

**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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**2014-2015**

# CERTIFICATE



**I Mr. Kenaz Varghese Mathew 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 :.....**

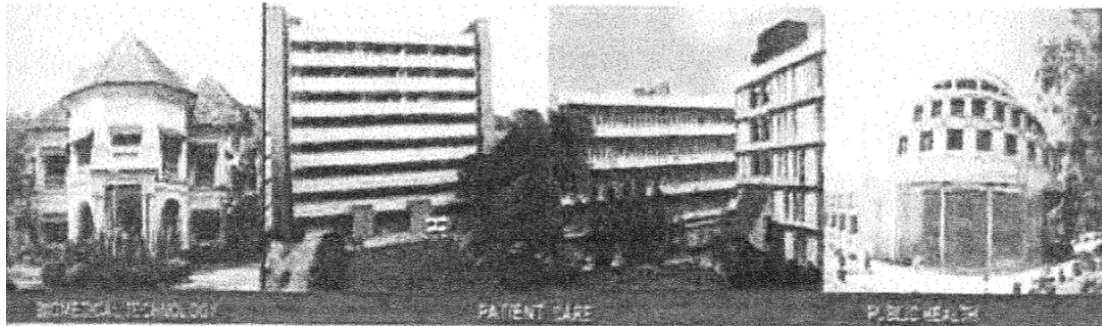
**Head of the department  
SCTIMST, Thiruvananthapuram**

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

## Introduction

The electroencephalogram (EEG) is a recording of the electrical activity of the brain. Hans Berger made the first recording in 1929 although similar studies have 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 from 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.

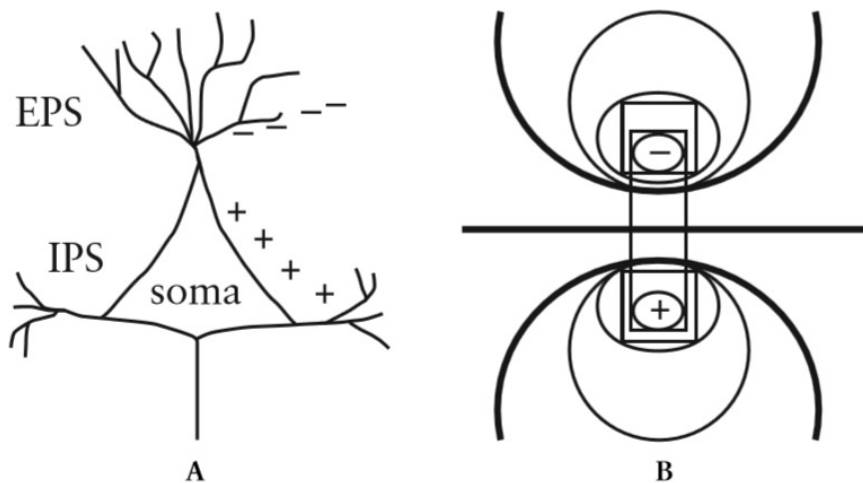
For the successful interpretation of an abnormal EEG, one must first understand the criteria necessary to define normal patterns. While a normal EEG does not exclude a clinical diagnosis (i.e., epilepsy), an abnormal finding on EEG may be supportive of a diagnosis (i.e., in epilepsy), be indicative of 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 (EPSPs) flow inwardly (extracellular to intracellular) to other parts of the cell (sinks) via sodium or calcium ions. Inhibitory postsynaptic potentials (IPSPs) 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  $>6 \text{ cm}^2$ , although probably  $>10 \text{ cm}^2$  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.

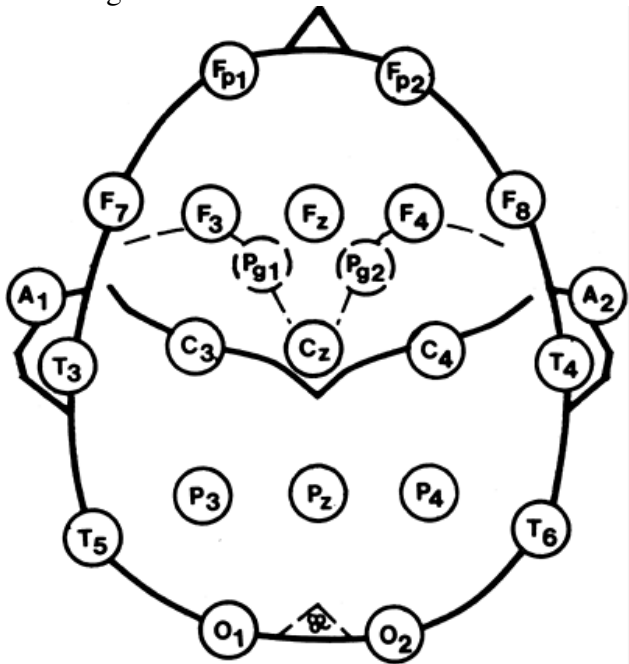


**FIGURE 1.1.** (A) A pyramidal cell with excitatory postsynaptic potentials and inhibitory postsynaptic potentials. (B) Dipole depicting a field of charge separation.

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 of 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.



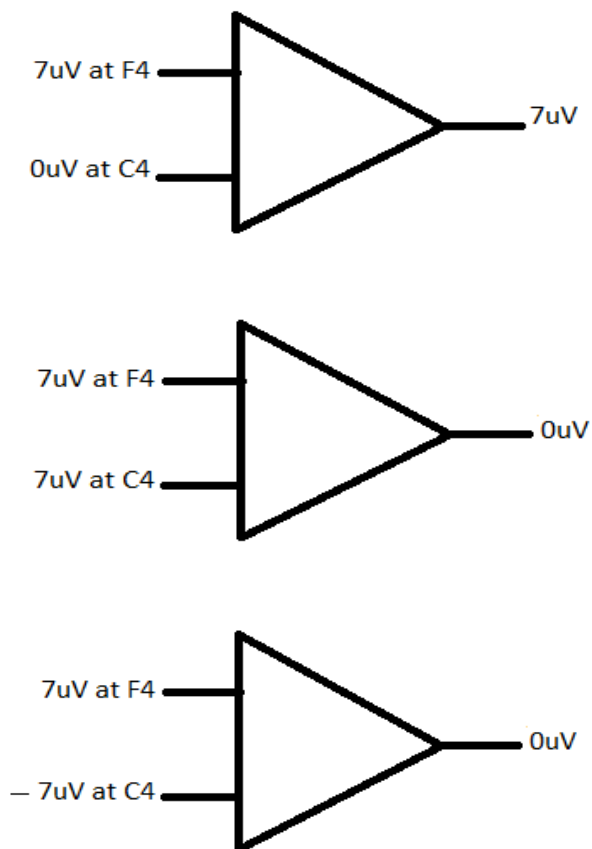
There are a great variety of electrodes that can be used. the majority are small discs of stainless steel, tin, gold or silver covered with a silver chloride coating. These normally have a lead attached. Alternative methods consist of a cap in which the electrodes are already embedded.

## 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

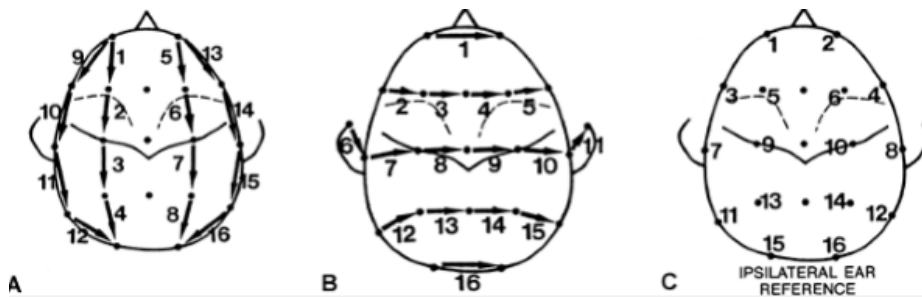
Differential amplifiers measure the voltage difference between the two signals at each of the inputs. The resulting signal is then amplified and then displayed as a channel of EEG activity.



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

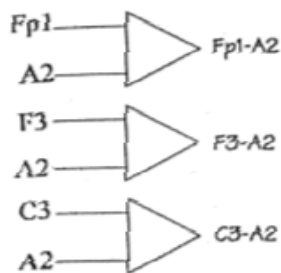
If one is mainly interested in a striking pattern, such as a spike-and-wave complex, then the display of such a pattern in a way that makes it stand out at a fairly large amplitude is desirable. For such a pattern, which is generally predominantly parasagittal, the use of the earlobe, whether ipsilateral 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 anteriorly and posteriorly (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).

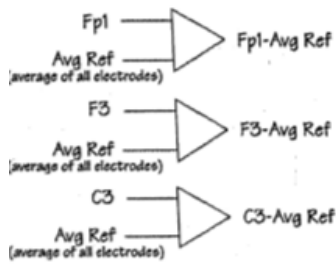


A: A representation of bipolar (scalp-to-scalp) montage in a longitudinal or anteroposterior (AP) direction. This montage is designed for easy comparison of left-right differences in the parasagittal or temporal area, but a montage with strict left-to-right sequence of sets of four could provide the 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 A1, A2, the ear electrodes, or A1 and A2 linked together.

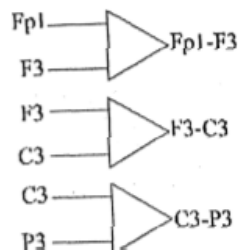


Average reference derivation: Activity from all the electrodes are measured summed together and averaged before being passed through a high value resistor. The resulting signal is then used as a reference electrode and connected to input 2 of each amplifier and is essentially inactive. All EEG systems 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 Fp1 and F3 connected to it and the second amplifier F3 and C3 connected to it



## Analogue EEG instruments

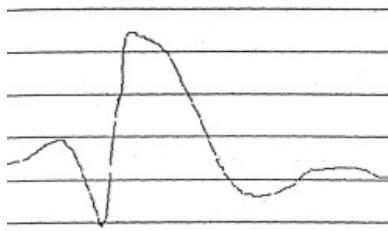
Conventional analogue instruments consist of an amplifier, a galvanometer and a writing device. A galvanometer is a coil of wire inside a magnetic field. The output signal from the amplifier passes through the wire causing the coil to oscillate. A pen mounted on the galvanometer moves up and down each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

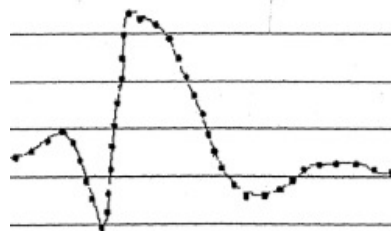
The speed at which the paper moves on will also affect the appearance of the waveforms.

## 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



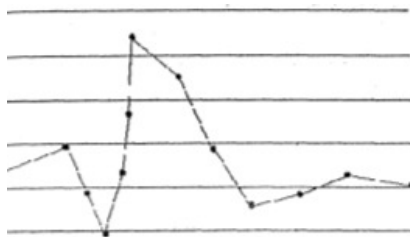
Sampling rate of 240 Hz

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 than 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 1, then sample channel 2, then channel 3 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 480 pixels while a monitor with a Super VGA display will have a screen resolution of around 1024 x 768 pixels. A typical page of EEG contains 10 seconds of data. A digital EEG system, sampling at rates of 240 Hz 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 electrode. 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 A1 is common to both channels on input 2. It has the identical value in each channel. Remontaging 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).

$$\begin{array}{rcc}
 \text{Input 1} & & \text{Input 2} \\
 (F3 - A1) & - & (F4 - A1) \\
 & \downarrow & \\
 F3 - A1 & - & F4 - A1 \\
 & \downarrow & \\
 & & F3 - F4
 \end{array}$$

## 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 of 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)

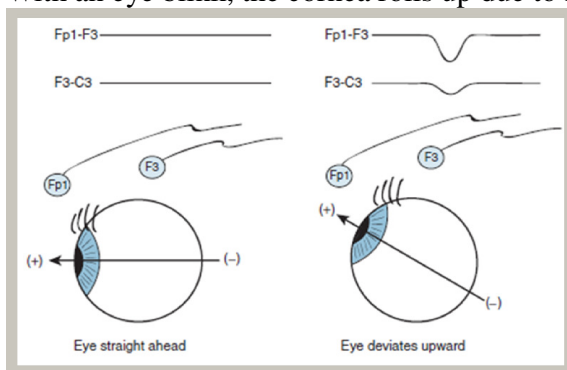
Artifacts originate from a variety of sources, and their recognition, identification, and possible elimination are a primary responsibility of the electroencephalographic (EEG) technologist. If unrecognized, can lead to misinterpretation of the recordings.

