep spindles in adults should be bilaterally and essentially symmetrical

POSTS

The positive occipital sharp transient of sleep (POSTS) are occur over the occipital regions on either side, being positive related to other areas, seen in stage II sleep. They occur singly or more cornmonly in runs. They are often bilaterally synchronous, but the sarne time they are asymmetrical on both sides.

The EEG in deeper stage of sleep

The stage m sleep is characterized by a background that consists of irregular and senilihythmic theta activity mixed with mostly rhythmic delta activity in this stage delta activity comprises more than 20% of the record. Occasionally, few sleep spindles are mixed with this activity.

The stage IV sleep is the deeper stage of sleep. The EEG contains high amplitude, rhythmic theta and delta activity. The delta activity which comprise more than 50% of the record.

REM sleep

The EEG during rapid eye movement (REM) sleep is different from the other stages of sleep. The background is paradoxica).ly similar to that seen in wakefulness with eyes open.

Paroxysmal epileptiform abnormalities

The term paroxysmal activity refers to activity that shows changes in amplitude or frequency which occur with sudden onset and offset and that stands out distinctly ftom the background activity. Paroxysmal abnormalities can be classified in to epileptiform abnormalities and periodic patterns.

Epileptiform Discharges

Spike discharges

A spike discharge is defined as a transient that is dearly distinguished from the background activity, has a pointed peak at a paper speed 30mm/sec./ and has a duration of 20 to 70 ms. It is usually surface negative and is of variable amplitude.

Sharp wave discharges

A spike discharge is defined as a transient that is dearly distinguished from the background activity, has a pointed peak at a paper speed 30mm/sec., and has a duration of 70 to 200 ms. Amplitude is variable, and like spikes, sharp waves usually surface negative.

Polyspikes or multispikes

Spike discharges are usually monophasic or biphasic. The term polyspike is used when several spikes comprise a single waveform. As with spike discharges, polyspikes discharges may also be accompanied by slow waves.

Spike and wave complexes

Spike and wave complex refers to the activity, which shows a spike or sharp wave is accompanied by a slow wave, usually of same polarity.

Focal epileptiform abnormalities

Interictal epileptiform abnormalities manifest themselves as focal spikes or sharp waves, or focal spike and wave discharges. Their presence helps to confirm the clinical suspicion of a focal or partial seizure.

Generalized epileptiform discharges

3 Hz spike and wave discharges

This pattern is classically seen in absence seizures. It consists of bilaterally symmetrical and synchronous complexes, each made up of a high amplitude spike and wave, surface negative spike and wave. The complexes repeat at a rate of 3 Hz and appear in a g.eneralized distribution, maximum amplitude on frontal and rarely in the occipital areas. At the onset of discharges, repetition rate may be faster (4Hz), whereas towards the end it becomes slower (2.5 Hz). They are precipitated in hyperventilation.

Generalized atypical fast spike and wave discharges

This pattern lacks the typical appearance .and repetition rate of 3 Hz spike. It consists of 3 to 5 Hz spike and wave discharges that show variations both in rate and morphology. The waveform may be mixture of spike and wave complexes and polyspike and wave complexes.

Generalized slow spike and wave discharges

This pattern consists of sharp and slow wave complexes occurring at a rate of less than 2.5 Hz. It also has been called "petit mal variant". The discharges are bilaterally synchronous and generalized, although fluctuating asymmetry is not uncommon. It may last for several seconds. This patttfm is the features of the diagnosis of Lennox - Gastaut syndrome.

Generalized polyspike and wave discharges

Generalized polyspike or polyspike and wave complexes may occur as an Interictal phenomenon in patent with primary generalized epilepsy (often myoclonous) and in Lennox- Gastaut syndrome. Sometimes the discharges may accompanied by myoclonic jerks.

Hypsarrhythmia

This pattern consists of continuous, high amplitude 1 to 3 Hz irregular, disorganized background activity and shifting spike foci. It occurs in most patients with infantile spasms. During sleep, the activity may become discontinuous.

Abnonnal periodic paroxysmal patterns

These are defined as stereotyped recurrences of paroxysmal complexes at relatively fixed intervals. They should be present throughout the entire tracing or a major portion if it. The discharges should stand out from the background.

Generalized periodic paroxysmal patterns

SSPE

The EEG in SSPE may be sufficiently specific to suggest the diagnosis. The periodic discharges consist of high amplitude (100 to 1000 uV) complexes - each consists of one or two slow waves - that recur at intervals of several seconds. The interval between the complexes may have a range of 4 to 14 seconds. Eaeh complex may last from 0.5 to 3 seconds. The activity is often fronto centrally dominant and may accompany the myoclonic jerks.

Jackob-creutzfeld Disease

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The periodic complexes consist of sharp waves of 100 to 300 ms duration that occur at much faster rate than those in SSPE. The interval remains constant in a given patient. Sometimes the complex have a triphasic configuration.

Herpes Simplex Encephalitis

The periodic discharges may be focal or liberalized to start with, but later they become generalized. The usual site is temporal or temporo frontal. The discharge consists of large sharp waves1 100 to 500uV in amplitude, having a duration of up to 1 second and occurring at a interval of 2 to 4 seconds.

Suppression Burst Pattern

This may be considerd as a periodic pattern, since it consists of periodic burts of . activity with intervals between in which the background activity is markedly attenuated to less than 10 uV. The pattern is indicative of severe encephalopathy and rnay result from cerebral anoxia, head trauma. and severe drug intoxication as well as deep anesthesia.

Triphasic waves

Triphasic wave may some tim~ occur in periodic fashion. Each waveform shows a prominant positive phase preceded and followed by negative phases. Classic triphasic waves show a time delay between their appearance in the frontal and occipital area. The wave are usually frontally dpminant, bilaterally synchronous and generalized.

Periodic discharges

Stereotyped activities in the form of complexes repeating in constant time intervals and persisting for a substantial period of the record

They are commonly classified as :

- ✓ Periodic lateralized epileptiform discharges (PLEDs)
- ✓ Bilateral independent PLEDs(BIPLEDs)
- ✓ Generalized periodic epileptiform discharges (GPEDs)
- ✓ Triphasic waves

High potential for SZs & convulsive / nonconvulsive status epilepticus, So authors recommended the use of AEDs to manage these discharges. Sometimes, these discharges are not epileptogenic in nature

PLEDS

- ✓ Term PLEDs was first used by Chatrian et al. in 1964, described as "periodic complexes occurring every 1-2 s, consisting of spikes or sharp waves followed by a slow wave."
- ✓ Kuroiwa and Celesia(1980) defined PLEDs as complexes which occur at approximately regular intervals and which persist for greater than 10 min or are continuous in a specific behavioral state.
- ✓ Recurrence frequency – once in every 0.5 to 4 sec (usually recur at least every 2 sec)
- ✓ Metronomic periodicity - the recurrence of discharges at constant intervals
- ✓ Stating periodicity as metronomic or relative bears no relation to any particular etiology
- ✓ **Unaffected by :** Sensory stimuli, HV, REM/NREM sleep, Eye opening, IPS
- ✓ **Increased in frequency with** – Drowsiness
- ✓ **BGA** – Normal / low voltage delta activity
- ✓ **Duration of complexes** – 100-400 msec, later in evolution they get prolonged
- ✓ Morphology varies over time
- ✓ Decreasing frequency of discharges over time
- ✓ Painful stimuli can result in an increase in the interval between the complexes
- ✓ Maximal on the side of the structural involvement
- ✓ 90% of the diagnosed PLEDs disappear within 4 weeks. (Schwartz et al. 1973)
- ✓ Chronic PLEDs described from 18 months to 20 years (Westmoreland et al 1986)
- ✓ Recurrent PLEDs also described in patients with TIAs and symptomatic epilepsy.

PLEDs – Etiologies

PLEDs are usually a response to an acute process. Stroke, infections, tumors, metabolic conditions –majority of causes.

Pathophysiology

A nonspecific result of acute partial and transient functional denervation in a localized area of the cortex Disappearing of PLEDs – cells would either Recover and respond normally or Die after a period of time, no longer responding.

PLEDs may be sub classified into :

PLEDs-proper and PLEDs-plus

Reiher's classification

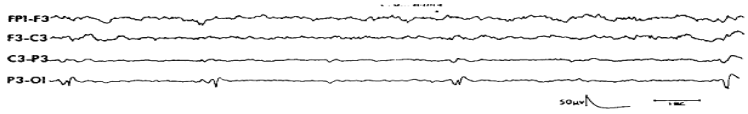
PLEDs proper (without rhythmic discharges)

- ✓ Class I - aperiodic, throughout
- ✓ Class II - metronomic, intermittent
- ✓ Class III - metronomic, throughout

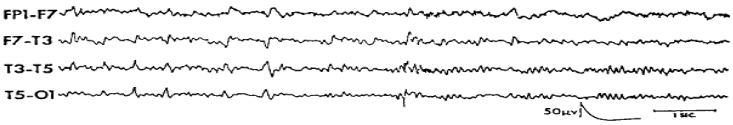
PLEDs plus (with rhythmic discharges)

- ✓ Class IV - brief RDs < 1 sec
- ✓ Class V - prolonged RDs

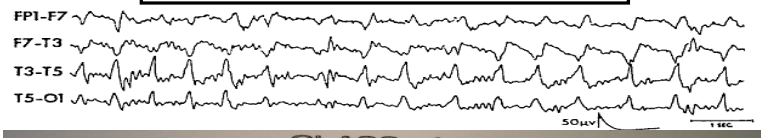
Class I - aperiodic,

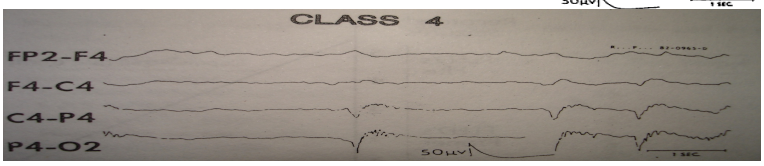


Class II metronic, intermittent

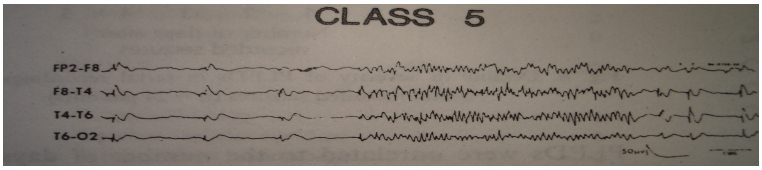


Class III -





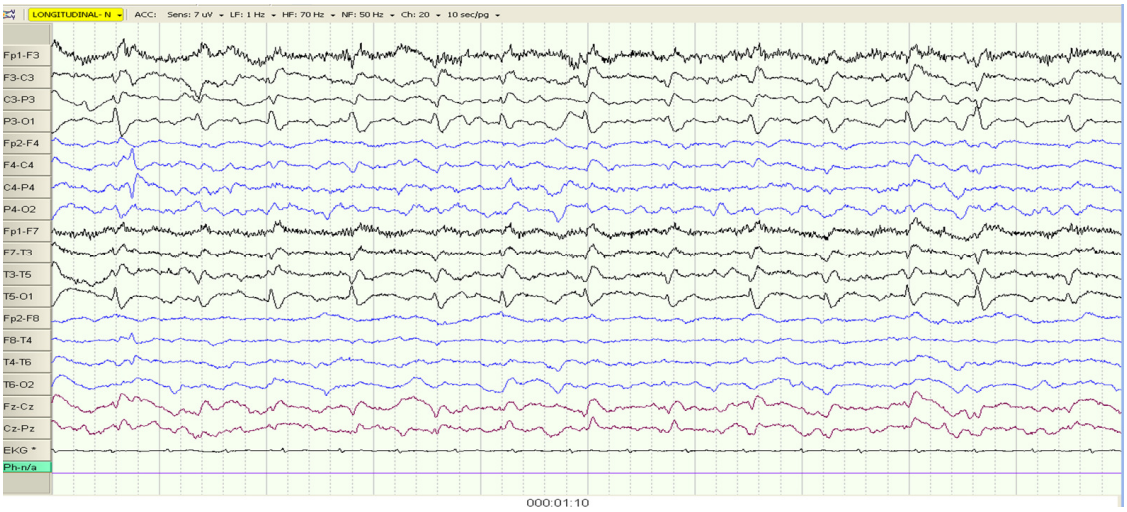
brief RDs < 1 sec



Class V - prolonged RDs

NOTE :

EEG Typically fluctuating b/w PLEDs-proper and PLEDs – plus, indicating high seizures risk , PLEDs plus are less common in chronic lessions



PLEDs - Evolution

With time PLED Plus evolve into periodic PLEDs (class 2 and 3) & then into aperiodic PLEDs (class 1). This transformation is also accompanied by a decreasing frequency of clinical seizures. Usually disappears with in 2-3 weaks. Discharges → ↓ Amplitude, ↓ Repetition rate. PLEDS may change to seizures, Isolated high voltage slow waves with delta or theta activity, Sporadic spikes or sharp waves, Hypsarrhythmia in infants. May persists as chronic PLEDs, Recurrence of PLEDS – Recurring pathology e.g. TIA.

NOTES

- ✓ Obtundation in 95% of PTs
- ✓ Focal seizures & focal neurological signs - 80%
- ✓ Epilepsia partialis continua – 30%
- ✓ SZs are not usually generalised
- ✓ 20% - comatose
- ✓ 80% - impaired consciousness
- ✓ Prognosis depends on the underlying etiology
- ✓ The worst prognosis noted for acute severe stroke

Bilateral and independent Periodic lateralized epileptiform discharges

BIPEDs

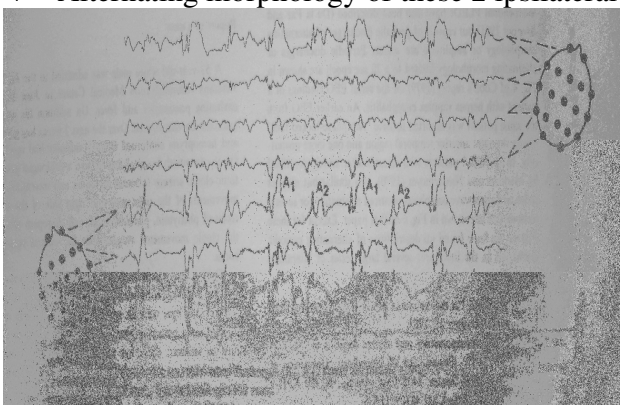
- ✓ Seen in both hemispheres

- ✓ Independent
- ✓ Asynchronous
- ✓

Pattern is less common than PLEDs, Highly associated with SZs, Typically associated with acute structural lesions, with or without metabolic disturbances

Cerebral Bigeminy

- ✧ 2 separate foci are present in the same area of an hemisphere
- ✧ Alternating morphology of these 2 ipsilateral PLEDs



Multifocal PLEDs

- ✧ 3 or more independent foci of PLEDs located over both hemispheres.
- ✧ 3 foci are also called TriPLEDs.
- ✧ Detection may be facilitated by use of Laplacian montages
- ✧ Reflect severe brain dysfunction and are associated with a significant mortality rate

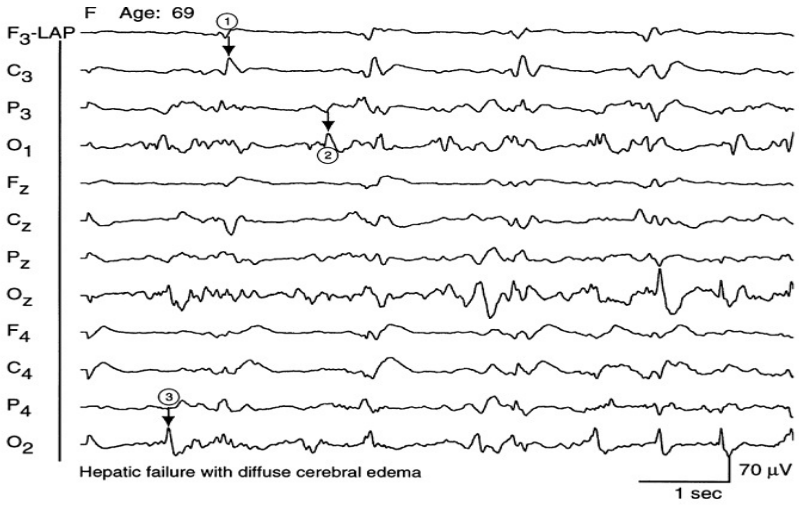
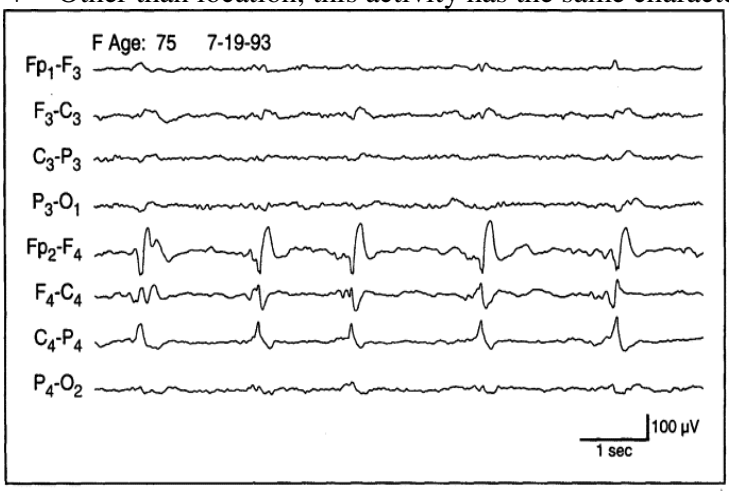


Fig. 1. Typical example of multifocal PLEDs (arrows) using the Laplacian montage, recorded from a 69-year-old woman with liver failure, sepsis, and hypoxia. Focus 1 (C_3) occurring every 1.5 s; focus 2 (O_1) occurring every 0.5–1 s; focus 3 (O_2) occurring every 0.5 s.

From Lawn et al. Clin Neurophysiol 111:2125-2129, 2000.

PEDIMs

- ✧ Periodic epileptiform discharges in the mid line.
- ✧ Association with underlying stroke & SZs
- ✧ Other than location, this activity has the same characteristics as PLEDs.



Benign EEG variants and patterns

Benign patterns with 'epileptifonn morphology abnormalities'

Small sharp spikes/BSSS

Small sharp spikes (SSS) the original name also have been referred as 'benign sporadic sleep spikes'(BSSS) and 'benign epileptiform transients of sleep' (BE'TS); The BSSS are mainly seen in adults during arowsiness and light sleep. They are usually of low amplitude, usually less than 50uV and of short duration, less than 50 ms. The waveform consists of single monophasic or biphasic spikes with abrupt ascending limb and steep ascending limb. The BSSS may. have a single aftercoming slow wave component or may be associated with dip in background. They are best displayed in the temporal artd ear leads.

Wicket spikes

Wicket spikes occur either in brief runs or as isolated transi.ents. It look like an arch. they are not accompanied by a slow wave. They occur over the temporal areas and may be unilateral or bilateral. Wicket spikes are seen best during stage I and II sleep, although they may rarely occur on wakefulness.

14 and 6Hz positives spikes

The pattern consists of bursts of comb shaped waves having a frequency of 13 to 17Hz and/or 5 to 7Hz. The pattern is seen generally over the posterior temporal region and adjacent areas on one or both sides during the sleep. The sharp peaks of the wave are surface positive.

6 Hz spike and waves (Phantom spikes)

1hls pattern consists of brief burst of 5 to 7 Hz spike and wave .complexes. The spikes are of very small in amplitude and much smaller than the waves. The discharges, which are usually bilateral with frontal or occipital dominance, may occur during wakefulness and sleep, bit it is seen mostly in drowsiness.

Rhythmic patterns

Rhythmic Midtemporal Theta of Drowsiness (Rmtd)

This pattern consists of runs of sharply contoured theta activity. It may last for several seconds without significant change in frequency of the waves. They occur mainly in midtemporal area, although the activity may spread to the more anterior and posterior temporal areas and to the parietal regions.

Subclinical Rhythmic Electroencephalographic Discharges of Adults (SREDA)

This pattern is reported to occur mostly during drowsiness in adults. Often the discharges starts with a sharp or slow wave of high amplitude and is followed by build of sharp waves to a sustained frequency of 4 to 7 Hz. The activity may last for several seconds to minutes and end abruptly. The most common areas to involved includes temporal and parietal regions

EEG Recording

The EEG recording can last from anything between 15 minutes to 1 hour or longer depending on the situation. Typically the patient will be lying down or sitting relaxed in a chair. Most of the recording is taken with the eyes closed, although the patient will be frequently asked to open them for short periods,

Most patients will be asked to carry out a period of deep breathing for approximately 3 minutes. This may produce some abnormal activity, which would not be seen while· the patient is relaxed. The physiological effect of deep breathing is to increase the amount of carbon dioxide (C02) being removed from the bloodstream. This fall in C02 produces a fall in blood pressure and at the same time blood vessels in the brain become constricted. This reduces blood flow and the delivery of oxygen and glucose to the brain. This in tum may produce some abnormal brain activity not seen in the resting record;

Photic stimulation is also carried out. A strobe lamp is placed 30cm from the patient's eyes. Brief flashes of light (2-5 seconds in duration) at a number of different flash frequencies are delivered to the patient with both eyes open and eyes closed. A continuous flash with increasing and decreasing flash frequencies is sometimes used. Some patients who are sensitive to flashing lights may show abnormal activity in. the EEG.

Throughout the test the technologist is constantly annotating the record with any patient movements, or tasks that they are carrying out. Other signals may also be recorded in conjunction with the EE;G such as- heart rate (ECG), respiration, eye movements (EOG)

EEG Analysis

The EEG reports consist of a number of different sections. The technologist may prepare a report describing the type of activity seen in the record together with changes produced by deep breathing and photic stimulation. They will also comment on the patient's state during the recording. The physician will then interpret these changes with regard to the medical problem being investigated.