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

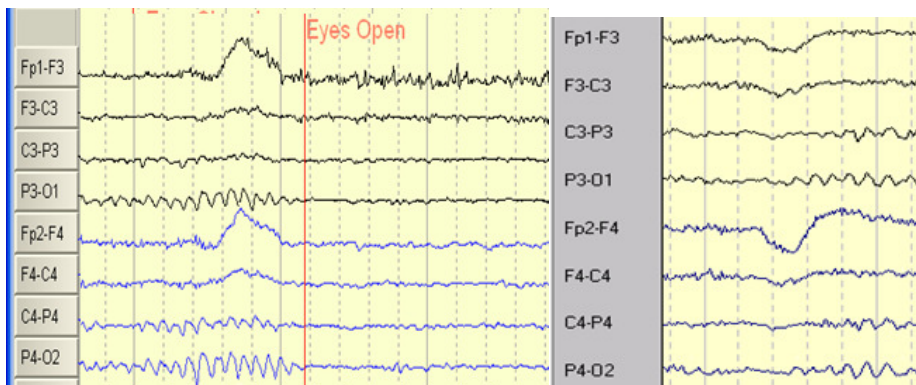
## Physiological artifacts

### 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 .

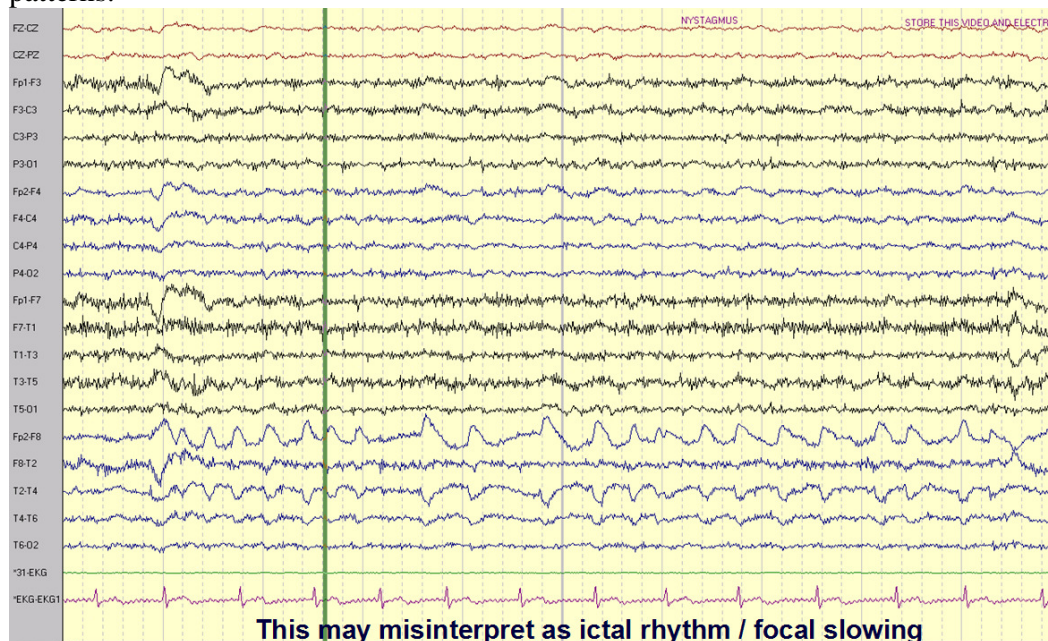


## Eyelid flutter

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)

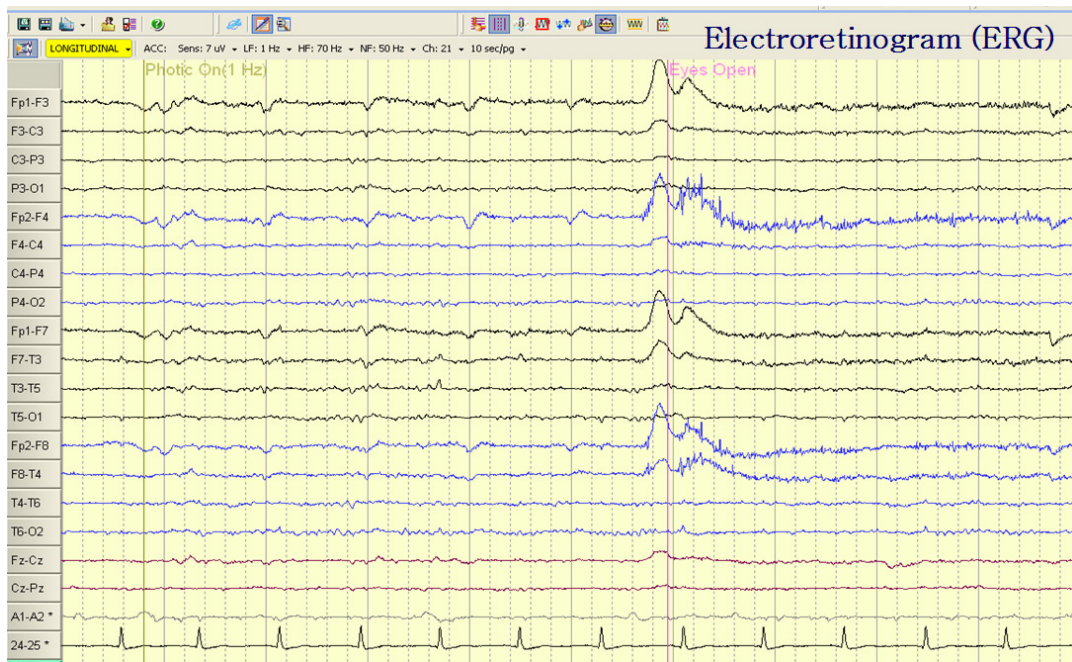
## Nystagmus

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 (<50 microV) 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.

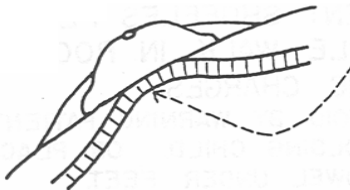
### **Electrocardiographic Artifacts**

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.

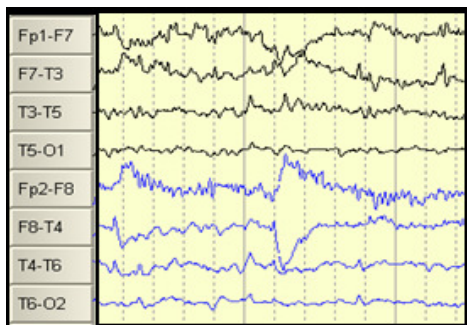
### **Cardiac arrhythmias**

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 automatism prior to or during an ictal event or who have orofacial dyskinesias.

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



## **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  $< 5\text{kohm}$  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 dysfunction 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 extremity.

**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 synchronous and symmetrical. Posterior slow waves of youth attenuating or blocks with eye opening and disappears during drowsiness along with alpha rhythm.

## **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 EEG. 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 commonly 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 II sleep. The waves are aptly named, as their focus lies in Cz, the vertex. When the waves are larger amplitude, they also are 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 commonly in runs. They are often bilaterally synchronous, but the same time they are asymmetrical on both sides.

### **The EEG in deeper stage of sleep**

The stage III sleep is characterized by a background that consists of irregular and senilhythmic 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 paradoxically 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 from the background activity. Paroxysmal abnormalities can be classified into epileptiform abnormalities and periodic patterns.

### **Epileptiform Discharges**

#### **Spike discharges**

A spike discharge is defined as a transient that is clearly 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 clearly 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 generalized 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 pattern 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 patient 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.

## **Abnormal 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 of 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. Each 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**

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 generalized to start with, but later they become generalized. The usual site is temporal or temporo frontal. The discharge consists of large sharp waves 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 considered as a periodic pattern, since it consists of periodic bursts 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 may result from cerebral anoxia, head trauma, and severe drug intoxication as well as deep anesthesia.

### **Triphasic waves**

Triphasic wave may sometimes occur in periodic fashion. Each waveform shows a prominent 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 waves are usually frontally dominant, 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 (BIPLLEDs)
- ✓ 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**

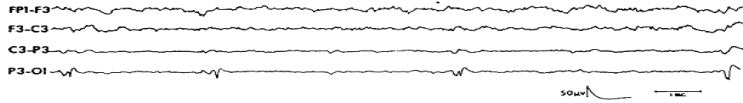
PLEDs proper (without rhythmic discharges)

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

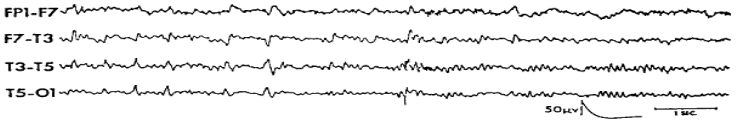
PLEDs plus (with rhythmic discharges)

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

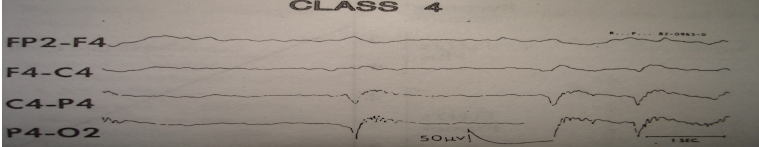
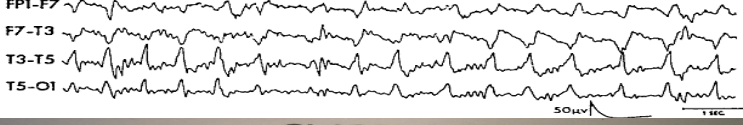
Class I - aperiodic,



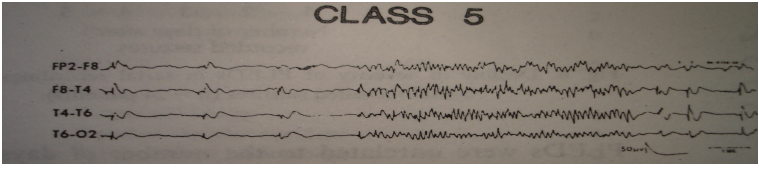
Class II metronomic, intermittent



Class III - metronomic, throughout



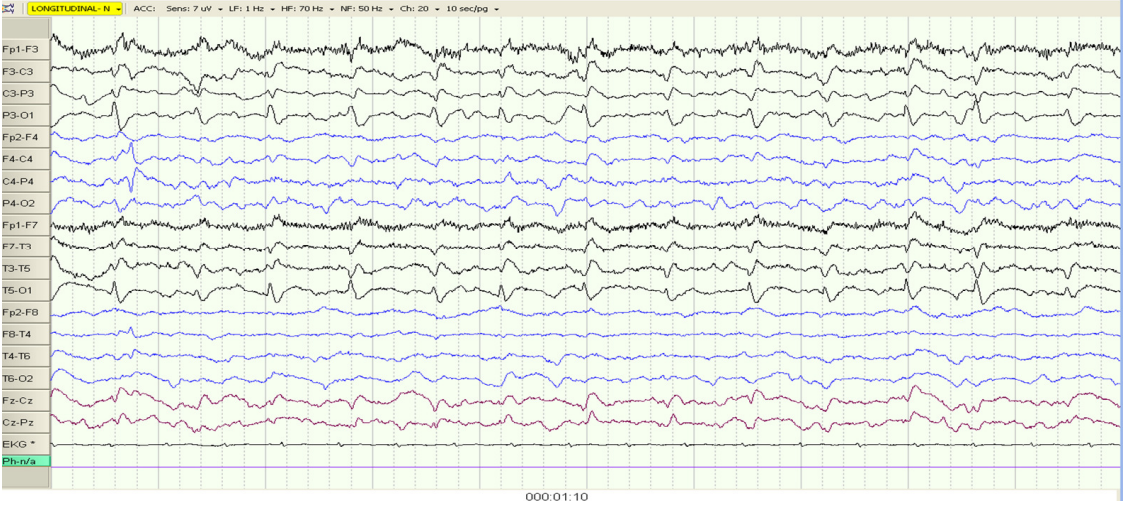
brief RDs < 1 sec



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 lesions



## 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 within 2-3 weeks. 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 persist 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

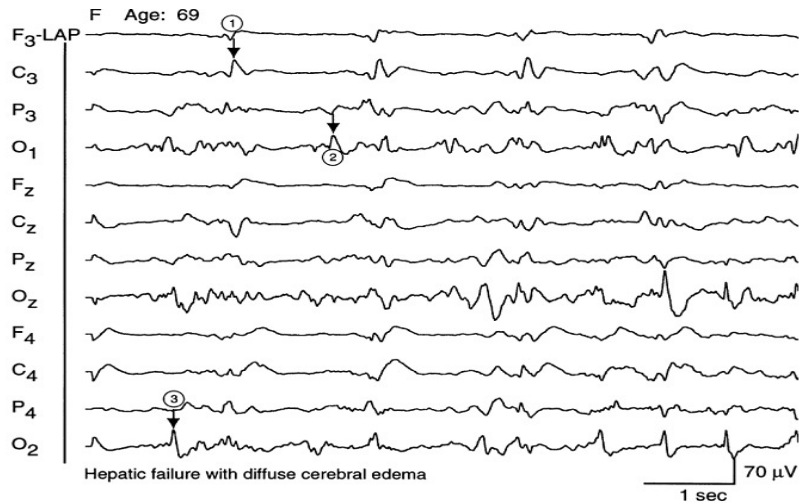
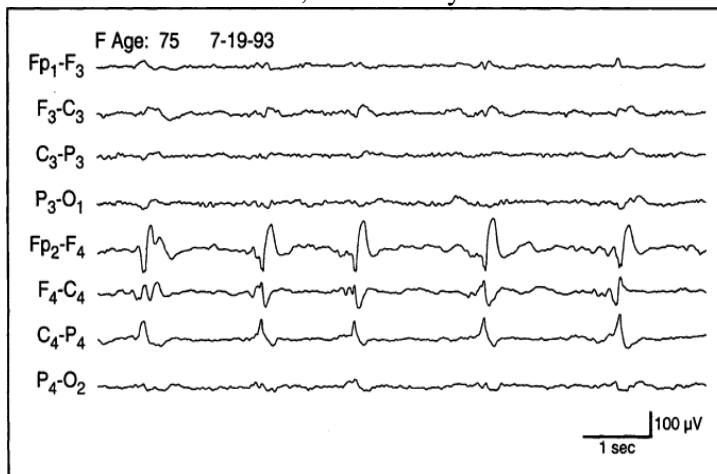


Fig. 1. Typical example of multifocal PLEDs (arrows) using the Laplacian montage, recorded from a 69-year-old woman with liver failure, sepsis, and hypoxia. Focus 1 (C<sub>3</sub>) occurring every 1.5 s; focus 2 (O<sub>1</sub>) occurring every 0.5–1 s; focus 3 (O<sub>2</sub>) occurring every 0.5 s.

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 'epileptiform 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 arousal and light sleep. They are usually of low amplitude, usually less than 50 $\mu$ V and of short duration, less than 50 ms. The waveform consists of single monophasic or biphasic spikes with abrupt ascending limb and steep descending 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 and ear leads.

### **Wicket spikes**

Wicket spikes occur either in brief runs or as isolated transients. They 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 positive 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)**

This 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, but 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 (CO<sub>2</sub>) being removed from the bloodstream. This fall in CO<sub>2</sub> 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 EEG 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 the 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 (hypocarbica). 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 of 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 known 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 EEG activity over 2  $\mu$ V in amplitude when recording from electrodes on the scalp that are 10 cm 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 cm. 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  $\mu$ V/mm are required. However digital EEG systems have the added advantage of having sensitivity values of 1.5 and 1  $\mu$ V/mm. This 50-100 % increase in sensitivity will allow a more confident assessment of the presence or absence of a 2 $\mu$ V 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 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 re activity, 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 EEG 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 be caused by liver dysfunction or toxic substances. From clinical and neuropsychological point of view, it is important to test reactivity of EEG. 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 1 and T2 electrodes located below F7-F8 or T3 -T4 positions. Some used T 1 &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 interictal period, but increasingly postictal and ictal studies are being carried out.

Usually the gamma-emitting tracer used in functional brain imaging is  $^{99m}\text{TcHMP AO}$  (hexamethyl propylene amine oxime).  $^{99m}\text{Tc}$  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  $^{99m}\text{Tc}$  to be taken up by brain tissue in a manner proportional 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 during 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, epiduralelectrodes, 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**

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. Electrocortigraphy during epilepsy surgery has been performed for several years. In particular is this true 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 rhizotomy procedure to reduce spasticity in children with cerebral palsy and spastic diplegia. The function of the facial nerve is monitored in the resection of acoustic neuroma.

# POLYSOMNOGRAPHY

## Introduction

A polysomnogram consists of a simultaneous recording of multiple physiologic parameters related 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 disorders' 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 sleep 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
  - o Sleep apnea syndrome
  - o Upper airway resistance syndrome

### **Parasomnias**

- Disorders of arousal
- Disorders of sleep-wake transition
- Disorders that occur during REM sleep
  - o Nightmares
  - o REM behavior disorder
- Medical-psychiatric sleep disorders
  - o Medical ~ Sleep-related asthma
  - o 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 can 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 amyotrophic 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 increases 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 0.51.8 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 for 10 cm 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 axons conduct impulses, and a smaller than normal muscle response 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 mm 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.

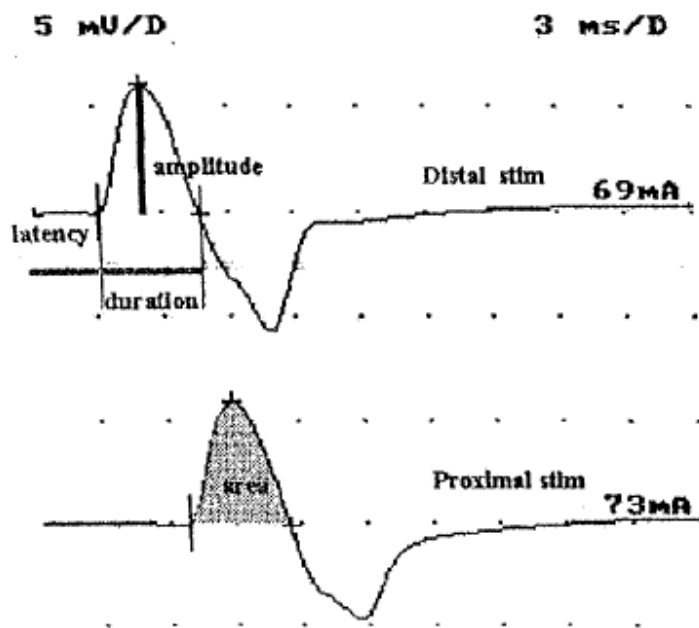
They are fixed on to a plastic bar a fixed distance apart. In a few situations it is preferable to use needle electrodes. A pair of monopolar needle electrodes is used in these cases. Alternatively a surface electrode is used as an anode.

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.

## Parameters

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) / LAT<sub>prox</sub> - LAT<sub>distal</sub>**

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 (DU<sub>prox</sub> - DU<sub>distal</sub>) / DU<sub>distal</sub>**

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.

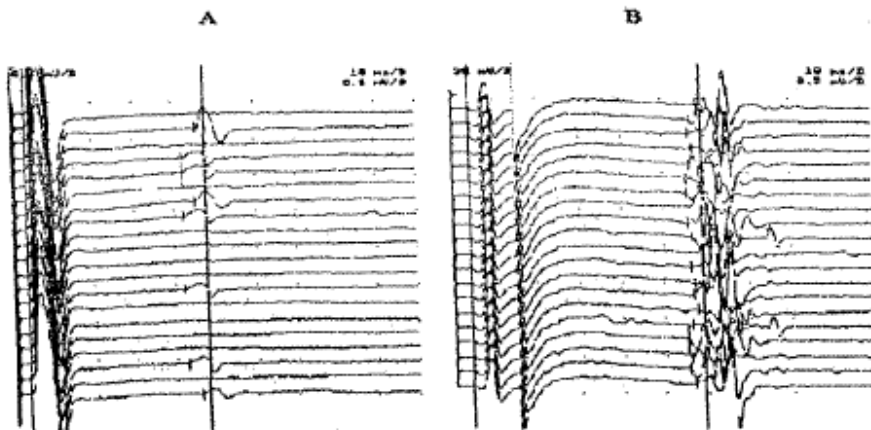
$$\text{AMPLDECAY} = 100 \times (\text{AMPL}_{\text{distal}} - \text{AMPL}_{\text{prox}}) / \text{AMPL}_{\text{distal}}$$

$$\text{AREADECA Y} = 100 \times (\text{AREA}_{\text{distal}} - \text{AREA}_{\text{prox}}) / \text{AREA}_{\text{distal}}$$

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 AMPLDECAY in 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.

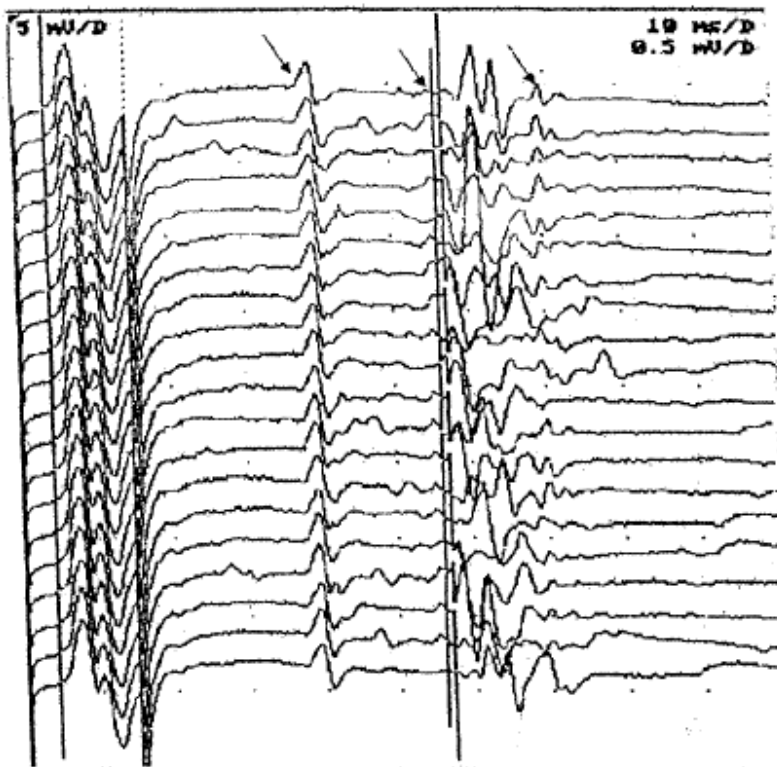
The F-waves travel from the stimulation point on the nerve to the neuron and back to the muscle. By subtracting the distal latency, the time taken from the stimulus point to the neuron and back again to the stimulus point can be obtained. This time depends on the conduction distance involved. Instead 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 to an anastomotic 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 first 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 of F-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 S1 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 \With 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 Jendrassik'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
<b><u>CMAP</u></b>			
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
<b><u>F-waves</u></b>			
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
<b><u>M-satellites</u></b>			
Presence	Abnormal excitability or slowly conducting axons	present or not	M
<b><u>H-reflex</u></b>			
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 polyneuropathy, 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% AMPLDECAY or 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:

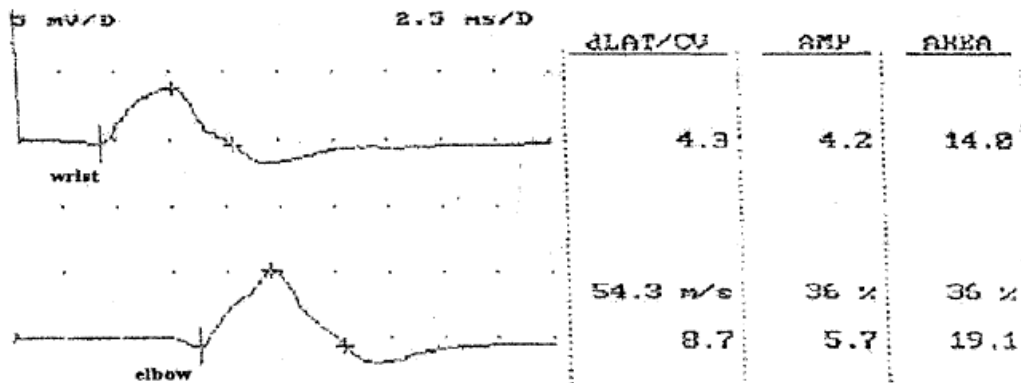
>25% (arm) or >40% (leg) AMPLDECAY and <15% DISPERSION or if there is >50% AMPLDECAY, independent of DISPERSION (in this case there is a combination of CB and demyelination)

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 of F-waves: ulnar 18, median 15, peroneal 10, 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 is 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 of F-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%. 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 be divided into three types according to 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 pollicis 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 CMAP 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.