With an increase in the number of long recordings being carried out, many departments make use of detection algorithms such as spike and seizure detection. Although it is still necessary for the clinician to review the complete record, such programs will mark and highlight sections of interest. The most efficient method of implementing these algorithms is for the detection to be carried out on-line.

Other methods of analyzing EEG data include Power Spectrum Analysis. A Fast Fourier Transform (FFT) is performed on sections of EEG data to determine the power content of t.la.e four main frequency bands. The resulting waveforms can be displayed as a brain map)' which will show the scalp distribution of the power within each frequency band. The amplitude of the different waveforms at a single point can also be displayed in a similar format. This type of display provides a more objective analysis of the EEG activity compared to a subjective visual analysis by a physician.

ACTIVATION PROCEDURES

Various activation procedures can elicit or enhance certain normal as well as abnormal activity in the EEG. The following are the most commonly used activation procedures performed in most of the laboratories.

Hyperventilation

Hyperventilation is perhaps the most widely used activation procedures in EEG laboratories. This is a very simple and relatively safe procedure, consisting of three to five minutes deep breathing at the rate of about 20 per minute. During hyperventilation technologist should carefully observe the patient to detect the occurrence of absence seizure.

The normal response to the hyperventilation consists of the occurrence of symmetrical slowing on both sides. Although this slow activity may be diffuse slow wave activity. The major biochemical influence of hyperventilation is a drop in carbon dioxide content of the blood (hypocarbia). It leads to vasoconstriction. This presumably alters the metabolic rate of the neurons and leads to the slow activity.

The most striking EEG abnormality seen during hyperventilation is the 3 Hz spike and wave discharges often brought on in patients with absence seizures. It is preferable to avoid in patients with recent stroke or subarachnoid hemorrhage, myocardial infarction, chronic obstructive pulmonary disease and other conditions causing difficulty in breathing. Sometimes, other type o£ epileptiform abnormality, such as generalized spike and wave discharges or even focal spikes# may be brought on by hyperventilation

Intermittent Photic Stimulation

Visual stimuli are perhaps one of the most effective means of stimulation of the brain. Stroboscopes otherwise know as Photic stimulators are used for the purpose. Single or continuous bright flashes of light at frequencies ranging from 1 to 50 flashes per second are used.

Flashes of duration 10 micro second with an intensity of 1.5 million foot candles are routinely given. The flash lamp is positioned 30cm in front of the eyes. Each flash is presented for duration of 10 seconds, and the eyes are kept closed in first 5 seconds and open in the next 5 seconds.

Photic stimulation is most valuable in documenting photosensitivity, which has a correlation with primary generalized epilepsy. The flash lamp is positioned 30 em in front of the eyes. Each flash rate is presented for a duration of about 10 seconds, and the eyes are kept closed in the first 5 second and are kept opened in the next 5 seconds .. The IPS should be stopped if photo paroxysmal responses are obtained in order to avoid the of seizure.

Sleep deprivation and Sleep

In the last several years sleep recording have become routine procedure in many EEG laboratories for eliciting epileptiform abnormalities. The augmenting effect of sleep is both in generalized as well as in focal epilepsies, especially in patient with temporal lobe foci. In the majority of patients with epilepsy a generalized epileptiform discharges may become may be evident only during sleep. A dramatic increase in spike discharges during drowsiness and light sleep is a characteristic feature of benign rolandic epilepsy.

Pharmacological Activation

A number of pharmacological agents have been used to induce epileptiform activities in patients with seizure disorder, the purpose being to determine whether one is dealing with a primary generalized or a focal onset seizure. It is not commonly employed in routine EEG although it finds its place in Electrocorticography.

EEG Applications

One of the major roles of EEG is as an aid to diagnose epilepsy. Abnormal patterns such as spikes, sharp waves and/or spike and wave complexes can be seen. The type of activity and the area of the brain that it is recorded from will assist the physician in prescribing the correct medication for that type of epilepsy.

Patients with epilepsy that cannot be controlled by medication will often have surgery in order to remove the damaged tissue. The EEG plays an important role in localizing this tissue. Special electrodes can be inserted through the cortex or alternatively a grid of electrodes placed directly on the surface of the cortex. These recordings, often called Long Term Monitoring for Epilepsy (L TME), can be carried out for periods ranging from 24 hours to 1 week. The EEG recorded will indicate which areas of the brain should be surgically removed.

EEG studies can also be used in patients who are deeply unconscious, to distinguish between brain death and possible reversible conditions.

Electro cerebral inactivity (ECI) or electro cerebral silence (ECS) is defined as no BEG activity over 2 }-l V in amplitude when recording from electrodes on the scalp that are 10 em or more apart.

Using the 10/20 International System of electrode placement, the average distance between electrodes in an adult is 6 to 6.5 em. Activity recorded using these distances and at a normal display sensitivity may suggest ECS. However if the some activity was recorded using longer inter-electrode distances, some· activity might be seen. Therefore some double distance electrode linkages are recommended for example FP1-C3, F3-P3, and C3-Q1 etc.

Display sensitivities of a minimum of 2 uV/mm are required. However digital EEG systems have the added advantage of having sensitivity values of 1.5 and 1uV/mm. This 50-100 % increase in sensitivity will allow a more confident assessment of the presence or absence of a 2uV signal The EEG is also used to investigate other conditions that may affect brain function such as strokes, brain injuries, liver and kidney disease and dementia.

EEG MONITORING IN INTENSIVE CARE SETUP

EEG MONITORING IN INTENSIVE CARE SETUP

EEG provides a unique way of monitoring cerebral function in the intensive care unit and it is useful both for recording short and long acting events. Typically, epileptic spikes may last for a fraction of seconds, but recordings for many hours, for example during sleep cycles may also give important information. Recordings lasted for many days also be useful, to reflect slow trend changes in the EEG.

One basic requirement for an EEG monitor in the ICU is that has to present the original EEG on a monitor display and also has to provide a paper printout. Another additional benefit would be to have the system continuously store signals. This would allow access to previously stored EEGs for inspection if unexpected, rapid .changes, such as seizures, should occur.

It would be beneficial for the ICU clinician's daily work to understand the tasks of EEG. It would be even better if the clinicians were able to identify typical EEG patterns such as physiological activity, pathologically slowed rhythms, seizure activity in its various forms, periodic patterns, and burst suppression. It discusses the utilization of the EEG monitoring comatose patients in the ICU .The selected aspects include reactivity, periodic patterns, and burst suppression that may characterize an EEG in the ICU.

Recording of an EEG should be utilized more in the monitoring of comatose ICU patients. The BEG recording, repeated at intervals, can help with broad diagnostic categorization. In special clinical situations, for example in monitoring of the effectiveness of status epilepticus treatment, continuous monitoring of EEG could be deemed obligatory. In the assessment of the clinical importance of EEG patterns, clinical picture, age, etiology, acuity, and the integrity of the brainstem reflexes must be taken into consideration.

Another factor is that special EEG patterns, like triphasic waves, may suggest Metabolic disturbances, which could because by liver dysfunction or toxic substances. From clinical and neuropsychological point of view, it is important to test reactivity of BEG. In general reactivity can be considered as a feature of the lighter stages of coma. However, even .during deep anesthesia, with EEG at the burst suppression level, reactions to minor somatosensory, auditory, or visual stimuli can sometimes be seen.

Prolonged Video EEG Monitoring

Introduction

Video-electroencephalography (VEEG) monitoring provides long-term recording of the electroencephalogram (EEG) and time locked video of the patient in a dedicating recording room. It is the most definitive method for the differentiation of seizure versus nonepileptic events, classification of seizure types and localization of seizure onset. The clinical features helpful in distinguishing between temporal and extra temporal complex partial seizure. VEEG has an important role in the pre -surgical evaluation of patients with medically refractory epilepsy.

Scalp disk electrodes placed according to 10/20 system of placement. Special electrodes may be placed when indicated (e.g.: anterior temporal electrodes, sphenoidal electrodes) At least 16 channels of EEG and one channel of EKG should be monitored. The events can be detected by patient's caregivers triggered an event signal, a trained EEG technologist who visually scanned the VEEG or a computer equipped spike and seizure detection programme. Samples of inter ictal EEG and events are identified and then reviewed.

Analysis of some paroxysmal events may suggest alternative diagnosis-including e.g.; syncope, psychogenic seizures, sleep disorders, paroxysmal movement disorder and other causes of episodically distributed behavioral surgical management.

Recording method

Facilities

A room, close friends or relatives, medical team

Duration of monitoring

3 to 7 days

Scalp electrodes

10-20 international electrode placement is used T l and T2 electrodes located below F7-F8 or T3 -T4 positions. Some used T l &T2 electrodes to differentiate activity arising from the focus rostral to the sylvian fissure in the frontal lobe and an anterior temporal source. The maximal field of epileptiform activity in patients with complex partial seizure can be seen.

Ictal SPECT

The Single Photon Emission Computed Tomography (SPECT) is a nuclear medicine tomographic imaging technique using gamma rays. SPECT shows distinctive and rapidly changing blood flow pattern at the time of seizure. The use of SPECT in epilepsy centers on localization of the epileptogenic zone usually performed so that surgical treatment can be carried out. Most work has been carried out using tracers of regional cerebral perfusion. Early work concerned the iriterictal period, but increasingly postictal and ictal studies are being carried out.

Usually the gamma-emitting tracer used in functional brain imaging is 99mTcHMP AO (hexamethyl propylene amine oxime). 99mTc is a metastable nuclear isomer which emits gamma rays which can be detected by a gamma camera. When it is attached to HMP AO, this allows ^{99}mTc to be taken up by brain tissue in a manner proportial to brain blood flow, in turn allowing brain blood flow to be assessed with the nuclear gamma camera.

Neurotechnologist's role in ictal SPECT

The tracer is injected to the patient for an ictal SPECT, immediately at the onset of the seizure. Some patients have electrographical onset, before the clinical features begins. So the procedure should be done durintg VEEG monitoring. As the seizure begins the technologist give instruction to the nurse for injection of the tracer.

Sphenoidal electrode placement

These electrodes are used to record discharges from the anterior tip of the temporal lobe. Electrodes are usually made from thin, straight insulated stainless steel wire about 50 mm long and 0.5 mm diameter, with a small uninsulated ball at the tip. Sphenoidals are introduced via a needle cannula into the temporal and masseter muscle with insertion point between zygoma and sigmoid notch of the mandible. Penetration is directed slightly anterior so that the tip rests lateral to the foramen ovale at the greater wing of the sphenoid. Infection is risk but very rare. Sphenoidal electrodes wire can stay for several days for prolonged recording. The activity from the mesial and basal temporal cortex can be recorded without too much artifact.

Invasive monitoring

Patients may require invasive monitoring when the results of noninvasive methods such as scalp EEG, VEEG and MRI are conflicting. Placing sphenoidal electrode under fluoroscopy directly below the foramen ovale resulted in better detection of interictal and ictal epileptiform activity of mesial. Basal-temporal origin. Several types of intracranial recording_ electrodes. Such as subdural strip and grid electrodes, epiduralelctrodes, intracerebraldepth electrodes or combination of each are used.

Depth electrodes

Depth electrodes are arrays of electrodes designed for introduction directly into the substance of the brain by a neurosurgeon. They are used to detect and localize activities not visible by scalp EEG recording. Depth electrodes are composed of fine array of thin stainless steel, platinum or gold insulated wire of different ending in insulated tips. The depth electrodes are implanted stereotactically, free hand, or with x ay guidance, under sterile protocol. Electrodes are remaining in place for several days or weeks. Amygdala, hippocampus, orbitofrontal cortex and supplementary motor areas of frontal lobe are the popular placement targets.

The depth EEG clearly increases the ability to detect and localize epileptiform activity in selected patients, but it has disadvantages. All the brain sites cannot be studied with this technique, resulting in possible sampling error.

Subdural electrodes

Subdural electrodes are designed to be in contact with cortex of awake, behaving patients for a few days or weeks. The goal of subdural recording is localizing the seizure foci in relation to important functional areas of the brain. Areas of the cortex involved in sensorimotor, speech, reading, or cognition are identified by stimulation. In order to study a large area of the cortex, subdural grids may be assembled in an approximately hand - sized array, with up to eight rows and eight columns of the electrodes. These electrodes are usually 3 mm disks fabricated from stainless steel or platinum. Electrodes are embedded in a sheet of flexible plastic, with center to center electrode separation of 1 mm. grids may be cut to size during implantation.

Epidural electrodes

Epidural electrodes in single or double row strips are less invasive than subdural grids (they can be placed through a burr hole) and often provide important information about seizure foci. Because a smaller area is covered, the

true foci.Because a smaller area is covered, the true focus is likely to be in the field of recording.

ELECTROCORTICOGRAPHY

Electrocorticography

It is one of the first important applications of intraoperative recording of cerebral electrical activity; later, depth recordings were made acutely during the course of epilepsy. Electrocorticography during epilepsy surgery has been performed· for several years. In particular is this tnte for the tailored resections both temporal and extra temporal in order to identify the epileptogenic zone. Techniques for preoperative localization of motor and sensor gyri with electrical cortical stimulation and SEP recordings with subdural strip electrodes have been developed and are also used in the resection of intracranial tumors close to these eloquent cortical regions.

Preoperative nerve stimulation is used in the neurosurgical reconstruction of peripheral nerve and plexus brachialis lesions. SEP recordings in patients operated for tethered spinal cord, scoliosis or intraspinal tumors monitor the spinal cord function. The method of preoperative electrical cortical stimulation in spinal cord monitoring will be introduced shortly. Dorsal root stimulation is used during the dorsal rizotomy procedure to reduce spasticity in children with cerebral palsy and spastic diplegi. The function of the facial nerve is monitored in the resection of acoustics neurinoma.

POLYSOMNOGRAPHY

POLYSOMNOGRAPHY

Introduction

A polysomnogram consists of a simultaneous recording of multiple physiologic parameters re1ated to sleep and wakefulness. The interaction of various organ systems during sleep and wakefulness is also evaluated.

Polysomnography (PSG) is used to evaluate abnormalities of sleep and/or wakefulness and other ·physiologic disordets' that ·have-an impact on-or -are' related to sleep and/or wakefulness.

By international standards, a polysomnogram must have a minimum of 4 neurophysiologic channels.

• One electroencephalography (EEG) channel (central with an ear reference provides the best amplitude) to monitor sleep stage
• Two electrooculogram (EOG) channels to monitor both horizontal and vertical eye movements (electrodes are placed at U1e right and left outer canthi, 1 above and 1 below the horizontal eye axis)
• One electromyography (EMG) channel (usually chin or mentalis and/or submentalis) to record atonia of rapid eye movement (REM) sleep

Other parameters often monitored include the following:

• Additional EEG channels, particularly in patients with sleep-related epilepsy
• Additional EMG channels, particularly anterior tibialis, to detect periodic limb movements of sleep
• Airflow
• Electrocardiography
• Pulse oximetry
• Respiratory effort
• Sound recordings to measure snoring

Optional parameters include the following:

• Continuous video monitoring of body positions
• Core body temperature
• Incident light intensity
• Penile tumescence
• Pressure and pH at various esophageal levels

In 1992, the Office of Technology Assessment of the Agency of Health Care Policy and Research recommended, in an evidence-based assessment.Two tests have been studied sufficiently. Both tests are performed in a sleep laboratory.

• Overnight polysomnography (PSG) is an overnight recording of the patient's sleep.
• Multiple sleep latency testing (MSLT) records multiple naps throughout a day.

Standard sleep studies usually include both tests, PSG (may be performed over several nights} followed by MSLT the next day. Limitations usually stem from the fact that recording conditions may not reflect what happens during a regular night in the patient's home.

Although diagnosing a sleep problem on the basis of a recording over a single night is common practice, some authorities caution that more than 1 night of recording may be necessary, so the patient may become comfortable with unfamiliar surroundings and sleep more naturally. This effect is greatest on the first night in the sleep laboratory ("first night effect").

Sporadic events may be missed on a 1-night PSG. External factors that disturb the subject's sl~p may be present in the home but absent from the controlled environment of the sleep lab. Patient preparation is important so that the patient sleeps naturally. Patient instructions include the following:
• Maintain regular sleep-wake rhythm
• Avoid sleeping pills • Avoid alcohol
• Avoid stimulants, including medications for narcolepsy
• Avoid strenuous exercise on the day of PSG testing

High costs and long waiting lists have prompted the exploration of alternative methods of evaluation. Although the following studies may have usefulness in specific clinical situations, Bloch concludes that their role compared to conventional sleep studies remains controversial.
• Ambulatory monitoring with portable equipment
• Daytime PSG

Simplified sleep studies with limited subsets of monitored parameters Automatic, computer-based systems often are employed in clinical and research settings. However, standard analysis still consists of tedious and time-consuming review and scoring of either paper tracings or recordings projected on a computer monitor.

Overnight parameters (e.g.: times of lights on/off, total time in bed, total sleep time) are collected. The overnight recording is divided into epochs of approximately 30 seconds. The standard EEG, EMG, and EOG recordings are evaluated, and the predominant stage of sleep then is assigned to the entire epoch.

Total time and relative proportion of the night spent in each of the 6 stages and in REM and non-REM sleep are calculated. Latencies to REM and slow-wave sleep (SWS) are reported.

Special neurophysiologic events (e.g.: epileptic events, intrusion of alpha into sleep, periodic activity of tibialis anterior) are reported. Respiratory activity (e.g., apneic or hypopneic episodes, oxygen saturation) is correlated with sleep stages. Other parameters such as body position, gastro esophageal reflux, bruxism, and penile tumescence are recorded. If a sleep apnea syndrome is diagnosed, a trial and titration of continuous positive airway pressure or a trial of an oral appliance may be undertaken, either in a partial-night or second-night PSG recording.

Dysomnias (disorders of initiating or maintaining sleep)

• Circadian rhythm disorders
• Narcolepsy
• Idiopathic hypersomnia
• Inadequate sleep hygiene

- Sleep-related respiratory disorders
    - Sleep apnea syndrome
    - Upper airway resistance syndrome

Parasomnias

- Disorders of arousal
- Disorders of sleep-wake transition
- Disorders that occur during REM sleep
    - Nightmares
    - REM behavior disorder
- Medical-psychiatric sleep disorders
    - Medical ~ Sleep-related asthma
    - Psychiatric
        - Depression
        - Panic disorder
- Neurologic- Sleep-related epilepsy
- Others
    - Bruxism

Restless legs syndrome and periodic limb movement disorder

NERVE CONDUCTION STUDY

Introduction

Nerves control the muscles in the body using electrical impulses. Whenever a. muscle contracts (tenses up) in response to a signal from the brain, it produces an electrical discharge of its own. Electromyography (EMG) measures the electrical discharges made by the muscles. Nerve conduction studies measure how well individual nerves car£ transmit electrical-signals.

Measuring the electrical activity in muscles and nerves can help detect the presence, location and extent of diseases that can damage muscle tissue (such as muscular dystrophy) or nerves (such as amytrophic lateral sclerosis). In the case of nerve injury, the actual site of nerve damage can often be located. EMG and nerve conduction studies are often done together to provide more complete information.

The myelinated nerve axon conducts impulses in a saltatory fashion i.e. depolarisation occurs at the nodes. The currents are prevented from penetrating the membrane between the nodes in the normal nerve due to an isolating myelin sheath. This means that the impulse propagation is much faster than if there was a continuous depolarisation. The conduction velocity is also dependent on the axonal diameter and the properties of the membrane. A normal axon conducts with a speed of 35-60 m/sec. The velocity is reduced if the myelin is defect due to pathological changes, if the ion- channels at the nodal areas are blocked or if the axon diameter is smaller than normal. It is also dependent on temperature.

When the correlation between neurophysiological and morphological parameters was established, the nerve conduction study become an important method used in, clinical routines [14; 15; 18;20;26;31]. Such studies are performed in most EMG laboratories since the 1960's and have since then become more sophisticated, sensitive and specific.

This summary will give a brief update of the nerve conduction studies, which are performed in clinical routine today.

General guidelines for performing nerve conduction studies

Before attempting nerve conduction studies on patients one must consider a number of factors. First, the patient must be aware of what to expect. In addition the tester must prepare the anatomical part to be tested and must understand the potential problems that could arise.

1. Explain the procedure to the patient. It is important to explain the procedure to the patient in the simplest language to make the patient less anxious and more co-operative.
2. Place the limb to be tested in a relaxed and comfortable position both for the patient and for the examiner.
3. The stimulating and recording electrodes should be used as per the guidelines.
4. Measure the skin temperature to avoid errors. Motor and sensory nerve conduction velocities decrease at the rate of 1.3 to 2.4 m/sec per degree drop in skin temperature. During the nerve conduction studies the skin temperature is kept at 31 to 34 degree Celsius whenever possible or else the correction should be introduced.
5. The electrode impedance should be kept at minimum to avoid unnecessary artifacts. 6. A ground electrode should be attached to the lead being tested and is ideally placed between the stimulating and the recording electrodes
7. Pediatric stimulating and recording electrodes are used in infants.
8. Motor nerve conduction studies are performed prior to sensory conduction study to locate nerves.
9. The patient should be warned before giving each stimulation.
10. The pain should be reduced as much as possible by adjusting the intensity and duration of the stimulus, but still within the supramaximal range

Factors influencing nerve conduction parameters

Temperature

The temperature affects the conduction velocity (CV), both locally at the recording site and generally along the nerve.