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 behind the malleolus."

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

**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 biphasic 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 recording may show multiple peaks indicating the difference in conduction velocity among individual axons. This is particularly apparent in cases of pathology. Amplitude 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

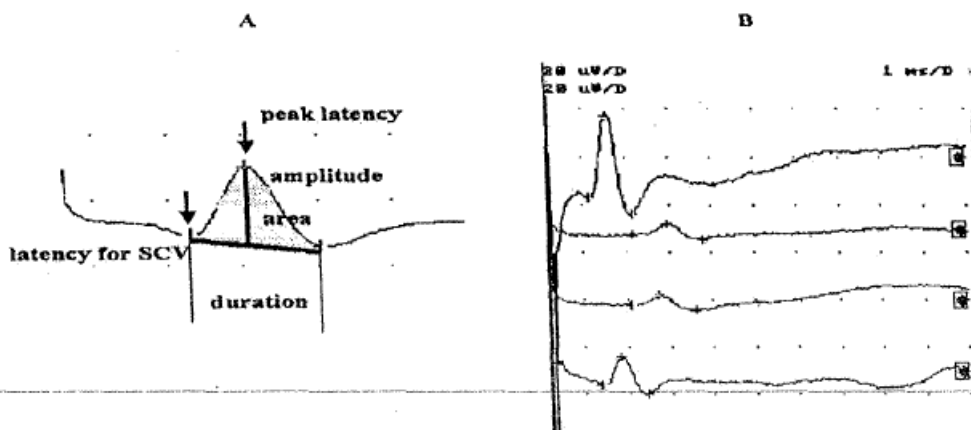
reproducible in pathology. We use needle electrodes in Morton's metatarsalgia and meralgia paresthetica.

**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

10



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

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  $\mu$ m 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.

$$\text{CV (m/s)} = \text{distance (mm)} / \text{LAT}$$

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.

### **Analysis**

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

**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 (• V)	a/m	
Area	# axons, temporal disp	total area (• 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.*

## Autonomic nerve testing

It should be 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 the 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 electrodermal 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 the 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 recorded 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**

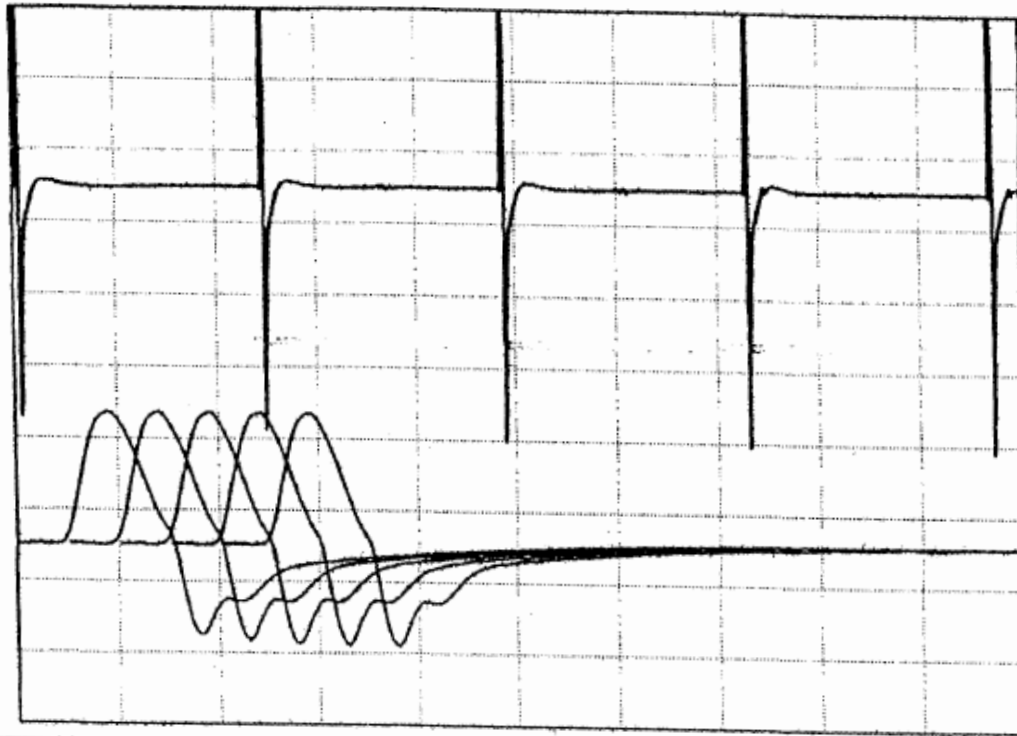
Repetitive nerve stimulation (RNS) is used in the evaluation of patients with suspected neuromuscular transmission disorders (NMTD) such as myasthenia gravis (MG) or Lambert-Eaton myasthenic syndrome (LEMS). RNS is a modified motor NCS where instead of recording CMAPs with single supramaximal electrical 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 cleft. 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 it 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 corner 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 VIIth 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  $\mu$ V/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 polyphasic 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 VIIth 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 sclerosis 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 reflexes 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 conduction 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 cystometry. 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 severely 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 voluntarily 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 innervated 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

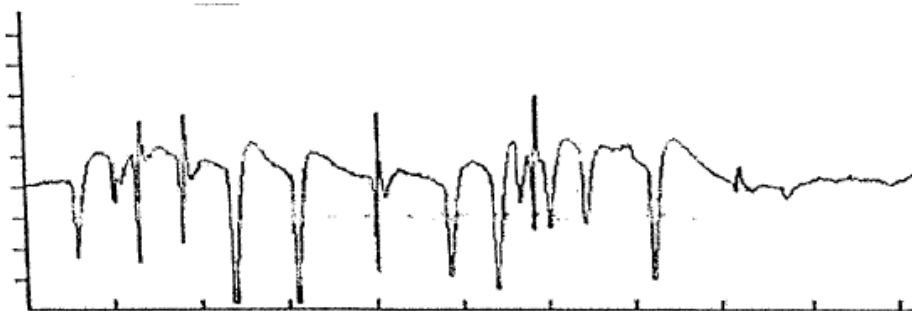
caused by emphatically activated muscle fibers as a result of discharge in pacemaker fibers in either nerve or muscle. Although very little tension is produced, fasciculation can sometimes be observed through the skin. Fasciculation can be seen in a variety of diseases affecting nerve and muscle, and 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 one of the electrical hallmarks of partially or completely denervated muscle and is produced by the discharge of a single muscle fiber. The amplitude of fibrillation potentials may range from 50-350  $\mu\text{V}$  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 than fibrillation potentials and usually have a lower discharge rate.



**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  $\mu\text{V}$ .

### **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-bomber. For this reason, myotonic potentials are referred to as "dive-bomber potentials."

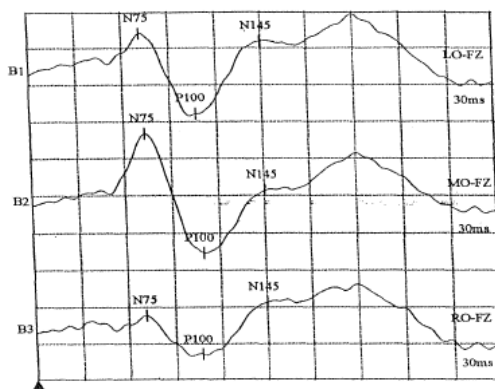
## VISUAL EVOKED POTENTIAL

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 to 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 females 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 P100 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 standard EEG electrode placement. Monocular stimulation is used to avoid masking of unilateral conduction abnormality. Sedation should 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 extent 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**

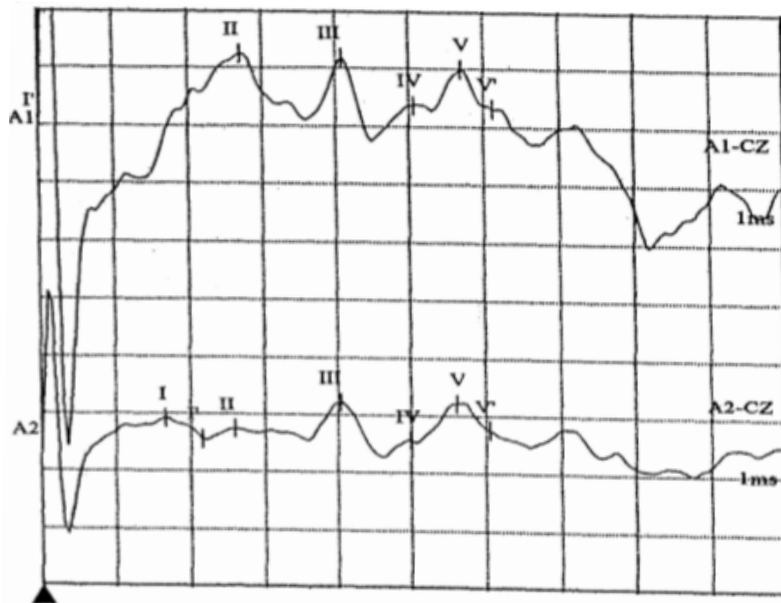
## **Introduction**

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 10Hz 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) VIII
- . Wave II- Cochlear nucleus (and CN VIII)
- . Wave III- Ipsilateral superior olivary nucleus
- . Wave IV- Nucleus or axons of lateral lemniscus
- . 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 than 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 spinal cord), 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 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 cm 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-T1, whereas the median nerve originates from C6-T1.

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 latency, 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. '

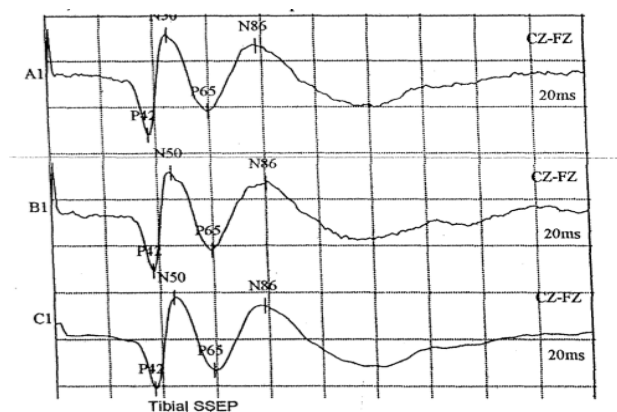
## Recording electrodes sites

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, T10S.

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 of being 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 sternocleidomastoid muscle and the clavicle), both ipsilateral and contralateral to the stimulus (labeled EPI and Epc),. or 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. 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 motor 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 pathways 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  $\cdot$  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) is recommended. 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 impedance.

# 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 in 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.

## **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 patient's neck, there will be a small scar where they are inserted, usually in the fold of patient'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 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 patient's wrist like a watch, or on patient'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 patient'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 patient's head rather than scanning patient's whole body and if they do not scan patient'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 determine 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 Jaeger 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 Jaeger type card must be held one foot from the patient's eye and a similar test is then carried out. The different types are labeled as N6, N24 etc 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 blepharospasms, 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 the 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 an electrophysiological procedure. The electricity can kill a patient if the equipment is not properly maintained or grounded and if adequate precautions are not taken.

The following precautions are to be 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 a 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 deliberate grounding of 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. If this results in unacceptable high levels of 60/50 Hz artifacts, remove the existing ground lead and replace it with the standard EEG electrode placement usually the center of the patient's forehead.

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 in correlation 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 and synchronized 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 Geodesics, 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 (IED, or interictal spikes), and their generators, and of seizures. IEDs are unpredictable and subclinical 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 IED and seizures, and has proven a powerful scientific tool. The simultaneous and synchronized video recording identifies clinical seizure activity along with electrophysiological 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,, wakefulness and sleep.