Locally the amplitude increases as the temperature in the recording site decreases. The amplitude incr~ases by 1. 7% per degree Celsius

The temperature also affects the conduction along the nerve segment. The CV decreases as it cools with a factor ranging from 1.2 to 2.4 m/s per degree Celsius. This varies for different nerve [8]. This will reduce the amplitude. These two effects of the temperature on the amplitude neutralize each other. In order to standardize CV and amplitude measurements, it is recommended to keep the skin temperature at above 29° C for the dorsum of the hand and 27° C for the dorsum of the foot.

Age

Conduction velocity is age dependent. Full term infants have conduction velocities, which are approximately half of that seen in adults. Conduction velocities rapidly increase from the values recorded in infants to near adult values at around 3-5 years of age. Furthermore pre-term infants have slower values at around 14-28 m/s. In the teens conduction velocities are almost the same as those of adult values

After the second to fourth decade, conduction velocities start to decrease very slowly. cv decrease by o.5-1.8 m/s for each decade

Length of segment and height

Longer nerves generally conduct more slowly than shorter nerves [5]. It has been shown that there is a good correlation between CV and estimated axonal length in the peroneal and sural nerves, but not in the motor or sensory fibers of the median nerve. Based on a good correlation between the height of the patient and the length of the nerve, the CV in lower limbs decreases by 2-3 m/s forlO em increase in height. Nerve impulses propagate faster in the proximal than in the distal nerve segments.

Gender

It has been reported that CV is slower in women than that in men, but the correlation is complex since gender and height are not independent of each other. In our routine, we use the same reference values for women and men.

Reference values

With standardized methods, the technique is sufficiently reproducible to allow the transfer of reference values from one laboratory to the other. A number of techniques and related reference values are given in the literature.

Pathophysiology

The principle changes in nerve function are related to demyelination, axonal degeneration and conduction block. There are no absolute dividing lines between these situations; they show some overlap and also dynamic changes from one stage to another due to the interaction between Schwann cells and the axonal condition. In cases of demyelination, the conduction velocity is reduced. In cases of axonal degeneration, there may be normal velocity in the remaining axons, but a weaker muscle response is evoked. In cases of conduction block, no axonal degeneration occurs and therefore a normal response is obtained when stimulating distal to the lesion. When stimulation is performed proximal to the site of abnormality, a reduced number of axon conducts impulses, and a smaller than normal muscle respc:mse is obtained.

PROCEDURES

Motor Conduction Studies

Recording electrode

For motor conduction studies (MCS), recording is formed over the belly of the muscle using a surface electrode. The active recording electrode· is placed· over the endplate zone of the muscle in order to record muscle activity at the moment of depolarization after the nerve impulse has arrived at the endplate. The muscle response, obtained after nerve stimulation is called the compound muscle action potential, CMAP, should have an abrupt negative take-off. If the electrode is away from the endplate, it will show an initial positive phase corresponding to the approaching electrical field of the impulses in the individual muscle fibers. The start of the positive phase corresponds to the start of the depolarization. However, because of the gradual increase in amplitude from the baseline it may be difficult to determine the exact start. If later part of the CMAP is used for latency measurements, the latency value will be contaminated with some conduction time along muscle fibers and not express only the nerve conduction time. However, when comparing distal and proximal latencies for CV calculations, other points than the take-off can be used, as long as the same parts of the CMAPs are used. It should be mentioned that in some situations, particularly in recording from the abductor digiti minimi (ADM) muscle of the foot in the study of the tibial nerve, underlying muscles with different positions of the endplate zones would contribute to the recording and give rise to positive components. If possible, measurements should be made to the point where the signal leaves the baseline

Reference electrode

The reference electrode should be placed in such a way that no recordings are taken from the muscle under study. If the reference electrodes are too close to the muscle, e.g. in its tendon, it will contribute with a significant amount of activity. Therefore, more distal positions are preferred, e.g. over the distal interphalangeal joint (from the thumb for the median nerve, dig V for the ulnar nerve, toe one for the tibial nerve and toe V for the peroneal nerve).

Stimulation

Usually stimulation is performed at two or more sites along the nerve. It is not adequate to stimulate at only one point and calculate the conduction velocity from the obtained latency between the stimulus and motor response and the distance. This is because the conduction time includes the slower in the conduction in the last segment of the nerve, time in the neuromuscular junction, and possibly some conduction along muscle fibers. Therefore, two stimulation sites well separated from each other along the nerve are used.

In situations of local nerve lesion, short segments should be tested in order to localize the site of the abnormality. This technique has been called "inching" using small distance between stimulation' points.We often use a distance· of 10mm ("centimetring"). The recording site is kept constant, and the stimulation is performed

every 10 mrm across the area of suspected lesion. The analysis should focus on sudden jumps with prolonged latency values with more proximal stimulation or abrupt drop in amplitude.

Sometimes somewhat longer segments of the nerve are studied as part of the routine. In ulnar nerve studies, stimulation is often made at the wrist, and above and below the elbow. In studies of the peroneal nerve, stimulation is made at the ankle, and below and above the fibula head. In cases of difference in conduction along the two segments, centimetring is performed.

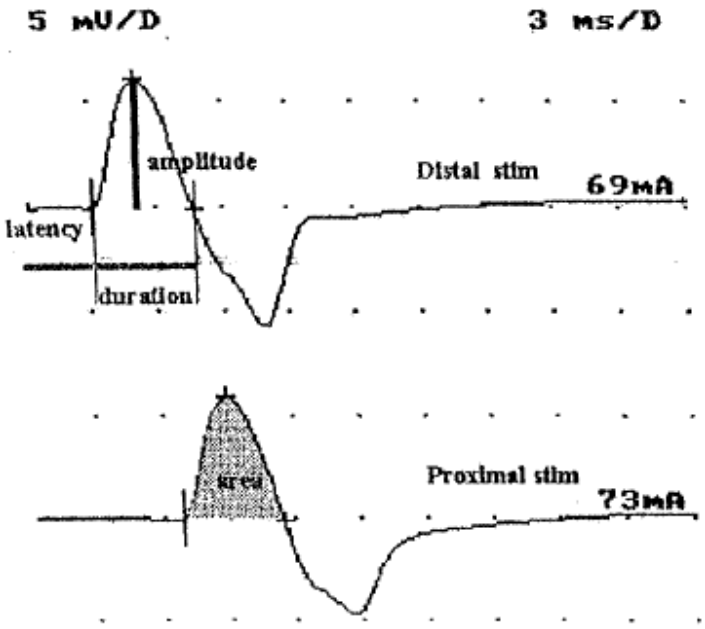
Generally surface electrodes (felt pad or steel) are used in motor nerve conduction studies. They are fixed on to a plastic bar a fixed distance apart. In a few s~tuations it is preferable to use needle electrodes. A pair of monopolar needle electrodes is used these cases. Alternatively a surface electrode is used as an anode.

It is necessary to use needle electrodes when stimulating a nerve that is located very deeply. Therefore the muscle response can be obtained with less stimulus strength than the one necessary in surface electrodes. In short segment study the use of needle electrode provides more certain localization than that of surface electrodes.

The output impulse used for MCS is a rectangular wave with a duration of 0.1 or 0.2 ms. Sometimes it may be necessary to increase the stimulus duration to 0.5 or 1 ms in order to get maximal amplitude. In order to ascertain reliable maximal amplitude of the CMAP, it is advisable to increase the stimulus strength by 10-25% of that which is necessary to obtain maximal amplitude. In some situations a biphasic stimulus pulse is used in order to suppress stimulus artifacts.

Parmeters

A number of parameters are of interest in the assessment of different aspects of motor nerve functions.



Measured parameters of the CMAP. Distal (trace 1) and proximal (trace 2) stimulations are shown.

Measured parameters

Latency (distal and proximal)

The latency is the time between the stimulus and the response. In motor nerve studies, this latency includes the nerve conduction time and also the neuromuscular transmission time. Distal latency is measured from the distal stimulation point to the first deflection from the baseline. Proximal latency starts at the proximal stimulation point and ends at the first deflection from the baseline.

Amplitude

The amplitude (AMPL) of the evoked motor response carries important information. It is dependent on the number of axons that conduct impulses from the stimulus point to the muscle, the number of functioning motor endplates and the muscle volume. The amplitude is measured from the baseline to the negative peak or from peak to peak.

Area

The area represents a combination of the amplitude and the duration. It therefore reflects the number and synchrony of the muscle fibers activated. A prolongation of the duration can cause a decrease in the amplitude and may be misinterpreted as a conduction block. In this situation there may not be significant difference in the area. Area is the integrated area between the CMAP and the baseline.

Duration

The duration (OUR) reflects the synchrony of individual muscle fiber discharges. If there is a significant difference in the conduction velocity among nerve fibers, the duration will be prolonged. This is mainly related to the range of the conduction velocities of the large myelinated fibers. Duration is measured from the onset to the first negative to positive baseline crossing.

Conduction velocity

The conduction velocity (CV) is calculated by dividing the length of the nerve segment between the two stimulation points by the difference between the proximal and distal latency. In this way the slow distal conduction and any delay in the neuromuscular transmission is eliminated. It is calculated as follows.

CV (m/s) =distance (mm) I LATprox-LATdistat

When motor conduction velocity is calculated in this way it reflects the fastest motor axons.

Temporal dispersion

Since nerve fibers have different conduction velocities, a more proximal stimulation site will give an increased duration of M wave. The change in duration with a proximal stimulation site is called temporal dispersion and is calculated as follows:

DISPERSION = 100 X (DURprox- DURdistal) / DURdistal

In healthy subjects, the maximum dispersion in the ulnar nerve is 10-15%. In long nerve segments the CV may be lower and the dispersion higher than that seen in short segments.

Amplitude and Area Decay

With proximal stimulation, when the duration of the M wave gets longer due to the temporal dispersion, the amplitude and the area of the M wave changes Decay is calculated as shown in these formulas.

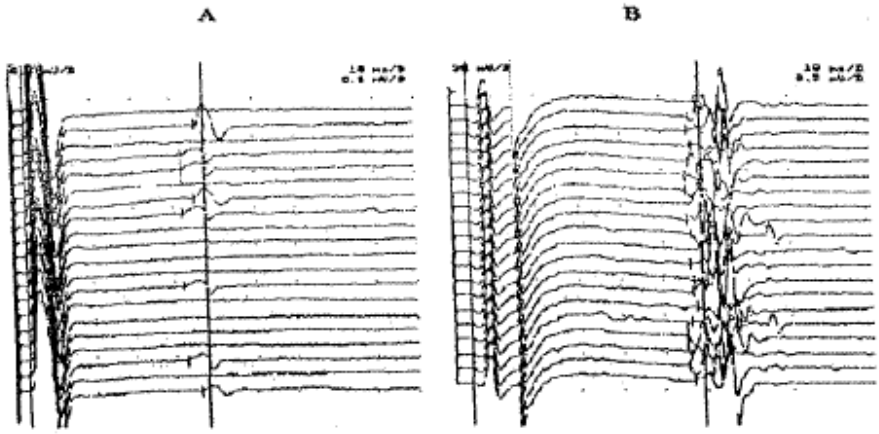
AMPLDECA Y =100 X (AMPLdistal AMPLprox) I AMPdistal

AREADECAY =100 X (AREAdistatl AREAprox) I AREAdistal

In healthy subjects, the mean value of the AMPLDECAY is 4.5-6.2% in the ulnar nerve [and 5.6-7.7% in the median nerve]. The AMPLDECAY is larger in the lower extremities than in the upper extremities. The peroneal nerve has a mean value of 11% and an upper limit of 29% in the AMPLDECAY. On the other hand the AREADECA Y is smaller than the AMPLDECA Yin peroneal nerves. F-waves

F WAVES

When the motor nerve is stimulated, nerve action potentials propagate both in the distal direction to evoke a muscle response, and in the proximal direction as a non- physiological event. Occasionally, the motor neuron depolarisation may evoke a recurrent response by stimulating the first node distal to the neuron. There is only a small chance that the timing of the depolarisation/repolarisation allows this to happen. Normally a recurrent response is evoked in 0.5-5% of the stimulations with some differences between nerves.



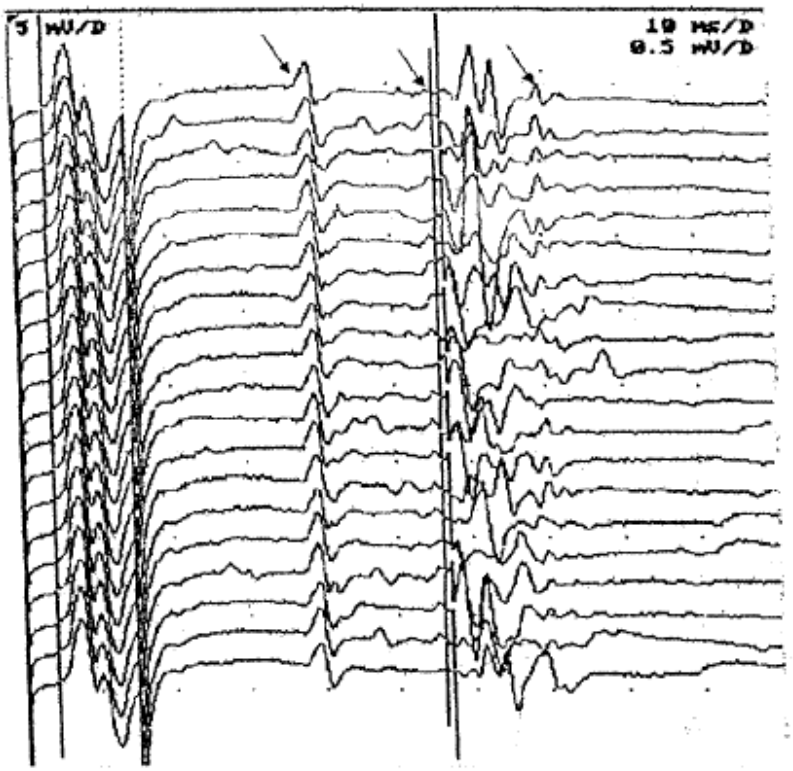
Studies of F-waves in normal peroneal (A) and tibial nerves (B). Note the different occurrence of frequency of the F-waves in these two nerves.

The F-waves travel from the stimulation point on the nerve to the neuron and back to the muscle. By subtracting the distal latency, the time taken from the stimulus point to the neuron and back again to the stimulus point can be obtained. This time depends on the conduction distance involved. Instead of measuring the extremity length~ we relate the reference values to the height of the patient since arm and leg length are normally correlated to height.

Since each normal nerve contains hundreds of motor axons, it is usual to obtain 5-15 F-waves from 20 stimulations. They differ in latency and shape since they normally represent activity from different motor units. The frequency of occurrence is reduced when there is a conduction block anywhere along the nerve. F-wave measurements thus reflect conduction along the entire nerve and are therefore particularly useful in the study of general polyneuropathy and also when proximal segments are preferentially involved, as in Guillain-Barre syndrome, GBS.

M-satellites (often called A-waves)

M-satellites are responses usually occurring between the CMAP and the F-wave They have a constant shape and latency and occur in at least 10 per 20 stimulations. In normal conditions they are only seen in the tibial nerve. They are present in various pathological conditions but are unspecific in relation to a given diagnosis. An M- satellite may be generated as an extra discharge in the stimulated axon (intermediate double discharges, IDD), be due-" t~ emphatic .transmission-between two axons, axon reflex or represent the response from one axon with exceptionally slow conduction velocity. They are for example seen during the fist days of GBS representing IDD



M-satellites (arrows) seen in the tibial nerve in a patient with normal conduction velocity. The two vertical lines indicate upper normal limit (right) and the estimated shortest F-latency among the obtained responses (left line). The F-wave latency is thus minimally increased. The number ofF-waves is normal.

H WAVE

An H-reflex is a monosynaptic reflex that can be elicited by the stimulation of muscle spindle afferents in the limbs. It is possible to evoke H reflexes on most nerves during the first year of life. In adults it can most easily be elicited in the calf muscles and flexor carpi radialis. The H reflex recorded from calf muscles - gastrocnemius, soleus - is mediated via the St root. The H reflex recorded from the flexor carpi radialis is mediated C1 root .

The H-reflex and F-wave differ in some aspects. The H-reflex contains a sensory and a motor branch. The H-reflex is studied only with a sub maximal stimulus and is abolished by supramaximal stimulation. Although consecutive F waves vary in latency and waveform, H reflexes remain constant in response to repetitive stimuli. This is because H reflexes occur from activating the same motor neuron pooL In contrast F- waves represent recurrent discharges from different groups of motor neurons \Vith different conduction characteristics.

H-reflexes may be obtained more easily if a long stimulus duration is used i.e. 0.5 or 1 ms. The H reflex habituates and decreases in amplitude with stimulation rates> 0.5 Hz. The voluntary activation of the investigated muscle or Jendrassic's maneuver will enhance the H-reflex amplitude and shorten the latency.

Summary of parameters

The most common motor neurography parameters are summarized in Table

Parameter	Significance	Usually measured as	Analysis mode
CMAP			
Ampl	# axons, synchronisation	neg. amplitude (mV)	a/m
Area	# axons, "	neg. area (mV *ms)	A
Dur	neg. peak duration	(ms)	
Ampl decay	Cond. block + dispersion	% reduction in ampl	a
Dispersion	axonal velocity disp	% increase in dur	A
CV	velocity of fastest axons	latency diff. (m/s)	a/m
Distal latency	velocity of fastest axons	latency	a/m
F-waves			
Latency	cond. of axons along entire nerve	Lat (min, mean in ms)	
Dispersion	axonal velocity dispersion	Min and max lat (ms)	a/m
# Of F-waves	# axons and MN excitability	# F-waves 20 stimuli	a/m
Amplitude	MUP shape +# F-waves	peak-peak ampl (• V)	a/m not often used
M-satellites			
Presence	Abnormal excitability or slowly conducting axons	present or not	M
H-reflex			
Latency	cond. along reflex arc	H-lat minus M-lat (ms)	a
Amplitude	excitability	M ampl / H ampl	a

Table 1. Note: CMAP = compound muscle action potential; CV= conduction velocity; MN = motor neuron.

Analysis

All modern EMG systems have programs for neurography. Many have algorithms for automatic measurements. These algorithms vary and reference values may therefore differ somewhat between laboratories.

Motor conduction block

One of the parameters in nerve conduction studies concerns the presence of impulse conduction blocks, CB. A Conduction block is the failure of an action potential to propagate throughout the length of a structurally intact axon. This may be seen at the site of a local nerve entrapment and is typical of autoimmune neuropathies (Guillain- Barre, GBS; chronic inflammatory demyelinating pblyneuropathy, CIDP) and in multifocal motor neuropathy with persistent conduction block, MMN. In the hereditary demyelinating polyneuropathies, conduction block is seen only to a slight degree. Other . polyneuropathies do not usually show conduction block

The principal finding is the blocking of an impulse across a local segment without axonal damage. Therefore, stimulation distal to the block gives a normal CMAP whereas a proximal stimulation produces decreased amplitude.

This is normally studied in two ways in the motor nerve; by comparing CMAP at distal and proximal stimulation and by the assessment of the F-wave frequency.

In MCS, there is normally a slightly lower amplitude for proximal stimulation compared with distal and also a slight temporal dispersion. This can be explained simply as the difference in the conduction velocity between individual axons. This gives rise to an incomplete summation of signals and even some degree of so called phase cancellation. The longer the distance, the more pronounced these changes are. These effects are more pronounced in cases of slow conduction velocity due to demyelination, particularly when there is an increased spectrum of velocities among the axons. This means that when a reduction of the CMAP is obtained at proximal stimulation compared to distal response, the differential diagnostic problem is demyelination with general slowing causing abnormal temporal dispersion vs. a pure conduction block. In the first case, the distal-proximal amplitude drop is parallel to the temporal dispersion as expressed by the change in CMAP duration. With pure CB, there is no increase in temporal dispersion.

There have been several attempts to define criteria for conduction block. To discriminate between "pure" demyelination and a conduction block, the following criteria have been suggested (Ad Hoc Subcommittee of the American Academy of Neurology AIDS Task Force 1991}:

Conduction block is present if there is
>20% AMPLDECAY or AREADECAY and <5% DISPERSION or if there is
>50% AMPLDECAYor.AREADECAY, independent of DISPERSION.
'

It has been demonstrated that both these criteria are equally sensitive in detecting a block.

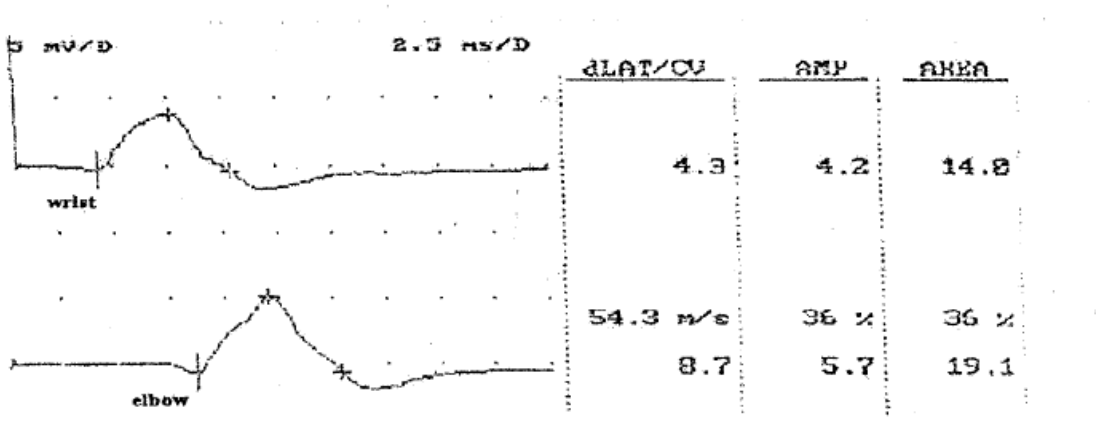
Our own(SCTIMST) modifications of these rules are:

>25% (arm) or >40% (leg) AMPLDECAY and <15% DISPERSION or if there is
>50% AMPLDECAY, independent of DISPERSION (in this case there is a combination of CB and demyelination)

The other parameter, which indicates CB, is the frequency of occurrence of F- waves. In cases of proximal CB, there may be no drop in amplitude in the distal part of the nerve, e.g. in early GBS. In this case, the CB can be seen by a reduction in the occurrence of F-waves. The normal frequency of F-waves varies for different motor nerves. When 20 stimuli are given in a normal situation the different nerves normally show the following numbers ofF-waves: ulnar 18, median 15, peroneal10, and tibial 18. As can be seen, it is impossible to detect a slight reduction in the number of F-waves in the ulnar and peroneal nerve. Lack of response i~;> usually taken as an abnormal finding. In the tibial nerve, a better assessment of pathology can be made. It should be noted that in the interpretation of the number of F-waves, the CMAP amplitude needs to be considered. If the CMAP is reduced to half due to axonal degeneration, then the expected number ofF-waves is correspondingly reduced.
The number of F-responses should also always be reduced when conventional MCS has shown a conduction block; otherwise a technical error is to suspect in the measurements.

Anatomical variants

In normal subjects, there could be some anatomical variations in the muscle innervation. Martin-Gruber anastomosis is the most common anomalous innervation of the hand with an incidence of 15-28% [17]. The fibers innervating intrinsic muscles of the hand cross from the median nerve to the ulnar nerve. Sensory fibers are not involved. Martin-Gruber anastomosis can 15e diVided .. irito three types according fo the muscle innervated by the crossing fibers. The most common type is type II, in which the crossing fibers innervate first dorsal interosseus muscle. When the anastomotic fibers end in the ADM and abductor pollids brevis the MG is classified as type I and type III respectively. type III is the least common. Stimulating the median and ulnar nerves and recording from the muscles mentioned above can reveal martin-Gruber anastomosis. The amplitude of the CMAP wave evoked by the median nerve stimulation at the elbow is found higher than the one evoked by stimulation at wrist level when recording is performed by muscle with anomalous innervation. The CAMP obtained at the proximal site also has an initial small positive deflection because of the volume of conduction from the deep ulnar innervated muscles. In type I the amplitude of the M wave from the ulnar nerve stimulation shows a reverse discrepancy; lower with elbow stimulation and higher with wrist stimulation. 8



Median nerve MCS in a case of Martin-Gruber anastomosis. Note the higher amplitude and the initial positive going phase at stimulation at elbow.

When this anastomosis accompanies the carpal tunnel syndrome (CTS), an abnormally fast conduction velocity value, in the forearm segment of the median nerve is found, due to delayed DLAT but a "normal" PLAT. Another common anatomical variation is the innervation of the extensor digitorum brevis by the accessory peroneal nerve. The deep peroneal nerve normally supplies this muscle. In 23 to 28 percent of population, the superficial branch, behind the lateral malleolus, innervates it. When a low CMAP is obtained distally, stimulation should always be performed behindthe,mnlleolus."

Schematic summary of the relationship between CV and amplitude parameters in axonal (low amplitude) and demyelinating (low CV) neuropathy. Table 2..

Table 2.

	Demyelination	Axonal degeneration	Conduction block
CV and dist latency		n/	N
Amplitude	n/		
Amplitude decay	n/	N	
Dispersion		N	
F-wave latency		n/	N
# Of F-waves	n/		

Table 2. Note: Classical findings in neurography at different types of pathology. = increased; ¯ = decreased; n= normal

Sensory Conduction Studies

The pathophysiological principles regarding sensory nerves are the same as those discussed for motor nerves. Sensory neurography differs in some aspects: The amplitudes are much smaller than those in MCS and since the recordings,are carried out on the nerve itself. Furthermore, only one stimulation site is necessary for calculating CV. The stimulation recording can be performed in both the orthodromic or antidromic direction. Some of these things will now be discussed.

Recording electrode

The recording may be performed orthodromically or antidromically. The conduction velocity is the same but other parameters are different. In some instances one choice is preferred, but in, other cases the choice is based on tradition. Table 3 shows differences between ortho and anti-dromic methods.

Table 3. Differences between orthodromic and antidromic nerve stimulation

Orthodromic	Antidromic
No muscle artifact (+)	Less painful (+)
More painful	Larger amplitude (+)
Lower amplitude	Muscle artifact in mixed nerves

Usually surface electrodes are used for recording purpose. The electrodes may be so called ring electrodes, e.g. around the digits. More commonly we use felt pad electrodes with a fixed interelectrode distance between the two recording poles. The electrode is placed along the nerve with the "active" towards the point of stimulation.

If the electrode is placed along the nerve, the two poles record an identical signal, often biphask in shape with an initial smaller positivity and a larger positive part. In the amplifier the two signals are subtracted, giving rise to a triphasic configuration. The shape, in particular the duration and amplitude of the recorded signal is dependent on the conduction time between the two poles (distance and CV). This means that, the interelectrode distance has to be kept constant within the laboratory.

Reference data must be collected using the same technique that is used in routine studies.

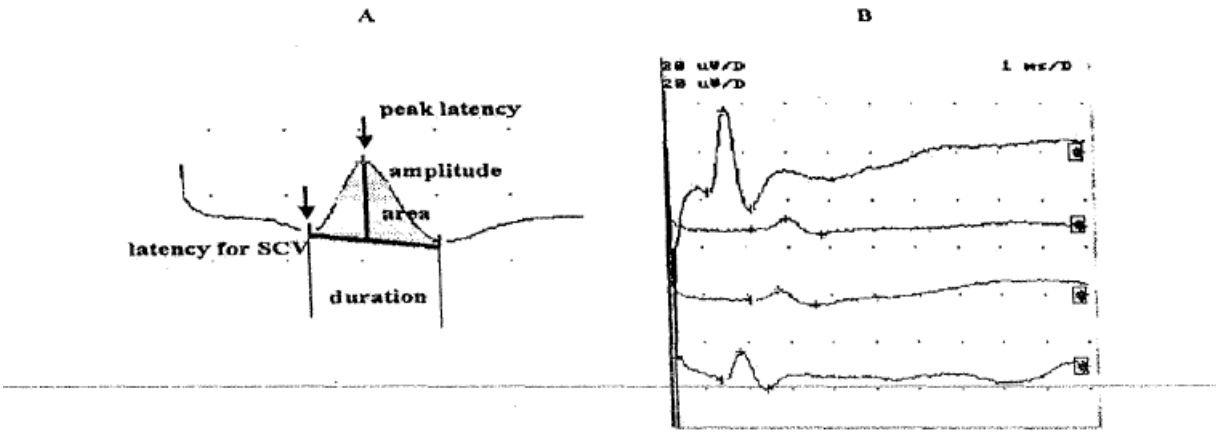
In cases when near nerve needle electrodes are used, all the parameters except CV are different. In this case a special needle electrode is inserted just outside the nerve (the position may sometimes be tested by using the electrode for stimulation. An optimal position is found when a muscle response is obtained with minimal current, often less than 2 mA). The r.e_~ording_may sho~ multiple peaks indicating the difference in conduction velocity among individual axons. This is particularly apparent in cases of pathology . .Anlplitude parameters are dependent on both the needle position in relation to the nerve, and the distance between the stimulation and recording. It is often not very reproducible in pathology. We use needle electrodes in Morton's metatarsalgia and meralgia paresthetica.

Stimulation

Stimulation can be performed with a surface electrode, or with a needle electrode. In the first and more common case, the electrode is placed over a sensory nerve, or sometimes over the relevant skin area. The stimulus duration is often 0.1 ms and the frequency around 1 Hz. The stimulus strength is increased, as long the recorded response is increases. With higher stimulus strength, the increase in pain and, depending on the stimulus site, the motor artifacts may significantly disturb the recordings. Here an optimum must be found. When a needle electrode is used, similar considerations should be made. In our study we used needle electrodes to obtain high selectivity, e.g. in the stimulation of individual digital nerves in cases of Morton's metatarsalgia.

Parameters

Parameters and a typical recording from the ulnar sensory nerve are shown



below. Sensory nerve action potential. A= parameters, used in all nerve studies. B= Sensory conduction studies in the ulnar nerve. Averaged responses obtained orthodromically at the wrist with stimulation at palm, dig IV and dig V (traces 1-3). Antidromic sensory response obtained in the interdigital space between metacarpals IV and V and stimulating the dorsal ulnar branch. In cases of entrapment at the Guyon's canal, this response is normal, while the digital responses are abnormal.

Latency (distal and proximal)

The latency is the time from the stimulus to the first positive peak of sensory nerve action potential (SNAP). If there is no clear positive peak in antidromic recording, the latency is measured from the take--off from baseline. For the normal latency of a nerve there should be approximately two hundred nerve fibers conducting normally and having 10 m V or more of diameter [26].

Some laboratories have a tradition of measuring latency to the first negative peak. Since negative peak latency includes the rise time of SNAP and indicates the temporal dispersion, it is not recommended to use negative peak latency to calculate the cv.

Amplitude

The amplitude of the SNAP should be measured from the first positive peak to the highest negative peak. Some authors measure the amplitude as the maximum peak- to-peak amplitude or as the amplitude between a line joining the positive peaks as the positive value and the negative peak The amplitude reflects the number of nerve.fibers having. a diameter; of 9 m V or more.

Area

The area is the integrated area between the signal and baseline over the DUR. The area represents the combination of amplitude and duration; therefore this reflects the number and synchrony of the activated nerve fibers.

Duration

The duration is measured from the first positive peak to the last positive peak. When there is no presence of the initial positive peak, the duration is measured from take-off on the baseline. **Conduction velocity**

The conduction velocity (CV) is calculated by dividing the length of the nerve segment from the stimulus point to the recording point by the positive peak latency. It should be calculated as follows.

CV (m/s) =distance (mm) / LAT

When the sensory conduction velocity is calculated in this way, it reflects the conduction velocity of the fastest sensory fibers.

Temporal dispersion and decay

Since physiologic temporal dispersion affects the sensory action potential more than the muscle response, these parameters are not easily used in the routine studies. This is due to the difference in duration of individual unit discharges between nerve and muscle. With short-duration diphasic sensory spikes, a slight latency difference could line up the positive peaks of the fast fibers with the negative peaks of the slow fibers, cancelling both.

Table 4.

Parameter	Significance	Usually measured as	Analysis mode	Comment
Latency	conduction velocity	positive peak (ms)	a/m	
CV	conduction velocity	distance/latency (m/s)	a/m	
Amplitude	# axons, temporal disp	peak-peak (μ V)	a/m	
Area	# axons, temporal disp	total area (μ V * ms)	A	
Duration	dispersion	pos.-pos. peak dur (ms)	a/m	
Late components	conduction dispersion	shape	M	in needle rec.

Table 4 showing parameters usually measured in sensory neurography. Note: for explanation, see Table 1.

Analysis

Modem EMG equipment has programs to measure these parameters. Often averaging is necessary in order to obtain a good signal to noise ratio.

Autonomic nerve testing

It should noted that this is important in the general neurophysiological investigation of a patient with neuropathy. Some of the most commonly used methods are indicated in table given below

Autonomic tests

Autonomic testing includes Heart rate variation	
(At deep breathing, Valsalva, tilt test)	parasympathetic (sympathetic)
SSR	Sympathetic
Pletysmography	Sympathetic

Sympathetic Skin Response

The sympathetic skin response is the elctrodermal activity caused by alteration in the electrical skin resistance from minute changes in the sudomotor activity and serves as an indirect assessment of the autonomic nervous system. A variety of sensory stimuli (auditory, tactile and deep inspiration) can elicit an SSR.

Procedure

The active recording electrode is placed over palm on the hand or the plantar surface of the foot and the corresponding reference electrode is commonly placed over the dorsum of the hand or foot. Electrical stimuli 0.1 to 0.2 ms duration and 10 -20mA in intensity can be applied to the wrist or ankle on either side to depolarize the sensory nerves. Random frequency of stimulation is preferable to avoid the habituation of the response. Filter setting 0.5 Hz to 2 KHz is optimum for the recording.

The recoded response may have one to three phases. The latency is not useful in clinical practice. The sympathetic skin response is normal when it is present and abnormal when absent, The absent SSR in patients with neuropathies correlates with involvement of unmyelinated autonomic fibres.

REPETITIVE NERVE STIMULATION

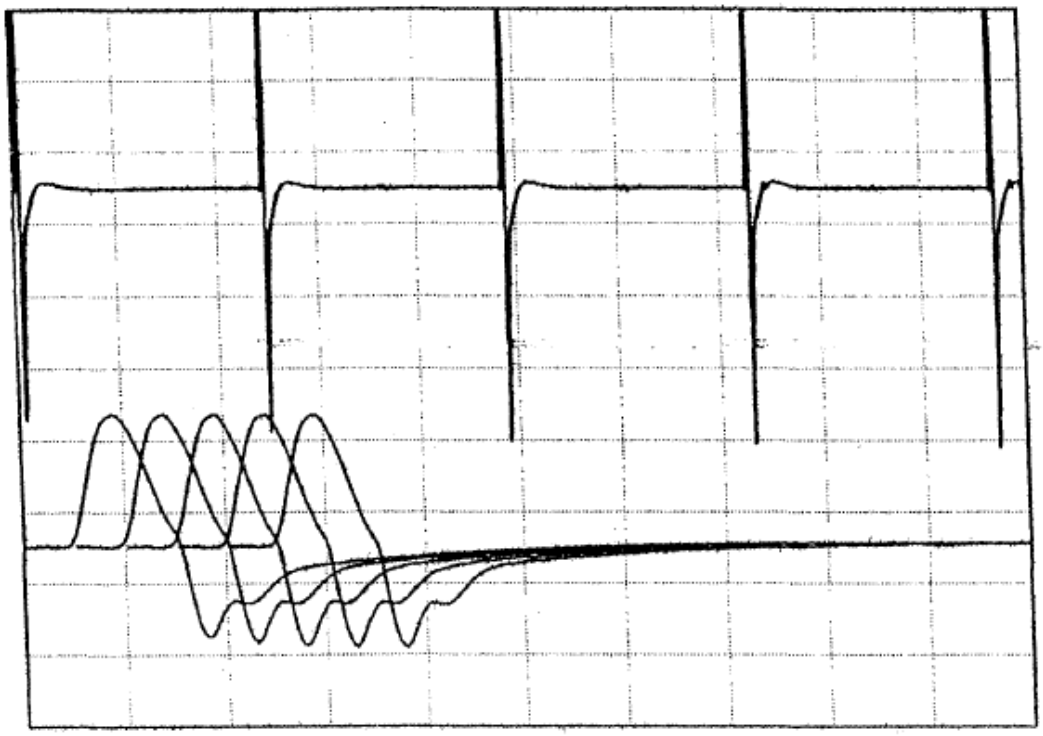
Repetitive nerve stimulation (RNS) is used in the evaluation of patients with suspected neuromuscular transmission disorders (NMTD) such as myasthenia gravis (MG) or Lambert-Eaton myasthenic syndrome (LEMS). RNS is a modified motor NCS where instead of recording CMAPs with single supramaximal electrical stimuli, a train of 8-10 stimuli is applied and· ·the sequential response--o amplitudes and/or areas measured. This may be carried out at low (3-4Hz) or high frequency stimulation (20-50 Hz). In the latter case the train is prolonged to allow 2-10 seconds of continuous data to be measured. Both distal and proximal muscles/nerves should be studied in every patient suspected of an NMTD as the sensitivity of the test is greatly increased by this means.

With low frequency stimulation in normal subjects, the CMAP amplitude and/or area falls over the first 4-5 stimuli by a maximum of 10-12%. The maximum fall should be between the potentials 1 and 2.A number of departments with specific studies have published studies on RNS over time both before and after a period of maximum voluntary contraction of the muscle to pick up early or late NMT failure. High frequency stimulation may be used to discover evidence of a post-synaptic transmitter release disorder like LEMS. It is painful and requires considerable patient tolerance . There is evidence that recording low frequency RNS immediately before and after a 20- 30 second period of maximum voluntary contraction by the patient is equally sensitive and is more humane.

There are many pitfalls in the RNS test and artifact almost always gives rise to an abnormal test. Thus adherence to a strict protocol and heightened suspicion on the part of the CN to an abnormal result is essential as are repeated studies for reproducibility of abnormalities.

Physiological basis for the RNS

The neuromuscular junction consists of the motor axon terminal, the synaptic cleft, and the post-synaptic muscle membrane. As the motor axon potential depolarizes the nerve terminal, voltage gated calcium channels open increasing the concentration of calcium in the pre-synaptic nerve terminal. This in turn facilitates the release of quanta of acetylcholine (ACh) from the nerve terminal into the ,synaptic deft. ACh binds to receptors on the post-synaptic membrane causing depolarisation (end plate potential). The size of the end plate potential is dependent on the amount of ACh released and its binding to receptors. In the healthy state, the end plate potential reaches



a threshold level and causes an action potential to be propagated along a muscle fibre resulting in muscle contraction. Normally there is a large safety factor for neuromuscular transmission with the amount of ACh released per impulse several times that required to generate a threshold level end plate potential.

In low frequency RNS, the rate of stimulation is such that the end plate physiology is stressed, but not to the level that produces the natural facilitation of NMT at greater stimulation frequencies. Thus an abnormal fall (decrement) in CMAP amplitude and/or area at low stimulation rates indicates a drop in the safety factor for transmission whether from a pre- or post-synaptic cause.

In high frequency stimulation natural facilitation is enhanced by pre-synaptic Ca++ influx and this may counteract a process such as LEMS where quantal release is depressed.

BLINK REFLEX

The cranial nerves that can be readily tested are the trigeminal (V), facial (VII), and spinal accessory (XI).

Facial (VII)

The facial nerve is examined by recording the latency and amplitude from a stimulus at only one site along the course of the nerve. Nerve conduction velocities are not calculated.

Stimulation site: Place the electrodes behind the angle of the jaw, with the cathode posterior to the earlobe and the anode behind. This placement stimulates the nerve just before its enters the parotid gland. Alternatively, you may place the cathode over the stylomastoid and anode over mastoid.

Ground: Usually the ground is placed over the parotid area, but you may place it on the chin or forehead also.

Recording sites: Place the active recording over the orbicularis oris at the comer of the mouth, over the orbicularis oculi on the outer canthus of the eye, over the frontalis in the forehead, or over the nasalis muscle on the nasolabial fold. Place the reference electrode on the nose. Either a needle or surface electrode may be used for recording. The facial nerve may be evaluated differently - by using the blink reflex, which will be discussed with the trigeminal nerve (below).

Trigeminal

Using reflex activity and extrapolating information from it evaluate this nerve.

As the sensory fibers of the Vth nerve enter the brain stem, they establish three kinds of synaptic connections with the VIIth nerve nuclei: -One, a direct and monosynaptic with the ipsilateral VIIth nerve nucleus. - Another, indirect and polysynaptic with the contralateral VIIth nerve nucleus. - A third, also polysynaptic, again to the ipsilateral Vllth nerve nucleus.

These connections are demonstrated clinically by the fact that when the glabella is lightly tapped with a reflex hammer or a finger,a brisk blinking reaction is seen bilaterally. The blink reflex is the electrical equivalent of this reaction referred to clinically as the glabellar reflex. Stimulation of the supraorbital branch of the Vth nerve as it enters the skull through the supraorbital foramen will result in contraction of the orbicularis oculi muscles bilaterally.

Using two channels on the cathode ray tube to study both sides simultaneously best performs the test. One each side an active electrode is placed over the orbicularis oculi muscle on the outer canthus of the eye and the reference on the lateral aspect of the nose. One ground is used and is placed over the chin.

The Vth nerve is stimulated via its supraorbital branch over the supraorbital foramen; the sweep speed used is 10 msec/division and the gain set at 200 1-.lv/division. On the ipsilateral channel, both direct and indirect responses are seen, the direct of a short latency and mono or biphasic configuration, the indirect of a long, usually variable, latency and polrphasic configuration. On the contralateral channel, only the indirect, long latency polyphasic response is seen.

Blink Reflex Findings

In unilateral Vth nerve lesions, all three responses are equally affected. In unilateral Vllth nerve lesions, stimulation on the same side of the lesion will result in delayed or absent direct and indirect responses ipsilaterally but a normal indirect response contralaterally. When the nerve is stimulated on the healthy side, both the direct and indirect responses are spared while the contralateral indirect response is affected.

The blink reflex can be used in the evaluation of toxic neuropathies and in comatose patients and multiple sderosis as a means of evaluating brain stem functions.

ELECTROMYOGRAPHY

Volitional Activity in Muscle

Electromyography (EMG) is the science involved with the study of electrical activity in muscle. The basic physiologic unit of normal skeletal muscle function is the motor unit, which consists of a lower motor neuron (LMN) and a finite number of muscle cells (fibers). When a LMN discharges in response to volitional or reflexive activation, an action potential is propagated'along its·axon -{nerve fiber")_chemically recreated at end plates, and then propagated along muscle cell membranes just prior to muscle contraction.

Motor Unit Potentials

The composite electrical activity in muscle cell membranes when a motor unit discharges is called a motor unit potential (MUP). The size of a single MUP depends upon the type and size of the motor unit and the proximity of the unit to the recording electrode. Primary muscle disease may cause MUP durations and amplitudes to decrease (due to loss of individual fibers).

Manipulating posture or using relfexes to induce movement can record motor unit potentials. In some neurogenic diseases, the duration and amplitudes of MUP increase due to increases in innervation ratios caused by collaterization and reinnervation. These unusually large MUP have been referred to as giant motor unit potentials. During periods of active degeneration, MUP may show slightly increased durations and amplitudes. The presence of polyphasic MUP may point to cond,uction impairment in smaller intramuscular nerve terminals.