## NERVE CONDUCTION STUDY

Sl.No	Name	Age/Sex	Hospital No	Diagnosis
1	Babu B	46/M	392151	Right L5radiculopathy & right cervical radiculopathy
2	Raju C K	59/M	391978	Axonopathic sensorimotor polyneuropathy
3	Jeena Cherian	27/F	231706	Mononeuritis Multiplexes
4	Shaduli T P	63/M	392959	Anterior Horn Cell Disease
5	Francis Xavier	62/M	392087	Left ulnar neuropathy
6	Mayadevi P	40/F	360126	Bilateral CTS
7	Sachu Sajeevan	9/M	299243	Myopathic Process
8	Mohanan J	54/M	252983	AMAN
9	Soundha L	47/F	380652	Right lower lumbar radiculopathy
10	Affan Ali	3/F	382403	Myopathy, mixed sensory motor polyneuropathy
11	Sussanna G R	59/F	380894	Right peripheral facial nerve lesion
12	Bindhu Sunil	40/6	382678	Irritative myopathic process
13	Lekenath Bhagat	47/M	383319	S1 radiculopathy
14	Joby Raj	21/M	382375	Thorasic outlet syndrome
15	Johnson T	35/M	381728	Lumbosacral radiculopathy
16	Niyas B N	25/M	266727	Right cervical radiculopathy (C8)
17	Raju M S	54/M	383489	Left lower brachial plexopathy
18	Shobika K	24/F	317653	Fredrich's ataxia
19	Biju Kumar D	34/M	383336	AMSAN
20	Ragunathan Pillai	59/M	333239	Neuralgic Amyotrophy (upper trunk of left Brachial plexus)
21	Purushothaman K	61/M	376548	Cauda Equima Syndrome
22	Safiya Beevi S	45/F	383752	Left L5-S1 radiculopathy
23	Abdul Rahim	53/M	379122	C7 radiculopathy
24	Shinto V	23/M	357643	Lower motor neuron myotones bilateraly
25	Prem Kumar	48/M	378590	Bilateral Meralgia Parasthesia
26	Sruthy S	26/F	384245	Post Synaptic NMJ Transmission defect
27	Rajendran Pillai K R	51/M	382754	Pre ganglionic Axonpathic process
28	Sreerag S	13/M	383666	Myopathic Process
29	Muneera Beevi	38/F	384567	Bulbar MG, absent blink reflex
30	Radhakrishnan P	53/M	385160	Proximal Involment of right upper brachial plexus
31	Subramanaian	67/M	385197	MND
32	Aleema S	6/F	384899	Zelleger Syndrome

33	Chellapan Pillai P	90/M	385491	Late Onset MG
34	Edward M	47/M	347216	Abnormal Phrenic nerve conduction, Diaphragm palsy
35	Khareem T	32/M	363244	L3-S1 neurogenic process
36	Joykutty Y G	57/M	385402	Acute peroneal neuropathy
37	Chandran S	49/M	383743	Ulnar Neuropathy
38	Siji George	44/F	386172	Bilateral radial neuropathy
39	Sadanandan K K	63/M	382109	Early radiculopathy
40	Soumya P S	32/F	386296	Left afferent trigeminal nerve lesion
41	Prabakaran Nair S	67/M	384350	Chronic denervation process involving b/l L4,L5,S1 segments
42	Raju George	58/M	385407	Meralgia Parasthesia
43	Gopalakrishna Pillai	75/M	340015	Post surgical median nerve neuropathy
44	Jubia Prince	37/F	386585	Ocular MG
45	Sinimol O	25/F	386364	Mononeuritis Multiplexes
46	Leena V	28/F	378592	Bilateral S1 radiculopathy
47	Sangeetha M Nair	43/F	377704	focal AHC (Hirayan's disorder)
48	Athul Ramesh	18/M	313628	Metachromatic Leukodystrophy
49	Suresh S	1/M	386610	Krobbel Leukodystrophy
50	Sreelatha O	45/F	386919	Left ulnar neuropathy
51	Shanza Mehrin E	1/F	399311	Leukodystrophy
52	Balachandran C	47/M	400396	GBS
53	Kurien K G	60/M	398601	Left Cervical Radiculopathy
54	Gopakumar	50/M	399759	Myopathy
55	Varghese	67/M	399703	GBS
56	Joseph	60/M	400180	Stiff person syndrome
57	Shilpa K O	17/F	324532	Mitochondrial cytopathy
58	Devis V P	52/M	399586	Anterior Horn Cell Disease
59	Girija V	44/F	400195	Mylopathy
60	Venukumar S S	54/M	400181	Left lumbosacral plexopathy
61	Sreedaran K	71/M	400877	MND
62	Muhammed Rishal	9/M	391963	Irritable myopathic process
63	Siva Balan	59/M	396481	L5-S1 radiculopathy
64	Padmanaban N	57/M	331951	L5-S1 radiculopathy
65	Julianna Nedumpara	38/F	400950	Stiff person syndrome
66	Sasidharan R	60/M	401317	Left ulnar neuropathy

67	Jaisni PV	40/F	395765	Vasculitic neuropathy(phrenic nerve study)
68	Rema S Nair	60/F	400495	Left S1 Radiculopathy
69	Santhosh Lal S	35/M	396814	Right S1 L5 Radiculopathy
70	Ziya M U	7/F	400778	Left facial nerve pathology
71	Devaraj A	45/M	401348	Myotonic dystrophy
72	Vishnu Sarma	50/M	401364	Myopathic Process
73	Ali K	61/M	401706	Peroneal mononeuropathy(conduction block)
74	Andi Muthu M	34/M	388566	Afferent response from right trigeminal nerve stimulation absent
75	Gopikrishnan S	47/M	9510082	Left Meralgia Parasthetica
76	Shameed Ali K K	16/M	368935	Facial nerve palse (Blink)
77	Venu Kumar K S	54/M	400181	Left lumbosacral plexopathy
78	Vijayan C	56/M	184861	Right cervical radiculopathy (C7,C8 roots)
79	Anirudan Pillai M	48/M	399254	B/L lumbosacral plexopathy
80	Balakrishnan Nair S	72/M	399328	piriformis syndrome(Probable Sciatic neuropathy)
81	Rajesh P	33/M	394572	Diffuse anterior horn cell disease
82	Ramanathan S	61/M	394774	Diffuse anterior horn cell disease (conduction block )
83	Indira Kumari	59/F	238164	S1 radiculopathy
84	Lilly Kutty	47/F	392364	Thorasic outlet syndrome
85	Vimala Soman	49/F	394863	Tarsal tunnel syndrome
86	Bhuvanendran Nair P	59/M	394364	left facial palsy
87	Arunima	16/F	382966	Mononeuritis Multiplexes
88	Omana B	65/F	381988	Bilateral Lumbosacral radiculopathy
89	Susanna G R	59/F	380894	Rgiht peripheral facial nerve lesion
90	Muthumari P	22/F	376905	Friedrieich's Ataxia
91	Shivani J	5/F	376706	AMAN
92	Saniya Mariam Binu	9/F	372354	Myopathy
93	Manoharan V	45/M	236635	Left peroneal axonopathy
94	Sunil M	41/M	368086	Benign fasciculation cramp syndrome
95	Shamnad A	27/M	375775	Myotonia Congenita
96	Sageetha M Nair	43/F	377704	focal AHC (Hirayan's disorder)
97	George Varghese	33/M	403588	LMN Fasial palsy
98	Vijayan Nair B	54/M	402006	Left Bell's palsy
99	Kunju Krishnan	81/M	402846	AIDP
100	Reji R	28/F	400444	Left AIN

## REPETITIVE NERVE STIMULATION

Sl. No	Namr	Age/Sex	Hospital No	Diagnosis
1	Rekha Warriar	25/F	375354	Rule out myasthenia gravis
2	Leetha Kumari C	50/F	373427	Rule out myasthenia gravis
3	Sandanandan V	65/M	363991	Rule out myasthenia gravis
4	Usha Kumari	40/F	375788	C/o Thymoma ; R/o MG
5	Aminath Hussaian	38/F	339314	Myasthenia Gravis (36% decrement)
6	Abdul Rahman	47/M	376795	Ocular Myasthenia
7	Paramasivam K	40/M	376785	Myasthenia Gravis
8	Vivek John Abraham	20/M	382844	Myasthenia Gravis (20% decrement in facial)
9	Thrisha Jayaprakash	10/F	381321	Myasthenia Gravis
10	Abraham Y Babu	59/M	381184	Ocular Myasthenia
11	Melvin Samuel	14/M	383465	Myasthenia Gravis
12	Prasanna S	51/F	383630	Double Vision
13	Ramala K	38/F	383939	Rule out myasthenia gravis
14	Resmy K G	31/F	382996	Myasthenia Gravis
15	Sarojini V	65/F	383268	Rule out myasthenia gravis
16	Sruthy S	16/F	384245	Myasthenia Gravis
17	Muneera Beevi	38/F	384567	Bulbar MG
18	Krishna Veni	44/F	384608	Rule out myasthenia gravis
19	Jayachandran D	62/M	384611	Rule out myasthenia gravis
20	Thresiyamma	78/F	384082	Motor Neuron Disease
21	Latha P	48/F	385362	Myasthenia Gravis
22	Samson P	67/M	382849	Rule out ocular myasthenia
23	Chellapan Pillai P	90/M	385491	Late Onset MG
24	Helen A	46/M	400250	Ocular Myasthenia
25	Adityan	5/M	343810	An early myopathy
26	Rahana V R	12/F	347530	Myasthenia Gravis
27	Jiju Mathew	42/M	9803645	Generalised myasthenia gravis
28	Manu M	23/M	395853	Myasthenic syndrome(median - 22%; ulnar - 24 %)
29	Fairoj I	37/F	377138	Rule out myasthenia gravis
30	Syamala M K	60/F	402398	Myasthenia Gravis

## BLINK REFLEX

Sl. No	Namr	Age/Sex	Hospital No	Diagnosis
1	Gowri S	45/F	376237	Probable C8 T1 radiculopathy
2	Susanna G R	59/F	380894	Right peripheral facial nerve lesion
3	Kuriakose	38/M	383112	Foreign body sensation in eyes (normal study)
4	Yashoodha Amma S	58/F	383326	Trigeminal nerve palsy
5	Madhavan Pillai P	64/M	383061	Dysarthria
6	Muneera Beevi	38/F	384567	Bulbar MG
7	Shamseed Ali K K	16/M	368935	Facial nerve palse (Blink)
8	Ajeesh P S	29/M	399983	
9	Thavasiappan K	63/M	400278	Right cervical lesion
10	Ziya M U	7/F	400778	Left facial nerve pathology

## SINGLE FIBER EMG MECHINE OPERATION

Sl. No	Namr	Age/Sex	Hospital No	Diagnosis
1	Sreedharan Nair	76/M	400983	Bilateral Ptosis
2	Rajalakshmi	35/F	401079	Myasthenia Gravis
3	Elizabeth Mamman	58/F	399707	Myasthenia Gravis
4	Sumayya C	16/F	398106	Myasthenia Gravis
5	Rajakumar M	28/M	398371	Ocular MG

## BAEP

Sl.No	Name	Age/Sex	Hospital No	Diagnosis	Rseult
1	Sai Krishna K R	8/M	402569	Static encephalopathy, GDD, Symptomatic Seizures, CPS ET, Probalbe Miller Dieker syndrome	Absent II to V waves from left side and normal peak latencies from right ear
2	Rathish Kumar	8/M	398551	Leucodystrophy - Krabbe vs Mitochondrial v/s Leucodystrophy	Prolonged peak latencies of III,IV,V waves bilaterally and reduced V/I ratio over left side
3	Akhil M R	16/M	385183	Probable Relapsing Remitting multiple sclerosis	prolonged latencies of II, III, V waves on the right ear and I,II,IV,V waves on the left ear respectively
4	Isaac Lalnunfela	1/M	395515	Benign myoclonous of infancy V/s symptomatic myoclonus with GDD	Normal
5	Anvar P	24/M	372496	Primary CNS Demyelination	Inelicitable latencies from left side on ipsilateral and contralateral stimulation and essentially normal latencies from right side