Interference Patterns

Normal volitional muscle contraction is brought about by the repetitive asynchronous activation of large numbers of motor units. Recruitment is the process of adding motor units to ones that are already active thus increasing the force of contraction. Electromyographically, the pattern of muscle contraction during normal physiologic activity is called an interference pattern because the individual MUP are so numerous, they seem to "interfere" with each other in a recording Depending upon the intensity of muscle contraction, the interference pattern may be called complete or incomplete. The interference pattern can be recorded from single muscles with intramuscular monopolar needle electrodes or fine wire electrodes. Both type of electrodes are insulated except for the tip of the needle or wire. These procedures allow for an evaluation of a specific muscle with little to no interference from other muscles.

Fine wire electrodes, for example, have been used successfully in small animals to assess the function and synergistic action of the external urethral sphincter during cystometrography. In peripheral neuropathies, the interference pattern is markedly reduced or completely lost.

Evoked Activity in Muscle

In clinical settings, skeletal muscle activity is usually evoked by .electrical stimulation of motor nerves while intramuscular needle or surface electrodes are used as recording electrodes. In other evoked responses, receptors are physiologically stimulated and muscles are reflexively activated. Muscle activity may also be produced by electrical or electromagnetic stimulation of the motor cortex. Mechanical injury to muscles by needle recording electrodes will also evoke activity. Electrical or reflexive activation of muscles will be described in other briefs.

Insertion Potentials

Other than end-plate noise, normal muscle membranes are electrically silent if there is no LMN activity. However, when a needle electrode is inserted into or moved in a normal healthy muscle, it is accompanied by electrical activity called insertion potentials. Insertion potentials are caused by the mechanical stimulation of muscle fibers and usually cease when needle movement ceases. As described below, insertion potentials may be prolonged in neuropathic ,or myopathic disorders and mixed with other abnormal potentials such as positive sharp waves and fibrillation potentials. Insertion potentials may be reduced in severly atrophied muscle.

Spontaneous Activity in Muscle

Spontaneous activity in muscle, when it occurs, may be initiated in the perikaryon of the LMN, its peripheral axon, the end plate, or the muscle membrane itself. As mentioned previously, if skeletal muscle is not volitionally or reflexively activated, it is electrically quiescent, therefore, most spontaneous activity in muscle is often associated with neuromuscular abnormalities

Miniature End-Plate Potentials

Each skeletal muscle cell is hmervated by a single branch of the LMN axon at a synapse referred to as the end plate. In the absence of LMN activation, spontaneous activity in muscle is only recorded in the motor point, an area in the muscle where there is a high concentration of end plates. This activity may be recorded by needle electrodes and referred to as end-plate noise, which consists of large numbers of miniature
end-plate potentials (MEPP). A single MEPP is a non-propagated calcium-dependent potential caused by a small number of neurotransmitter packets being released spontaneously at the end plate. Motor end-plate noise as well as other muscle potentials described below can be recorded with intramuscular needle electrodes.

Fasciculation Potentials

Fasciculation is a spontaneous twitch when motor unit& or parts of neighboring motor units discharge. Electrically, fasciculation's have durations, amplitudes and other characteristics similar to MUP. Recent findings suggest that benign fasciculation's are caused by emphatically activated muscle fibers as a result of discharge in pacemaker fibers in either nerve or muscle. Although very little tension is produced, fasciculation can sometimes be observed through the skin. Fasciculation can be seen in a variety of diseases affecting nerve and muscle, and infrequently in degenerative diseases affecting the spinal gray matter.

Fibrillation Potentials.

Fibrillation potentials (fibs) are brief spontaneous bi- or triphasic potentials that are seen in neurogenic and primary muscle disease. This potential, also called a denervation potential, is qne of the electrical hallmarks of partially or completely denervated muscle and isproduced by the discharge of a single muscle fiber. The amplitude of fibrillation potentials may range from 50-350 mV with durations of 1-2 msec. Fibs appear in denervated muscle after a latent period that is proportional to the length of remaining axons distal to the site of a nerve lesion. Their onset may be preceded by periods of increased insertional activity. Once these potentials appear in denervated muscle, their rate of occurrence increases over a period of several weeks. They will persist until muscle is reinnervated, or until no viable muscle fibers remain. Fibrillation potentials are also seen in myopathic disorders such as muscular dystrophies, polymyositis, and dermatomyositis. Their origin is related to oscillating membrane changes or irregular prepotentials caused by membrane instability. In neuropathies, where the primary feature is demyelination, fibrillation potentials may not be found. The sound of these potentials from a loudspeaker has been likened to crackling cellophane.

Positive Sharp Waves.

Positive sharp waves (PSW), like fibrillation potentials, occurs when muscle is denervated, but also occur in a variety of myopathic disorders. In denervation, PSW may precede fibrillation potentials by one or more days. The special feature of the PSW is the initial positive phase followed by a more gradual negative-going phase. By convention, EMG recorders are configured such that positive potentials produce a
downward deflection in the recording. These potentials make a lower pitched sound

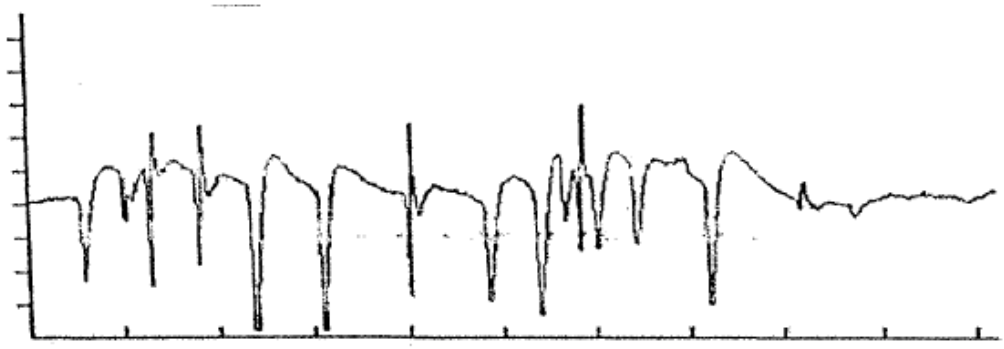


Figure 4- Positive sharp waves (PSW) mixed with fibrillation potentials recorded from a forearm flexor muscle in a dog. Arrow identifies one of several PSW Horizontal division = 10 msec; vertical division = 97.7 uV.

than fibrillation potentials and usually have a lower discharge rate.

Bizarre High Frequency Discharges.

Bizarre high frequency (BHF) activity consists of polyphasic potentials that discharge spontaneously at a high frequency. Within the train of discharges, each potential may have precisely the same appearance. Such behavior suggests the presence of pacemaker muscle fibers that oscillate. The onset may be associated with needle movement and the discharges start and stop abruptly. These potentials can occur in a variety of myopathic conditions including hyperadrenocorticism, and polymyositis. Some have referred to these as pseudomyotonic potentials because they do not wax and wane like true myotonic potentials. From the loudspeaker of the electromyograph, these discharges have continuous high-pitched motor-like sounds.

Myotonic Discharges.

Myotonic potentials occur in muscles as a result of permeability abnormalities in muscle fiber membranes. Muscles continue to be electrically activated even after the cessation of volitional contraction. These high frequency (100 to 200/sec) potentials spontaneously wax and wane in amplitude and rate in an EMG pattern that has become the electrical signature of myotonia. In addition to normal presynaptic nerve activity, mechanical muscle movement, or needle displacement may precipitate the onset. Spontaneous activity may last for a second or more, which persist in the presence of depolarizing and non-depolarizing muscle relaxants. In some types of myotonia, the repetitive discharges may be explained by altered chloride conductance in muscle membranes while in other types, the malady may be related to a disorder in cation conductance. Audio ,monitoring of myotonic potentials reveal the most characteristic EMG sounds, that of a dive-boinber. For this reason, myotonic potentials are referred to as "dive-bomber potentials."

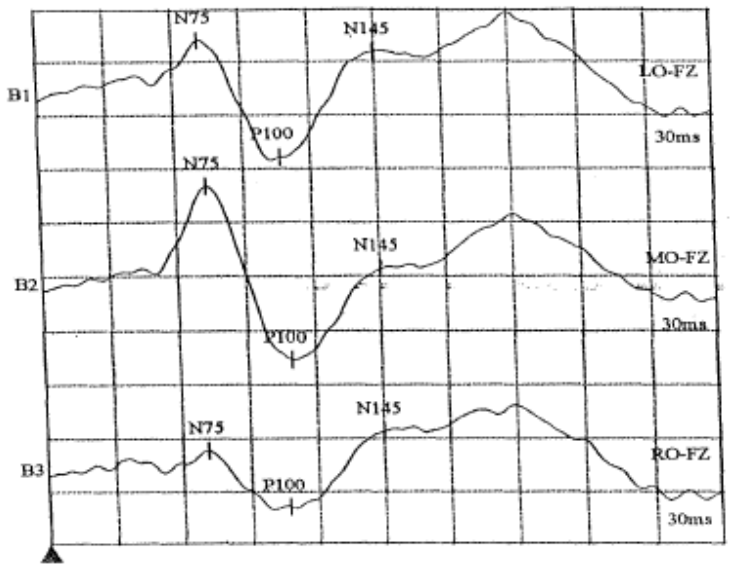
VISUAL EVOKED POTENTIAL

Visual Evoked Potentials

The VEP tests the function of the visual pathway from the retina to the occipital cortex. It measures the conduction of the visual pathways from the optic nerve, optic chiasm and optic radiations to the occipital cortex. The most important fact is to consider that, although the axons from the nasal half of the retina decussate at the optic chiasm, the temporal"axons do not. Therefore retrochiasmatic lesions may not be detected by full field checkerboard stimulation. VEPs are most useful in testing optic nerve function and less useful in postchiasmatic disorders. In retrochiasmatic lesions, the MRI is a more useful test. Partial field studies may be useful in retrochiasmatic lesions; however, they are not performed routinely in clinical settings. Also, note that the macula projects to the occipital pole, while the rest of the retina projects o the mesial calcarin cortex.

The VEP is very useful in detecting an anterior visual conduction disturbance. However, it is not specific with regard to the etiology. A tumor compressing the optic nerve, an ischemic disturbance, or a demyelinating disease may cause delaying the P100; only additional clinical history and, often, MRI are needed to uncover the etiology. The usual waveform is the initial negative peak (N1 or N75), followed by a large positive peak (P1 or P100) followed by another negative peak (N2 or N145).
Maximum value for P100 is 115 ms in patients younger than 60 years; it rises to 120ms thereafter in femal~s and 125 ms in males. Eventhough, published norms are available in the medical literature, each individual laboratory should have its own norms to control for lab-to-lab variability in technique.

The W morphology, is most often a individual variation, although decreasing the stimulation frequency from the ubiquitous 2Hz -1 Hz usually converts the W shape into a conventional P100 peak Check size and alternation rate are the factors in this; the response can be manipulate to a W or a conventional PlOO response by changing these parameters. Large checks tend to produce VEPs similar to those produced by flash stimulation.



Technical Aspects

Checkerboard pattern (or less often, flash) is used as stimulation. Responses are collected over OZ, 01, and 02 and with hemi field studies at T5 and T6 electrodes using the staridard EEG electrode placement. Monocular stimulation is used to avoid masking of unilateral conduction abnormality. Sedation should be not be used, and note should be taken of medications that the patient is taking regularly. Testing circumstances should be standardized, including seating distance of 70-100 cm from the monitor screen, giving a check size of approximately 30 seconds of visual angle. The vision should be corrected to the extend possible in case of a visual problem. Pupil's size and any abnormality should be noted. The P100 waveform is at its maximum in the mid occipital area. Stimulus rates of 1-2Hz are recommended, and filter setting should be 1-200 Hz bandwidth.

The recommended recording time window (sweep length) is 250ms; 50 - 200 responses are to be averaged. A minimum of 2 trials should be given. The responses are averaged and the P100 positive polarity waveform that appears in the posterior head region is analyzed. The mean latency is about 100 ms. Normative data should be assembled on a lab-by-lab basis.

BRAINSTEM AUDITORY EVOKED POTENTIALS

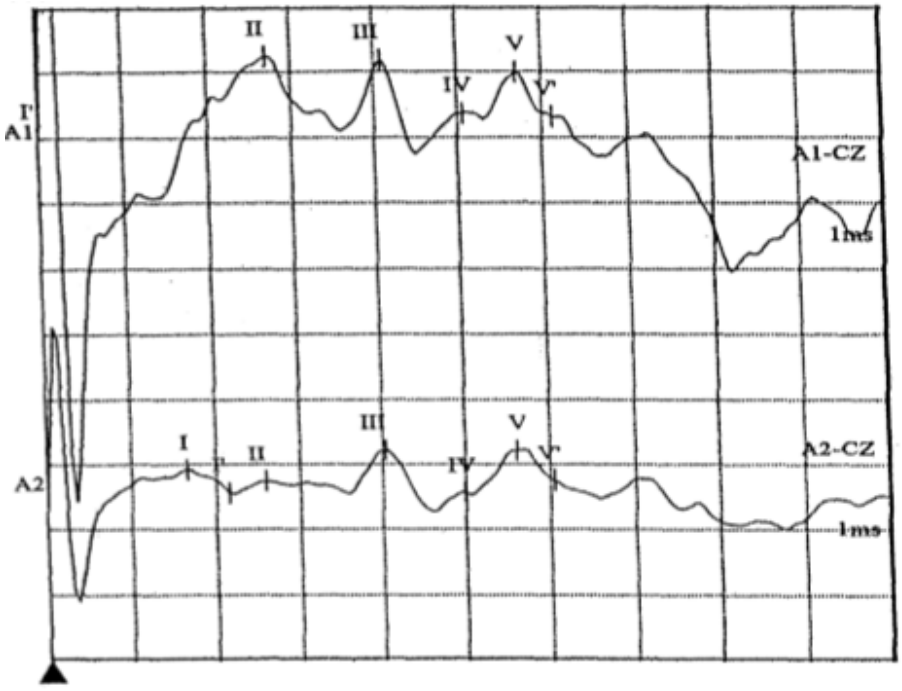
Introduction

BAEP or BAER measures the function of the auditory nerve and auditory pathways in the brain stem. The short latency BAER generally is used for clinical purposes. The test can be performed under sedation or under general anesthesia. Standard broadband click stimulation is used on the ear tested, while the contralateral ear receives masking noise of 30 to 40dB lesser intensity. Monaural stimulation is Used. The click intensity should be 65 - 70 db above. Click perception threshold. A repetition rate of about 1OHz should be used.

Electrode placement

An electrode is placed on each earlobe and at Cz. whether nuclei or tracts, or both, generate the peak latencies is not known. Generators currently are postulated to be as follows:

. Wave I- Action potential of the cranial nerve (CN) Vlll
. Wave II- Cochlear nucleus (and CN VIII)
. Wave Ill- Ipsilateral superior olivary nucleus
. Wave IV- Nucleus or axons of lateral lemnicus
. Wave V- Inferior colliculus



Factors influencing peak latencies of BAERs include age, sex, auditory acuity Stimulus repetition rate, intensity, and polarity. Rarefaction (ie, earphone diaphragm moves away from the eardrum) produces an increase in wave I amplitude. In severe hearing loss,all wave forms may be delayed, wave I may be absent with waves II though V delayed, or all waveforms may be absent.

Patients with hearing loss BAER still can be obtained to assess central conduction time by increasing stimulation intensity.

BAEPs are useful in estimating or aiding in the assessment of hearing loss. The most commonly used method for this is evoked response audiometry. The frequency of stimulation is 50-70Hz, and at least 3 different intensities should be used. Wave V latency shifts are used to estimate the amount of hearing loss.

In children, especially those younger than 2 years, the BAEP can be used to Screen those who might benefit from auditory amplification in order to achieve more normal speech and language development. However some children with a normal BAER have abnormal hearing. Kileny showed middle latency abnormalities in some of these cases. The role of BAEP nevertheless is to identify those patients who could benefit from a hearing aid. Obviously with normal BAEP a hearing aid would not be useful to correct the hearing loss. Kern et al studied effects of insulin-induced hypoglycemia on the auditory brain stem response (ABR) in humans. ABRs were examined in 30 healthy men during euglycemia and after 20 minutes and 50 minutes of steady state hypoglycemia of 2.6mm induced with insulin. Hypoglycemia increased interpeak latencies III-V and I-V, whereas changes in the latency of the wave I are not significant.

Technical aspects

Filter band pass of 100-3000Hz is used. The first 10 ms are averaged, and 2-4000 responses may be averaged. At least 2 separate trails should be performed. The recording montage is at least, and usually, a 2 channel montage- channel 1 is ipsilateral ear to vertex and channel 2 is contralateral ear to vertex. Because of relative vertex positivity, the waveforms are recorded as upward deflections. The normal response is a series of waveforms within a time window of 10ms.

Clinically, the first 5 waves are used, with more significance placed on waves I,III and V. Peak and interpeak latencies are measured, side-to-side differences are calculated, and wave I-V ratios may be used. Audiometry is very helpful and should be done within a reasonable time interval of the BAER. This helps the delineate any hearing loss that might influence the test results. Hearing loss in the 2000 - 4000Hz frequency range is especially important, since it may delay the BAER.

Neonatal BAEP: recording the neonatal BAEP is technically different from recording that of the adults. The skin is very sensitive, and special non-allergic tape should be used to fix the electrode. Collodion or other irritant chemicals are to be avoided. To avoid collapse of the earlobe and obstruction of the auditory canal of the premature babies, the earphone should be held above the ear. The earphone is

best held by hand, and recording preferably should be performed with the neonate asleep. This helps reduce the high frequency components of the EEG that might interfere with BAEP recording. Because of the slower response, sweep should be set at 15-20ms and the low frequency cutoff filter at 20-30Hz.

BAEPs predominantly activate the pathways in the brain stem ipsilateral to the side of click stimulation. In particular, mid-upper pontine lesions tend to lead to ipsilateral BAER abnormalities. The structures involved in generation of BAER may be more concerned with sound localization that with hearing itself.

SOMATOSENSORY EVOKED POTENTIALS

Introduction

Somatosensory evoked potentials (SSEPs) consist of a series of waves that reflect sequential activation of neural structures along the somatosensory pathways following electrical stimulation of peripheral nerves. In clinical practice, SSEPs are elicited typically by stimulation of the median nerve at the wrist, the common peroneal nerve at the knee, and/or the posterior tibial nerve at the ankle and recorded from electrodes placed· over the scalp spine, and peripheral nerves. The dorsal column lemniscal system is the major anatomical substrate of the SSEPs within the CNS.
SSEPs are used for clinical diagnosis in patients with neurologic disease and. for intraoperative monitoring during surgeries that place parts of the somatosensory pathways at risk. Abnormal SSEPs can result from dysfunction at the level of the peripheral nerve, plexus, spinal root, spinal cord, brain stem, thalamocortical projections, or primary somatosensory cortex. Since individuals have multiple parallel afferent somatosensory pathways (e.g., anterior spinothalamic tract, dorsal column tracts within the spinalcord),recordings Of SSEPs can be normal even in patients with significant sensory deficits.

SSEPs depend on the functional integrity of the fast-conducting, large-diameter ~ group IA muscle afferent fibers and group II cutaneous afferent fibers, which travel in t the posterior column of the spinal cord. When a mixed peripheral nerve (with both sensory and motor components) is stimulated, both group IA muscle afferents and group II cutaneous afferents contribute to the resulting SSEP. Selective ablation of the dorsal column of the spinal cord abolishes the SSEPs generated rostral to the lesion. Diseases of the dorsal columns in which joint position sense and proprioception are impaired invariably are associated with abnormal SSEPs.

Stimulus Location

For recording median nerve SSEPs, the nerve is stimulated at the wrist. The anode is placed just proximal to the palmar crease, and the cathode is placed between the tendons of the palmaris longus muscle, 3 em proximal to the anode.
Ulnar nerve SSEPs are preferred to median nerve SSEPs for assessing the lower cervical spinal cord segment since the ulnar nerve originates from spinal roots C8-Tl, whereas the median nerve originates from C6-Tl.

For recording posterior tibial nerve SSEPs, the nerve is stimulated at the ankle, with the cathode midway between the Achilles tendon and the medial malleolus and the anode 3 cm distal to the cathode.
peroneal nerve SSEPs, the common peroneal nerve is stimulated at the knee, with the cathode inferior to the leg crease just medial to the tendon of the biceps femoris muscle and the anode 3 cm distal to the cathode.

In the lower limb, posterior tibial SSEPs are preferred because of the following:

• In clinical diagnostic use, they are larger and less subject to variability.
• In intraoperative settings, they produce smaller muscle contractions with larger SSEP amplitudes
. • In intraoperative settings, electrodes at the ankle are more easily accessible than those at the knee The peripheral compound action potential (CAP) is recorded easily at the popliteal fossa.

Stimulation

Intensity of the selected nerves are stimulated with monophasic square pulses, 100-300 microseconds in duration. Stimuli are delivered by using either a constant voltage or a constant current stimulator. The contact impedances of the stimulating electrodes should be kept low for the following reasons:
• To minimize patient discomfort
• For more effective nerve stimulation, if a constant voltage stimulator is used

To avoid electrical artifacts with constant current stimulation in the clinical setting, the stimulus intensity is set high enough to produce a consistent muscle twitch, which usually is tolerable to the patient. Because the patient is anesthetized during intraoperative SSEP monitoring, higher stimulus intensities can be used and are advisable to provide a safety margin in case the efficacy of nerve stimulation decreases during surgery.