6	Asha Thuhin V R	27/F	304456	Multiple Sclerosis	Normal
7	Antriya Thankachan	1/F	399178	Genetic Syndrome (? CRI DU CHAT) vs cerebral malformation (pachygyria)	Normal
8	Asha Gopan	40/F	400122	CNS Demyelinating Disease , Pachymeningitis	Normal
9	Suja Varghese	30/F	402570	Relapsing Remitting multiple sclerosis	Normal
10	Rejith R S	36/M	384263	Recurrent Brain stem syndrome? Autoimmune	Normal
11	Fahim Faisal	4/M	396492	Probable doose syndrome	Normal
12	Gopi O S	37/M	395615	Logitudinal Extensive Transverse myelitis	Normal
13	Jamaludeen Kunju	54/M	397224	B/L Sequential Abducens Nerve palsy, Diabetic mellitus, Peripheral neuropathy.	Normal
14	Vijayakumar B	16/M	399049	Generalised Dystonia, Spastic Paraplegia, Sequele of probable encephalitis	Normal
15	Smitha Santhosh	31/F	399232	Spastic paraparesis: tropical spastic paraparesis vs primary progressive multiple sclerosis vs complicated hereditary spastic paraparesis	Normal
16	Lakshana	6/F	392338	GDD with autism spectrum disorder, under evaluation	Normal
17	Prasanth B P	28/M	401714	Spastic Ataxic syndrome probable hereditary spastic paraparesis with thin corpus callosum	Normal
18	Precilla Ann Thomas	25/F	403298	Spino Cerebellar Ataxia	Normal
19	Sajeesh P	37/M	399880	? Mitochondrial cytopathy	Inconsistent
20	Sugaina Beegum	13/F	400757	To rule out brain stem involvement	Prolonged III.IV.V waves bilaterally

### VEP

SL.No	Name	Age/Sex	Hospital No	Diagnosis	Stimulation Type	Result
1	Akhil M R	16/M	385183	Probable Relapsing Remitting multiple sclerosis	Pattern	prolonged P100 latencies bilaterally
2	Isaac Lalunfela	1/M	395515	Benign myoclonous of infancy V/s symptomatic myoclonus with GDD	Goggle	normal
3	Sai Krishna K R	8/M	402569	Static encephalopathy, GDD, Symptomatic Seizures, CPS ET, Probalbe Miller Dieker syndrome	Goggle	normal
4	Asha Gopan	40/F	400122	CNS Demyelinating Disease , Pachymeningitis	Pattern	Normal
5	Suja Varghese	30/F	402570	Relapsing Remitting multiple sclerosis	Pattern	prolonged
6	Rejith R S	36/M	384263	Recurrent Brain stem syndrome? Autoimmune	Pattern	Normal
7	Gopi O S	37/M	395615	Logitudinal Extensive Transverse myelitis	Pattern	Normal
8	Jamaludeen Kunju	54/M	397224	B/L Sequential Abducens Nerve palsy, Diabetic mellitus, Peripheral neuropathy.	Pattern	Normal
9	Smitha Santhosh	31/F	399232	Spastic paraparesis: tropical spastic paraparesis vs primary progressive multiple sclerosis vs complicated hereditary spastic paraparesis	Pattern	Normal
10	Lakshana	6/F	392338	GDD with autism spectrum disorder, under evaluation	Goggle	Normal
11	Antriya Thankachan	1/F	399178	Genetic Syndrome (? CRI DU CHAT) vs cerebral malformation (pachygyria)	Goggle	Normal
12	Prasanth B P	28/M	401714	Spastic Ataxic syndrome probable hereditary spastic paraparesis with thin corpus callosum	Pattern	Normal
13	Gokul Das	11/M	399540	Psychogenic movement disorder	Pattern	Normal
14	Renu Mathew	52/F	9803794	Relapsing Remitting multiple sclerosis	Pattern	LEFT: prolonged P100 latencies right: normal p100 latency

15	Vasudevan	49/M	402152	Spastic Paraparesis - Hereditary Spastic Paraparesis vs tropical spastic paraparesis vs primary lateral sclerosis varicella zoster myelopathy	Pattern	Normal
16	Sreenandhana	4/F	401162	Static encephalopathy, GDD, Symptomatic Seizures, CPS ET, PHR; congenital Rubella infection	Goggle	RIGHT: prolonged P100 latencies LEFT:normal
17	Rajendran R	49/M	402185	spinal DA VF with feeders from T8 intercostal artery	Goggle	Normal
18	Rathish Kumar	8/M	398551	Leucodystrophy - Krabbe vs Mitochondrial v/s Leucodystrophy	Goggle	normal P100 latencies bilaterally
19	Asha Thuhin V R	27/F	304456	Multiple Sclerosis	Pattern	Normal
20	Sreekutty	10/F	401881	Longitudinal Extensive transverse myelitis	Pattern	Normal

### SSEP

Sl.No	Name	Age/Sex	Hospital No	Diagnosis	Stimulation Point	Resut
1	Gokul Das	11/M	399540	Psychogenic movement disorder	median	Normal
2	Aamina Z	14/F	381332	PME	tibial and median ssep	Giant
3	Mohammed Arabi	15/M	400881	JME/PME	median	normal
4	Nandamithra	13/F	314680	Myoclonic ataxia syndrome	median	normal
5	Karthikayan S	16/M	377719	PME	median	normal
6	Jeswanth J	4/M	398690	?PME	median	normal
7	Vishnu Dut S	14/M	378829	Cerebellar ataxia	median	normal
8	Archana D J	18/F	386346	Tumefactive Demyelination	median	Normal
9	Rathish Kumar	8/M	398551	Leucodystrophy - Krabbe vs Mitochondrial v/s Leucodystrophy	median	Normal
10	Asha Gopan	40/F	400122	CNS Demyelinating Disease , Pachymeningitis	median	Normal
11	Nagulan A	12/M	403196	Probable SSPE	median	normal
12	Jigar Joshi	11/M	401501	Subacute sclerosing panencephalitis	median	normal
13	Sulochana Gopalan	54/F	399256	Idiopathic Sensory Ganglionopathy	median	normal
14	Keerthi S	6/F	398331	PME vs LGS	median	normal
15	Sreedevika Ravesh	5/F	397303	Progressive Myoclonic epilepsy	median	Normal
16	Jibin Varghese	29/M	397443	Mitochondrial cytopathy, Probable myoclonic epilepsy with Ragged Red fibers	median	gaint
17	Urmila	19/F	385504	Primary CNS Demyelination, Depression, Vascular Headache	median	normal
18	Ashik Sabu	74/M	386165	Symptomatic lennox gestaut syndrome	median	prolonged
19	Hari Krishnan	11/M	392334	Longitudinal Extensive transverse myelitis	median and tibial ssep	normal
20	Shilpa K O	17/F	324532	Mitochondrial cytopathy, Probable MELAS	tibial and median ssep	Giant

## VISUAL ACUITY AND VISUAL FIELD CHARTING

Sl.No	Name	Age/Sex	Hospital No	Diagnosis	Visual Field Report
1	Beena D	34/F	374322	Pituitary Adenoma	bitemporal hemianopia
2	Ramith Raveenran	27/M	388182	Right mesial temporal Ganglioglioma	Left eye showed normal visual field and right eye showed superior altitudinal field defect
3	Shajahan H	53/M	391103	Tuberculum Sella Meningioma	Peripheral field constriction on right eye and inconsistent visual field on left eye
4	Muhammadali	41/M	373803	Right parietal convexity lesion	Normal
5	Mathews Samuel	57/M	393179	Clival Chordoma	Essentially normal
6	Anil Kumar J	40/M	393615	Probable idiopathic intracranial hypertension, Hypothyroidism	Normal
7	Vasanthakumari B	47/F	390334	Pituitary Adenoma	Bitemporal visual field defect
8	Sarojini T K	69/F	391178	Left Coronal / High Frontal Convexity /meningioma	mild constriction of visual field on left eye and normal on right eye
9	Vijayan B	56/M	394077	Optic neuropathy	Severly constricted visual fields bilaterally (left > right)
10	Rajina J	34/F	371282	Idiopathic Intracranial Hypertension	Paracentral scotoma in the superior temporal field of left eye and right eye could not be done due to poor visual acuity
11	Mohanam J	60/M	391822	Pituitary Adenoma	Right incomplete Homonymous Hemianopia
12	Gopalakrishna Pillai	69/M	313811	Recurrent Left Pterional Meningioma	Right eye: nasal field defect ; left eye : not done (poor visual acuity)
13	Maharajan V	54/M	388245	Right CP angle lesion	Normal
14	Harish R	22/M	396284	Chronic Dural Venous Sinus Thrombosis S/P Thecoperitoneal Shunt (22/04/2015)	Normal
15	Biji Chandran	37/M	397874	Idiopathic Intracranial Hypertension	Left eye : mild nasal field defect; right eye : peripheral field defect
16	Shilpi Kumari	14/F	393206	Right posterior quadrantectomy on 19/06/2015	Not co-operative for visual field charting
17	Bessy Benny	45/F	244826	Recurrent left clinoidal meningioma	Right eye : essentially normal ; left eye : not done (poor VA)
18	Lekha Pavithran	44/F	391109	Recurrent right parasellar meningioma	Right eye : not done (poor visual acuity) ; Left eye : left superior quadrant field defect
19	Radha P K	52/F	389590	Pituitary Macroadenoma	Right: temporal field defect : Left eye : poor visual acuity (not done)
20	Subhadramma A	65/F	245272	Left CP angle lesion with hydrocephalus	Not co-operative for visual field charting
21	Kingsley G	18/M	326080	Recurrent craniopharyngioma	Not co-operative for visual field charting
22	Archana S	35/F	267512	Recurrent ACTH Secreting Pituitary adenoma	Left eye : Incomplete nasal field defect ; Right eye : Poor VA
23	Sreenandhana	11/F	402426	CPS ET ; PHR VS Frontal	incongruous left homonymous hemianopia
24	Usha S	43/F	232304	Autoimmune Hemolytic anemia - post splenectomy	Not done due to poor visual acuity
25	Rahul R	12/M	376326	Recurrent bilateral optic neuritis	constriction of peripheral field of vision
26	Vasanth D	14/F	376729	Raised Intracranial Pressure	Enlargement of blind spots bilaterally
27	Eleena Varkey	28/F	377651	Idiopathic Intracranial Hypertension	Normal
28	Anil Kumar S	47/M	378598	Probable VOGT-KOYANAGI-HARADA disease	Normal
29	Remya Jomon	23/F	380448	Post partum cortical venous sinus thrombosis, Hyperhomocysteinemia	left homonymous hemianopia
30	Haritha M K	32/F	379480	Benign Intracranial Hypertension, PCOD, Migraine	peripheral field constriction bilaterally
31	Cleetus	50/M	380822	Cortical venous thrombosis, chronic liver disease with portal hypertension.	Normal

32	Jagadeeswari S	24/F	381497	Idiopathic Intracranial Hypertension	constriction of visual fields (R>L)
33	Geetha S	28/F	380095	Cerebral venous sinus thrombosis	Normal
34	Nanditha Gopinath	19/F	232509	Idiopathic Intracranial Hypertension	enlargement of blind spot
35	Manikandan K	30/M	384526	Multisite DA VF at tentorium	Normal
36	Bindu Anil	43/F	384575	Subacute Bilateral Visual Failure	Normal
37	Sandra S	19/F	379858	Multiple Sclerosis	Normal
38	Manju C	31/F	388516	Idiopathic Intracranial Hypertension	Normal
39	Sukumaran K	61/M	386677	Right Tuberculum Sella Meningioma	Left superior temporal field defect on left eye ; right could not be done due to poor VA
40	Biju Kumar	44/M	366574	Post operative pituitary adenoma	Essentially normal
41	Kala S K	43/F	9501714	Supracellar archnoid cyst	bitemporal hemianopia
42	Marthandan A	48/M	372582	Left insular glioma	incomplete left homonymous hemianopia
43	Thulasirajan	32/M	349355	Pituitary Apoplexi	Normal
44	Akanshya Adhikary	17/F	357739	Left ATL + AH	Right superior Quadrant anopia
45	Muhammed S	19/M	305567	Left ATL + AH on 06/11/2013	Right incomplete superior quadrant anopia
46	Junaid K V	24/M	343371	Left frontal lesion	Right homonymous hemianopia
47	Ayisha Fida	11/F	335496	Left ATL + AH 17/2/2014	incomplete right homonymous hemianopia
48	Latha Vijayan	51/F	350689	Pachy Meningitis	Central field defect
49	Athika Ollakkan	20/F	312943	Left ATL +AH 20/2/2014	Right superior Quadrant anopia
50	Saroj Shaw	12/M	352599	Right occipital glioma	incomplete left homonymous hemianopia