Stimulus Rate Rapid stimulus delivery rates should be avoided, as they degrade the waveforms of SSEPs. In clinical settings, a rate of 3-6 stimuli per second usually is used. Rates that are exact subharmonics of 50 Hz (eg, 5Hz) should be avoided, since their use would lead to contamination of the mean SSEPs by large artifacts of 50-Hz line frequency SSEPs typically are recorded by using standard EEG electrodes affixed with tape or collodion; electrode caps containing multiple recording electrodes also can be used. Scalp needle electrodes are not used routinely because of their high impedance, risk of infection, and discomfort to the patient.. Recording electrode impedances should be kept below 5000 ohms and should be as uniform as possible across the electrodes to maximize common-mode rejection and minimize noise pickup. Also, placing the ground electrodes on the stimulated limb helps reduce the electrical stimulant artifact. Typical recording amplifier filter settings for SSEPs are 30-3000 Hz. Diagnostic SSEP studies should be performed using the same filter settings used to record normative data.

Several characteristics of SSEPs can-be measured including onset lateney ,interpeak latency, morphology (i.e., presence and absence of components), and dispersion. Onset latency is the easiest SSEP feature to measure and standardize, but it gives rather limited information. Other characteristics (i.e., morphology and dispersion) are more variable and difficult to interpret.

Absolute SSEP latencies vary with limb length. Interpeak (i.e., transit) times are reliable parameters that are independent of limb length and usually independent of peripheral nerve disease. Aging is associated with some prolongation of SSEP latencies. Latencies are considered abnormal when they are more than 3 standard deviations above the mean of the normative data. '

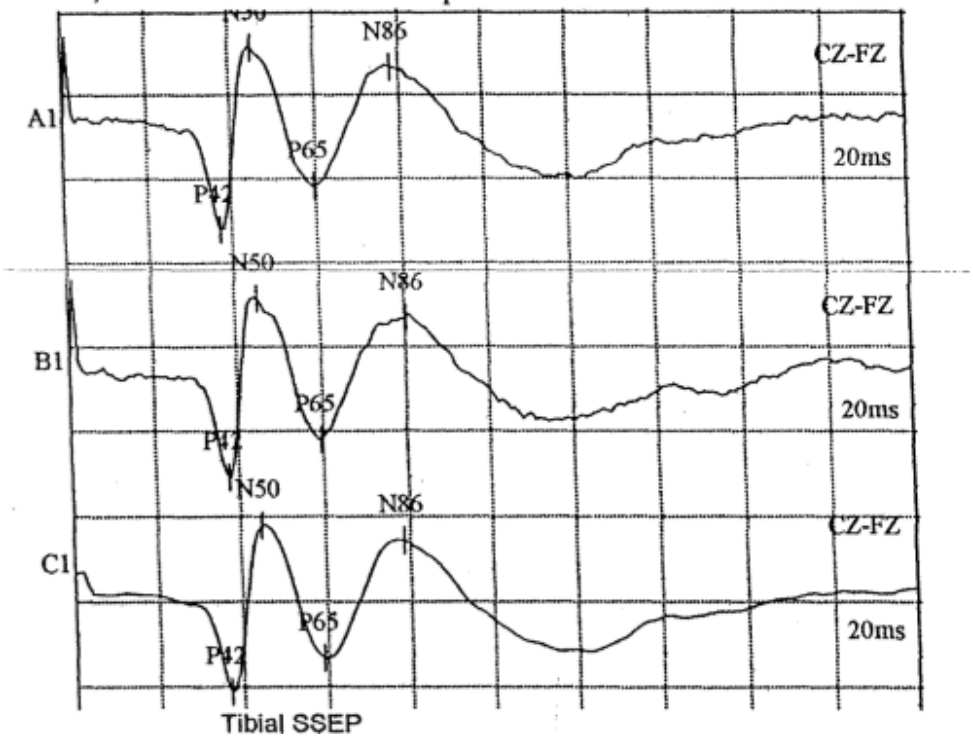
Recording electrodes sites

Anatomical landmarks identify recording electrode sites. Those on the head are defined using the international 10-20 systems, or its extension, the 10-10 systems. Electrode CP3 is midway between C3 and P3, and electrode CP4 is midway between C4 and P4. CPi denotes either CP3 or CP4, whichever is ipsilateral to the stimulated limb; CPc is the contralateral Centro parietal scalp electrode. CPz is midway between Cz and Pz.

Recording electrodes over the spine are placed in the midline, and they are labeled with the name of the vertebral body they are placed on followed by the letter S, for example, TlOS.

Recording montages for cortical SSEP components are either cephalic bipolar, in which both electrodes are placed over the head, or referential, in which a reference electrode is placed at a noncephalic site. Cephalic bipolar montages have the advantage o fbeing relatively free from noise and are preferred for routine clinical use.

For upper limbs SSEP studies, electrodes are placed over both Erb point (ie, the angle between the clavicular head of the sternocleido mastoid muscle and the clavicle), both ipsilateral and contralateral to the stimulus (labeled EPi and Epc). For lower limb SSEP studies,IC denotes an electrode placed over iliac crest.



Spinal SSEP

Electrodes placed over the spine as described above record spinal SSEPs. They are considerably smaller in amplitude than SSEPs recorded over the scalp. However, the difference in latency between the scalp and the cervical or limb SSEPs is a measure of central sensory conduction, assessment of which remains the chief clinical goal of recording SSEPs. Thoracolumbar spinal SSEPs are even smaller than cervical spinal SSEPs and can be difficult to record in obese subjects SSEP components typically are named by their polarity and typical peak latency in the normal population. For

example, N20 is a negativity that typically peaks at 20 milliseconds after the stimulus. The normal latency value for a component in a particular individual may be different from that implied by the component's name, because the lengths of the peripheral nerve and spinal conduction pathways, which vary with the patient's stature and age influence the latencies of the SSEP components.

The nomenclature of an evoked-potential component is inconsistent in the ,literature because the recording montage is not specified with the peak latency and 'polarity. For example, a CPi-EPc linkage following median nerve stimulation records a . P14, while an EPc-CPi linkage records an N14.

Intraoperative Monitoring

A comprehensive discussion of the interpretation of intraoperative evoked potential data is beyond the scope of this presentation and the reader is referred to other sources. When surgical maneuvers compromise neural tissue, SSEP components may show significant amplitude attenuation before their latencies become prolonged. Thus both amplitudes and latencies should be evaluated during intraoperative monitoring. monitoring. No universally accepted standard exists for what constitutes a significant change, but a 50% decrease in the amplitude of an SSEP component or a 10% increase in its latency often are used as threshold criteria (SSEP amplitudes generally show more run-to-run variability than SSEP latencies).

Anesthetic agents are probably the most common cause of intraoperative SSEP changes. In general, the longer the latency of an SSEP component and the more synapses between the stimulation site and component's neural generator, the greater is the degree to which that component will be affected by anesthetic agents. Thus, anesthetic effects may alter the cortical SSEPs while sparing the far-field SSEPs mimicking surgery-related dysfunction of the cerebral cortex or of the pathways from the brain stem to the cerebral cortex. Personnel performing intraoperative monitoring must pay careful attention to the anesthetic regimen and should record it periodically on their data logs.

Personnel performing intraoperative monitoring also should periodically note and log the temperature and blood pressure of the patient, which also can affect the electro physiologic signals. Anesthetic-induced changes typically are bilateral; this can help distinguish anesthetic-related from surgery-related SSEP changes when the latter are expected to be unilateral but not when surgical manipulations can damage afferent sensory pathways bilaterally (e.g., bilateral spinal cord damage during surgery for scoliosis).

Motor Evoked Potentials

Noninvasive elicitation of motor evoked potentials (MEPs) was made possible by Merton and Morton in 1980. They designed a high-voltage transcranial electrical stimulator that excited the motor cortex using cutaneous electrodes, which were placed over the scalp. After transcranial electrical stimulation (TES), a contraction of contralateral muscles is recorded in a conscious subject.

The usefulness of this method has remained limited by the local discomfort of the electrical currents that are applied over the scalp. An exception to this limitation is its use for intraoperative monitoring.

The development of transcranial magnetic stimulation (TMS) in 1985 opened new possibilities for MEP studies. Barker et al created a new type of cortical magnetic stimulator, based on the principle of electromagnetic induction. The device was composed of a main unit, which contains a bank of heavy duty capacitors the hand held part was freely moveable so that it could be placed over any part of the body . Although magnetic stimulation was used first to stimulate the peripheral nervous system (PNS) and muscles, cortical stimulation has become the focus of many studies.

Motor Cortex

The main motor cortical area is located on the anterior wall of the central sulcus and the adjacent portion of the precentral gyrus. This area corresponds to area 4 of Brodmann. It is rich in pyramidal neurons, which provide the anatomical substrates for the motor output function of area 4.

Electrical stimuli over area 4 produce activation of contralateral muscles; the face, mouth, and hand muscles occupy about two thirds of the primary motor area. The size of cortical representation of muscles is less a function of the muscle mass than of precision of the muscle movements. Secondary and tertiary areas of motor function can be mapped roughly around the primary motor cortex.

The primary motot cortex contributes more fibers to the corticospinal tract than any other region. Numerous observations support contributions from several other areas, including the frontal and parietal cortices. Ipsilateral projections are far less numerous than contralateral, estimated between 1.8-5.9% of corticospinal connections.

Pyramidal tract

Fibers of the corticospinal tract and corticobulbar tract originate from the sensorimotor cortex around the central sulcus. The human pyramidal tract contains over 1 million fibers. Most fibers are myelinated and have a small diameter (1-4 mm) only a small portion (3-5%) are large-diameter fibers (10-22 mm) that originate in Betz cells from area 4.

In humans, only 5% of the fibers of the corticospinal tract originate from Betz cells in area 4. The concept of pyramidal pathways with fibers originating only from Betz cells in the primary motor cortex has been invalidated. A large part of the corticospinal neurons have nonmotor function, especially those originating in sensory or associative areas.

Sub cortical projections of the pyramidal pathway

The Pyramidal fibers converge into the corona radiata toward the posterior arm of internal capsule. In the pons they divided into multiple longitudinal pathwayas which merge in the medulla oblongata to form the pyramidal tract after branching out efferences to motor nuclei of cranial nerves At the junction between the medulla oblongata and the spinal cord, 75-90% of the fibers cross through the midline to constitute the crossed (i.e., indirect) pyramidal pathway. The remaining fibers comprise the uncrossed (i.e., direct) pyramidal pathway. A large part of direct pyramidal tract fibers actually cross the midline at the spinal cord level (i.e., through the white anterior commissura), so that its projections are bilateral.

Magnetic Stimulation

Magnetic stimulation of the nervous system can occur only in the setting of a rapidly changing magnetic field. Subjects exposed to a constant field strength (e.g: magnetic resonance imaging [MRI]) do not experience stimulation of nervous tissue. The intensity of the secondarily produced electrical field in nervous tissue (and of the stimulation) is related to the speed of change in magnetic field strength.

Formation of the magnetic pulse starts within the main unit of the stimulator, where a large bank of heavy duty capacitors is electrically charged. When triggered, these capacitors rapidly discharge through a cable into the hand held coil, producing a brief burst of high current (up to 4000 volts [V] or several 1000 amperes [A]). The current that moves through the hand-held coil produces a large magnetic field (1-3 T) that lasts only 50-200 milliseconds.

The stimulating coil consists of tightly wound and well-insulated copper coil. As a result of the brief- magnetic field induced from the coil a secondary electric field circulates in the opposite direction to the magnetic field is produced. The strength of the electric field is related in part to the first derivative of the magnetic flux over time: the more rapid the change in magnetic field, the stronger the intensity of the secondary electric field and nervous stimulation.

Most commercially available stimulators can produce stimulations at a rate as high as 5 Hz, although some can produce repetitive stimulations as high as 50 Hz. A big advantage of magnetic stimulation over electrical stimulation is its ability to penetrate tissues regardless of electrical resistance. The drop-off is essentially the same for air, bone, fat, muscle, and saline.

The magnitude, waveform, and rise time of the magnetic field are important parameters of the stimulation. The diameter, shape, and thickness of the coil are also important. Because of these multiple variables, the measurement of intensity of · stimulation usually is expressed as a percentage of the maximal output of the stimulator.

In choosing coils, the trade-off is between strength and focality of stimulation. Coil diameter may vary between 5 cm and 15 cm. Large-diameter coils stimulate over a wider area but are less focal than small-diameter coils. With the round coils, the highest intensity electric field is measured at the edges of the coil with lower intensities in the center. To obtain more focality, use of a butterfly (also called "figure of 8" coil) isrecommended. Their focality makes them particularly suitable for the performance of mapping out the upper limb and hand musculature.

Electrical Stimulation

Electrical stimulators have a simpler design, than, magnetic stimulators. The stimulation is transmitted through cutaneous electrodes. The main advantage is a better depth of penetration, allowing direct spinal cord stimulation. The main limitation is the local discomfort that is created by the stimulation.

Electrical stimulators contain a capacitor that produces constant current, highvoltage pulses of brief duration for percutaneous stimulation. The output current range is 0-1000 milliamperes, from a source voltage as high as 400 V. The pulse width range can be varied from 50 milliseconds to 2 milliseconds. The voltage is kept constant during the stimulation but the intensity of stimulation depends on the skin im pedance.

VAGUS NERVE STIMULATION

Introduction.

Vagus Nerve Stimulation is a type of treatment for epilepsy that involves a stimulator - called a pulse generator - which is connected, inside the body, to the left vagus nerve. The stimulator sends regular, mild electrical stimulations to this nerve.

The electrical signals from the stimulator travel through the lead to the electrodes, which are wrapped around the vagus nerve in the neck. From here, the signals travel up into parts of the brain that are thought to be involved in causing seizures

The VNS Therapy system consists of the VNS Therapy Pulse Generator, the Bipolar Lead, the programming wand and software and the tunneling tool. The VNS Pulse Generator (the vagus nerve stimulator) and Bipolar Lead are surgically implanted in a procedure which takes from 30 to 90 minutes, during which time the patient is under general, regional or local anesthesia. The VNS Pulse Generator is surgically implanted in a subcutaneous pocket I the upper left chest. The Bipolar-Leads connected to the VNS Pulse Generator and attached to the vagus nerve in the lower left side of the patient's neck The patient is generally admitted to the hospital the day of surgery and discharged the same or following day

Vagus nerve

The vagus nerves are a pair of nerves that start in the brain and run through other parts of the body. They send and receive messages, between the brain and the body.

Aim of VNS

VNS aims to reduce the number, length and severity of seizures. For some people their seizures become much less frequent, for others it may reduce their seizures a little, and for others it has no effect. For some people VNS reduces the length or intensity of their seizures, but this does not happen for everyone. It may also reduce the time it takes to recover after a seizure. It is unlikely to completely stop seizures and it does not 'cure' epilepsy.

The effect of VNS does not happen straightaway; it can take up to 2 years for it to have an effect on someone' s seizures. VNS is used alongside anti-epileptic drugs (AEDs) not instead of them. If VNS works, it may be possible to reduce a person's AEDs over time.

VNS Pulse Generator

The Pulse Generator of the VNS Therapy system is an implantable, programmable, cardiac pacemaker-like signal generator designed to be coupled with the bipolar lead to deliver electrical signals to the vagus nerve. The VNS Pulse Generator (vagus nerve stimulator) employs a battery which has an expected life of approximately eight to ten years at standard stimulation parameters. Upon expiration of the battery, the VNS Pulse Generator is removed and a new generator is implanted in a short, out-patient procedure using local anesthesia.

Because of the size of the stimulator there will be a small lump where it lies, and a small scar where it was put in. A lead connects the stimulator in the chest to the vagus nerve in the left side of the neck. Because the electrodes are coiled around the nerve in patent's neck, there will be a small scar where they are inserted, usually in the fold of patent's neck

Bipolar Lead

The lead incorporates patented electrodes which are self-sizing and flexible, minimizing mechanical trauma to the nerve and allowing body fluid interchange within the nerve structure. The lead's two electrodes and anchor whether wrap around the vagus nerve and the connector end is tunneled subcutaneously to the chest where it is attached to the VNS Pulse Generator. The leads, for the VNS Therapy, are available in two sizes of inner spiral diameter to ensure optimal electrode placement on different size nerves.

Programming Wand and Software

The VNS Therapy system includes a proprietary programming wand and software, used to interrogate the device and to transmit programming information from a personal computer to the VNS Pulse Generator via electromagnetic signals. These products are compatible with both Pentium and non-Pentium based platform personal computers. Programming capabilities include modification of the VNS Pulse Generator's programmable parameters (pulse width, output current, signal frequency and stimulation duration and interval) and storage and retrieval of telemetry data. The VNS Therapy programming wand can be connected to a standard personal computer using a serial connector.

Tunneling Tool

The tunneling tool is a single use sterile, disposable surgical tool designed to be used during surgical placement of the Bipolar Lead. This VNS Therapy tool is used for subcutaneous tunneling of the lead assembly between the nerve site in the neck and the VNS Pulse Generator site in the chest.

Accessory Pack

The VNS Therapy system also includes an Accessory Pack comprised of one Pulse Generator resistor assembly used to test the function of the device prior to implantation, the VNS Bipolar Lead tie-downs, one hex screwdriver, two setscrews and setscrew plugs.

Working of VNS during seizure

Some people have a warning or aura a type of simple partial seizure-that tells them that they are going to have a seizure. When this happens, passing a special magnet over the stimulator gives extra stimulation. This may stop the aura from developing into another seizure, or may reduce the length of the seizure, or the recovery time afterwards. This magnet can be worn on patent's wrist like a watch, or on patent's belt. For people who have no warning before a seizure, someone else could use the magnet when a seizure happens

Side effects

VNS can cause side effects but usually only during the time that the nerve is being stimulated. Side effects may not happen for everyone but include discomfort in the throat, a cough, difficulty swallowing and a hoarse voice.
Side effects usually reduce over time and do not usually mean that the VNS has to be switched off. Your doctor or nurse can adjust the settings if the side effects are a problem to you. If the side effects cause problems at certain times, such as difficulty swallowing when you are eating, then holding the magnet over the stimulator for a few seconds briefly stops the stimulation, which should stop the side effects. VNS does not affect, and is not affected by, anti-epileptic drugs. Some people feel that VNS enhances their mood, memory alertness, and may also help reduce depression or have a positive effect on their quality of life.

Precautions

If patient have VNS and need an MRI, it is important that everyone involved with the scan is aware of patent's VNS, to decide if the scan can be done. Magnetic Resonance Imaging (MRI) uses strong magnetic fields to take images of the brain. As the VNS includes metal, there is a risk that the magnetic field in the MRI machine could cause the electrodes to heat up. The risk of this depends on the MRI machine used. Some types of MRI is may be possible. If they just scan patients head rather than scanning patent's whole body and if they do not scan patent's neck or chest - where the VNS lies.

If the scan can be done, the VNS will need to be switched off before the scan, and turned back on again afterwards. X-Rays and CF scans do not affect and are not affected by VNS

OPTOMETRY

Introduction

Optic nerve is the most important nerve which carries visual impulses from the retina to the optic chiasma and on the optic tract to the lateral geniculate body to act as the afferent pathway for the papillary light reflex by means of fibres traveling to the superior colliculus of the mid brain. The major test performed in the optometry is: (i) To measure the acuity of vision and to determined any defect is due .to local ocular disease. (ii) To chart the visual fields

VISUAL ACUITY

The standard Snellen's Type of charts are used for testing distant vision and the Jaegar types cards for near vision. The Snellen's type chart is placed, evenly illuminated, 6 metres (20 feet) from the patient, who covers one eye and is asked to read the smallest line he/she can see accurately. Acuity is recorded as a fraction (6/24). The numerator indicates the distance at which the patient has to be from the chart in order to read the same type that the normal person could read at a distance indicated by denominator. 6/5 - 6/6 are within the average normal range. The Jaegar type card must be held one foot from the patients eye and a similar test is then carried out. The different types are labeled as N6, N24etc according to their size. The average acuity lies between N6 and N9.

VISUAL FIELD

Charting the visual field is the most important method of locating a lesion in the visual pathways or of interpreting certain fundus appearances, yet in clinical notes it is often difficult to find evidence that any attempt has been made to examine them at all. The purpose of the test is to chart the periphery of the visual fields; to determine the position, size and shape of the blind spot and any abnormal scotomata; to compare any defects shown with those abnormalities known to be reproduced by lesions at specific points in the visual pathways. The principles by which lesions are located are thus illustrated by comparing a diagram of the visual pathways with the principal field defects.

BOTULINUM TOXIN INJECTION

Botulinum toxin, a toxic protein produced by the bacterium Clostridium botulinum, causes botulism, a deadly form of food poisoning. But in tiny quantities, the toxin acts as a muscle-relaxing agent by blocking the release of acetylcholine in the nerve endings. Acetylcholine transmits electrical messages between the brain and the muscles. When the botulinum toxin blocks its release, the messages are not delivered. As a result, muscle contractions and associated pain are reduced.

Minute amounts of the toxin are injected directly into the muscle responsible for the spasms. The toxin weakens the muscle, but does not paralyze it, and allows the affected area to resume a more normal position. Pain from prolonged muscle contractions is eased. The treatments can be used in the eyelid to treat blepharospams, and in muscles elsewhere in the body, such as the arm or leg.

Botulinum toxin types A and B are available and effective in treating focal or segmental dystonias. Botulinum toxin has seven forms (serotypes): A, B, C, D, E, F, and G.
Botulinum toxin injections are not a cure, but they usually ease symptoms in the injected muscle in seven to 10 days. Patients usually receive maximum benefit one to two weeks after the injections. The effects can last several months, and the treatments can be repeated, often indefinitely.

A very fine needle is used for the one to three injections that are normally given per muscle. Discomfort from the injections, if any, is usually minor and temporary. Sometimes the needle is instated with the assistance of EMG to correctly locate the muscle.

Because symptoms vary during the course of dystonia, the treatment's effectiveness and duration vary from patient to patient. Subsequent injections may produce results that are less dramatic than the first, and doses may have to be adjusted. Identifying and injecting the affected muscle is not always easy.
In rare cases, a patient can develop antibodies to tl1e botulinum toxin proteins, rendering the treatment ineffective.