## ELECTROENCEPHALOGRAM

Sl.No	Name	Age/Sex	Hospital No	Diagnosis
1	Sudhakaran K	54/M	275436	CPS Temporal
2	Ayyappan S	2/M	394355	Symptomatic LRE Right Frontal
3	Shiva Surya	11months/M	393380	Downs Syndrome
4	Suresh M	12/M	339715	LKS
5	Arpitha B	7/F	391723	Post encephalic sequelae
6	Mithra S Manoj	3/F	393020	Panayitopolous
7	Akhil S	24/M	393052	JME
8	Arun A S	15/M	390313	BOE
9	Kishore S	47/M	333156	CNS-TB vasculities right parietal infarct
10	Muhammed Rashid	6/M	394697	Stuege weter syndrome phaces syndrome
11	Sreelekshmi P	15/F	394748	IGE-IPOE
12	Ann Rose Chandy	7/M	389840	Gefs plus
13	Prannav P	18/M	262546	Fronto Parietal Craniotomy on 14/1/15
14	Sathosh Kumar	47/M	296966	Right ATL + AH 8/1/2015
15	Archna P	10/F	311651	Hypothalamic Hamartoma resection 22/3/2013
16	Rajeshwari S	4/F	394981	Static encephalopathy
17	Muhammed S	15/M	392433	MELA
18	Jacob V	79/M	394933	Alzheimer's Disease
19	Essakki Pandi M	24/M	351526	MELA'S
20	Subhadep Das	10/M	387411	Atypical BECTS
21	Anju Lal	18/F	357775	Autoimmune encephalities
22	Gokul M	3/M	395139	SSPE
23	Aswin A	1/M	395193	West Syndrome, (tonic seziures)
24	Gokul Krishna S	4/M	394775	GEFS + ?IGE
25	Vishnu K V	22/M	320559	Left temporal cavernoma H/O CPS
26	Madhavan Nadar	75/M	384156	Menegioma
27	Amal Muhammed	11/M	389764	Absence Seizure
28	Nithin S	12/M	316478	CPS ET,ADHD
29	Subin Sabu	17/M	390194	Superrefractory Status epilepticus
30	Krishnavenin A	22/F	394437	Probable FLE
31	Saketh Vidyadhran	8/M	353890	Prrimary status epilepsy
32	Jemina	11/F	289691	Jeavens Syndrome

33	Prasanna Raj A	47/M	393107	Pseudo temporal
34	Lopamundra Jena	10/F	372635	Sps-ET Left Pericelvian
35	Devi Shaji	18/F	381663	Temporal Lobe Epilepsy
36	Neethu S	19/F	9601004	Migraine with aura
37	Farhan Al Nafia	8/M	345558	Viral encephalitis
38	Ananya K	4/F	392561	Spastic CP
39	Sona Shiba	13/M	258153	Cerebellar AVM
40	Sumi	26/F	395385	Mitochondrial disease
41	Meera A	11/F	348029	Left occipital lobe epilepsy
42	Padmapriya N	3/F	392244	CSWS
43	Jeevan Biju	6/M	395386	Panyitopolous Syndrome
44	Santhama	65/F	182896	Late Onset Seizure
45	Indhu Gayathri	10/F	373636	Left mesial temporal glioma
46	Sibin Subath	12/M	395528	HDEM
47	Raegan R Pereira	4/M	392895	Idiopathic autism
48	Bincy B	14/F	275031	BLRE
49	John Thomas	63/M	395685	Alcoholic recurrent syncope
50	Ashbel Antony	1/M	395544	Global Development Delay with MJ
51	Chidambaram G	10/M	391500	Infantile hemiplegia CPS ET
52	Amal Das	9/M	9106353	Medullaoblaetus CPS
53	Gopika U P	6/F	306130	West Syndrome
54	Vanaja Kumari	48/F	394654	Late onset seizure
55	Thushar Mohan Narang	24/M	373691	Left temporla DNET
56	Ravikumar M	32/M	368306	Eating Epilepsy
57	Mubaris C M	14/M	373682	SSPE
58	Inu Durivedi	3/F	358216	Complex febrile seizure
59	Thomas Simon	65/M	241451	Late Onset Seizure;?NCSE
60	Gothai S	17/F	373825	Mediallary refractory epilepsy with underlining neuronal megrate disorder
61	Arun Kumar S	24/M	372655	Primary psychiatric disorder
62	Mohammed Sayeir	49/M	373315	Chronic renal failure
63	Soorya Gayathri	20/F	193185	Visual sensitivty epilepsy
64	Dhamodharan Nair R	81/M	28708	Super refractory Status epilepticus;Bihemisphere Stroke
65	Pradul P S	7/M	275670	LGS
66	Jannath Sherin	14/F	323965	Autoimmune encephalities
67	Lekha	11/F	279652	Jeavon's Syndrome

68	Sudheesh Gopi	26/M	366139	CPS occipitotemporal
69	Muhammed Fayaz	8/M	374348	Dravets Syndrome
70	Aryan M S	7/M	374365	LKS
71	Biddyath Das	41/M	280311	Eating Epilepsy
72	Sabi Susan George	47/F	8605694	JME
73	Vijaya Kumar S	45/M	374347	Adoloesent Onset epilepsy; Ataxia + MJ
74	Kasim Kunju V	71/M	374324	Dementia
75	Timiri Sai Suparna	28/F	250828	Multilobar resection 31/01/2008
76	Keerthi A	28/F	304689	Left parietal lesionectomy 6/01/11
77	Makkar Kunju	53/M	263695	Old MCA infarct; Symptomatic Seizure
78	Kavya M	8/F	374711	CPS PHR Vs PNES
79	Mohanapriya	18/F	369917	PME
80	Neenu P	24/F	291116	Myoclonic Epilepsy
81	Dharush N S	4/M	373819	ADEM
82	Ancy Prasad	10/M	357452	Localisation related epilepsy
83	Gopinathan Nair	60/M	365754	Nonconvulsive Status Epilepticus
84	Vasanthakumari	54/F	374224	PNES
85	Dhineswaran	2/M	355982	Symptomatic west syndrome
86	Lekshmi M	1/F	370422	Infantile Spasm (Hypsarhythmia)
87	Aruna A	17/F	330229	Autoimmune encephalities( Triphasic waves)
88	Krishnahari S Nair	5/M	374690	LKS
89	Vijayan B	64/M	9401010	Metabolic Encephalopathy
90	Thanshika S	6months/F	375512	?Retti Syndrome
91	Asif Muhammed	7/M	303491	Autism
92	Sanjay S	2/M	375712	Myoclonus with ataxia
93	Lekha M	30/F	371145	Post trumatic epilepsy
94	Blessy Mohan	17/F	347337	NMDA Encephalitis
95	Mohammed Yshir Arafath M	9months/M	376864	Tuborn errors of metabolism
96	Govindan Nair	73/M	371766	Atypical parkinsonism
97	Dhanalekshmi	16/F	377146	Childhood absence epilepsy
98	Vishnu S	18/M	377678	Jourith Syndrome
99	George N C	70/M	204662	Deliru.....
100	Aathira Suresh	15/F	294820	Juvenile Absence epilepsy

## VEDIO ELECTROENCEPHALOGRAM

Sl.No	Name	Age/Sex	Hospital No	Diagnosis
1	Anshina B	16/F	338077	Frontal lobe epilepsy (MRI - negative)
2	Hariharan A	25/M	353527	CPS ET left hemispheric, MRI negative
3	Kinjalben A	25/F	385523	Nocturnal frontal lobe epilepsy. MRI negative, CPS ET -SMA
4	Bhavik Mansukhbhai	24/M	385522	Post meningitis sequelae, static encephalopathy
5	Gautham Bhupabhai	20/M	385533	CPS ET right hemispheric, right schizencephaly with heterotopia(periventricular)
6	Nitaben Kantibhai	26/F	385531	CPS Temporal. MRI- left MTS
7	Srijay Poddar	9/M	355270	CPS ET-B/L PHR gliosis
8	Subaih P K	12/M	331339	CPS , left temporal neocortical, left MTS S/P left ATL+AMIGDALO HIPPOCAMPECTOMY 8/10/2014
9	Ahamed Zaad	5/M	385541	Left hemimegalencephaly, CPS nad SPS - left hemispheric refractory
10	Priyanka S Pillai	24/F	383294	CPS temporal neocortical, left inferior temporal ganglioglioma PGE trait
11	Shashank Saravagi	25/M	385724	Juvenile Myoclonic epilepsy
12	Aditya Kumar	8/F	365838	Benign rolandic epilepsy
13	Rudheer Sreebehara	8/M	326065	Mineralising microangiopathy with symptomatic seizures and GDD
14	Shameena P S	35/F	385712	CPS temporal plus vs temporal neocortical
15	Kashmira Pradeep	30/F	385738	CPS ET -left hemispheric - cryptogenic focal CSWS left hemispheric
16	Rahul R	19/M	385758	CPS ET
17	Yashmita	20/F	385771	Primary Generalised Epilepsy
18	Karthik Thampi	6/M	363031	Atonic variant of Benign rolandic epilepsy
19	Hephzibah Ruth	8/F	382605	Refractory Hypomotor CPS
20	Sharika Shulkla	31/F	382584	CPS left temporal-neocortical
21	Joshi Kalpesh	15/M	382628	Idiopathic generalised epilepsy, JME
22	Atreyee Saha	26/F	273548	Left hemispheric CPS with clustering, VNS done (30/4/2009). VNS battery removal done on 5/8/2014
23	Pavithran R	20/M	292139	Left temporal DBET VS FCD failed ATL + AH
24	Ancy Rajesh	25/F	279926	Idiopathic primary generalised epilepsy - JME
25	Geetha Ravi	35/F	351013	CPS ET VS temporal left periventricular nodular heterotopia
26	Abhishek M S	13/M	322243	CPS ET; left parieto-occipital gliosis with ulegyria parietooccipital lesionectomy done on 10/4/2013
27	Muhammed Sabir	9/M	348564	CPS right temporal ; right temporal DNET excision done on 6//8/2014
28	Jayalakshmi B	13/F	382916	CPS temporal
29	Daksh A A	0/M	382924	Symptomatic west syndrome
30	Nissy Jacob	17/F	320030	CPS ET, right hemispheric HHE syndrome, right hemispherotomy done on 7/8/2014
31	Karthik thampi	6/M	363031	Atonic variant of Benign rolandic epilepsy
32	Gokul G	27/M	380261	Right Mesial temporal epilepsy,Hippocampal sclerosis

33	Amit Singh	25/M	362336	CPS ET postencephalitic sequelae
34	Aiswarya Ravikumar	5/F	363505	Atypical BLRE
35	Vansh Dusad	13/M	382922	CPS ET SMA semiology possible left frontal, PET positive
36	Dipak Vijay	3/M	382943	CPS ET right frontal FCD
37	Ajeena Shajahan	33/F	382920	PNES
38	Safwan A	4/M	360155	Severe smptomatic localisation related epilepsy, B/L parieto occipital gliosis (right >left), right parieto occipital resection done on 8/8/2014
39	?Queency M	22/F	383026	CPS ET left frontal
40	Ramesh Kumar	19/M	331645	Right MTS with Neocortical change-post traumatic
41	Jeenal Jayprakash	10/F	383140	Psychogenic non epileptic events , to exclude previous frontal lobe epilepsy on follow up
42	Sonali Singh	41/F	383155	CPS ET PHR- likely visual sensitive epilepsy
43	Nasirkhan Hasankhan	42/M	354489	CPS Temporal neocortical, left MTS with left hemispheric atrophy, left ATL + AH (31/7/2014)
44	Minaxiben S	17/F	382969	PGE- Probable Juvenile absense epilepsy
45	Jiss Mon Thomas	17/M	382930	Probable Dravet syndrome
46	Anagesh Kannan A S	9/M	382815	SPS -right parietal FCD
47	Milan C Byju	2/M	382963	Extra temporal epilepsy, MRI neagative
48	Amal P S	15/M	190014	Post encephalic sequalae, CPS probable right extra temporal
49	Anil Kumar	34/M	8703399	CPS ET left hemispheric, parieto occipital gliosis , probable perinatal insult, probalbe drug induced ataxia, hypothermia and peripheral neuropathy
50	Himanshu Shekhar	24/M	383128	PGE- Probable myoclinic epilepsy
51	Samiya M	9/F	316855	Lennox Gastaut Syndrome
52	Krishna Harshadbhai Soni	9/F	383291	PNES
53	Rabi Shankar	17/M	282174	CPS b/l PHR gliosis
54	Shivanya Biju	5/F	382942	Symptomatic extra temporal epilepsy, perinatal insult
55	Ahana Das	5/F	317649	MRI nergative symptomatic LRE under evaluation; GDD
56	Shilpa K V	24/F	331060	IGE Possible JME-ipoe overlap
57	Chris George	9/M	353551	CPS ET left hemispheric
58	Anil Kumar	44/M	382590	probable symptomatic generalised epilepsy
59	Anamdhu H	16/M	298726	Left periventricular Nodular Heterotopia, symptomatic extra temporal epilepsy
60	Tinchu R J	28/F	216655	CPS Temporal plus , tuber cinerium hamartoma delalande II
61	Debayan Banerjee	13/M	308008	Landau Kleffner syndrome
62	Sreerag K V	4/M	383614	Static encephalopathy, possible neurometabolic syndrome
63	Piyush Guptha	17/M	345812	Idiopathic photosensitivie occipital epilepsy - IGE overlap
64	Shabib K P	14/M	370945	Right MTS, PNES
65	Ansh Mandhani	13/M	383597	BECTS
66	Ashwin S	6/M	383585	Atypical BLRE, Acquired epileptic aphasia