Botulinum toxin treatments should not be used by pregnant or nursing women or by people taking certain medications. Side effects to the treatment include a temporary muscle weakness and discomfort at the injection site.

Technologists' role in patient safety

It is important to understand that there is a potential hazard during the performance of a electrophysiological procedure. The electricity can kill a patient if the equipment is not properly maintained or grounded and if adequate precaution are not taken.

The following precautions are to taken for electrical safety while doing electrophysiological procedures.

> The power receptacle should have three holes. > Unnecessary electrical equipment should be kept away from the lab. >Whenever new equipment is installed, it should be thoroughly checked for safety.
) Use wooden examination table, avoid a metal table.
> The patient should not make any contact with a metal object, grounded or ungrounded and any part of the equipment.
> Never connect a second power line operated apparatus to the patient.

Any deliberately grounding the patient, we subject the patient to the risk of electrical shock into contact with a live voltage or with a current carrying wire. So ensure proper grounding for both the machine and the patient. When an electrical device already connected has a ground of its own on the patient, do not attach an EEG ground to the patient but use the already existing ground instead. Ground loop causes Current to flow through the patient's body. lf this results in unacceptable high levels of 60/EIJ Hz artifacts, remove the existing ground lead and replace it with the standard EEG electrode placement usually the center of the patient's forhead.

While doing nerve conduction studies and EMG place the ground electrode over the testing limb.

EEG-fMRI

EEG-fMRI (short for EEG-correlated/fMRI/ or electroencephalography-correlated functional magnetic resonance imaging) is a multimodal neuroimaging technique whereby EEG and fMRI data are recorded continuously and synchronously for the study of electrical brain activity incorrelation with haemodynamic changes in brain during the electrical activity, be it normal function or associated with disorders.

Principle

Scalp EEG reflects the brain's electrical activity, and in particular post-synaptic potentials (see Inhibitory postsynaptic current and Excitatory postsynaptic potential) in the cerebral cortex, whereas fMRI is capable of detecting haemodynamic changes throughout the brain through the BOLD effect. EEG-fMRI therefore allows measuring both neuronal and haemodynamic activity which comprise two important components of the neurovascular coupling mechanism.

Methodology

The simultaneous acquisition of EEG and fMRI data of sufficient quality requires solutions to problems linked to potential health risks (due to currents induced by the MR image forming process in the circuits created by the subject and EEG recording system) and EEG and fMRI data quality. There are two degrees of integration of the data -acquisition, reflecting technical limitations associated with the interference between the EEG and MR instruments

These are interleaved acquisitions, in which each acquisition modality is interrupted in turn (periodically) to allow data of adequate quality to be recorded by the other modality, continuous acquisitions, in which both modalities are able to record data of adequate quality continuously. The latter can be achieved using real-time or postprocessing EEG artifact reduction software. EEG was first recorded in an MR environment around 1993. Several groups have found independent means to solve the problems of mutual contamination of the EEG and fMRI signals. The first continuous EEG-fMRI experiment was performed in 1999 using a hardware-based approach. A predominantly software-based method was implemented shortly thereafter. An addition to EEG-fMRI set up is the simultaneous andsynchronized video recording without affecting the EEG and fMRI data quality for the most part, the combined EEG-fMRI data collection is now treated as a solved problem, and commercial devices are available from major manufacturers (e.g., Electrical Geodesies, Inc.), but issues remain. There are significant residual artifacts in the EEG that occur with each heartbeat. The traces in the EEG that record this are often referred to as a, "Ballistocardiogram (BCG)," because of their presumed origin in the motion of the EEG leads in the magnetic field that occurs with each heartbeat. In practice, the causes of this artifact are not well proven and may be the results of factors such as induced electrical fields with the movement of blood, etc ...

Applications

In principle, the technique combines the EEG's well documented ability to characterise certain brain states with high temporal resolution and to reveal pathological patterns, with fMRI's (more recently discovered and less well understood) ability to image blood dynamics through the entire brain with high spatial resolution. Up to now, EEG-fMRI has been mainly seen as an fMRI technique in which the synchronously acquired EEG is used to characterise brain activity ('brain state') across time allowing to map (through statistical parametric mapping, for example) the associated haemodynamic changes.

The initial motivation for EEG-fMRI was in the field of research into epilepsy, and in particular the study of interictal epileptiform discharges (lED, or interictal spikes), and their generators, and of seizures. IEDs are unpredictable and sub,clinical events in patients with epilepsy that can only be observed using EEG (or MEG). Therefore recording EEG during fMRI acquisition allows studying their haemodynamic correlates. The method can reveal haemodynamic changes linked to lED and seizures, and has proven a powerful scientific tool. The simultaneous and synchronized video recording identifies clinical seizure activity along with electophysiological activity on EEG, which helps to investigate, correlated hemodynamic changes in brain during seizures.

The clinical value of these findings is the subject of ongoing investigations. Outside the field of epilepsy, EEG-fMRI has been used to study event-related (triggered by external stimuli) brain responses and provided important new insights into baseline brain activity, scoring resting,, wakeTulness and sleep.

LOG BOOK

EEG

NO	NAME	AGE/SEX	HOS NO	DIAGNOSIS
1	Anuja A S	13/F	383141	? FLE
2	Nasiya M	13/F	382919	?SSPE
3	Ajeesha J	10/F	343847	BRE
4	Ramachandran Nair	77/M	383192	Transient global amnesia
5	Sasidharan Nair	63/M	382438	Metabolic Encephalopathy
6	Devpal Suresh	17/M	267788	CPS ET left Hemisphere
7	Binoy Bhaskar	17/M	9608818	CPS ET PHR gliosis
8	Syama Rajagpal	26/F	212168	JME
9	Abbay	12/M	334778	Maternal epilepsy
10	Sanmugham Ravi	19/M	383126	PME
11	Nicymol Simn	14/F	320526	Status Epilepticus
12	Sibin Jose	19/m	383279	PGE V/S SGE
13	Riya Unnithan	15/F	383162	IGE
14	Gokul m gopalan	11/M	374694	Syncope
15	Shobhana Devi L K	49/F	383134	PNES

16	Jeffrey Jasper	4/M	352124	LGS
17	Priyanka S Pillai	23/F	383294	SPS
18	Gopinathan Pillai	72/M	382705	SZ V/S SYNCOPE
19	Abhimod S	23/M	383041	Migraine
20	Parvathy S	23/F	383281	LRE
21	Muhammed Rifas	5/M	381739	GDD
22	Rohan Vijay	8/M	379430	OLE
23	Sasidharan Nair K	62/M	382438	Acute onset Encephalopathy
24	Albin George	11/M	383265	Static Myclonous
25	Muhammed Sali	74/M	353990	Rigth MCA Stroke

26`	Vishnu R Nair	20/M	381188	CPS ET Right
27	Ashik Ali	32/M	307714	TLE
28	Aswana	10/F	310110	LKS
29	Mahadevan Jayaghosh	26/M	344402	Doose Syndrome
30	Amina abdul Kabir	18/F	9710639	Tuberous Sclerosis

31	Disha Sunil	6/F	332883	BLRE
32	Kiruthik Hari Eswar K S	1/M	383385	Epileptic Encephalopathy
33	Prabha K	21/F	2999936	Autoimmune Encephalities
34	Jiswa M	3/F	382334	Symptomatic Seizures
35	Binu S	22/M	223593	LKS
36	Satyajit Rajan	53/M	383836	TLE
37	Sreelal L	19/M	379417	CPS
38	Reshma Chandran	22/F	382511	Migraine with PNES
39	Abhinand Sen	6/M	370764	JAE
40	Ganesh B	5/m	383595	Jeavons syndrome
41	Dhruv J	1/M	367720	HIE
42	Kiruba Elengelina	11/M	383983	Viral Encephalitis
43	Raja N	11/M	360989	West Syndrome
44	Aysha S	11/F	394030	Recurrent ADEM
45	Suby P Babu	25/F	374607	JME

46	Sruthy M	24/F	383674	CPS ET
47	Saravanan R	37/M	384043	CPS Pseudo Temporal
48	Gokul R	17/M	383443	CPS ET Right Parietal
49	Anand R	22/M	313374	Reflex Eating Epilepsy
50	Keerthana M nair	13/F	276464	BRE
51	Sudheer Khan	34/M	266150	Right Parietal AVM
52	Bala Babujee	38/M	346135	CPS Temporal Plus
53	Ansila Sherin	11/F	384299	B OE
54	Dr Rejith R S	35/M `	384263	EPC
55	Nayana A C	15/F	384098	Progressive Myoclonus epilepsy
56	Pallavi S	19/F	222260	Left MTLE
57	Rishana Shamsi	28/F	332985	CPS ET Frontal
58	Sheeja Beegum	38/F	324294	CPS Bilateral MTS
59	Muthulakshmi S	60/F	375994	Bifrontal Meningioma
60	Aliyas NAzrin Anas	11/F	384438	Symptomatic Seizure

61	Anusree K	6/M	379763	BLRE
62	Anadhu A	15/M	383438	Post Traumatic Epilepsy
63	Shameena	10/F	244407	Right Fronto Opercular FCD
64	Abdul Rasheed V K	22/M	375962	Insular Glioma
65	Azzam Saleh Hamed Al Amri	8/M	352472	GEFS
66	Mitapilli Ramu	25/M	384343	Parkinson disease

67	Raghunanthan C R	20/M	384354	Meningitis
68	Arsha Raj	10/F	384105	Autoimmune Encephalitis
69	Amirutham J	75/F	382253	Right MCA stroke
70	Muhammed Mussammil	9/M	337423	ADEM
71	Abhinav pramod	7/M	`240174	Atonic BRE
72	Merin Lenoy	16/F	217421	PGE
73	Pratheek Babu	9/M	336895	OLE
74	Ammini Samuvel	76/F	383577	Cerebellar Lesion
75	Bridgit A	19/F	302883	MTS

76	Anadan O	39/M	376236	Eating epilepsy
77	Mini A	39/F	384324	MTLE
78	Iyyathumma C	65/F	384618	Dementia
79	Anjali K	10/F	243726	FLE
80	Naseera V	27/F	379499	Astrcytoma
81	Jibin Subu	10/M	383960	Arachnid Cyst
82	Anand P	24/M	328810	JME
83	Petachaimmal M	18/F	376554	CPS ET
84	Jithin P Jose	27/M	384972	PGE
85	Karthykeyan G	65/M	384928	Hepatic encephalopathy
86	Dhanish G	02/M	384913	GDD
87	Akhila s	18/F	385084	PGE v/s SGE
88	Abhiram S k	5/M	319767	LKS
89	Akhila A	17/F	201297	CAE
90	Kunhu Muhammed	64/M	384990	R/o CJD

91	Sreedhanya C S	12/F	338438	FLE
92	Jayaram S	27/M	385021	CPS PHR
93	Rithin Krishna P R	1/M	385345	Tonic Status Epileptics
94	Shameena	7/F	385388	Absence Epilepsy
95	Issac C K	61/M	21732	TIA
96	Aclith Sekhar A V	6/M	324400	Autistic spectrum Disorder
97	Sujithra S	27/F	385366	PNES
98	Rahul C	16/M	384207	Static Encephalopathy
99	Geethu C P	18/F	385779	Syncope v/s Seizure
100	Dr Ravi Kumar	56/M	242761	TLE with IGE

VIDEO EEG

SL NO	PATIENT NAME	AGE/SEX	HOSPITAL NO	DIAGNOSIS
1	Daksh. A.A	1/M	382924	Jeevan syndrome
2	Chris George	9/M	353551	BRE v/s LRE
3	Anilkumar K.V	44/M	382590	IGE
4	Vidyavathy.S.M	44/F	384507	PNES
5	Nisha Negi	23/F	357578	Post encephalitic sequlae
6	Praveen kumar	26	384244	CPS-ET
7	Jyothimol M	33/F	384241	PGE v/s SGE
8	Vikas Baghel	7/M	368361	CPS-ET left
9	Saif Mohammed	23/M	384773	RIGHT-MTS
10	Jibin Subu	10/M	234332	PNES
11.	Saketh M V	11/M	833430	Atonic BLRE
12	Anju Prasad	11/F	357452	LKS
13	Adhulya S.P	11/F	385182	Post encephalitis Sequlae
14	Srijana.M	28/M	382745	PNES
15	Gokul.G	27/M	380261	Right MTS

16	Dipak Vijay	3/M	382943	Right frontal FCD
17	Minaxiben S	17/F	382969	JAE
18	Milan C.Byju	2/M	382963	CPS-ET
19	Vysakan I K	21/M	214961	FLE
20	Soumya G Nair	30/F	360634	CPS-Temporal
21	Himanshu Shekar	24/M	383128	PGE-JME
22	Saleemudheen V	27/M	381828	PGE v/s SGE
23	Jagadish Prasad	45/M	383167	Left MTS
24	Sanvi Reddy	7/F	383309	CPS-ET left
25	Samiya M Kherani	9/F	316855	LGS
26	Nikita Bhagath	14/F	304427	Post encephalitic sequlae
27	Krishna.H.S	9/M	383291	Bilateral PHR gliosis
28	Shivanya Biju	5/F	382942	Static encephalopathy
29	Silpa.K.V	24/F	331060	IGE
30	Vishakha Mishra	10/F	383408	CPS-ET+PNES

31	Apoorvaa S Nair	15/F	383289	Eating Epilepsy
32	Saraladevi Iyer	53/F	383322	Right MTS
33	Riya Fathima T	6/F	350643	LGS
34	Amina Noushad	1/F	386032	LRE left
35	Alpha Nath G N	23/M	370809	?JME
36	Biju B	18/M	304182	FLE
37	Dipankar Bhattacharya	41/M	263013	Left MTS
38	Karthuk.S	14/M	385999	PNES
39	Snadesh U S	23/M	382517	JME
40	Adithyan S Babu	9/M	371755	Right MTS
41	Sumat Kumar Jain	64/M	386145	Syncope v/s Seizure
42	Jovanna Joseph	12/F	385167	Epileptic encephalopathy
43	Akshaya Manoj	14/F	386177	PNES
44	Ashik Sabu	8/M	386165	Symptomatic LGS
45	Nidhina P V	25/F	379558	FLE

46	Ashik N U	12/M	383320	CPS-ET frontal
47	Roshnamol K	17/F	386163	CPS-ET ?PHR
48	Laksmi Deshmukh	10/F	386349	Reflex Epilepsy
49	Dawn Sabu	9/M	373944	Symptomatic LRE
50	Vani S Sivan	11/F	369380	Myoclonic seizures
51	Sebasatian Joshi	9/M	385746	Atypical BLRE
52	Keerthana S	3/F	385400	Static encephalopathy
53	Thasbeeha J N	19/F	378479	PGE v/s SGE
54	Shica Ann Joseph	25/F	231515	IGE with GTCS
55	Jismy K.B	22/F	386521	Right MTS
56	Devu P.S	10/F	386841	Atypical BRE
57	Havish Varma.N.S	3/M	386769	Epileptic encephalopathy
58	Ayush Kunwar	14/M	369164	Bilateral PHR gliosis
59	Sreeresh.C	32/M	244824	TLE with psychosis
60	Aby Abraham	34/M	387032	PGE ?JAE

61	Amijith Ramesh	14/M	388631	Static enecephalopathy
62	Naksh Hareshbhai	2/M	390364	GDD
63	Sritama Dey	10/M	390197	Pediatric TLE
64	Tanvi Chaturvedi	24/M	390232	IGE(Absence)
65	Suhavi Kaur Bhati	6/F	390485	Rasm encephalitis
66	Daiwik Babu T	1/M	390262	Symptomatic LRE
67	Moksh Chiragbhai Patel	5/M	390552	Post traumatic sequlae
68	Adarsh M P	15/M	383978	TLE
70	Sulaiman C.P	44/M	389831	Psychogenic myoclonus
71	Shruthy.M	24/F	383674	Probable IGE
72	Zeel Umesh K	13/F	30385	FLE
73	Kh Jarin Tasnim Momoka	10/F	390618	FLE
74	Anju Soye	15/F	296373	Right MTLE
75	Akshay N Borkar	17/M	390712	PME

76	Nidhi Dipak Kumar Vyas	9/F	349150	FLE
77	Visal Thivari R	21/M	373135	Left MTS
78	Ranga Suhas Sai.S	8/M	390716	CPS-Temporal
79	Utsab Chatterjee	25/M	347403	CPS-ET
80	Jisha.S	30/F	9800046	PGE
81	Muhammed Rameez	12/M	379601	CPS-Left frontal
82	Pavisri M.K	1/M	390715	West syndrome
83	Aadidev A	2/M	373212	West syndrome
84	Visakh.V	10/M	390817	PNES
85	Aamina.Z	14/F	381332	PGE v/s SGE
86	Anwar.M	24/M	389115	Right MTS
87	Jubal Joseph Joby	5/M	390947	CPS-Left PHR
88	Yash Pratap Singh	16/M	390972	PGE
89	Deepinder Singh	3/M	391061	Symptomatic LRE
90	Krish Harshbhai Vekariya	8/M	391066	CPS-Temporal

91	Poooja Sharma	17/F	363392	Rasmoosan encephalitis
92	Dinesh Deshmukh	42/M	391043	Right MTLE
93	Ram Shukla	10/M	391021	CPS-ET
94	Jenifer .A	11/F	386399	CPS-FLE
95	Mohammed Suhail	12/M	347918	FLE+PNES
96	Prabhu Devan.N	21/M	389509	CPS-Frontal
97	Farwa.K	9/M	321457	CPS-Temporal plus
98	Krushita Ashok	10/M	391332	Static encephalopathy

99	Muhammed Ali	8/M	363897	Symptomatic generalized epilepsy
100	Shanil.C.P	30/M	302841	TLE
101	Philomina K.J	31/F	391871	FLE
102	Arya.A.S	18/F	378067	PNES
103	Bhat Aijaz Ahmed	25/M	375381	CPS-Temporal plus
104	Heel SureshChangela	16/M	374310	CPS-ET
105	Tibin Alex	9/M	391688	FLE

106	Shashank Asshish Shek	7/M	297106	Symptomatic generalized epilepsy
107	Nazim Salim	17/M	392263	JME
108	Arnav Suryavanshi	12/F	392427	FLE
109	Prabhakaran.V	38/M	390437	Late onset seizures
110	Mini.P	38/F	387319	PNES
111	Vijiyachandran Menon.A.G	53/M	391412	CPS-Temporal
112	Kanchan Suresh Bhujade	21/F	392392	JME
113	Haasan.M.D	6/M	392431	Atypical BLRE v/s Symptomatic LRE
114	Daksh Kulshrestha	4/M	372703	Static encephalopathy

115	Jayasree	30/F	350132	CPS-Temporal plus
116	Rahul Kaurav	18/M	381611	CPS-ET
117	Mohammed Risaladar T.S	14/M	380778	Post encephalitis sequlae
118	Hiranmaya Lal	41/M	316690	Cryptogenic west syndrome
119	Nirmal.S	13/M	381163	Pseudotemporal v/s temporal
120	Jay Rajunayyer	15/M	381901	Reflex eating epilepsy

ELECTROCORTICOGRAPHY

NO	NAME	AGE/SEX	HOS NO	DIAGNOSIS
1	Hariharan K	10/ M	3299886	Left Occipital lobectomy
2	Vivek Kumar jha	22/ M	361237	Right ATL +AH
3	Naina Joseph	14/F	362589	Right frontal lesionectomy
4	Saukat SK	22/ M	356113	Right frontal FCD
5	Nandu Suresh	13/ M	282470	Right parietal lesionectomy
6	Tirumala Chokkapu	24/ M	357093	Left parietal gliosis
7	Hemalatha vagtaram suthar	14/ M	365284	Left ATL +AH
8	Abhishek Bhattacharya	23/ M	312486	Right frontal dysplacia
9	Jadeja Kuldeep singh	31/ M	373068	Right occipital gliosis
10	Sheikh Ahsan	7/ M	372022	Left hemisphereoctomy
11	Yasar C	26/ M	351010	Right Temporo parieto resection
12	Rahul K R	23/ M	366961	Insular Lesion
13	Firasha Shareef	30/F	371140	Temporal lesinoctomy
14	Sabari	22/ M	9501868	Occipital lobectomy
15	Vishnu P K	17/ M	346211	Right Parieto occipital lesinoctomy

16	Asish Verma	19/ M	348179	Perisylvian lesionctomy
17	Sagar Tyagi	12/ M	379187	Left frontal grid Placement
18	Sushil oli	20/ M	382424	Left temporal grid placement
19	Jagadish Prasad	45/ M	383167	Orbto frontal grid placement
20	Suparnika Baji	6/F	319653	Left parietal craniotomy

POLYSOMNOGRAPHY

SL NO	PATIENT NAME	AGE/SEX	HOSPITAL NO	DIAGNOSIS
1	Raj G	45/M	385938	EDS
2	Nalini K	64/F	385207	? OSA
3	Mohankumar	69/M	203717	Severe OSA
4	Pradeep A	39/M	387732	OSA
5	Lal J L	20/M	384950	OSA
6	Lissy Jacob	47/F	387542	EDS

7	Jessel Vinober	42/M	283781	OSA
8	Ananandan K	49/M	231612	OSA
9	Mathew P Rajan	52/M	390510	OSA
10	Habeepbulla M	59/M	391145	OSA
11	John Joseph	54/M	397277	EDS
12	Unnikrishnan V	39/M	395881	OSA
13	Anamika Sabu	10/F	391717	Narcolepsy
14	Salini S	40/F	397982	OSA
15	Sreelatha J V	41/F	397278	OSA
16	Rajesh V S	48/M	383226	OSA
17	Prasanth S	41/M	397280	OSA and EDS
18	Jyothi K S	49/F	397983	OSA
19	Leela G	54/F	397635	OSA
20	Shaijan A	44/M	398832	OSA

Continuous Positive Airway Pressure

SL NO	PATIENT NAME	AGE/SEX	HOSPITAL NO	DIAGNOSIS
1	Ramachandran Nair	67/M	397280	OSA
2	Mohandas	62/M	391214	OSA
3	Habeepbulla	59/M	391195	OSA

4	Sasidharan B	73/M	384407	OSA
5	Sameer Mustahafa	43/M	9804254	Severe OSA
6	Binod Hediya	53/M	394777	OSA
7	Shaji M	37/M	391483	OSA and snoring
8	Shaji Kumar S	47/M	384623	EDS and OSA
9	Jayachandran Pillai	48/M	392224	Snoring
10	Gopalakrishnan	49/M	389140	OSA

MULTIPLE SLEEP LATENCY TEST

SL NO	PATIENT NAME	AGE/SEX	HOSPITAL NO	DIAGNOSIS
1	Siddique	51/M	394634	Narcolepsy
2	Saj Murali	41/M	392156	Idiopathic hypersomnia
3	Jayarani K S	43/F	312468	Narcolepsy
4	Adith Jayaprasad	16/M	400622	Narcolepsy and cataplexy
5	Lissy jacob	47/F	387542	Narcolepsy
6	Anandu K R	7/M	381328	Hypersomnolence
7	Keerthi Devan	19/F	379775	Idiopathic hypersomnolence
8	Vijay Mohan	58/M	267190	EDS

9	Venugopalan K S	57/M	373393	EDS
10	Prahaladan	59/M	366541	Periodic hypersomnolence

EMG

SL NO	PATIENT NAME	AGE/SEX	HOSPITAL NO	DIAGNOSIS
1.	Sneha Philip	18/F	360002	CIDP
2	George Roxam.T.J	24/M	391427	To r/o neuropathy
3	Mohanan.G	47/M	389892	Bells Palsy
4	Jumaila.S	56/F	9809427	Thoracic outlet syndrome
5	Moli.G.K	49/F	391031	Lumbosacaral plexopathy
6	Chithresh P	18/M	390459	Motor neuron disease
7	Abdul Mohammed	37/M	390837	Thoracic outlet synodrome
8	Suresh.S	45/M	391516	Diabetic neuropathy
9	Sindhu.M	28/F	388071	Right C7 plexopathy
10	Arunima.V	16/F	382966	Mononeuritis multiplex
11	Vamadevan.Nair	61/M	391642	To r/o neuropathy

12	Nandi.M	60/M	385495	Left CTS
13	Narayanan.K.M	58/M	391846	Motor neuron disease
14	Jithesh Kumar Lia	50/M	391921	Diabetic neuropathy
15	Velammal	62/F	391932	AIDP
16	Tressa Pereira	67/F	392025	Miller fisher syndrome
17	Dricily.S	36/M	390500	Right C7 radiculopathy
18	Sreevalsan N.S	49/M	216380	Cervical radiculopathy
19	Sayanora.V	14/F	290487	CIDP
20	Rani.M	54/F	390469	?CTS
21	Mohan Das.P	54/F	390824	Probable diabetic polyneuropathy
22	Velayudhan Sambasivan	53/M	392140	Lumbar plexopathy
23	Adarsh.R	4/M	389398	Dysstrophenopathy
24	Velammal.K	62/F	391932	GBS
25	Vijayakumar.P	43/M	382719	Brachial Plexus
26	Selvam.M	34/M	392762	Mononeuritis multiplex
27	Chandra Mohanan P	50/M	319938	To r/o CTS
28	Valsala Bhas.J	59/F	358945	Left S1 radiculopathy
29	Vasudevan Nair.C	72/M	391352	Sensory motor polyneuropathy
30	Thomas Samuel	70/M	358600	Motor neuron disease

31	Salahudheen.K	64/M	396090	Diabetic peripheral neuropathy
32	Rema Devi.B	44/F	396511	Bilateral CTS
33	Vijayan.R	60/M	397264	Left brachial plexus
34	Bahulan.N	75/M	329980	Diabetic peripheral neuropathy
35	Sreedevika R	5/F	397303	GDD
36	Mala.P	35/F	397272	Posterior interosseus nerve palsy
37	Muralidharan	48/M	396245	Critical illness neuropathy
38	Shruthi Muniyappa	34/F	397708	CIDP
39	Lekha.S	38/F	397217	Cevical myopathy
40	Leelamony.P.C	64/F	397048	Bulbar onset MND
41	Chitirai Kani.M	50/F	398772	Bulbar onset MND
42	Sulochana	54/F	399256	Sensory neuropathy
43	Raja.P.C	67/M	399090	Mononeruitis multiplex
44	Gangadharan Pillai.G	65/M	397890	To r/o peripheral neuropathy
45	Anitha Chandrababu	47/F	399979	Bilateral lower limb weakness

46	Gopalakrishnan.S	47/M	9510082	Lumbar spondylosis

47	Shivani.J	6/F	376706	GBS
48	Sreelakshmi.K.S	8/F	397528	Myotonic dystrophy
49	Murugeshan.T	14/M	378829	CIDP
50	Padmini Nair	68/F	397997	?Meralgia Parasthetica
51	Raveendran.B	75/M	392949	Stiff leg syndrome
52	Sumadevi.G	29/F	378646	Bilateral CTS
53	Annamma Abraham	75/F	398398	Mononeuritis multiplex
54	Abhinav.S	9/M	398022	To r/o myopathy
55	Ponnu.M	46/M	398241	Motor neuron disease
56	Asha Johnson	17/F	396705	Peripheral neuropathy
57	Nalini.K	58/F	397553	ALS plus
58	Blaze Mary Varghese	28/F	396252	CTS
59	Jibin Varghese	29/M	397443	Neuropathy v/s Radiculopathy
60	Sulochannan.K	73/M	9608358	Peripheral neuropathy

61	Vijayamma B	64/F	381338	Right Ulnar neuropathy
62	Rihan R	1/M	385932	GBS
63	Sreelatha P N	47/F	386070	Bell's Palsy
64	Sijij George	44/M	386172	Superficial Radial neuropathy
65	Umaudeen A	61/M	384203	Mononeuritis multiplex
66	Sheeja K	44/F	362892	CTS
67	Joseph C	56/M	276505	C5-C6-C7 radiculopathy
68	Prabhakaran Nair	67/M	384350	L5 Radiculopathy
69	Sunil J V	43/M	386231	Diffuse AHC
70	Aatif Abdul Karim	19/M	384959	HMSN
71	Anitha S	50/F	209561	Peripheral Neuropathy
72	Madhukumar	50/M	381148	Right upper trunk brachial plexopathy
73	Sahadevan N	65/M	383889	Diabetic peripheral neuropathy
74	Lilly kutty Thomas	40/F	386311	MND
75	Raji George	58/M	385407	Meralgia Parasthetica

76	Chandra sekhar	37/M	386028	HNPP
77	Leena V	48/F	3785992	L5-S1 Radculopathy
78	Athul Ramesh	13/M	313625	Metachromatic leukoystrophy
79	Murugesan	32/M	386804	CIDP
80	Nandini	45/M	280174	Sensory Neuropathy
81	Joshi P C	54/M	386446	SCA
82	Anil Kumar	47/M	385407	C7 Radiculopathy
83	Gireesan V	58/M	386028	Lumbar Spondylosis
84	Jayasree	37/M	3785992	RRMS
85	Suvinraj	48/F	313625	Meralgia Parasthetica
86	Akhila G Das	13/M	386804	HNPP
87	Syed Ibrahim	32/M	280174	L5-S1 Radculopathy
88	Manikuttan	45/M	386446	Metachromatic leukoystrophy
89	Athul A S	54/M	160841	CIDP
90	Josemon Y	47/M	385639	Sensory Neuropathy

REPETITIVE NERVE STIMULATION

NO	NAME	AGE/SEX	HOS NO	DIAGNOSIS
1	Subash Kumar	20/M	371198	Myasthenia gravis
2	Mariamma Thomas	50/F	370419	Cranial Polyneuritis
3	Chellappan s	49/M	371149	GBS
4	Dr Jayadeva Das	52/M	9109267	R/o Myasthenia Gravis
5	Ranesh K	38/M	330564	Diplopia with generalized fatigue
6	Shankaran	67/M	373373	Suspected myopathy
7	Devaki Amma	65/F	373372	Fatigable Potosi's
8	Sasikala B	45/f	373990	Bulbar onset MND
9	Deepthy T K	42/F	374878	Myasthenia Gravis
10	Girija C	57/F	375323	Thymoma
11	Sujith Dev J R	21/M	370192	Pre ocular Myasthenia Gravis
12	Nafeesa K M	58/F	370621	Bilateral Ptosis
13	Anice Jose	48/F	374805	?MND
14	Abdul Rahman	47/M	376795	ocular Myasthenia Gravis
15	Rasheekhali O P	17/M	378052	Congenital Myasthenia

16	Manuvel Moj\han	60/M	378988	Rapidly progressive e Myopathy
17	Ansalin A R	16/F	367545	Myotonia congenita
18	Suja J	44/F	380252	Tymoma
19	Geetha Viswanathan	59/F	364227	MCTD with poly mitosis
20	Chellappan Pillai	90/m	385491	Late onset Myasthenia Gravis
21	Daisamma Y	56/F	314402	Bulbar Onset Myasthenia Gravis
22	Vaisakh lal	27/m	386348	Congenital Myasthenia
23	Arokiya Gaya Mary T	58/F	388643	Generalized Myasthenia
24	Jannath	36/F	388636	? Myopathy
25	Sree Kumar S A	19/M	389376	Myotonia Congenita
26	Thomas	60/M	390457	Ocular myasthenia
27	Lathika G	58/8F	390493	Ocular myasthenia
28	Jaini P V	39/F	395765	Myasthenia Gravis
29	Jayapraksh	44/M	395069	Congenital ptosis
30	Mariammal	32/F	403481	Myasthenia gravis

BLINK REFLEX

SL NO	PATIENT NAME	AGE/SEX	HOSPITAL NO	DIAGNOSIS
1	Latha Krishnan	45/F	382219	Right facial palsy
2	Yeshwini	39/F	374464	Progressive increase in size of pupil
3	Sosanna G R	59/F	380894	Right facial palsy
4	Kuriakose Arun	38/M	383112	Foreign body sensation on eye
5	Yasodha Amma S	58/F	383326	Trigeminal nerve palsy
6	Soumya P s	32/f	383296	Left trigeminal neuropathy
7	Kumar S A	59/M	387868	Facial parasthescia
8	Fathima Firoz	18/F	388954	Bell's palsy
9	George Kurian	65/M	387942	Left Bell's palsy
10	Mohanan	57/M	390825	Left LMN Bell's palsy

VISUAL ACUITY AND VISUAL FIELDS TESTING

1.	Anil Kumar.R	42/M	3703070	Pituitary adenoma
2	Vignwsh umar	16/M	371890	ADEM
3	Gabriyal K.J	58/M	373040	Pituitary adenoma
4	Shameera.P	32/M	369072	Pituitary microadenoma
5	Kasirajan	46/M	372894	Meningioma
6	Mohammed Nafil	7/M	371978	Vision loss
7	Jadeja Kuldeep Sing	30/M	373068	Right hemisphere gliosis
8	Sharmila.D	45/F	273463	Chronic optic granuloma
9	Safada.P.K	13/F	369632	Recurrent optic chiasmic glioma
10	Rajina.J	33/F	371282	Case of IIH
11	Ponomana.D	35/F	187600	Pituitary adenoma
12	Manju Mathew	45/F	358724	Right ATL+AH on 09/10/2013
13	Athulya E S	12/F	340619	Optic lesion
14	Ambili B S	35/F	373234	Pituitary adenoma
15	Rameshan.P.P	39/M	373238	Left parieto occipital lesion

16	Muhammed Anaz A	10/M	350306	Pituitary adenoma
17	Aiswarya C M	33/F	372950	Pituitary adenoma
18	Rajendran Pillai.G.V	58/M	372888	Pituitary adenoma
19	Jose	39/M	206633	Pituitary adenoma
20	Saroja	55/F	372075	Case of clenoid meningioma
21	Ramya.D	23/F	372906	Intraventricuar tumor
22	Thoufeera.T	21/F	355784	Right ATL+AH on 20/05/2014
23	Hegin Roj.C	22/M	872052	Pituitary adenoma
24	Pooja.A	33/F	355255	Right frontal glioma
25	Gangadharan Nair.P	58/M	371921	Right frontal glioma
26	Latha.S	27/F	357511	Right temporal cavernoma
27	Fousiya Neyyan	23/F	373318	Pituitary microadenoma
28	Amina Fadiya	12/F	373726	Optic chiasmatic glioma
29	Nagamanikkan	34/F	373777	Left frontal lesion
30	Manoj Thomas	46/M	9904661	Left parietal gliosis

31	Rajani.S	32/F	373808	Left thalamic lesion
32	Shanavas.E	25/M	373835	Pituitary adenoma
33	Bindu.S	55/F	369331	Pituitary adenoma
34	Lizy Chacko	55/F	373591	Left parietal lesion
35	Vinayakan.C	36/F	328851	Pituitary adenoma
36	Aysha.K.N	63/F	372214	Meningioma
37	Riyas.T	34/M	364746	Pituitary adenoma
38	Khadeeja Chorath	48/F	372578	Frontal meningioma
39	Kumaradas	66/M	9507282	Pituitary adenoma
40	Seema.R	37/F	304756	IIH
41	Kiran Gupta	44/F	351724	Right parietal lesionectomy
42	Animon.J	41/M	374216	Blurring of vison
43	Thilagavathi.R	35/F	374354	Pituitary adenoma
44	Geetha.K	338450	338450	IIH
45	Swathy Sathyan	21/F	373814	Multiple Sclerosis

46	Sridhar.N	8/M	354938	Hypothalamic glioma
47	Kunjumol	70/F	374042	Right temoral lesion
48	Ayisha Fida.M	11/F	335496	CPS-left frontal presurgical
49	Raseena Youoos	39/F	374334	Supra meningioma
50	Dayena Thomas	10/F	35841	Neeuromyelitis
51	Akshaya Adhikay	17/F	357739	Left ATL+AH on 17/05/2013
52	Kuriyakose.M.	56/M	374473	Pituitary adenoma
53	Malarvadi.P	49/F	374409	Recurrent optic neuritis
54	Rajeshwari.K	52/F	366106	Frontal lobe tumor
55	Sivaselvan.A	35/M	366120	Left frontal glioma
56	Saleena Beeevi	37/M	299356	IIH
57	Dipankar Roj	32/M	32/M	Left ATL+AH on 28/1/2015
58	Afsana Perveen	12/F	361972	Right atl+ah on 19/02/2014
59	Lathaa Vijayan	51/F	350689	Pachy Meningitis
60	Mohanan.S	55/M	8705915	Pituitary adenoma

61	Rajina.J	38/F	371282	IIH
62	Joseph Garwasis	58/3	240352	Pituitary adenoma
63	Sindhu.S	43/F	352180	IIH
64	Kamalanandan.P	65/M	383893	Pituitary adenoma
65	Jismi James	26/F	382672	Right temporal lesion
66	Mohammed.Jabir. K	16/M	349208	Lesionectomy on 07/05/2014
67	Nabeesa.Beevi.A	67/F	384323	Sellar lesion
68	Anas Abdul Majeed	29/M	384328	Pituitary adenoma
69	Abdul.Rasheed	22/M	375962	Insular glioma
70	Sankarraj.D	39/M	383759	Temporal glioma
71	Hassan Kunju	73/M	383964	Frontal lesion
72	Salim.K.P	45/M	227635	Recurrent frontal glioma
73	Unnikumaran.A	49/M	362886	Pituitary adenoma
74	Riyas.T	34/M	362886	Pituitary adenoma
75	Pothammal	51/f	342120	Optic nerve entrapment

76	Gayathri.N	25/F	383720	Supraselar elssion
77	Prameesh V.K	31/M	306466	Left frontal FCD
78	Serin Prasad	25/M	375542	Left optic neuritis
79	Sajith.S	28/M	360892	Frontocortical961 dysplasia
80	Rahul.K.R	23/M	366961	Ridathilght ATL+AH on 18/08/2014
81	Lali.Benny	46/F	387353	Pituitary adenoma
82	Ayisha.P.M	28/F	351360	Right ATL+AH on 29/10/2014
83	Nasirkhan	72/M	354489	Right ATL+AH on 31/07/2014
84	Devyani.C.S	59/M	387388	Pituitary adenoma
85	Sunil.Y	33/M	387277	Glioma
86	Maria Madathil	34/F	387346	Vascular glioma
87	Gopakumar,.S	38/M	255232	Pituitary adenoma
88	Amar Jyothy.R.K	34/M	282333	Ocular lesion
89	Aboobaekar	50/M	377307	Pituitary adenoma
90	Jyasree.S	41/F	365259	IIH

91	Abhishek.S	11/M	348422	occipital lesion
92	Sophia Praveen.A	28/F	310085	Blurring of vision
93	Yonus.E	18/M	381848	Left thalamic lesion
94	Chandra Prakash	26/M	363196h	Pituitary adenoma
95	Thankamani	58/F	313294	Pituitary adenoma
96	Shifanth.P	17/F	355306	Left ATL+AH on 12/11/2014
97	Kunheevi	42/F	387339	Mengioma
98	Dhayanthi.P	37/F	384741	Left vestibualar Schwannoma
99	Lalitha.K.V	49/F	316069	Boilateral optic atrophy.
100	Adarsh Krishnan	26/M	297630	NMO spectrum

VISUAL EVOKED POTENTIALS

NO	NAME	AGE/SEX	HOS NO	DIAGNOSIS
1	Lubina M	32/F	327133	Dystonia
2	Naushal A	39/M	391157	Optic Neuritis
3	Nikhil Benoy Abraham	12/M	391345	LETM
4	Lalitha Vijayan	52/F	389882	Multiple Sclerosis
5	Saleena	46/F	211801	NMO
6	Sathi R	56/F	383498	Patchy meningitis
7	Subin Sabu	17/M	390194	Static epileptics
8	Rugma B Rao	23/F	392124	IIH
9	Harikrishnan H S	11/M	392334	NMo spectrum disorder
10	Amravathy	32/F	304905	RRMS
11	Usain kutty P	52/M	393325	Mononeuritis multiplex
12	Athul Ramesh	9/M	313625	Mitochondrial Cytopathy
13	Vijayan B	56/M	394077	Optic Atrophy
14	Urmila M	19/F	385504	CNS Demylination
15	Athira S selvan	15/F	379482	Autoimmune encephalitis
16	Binu C	32/M	393660	Right Optic neuritis
17	Rani Mohan	30/F	396096	Multiple Sclerosis
18	Sibin Subodh	12/M	395325	ADEM
19	Jumna Fathima	18/F	396412	Leukodystrophy
20	Jamaludheen kunju	54/M	397224	Cranial Neuritis

BRAINSTEM EVOKED POTENTIALS

NO	NAME	AGE/SEX	HOS NO	DIAGNOSIS
1	Anamika	9/F	391462	Static encephalopathy
2	Gopi Shanth Nidhi	4/M	380451	GDD
3	Meera S	5/F	392676	Mitochondrial Cytopathy
4	Harikumar KN	53/M	393595	Cortical Myopathy
5	Dharmaraj K T	41/M	394126	HMSN
6	Chakki Chandran	70/F	393294	Tuberculosis CNS
7	Athul M R	16/M	385183	Multiple sclerosis
8	Anvar	24/M	372496	ADEM
9	Sabu D	47/M	374708	Large Fiber Neuropathy
10	Anila Joy	43/F	290832	? Demylination
11	Muhammed Faisal	4/M	396492	Doose Syndrome
12	Jibin Varghese	29/M	397443	Mitochondrial Cytopathy
13	Yamuna MT	48/F	399410	Probable NMO
14	Jigan Joshi	11/M	401501	SSPE
15	Rathish Kumar	8/M	398551	Leuko dystrophy
16	Murugeswary	11/F	401624	Auto immune Encephalopathy
17	Vairamuthu	1/m	401042	HIE
18	Adya Nair	6/F	401669	NMDAR with Sequale
19	Haizel Elze Joseph	17/F	402565	RRMS
20	Aswin S	1/M	402813	West syndrome

SOMATOSENSORY EVOKED POTENTIALS

NO	NAME	AGE/SEX	HOS NO	DIAGNOSIS
1	Urmila	19/F	385504	MS v/s vacuities
2	Krishna MS	23/F	385352	ADEM
3	Havish Varma N S	3/M	389796	Hypoxic ischemic Encephalopathy
4	Sudha V	48/F	386994	Optic neuritis
5	Abhijith Singh	23/M	281884	?PME
6	Abdul Manaf P A	55/` M	387931	Mylo radiculopathy
7	Susan George	42/ F	8900846	CPS ET
8	Ramadevi T P	52/ F	386278	Multiple sclerosis
9	Amina Z	15/ F	381332	PME
10	Muhammed Abdulla	22/ M	395486	SCA
11	Vishnu Dutt S	14/ M	378829	Cerebellar Ataxia
12	Gokul Das	11/ M	399540	? PNES
13	Nandamithra	13/ F	314680	Myoclonic ataxia syndrome
14	Rathish Kumar	9/ M	398551	Met achromatic Leuko dystrophy
15	Sreenanda SD	4/ F	401162	GDD
16	Varun	31 /M	385521	JME
17	Milapilli Ramu	25/ M	384343	Parkinson's Disease
18	Asharaf K	39/ M	384660	NMO
19	Krishna M S	23/ F	385352	ADEM
20	Bindhu B L	38/F	388755	Young Onset dementia