67	Rajashree Patidar	5/F	383767	Acquired epileptic aphasia, Probable Landau Kleffner syndrome
68	Harsha S	13/F	380967	PNES
69	Thanti Viren	13/M	383592	Static encephalopathy ,CPS ET, spastic Bladder
70	Lekshmi Priya	16/F	326596	Paroxysmal tonic up gaze disorder of childhood, dissociative disorder, IGE trait
71	Priyanka Musaddi	28/F	383598	Visual sensitivity epilepsy (IGE - IPS overlap)
72	Shwetanshu Sekhar	9/M	383591	PDP-ASD
73	Binu S	22/M	223593	Landau Kleffner syndrome, clustering of seizure
74	Albin George	11/M	383265	Autosomal dominant cortical tremor with myoclonic epilepsy
75	Vinu M	26/M	333405	CPS left temporal, left temporal lesion (?DNET), left lesionectomy with amygdalohippocampectomy (27/08/2014)
76	Al Zahra Alim Abdullah	21/F	358374	CPS right frontal , post op case of right insular resection for right fronto-insular dysplasia
77	Mirshan C	10/M	383269	Probable IGE syndrome
78	Virendra Kumar	35/M	383912	PNES
79	Murrlihar Tripathi	34/M	383909	PGE - JME
80	Dhrupad Vipin	5/M	369067	CPS Temporal ATL+ AH and insular cortical resection 28/8/2014
81	Prachi Saraf	4/F	363051	PUO- Brucellosis, secondary generalised epilepsy
82	Zakir Ali Khan	29/M	383972	CPS ET; left parieto occipital focal cortical dysplasia
83	Swathi Gundapaneni	29/F	383973	Probable PGE (JME)
84	Jannatul Ferdous Mon	29/F	358087	CPS Temporal plus - post encephalitic sequel
85	Shivam singhal	17/M	384048	CPS ET PHR, M RI -b/l occipital gliosis
86	Bandi Yashaswini	17/F	384017	CPS ET right subcortical band heterotopia
87	Piyush Sharma	7/M	339454	CPS ET right hemispheric
88	Preethi G	14/F	384115	CPS ET Right hemispheric , right parieto occipital gliosis
89	Animesh Dash	15/M	367677	CPS B/L PHR , b/l PHR gliosis
90	Guru Prakash	24/M	383937	CPS ET MRI multiple calcification, lesions(right frontal, right parietal, left PHR
91	Amur Mohamed Hamed Al	26/M	383990	Epilepsy with myoclonic absences (Tassinari syndrome)
92	Joanna Sarah	0/F	384726	Probable Dravet syndrome
93	Dilliraj Gyawale	71/M	385034	CPS ET right hemispheric Highh Grade Glioma right fronto temporal region.
94	Praveen Ravi	12/M	384778	CPS ET right hemispheric
95	Shilpa Dagor	42/F	384753	CPS temporal -left temporal meningioma; hypothyroidism on treatment
96	Lena Jojy	3/F	377061	Symptomatic west syndrome ,static encephalopathy -sequelae to perinatal insult.
97	Siddharth Jose	22/M	384788	Probable generalised epilepsy with febrile seizures plus syndrome
98	Sreelekshmi S R	12/F	384991	Sturge Weber syndrome-right occipito temporo parietal calcification
99	Puja Dash	21/F	385137	CPS ET with secondary GTCS - Probable left hemispheric
100	Ashik Sabu	8/M	386165	Lennox Gastaut Syndrome

### VEEG INVASIVE MONITORING

Sl .No.	Name	Age/Sex	Hospital No.	Electrode Placement	Diagnosis
1	Shabnam Anwar	18/F	369146	Bilateral 8 contact hippocampal depth electrode placement on 7/1/2015	CPS, left mesial temporal sclerosis; s/p left anterior temporal lobectomy with amygdalohippocamectomy on 17/6/2015
2	Sagar Sachdeva	21/F	378777	48-contact grid - right temporal neocortex, two 6 contact subtemporal strip (RATS and RPTS) and one 8 contact strip over right inferior frontal gyrus (RIFS)	Right temporal neocortical(MRI -VE, PET - +VE), s/p right extended anterior temporal lobectomy and AH on 1/4/2015
3	Manish Shantaram	41/M	395946	32 contact grid over right temporal region (contact 1 over anterior inferior, 32 over superior posterior); 6 contact strip - right frontal opercular; three 4 contact strip over right anterior subtemporal, right middle subtemporal, right posterior subtemporal	CPS temporal neocortical, non dominant, s/p right anterior temporal lobectomy and AH on 9/6/2015
4	Jagadish Prasad	45/M	383167	32 contact grid over left temporal region ; 6 contact strip - left sub frontal ; three 4 contact strip over left anterior subtemporal, left middle subtemporal, left posterior subtemporal	CPS ET,s/p Left ATL + AH with excision of gliotic areas in left orbitofrontal region on 10/7/2015
5	Sagar Tyagi	12/M	379187	64 contact grid over left frontal	CPS ET, left frontoparietal FCD, s/p left fronto temporoparietal craniotomy
6	Rohit Vallabhabhai	30/M	370771	20 contact grid - left frontal; 16 contact grid - left temporal; 6 contact depth - left hippocampus; 6 contact depth - right hippocampus	CPS temporal, left MTs, right ATL + AH & ight frontal porenephalic cyst wall excision on 4/12/2014
7	Ayisha Fida M	11/M	335496	20 contact grid-left lateral temporal; 4contact strip -left inferior frontal gyrus; 6 contact strip- left posterior subfrontal; 6 contact strip- left anterior basifrontal; 4 contact strip- left anterior sub temporal; 4 contact strip- left middle subtemporal ; 4 contact strip- left posterior sub temporal	CPS left hemispheric left MTS;S/P left ATL+ AH on 17/02/2014
8	Drishiya K	16/F	282470	32 contact grid - right parietal region; 16 contact grid- right temporal region; 6 contact strip- right mesial parietal strip; 6 contact strip - right basal margin; 6 contact depth - right hippocampal; 6 contact strip - left lateral occipital	CPS right PHR,right occipital lobectomy 14/1/2009,extended occipital lobectomy,posterior temporal and parietal lobectomy on 6/5/2015
9	Nahas K P	30/M	357875	6 contact depth - right hippocampus; 6 contact depth - left hippocampus	CPS temporal, right MTS, S/P right anterior temporal lobectomy and AH on 23/10/2015
10	Sara Hassan	27/F	370925	32 Grid- left temporo-occipital; 8contact strip-left mesial occipital strip; 6 contact-left anterior subtemporal;6 contact strip - left middle subtemporal; 6 contact strip - left posterior subtemporal; 6 contact strip - right anterior subtemporal; 6 contact strip - right posterior subtemporal	CPS temporal neocortical; post encephalitic sequelae; MRI- left mesial temporal sclerosis with left temporo-occipital gliosis. Left temporaloccipital gliosis excision done on 7/4/2014

### VAGAL NERVE STIMULATION

Sl .No.	Name	Age/Sex	Hospital No.	Diagnosis
1	Paresh O H Bandhiya	23/M	250220	CPS RHR,Bilateral perisylvian gliosis, VNS on 21/5/2009, Battery replacement on 19/11/2014

## CORTICAL STIMULATION

Sl .No.	Name	Age/ Sex	Hospital No.	Electrode Placement	Diagnosis
1	Sagar Tyagi	12/M	379187	64 contact grid over left frontal	CPS ET, left frontoparietal FCD, s/p left fronto temporoparietal craniotomy
2	Sagar Sachdeva	21/F	378777	48-contact grid - right temporal neocortex, two 6 contact subtemporal strip (RATS and RPTS) and one 8 contact strip over right inferior frontal gyrus (RIFS)	Right temporal neocortical(MRI -VE, PET - +VE), s/p right extended anterior temporal lobectomy and AH on 1/4/2015

## VEEG MONITORING AFTER SPHENOIDAL ELECTRODE INSERTION

Sl .No.	Name	Age/Sex	Hospital No.	Diagnosis
1	Sindu	14/F	354012	CPS-right MTS with right frontal subcortical gliosis
2	Rajesh Rakhuliya	46/M	364958	CPS temporal with left MTS
3	Satheesan K S	63/M	307712	CPS left temporal (left MTS)
4	Prakasan Gothi	30/M	380428	CPS left temporal neocortical, right hemiatrophy, (left MTS with neocortical changes in MRI)
5	Jameela K	46/F	268905	CPS right temporal

## ECoG

Sl .No	Name	Age/ Sex	Hospital No.	Diagnosis & Operation Procedure
1	Mohammed Fasmii	19/M	281474	Cps Temporal, Right Mesial Temporal Sclerosis, Underwent Right Anterior Temporal Lobectomy With Amygdalohippocampectomy on 07/01/2015
2	Santhosh Kumar	47/M	296966	Cps Right Temporal; Right Temporal Craniotomy, Anterior Temporal Lobectomy and Amygdalohippocampectomy(8/1/2015)
3	Sreeraj p	14/M	359402	Complex Partial Seizures-Right Posterior Head Region, s/p Post Right Parietooccipital Craniotomy With Occipital Lobectomy and Posterior and Temporal Resection on 8/1/2015
4	Shalini S Jamwal	28/F	365185	Complex Partial Seizurestemporal Lobe Epilepsy, Left Mesial Temporal Lobe Epilepsy, Left Anterior Temporal Lobectomy with amygdalohippocampectomy on 14/1/2015
5	Pranav p.	18/M	262546	Complex Partial Seizures - Right Frontal Focal Cortical Dysplasia, s/p Right Paracoronal Frontoparietal Craniotomy and Lesionectomy on 14/1/2015
6	Tejal K Gawade	14/M	277531	Complex Partial Seizurestemporal; Left Temporal Cortical Dysplasia, Underwent Left Anterior Temporal Lobectomy with amygdalohippocampectomy on 15/01/2015
7	Vagasiya	42/M	359650	Cps Temporal Lobe Epilepsy, Right Mesial Temporal Lobe Sclerosis, Right Temporal Craniotomy anterior Temporal Lobectomy with amygdalohippocampectomy on 15/1/201
8	Praveen Ravi	12/M	384778	CPS ET, Right Hemispheric, Right Frontal FCD, Underwent Right Frontal Craniotomy and total excision under neuronavigation on 21/01/2015
9	Mef:Ra .a	11/F	348029	CPS PHR, Left Occipital Gliosis, Left Occipital Lobectomy on 21/1/2015
10	Sangeeta Devi	41/F	365295	CPS Temporal Right MTS s/p Right Anterior Temporal Lobectomy and Amygdalohippocampectomy on 28/1/2015
11	Anu Karthika n	10/F	351538	CPS- Right PHR; Right Parieto-Occipital Gliosis Sip Right Occipital Craniotomy and Lesionectomy (Occipital Lobectomy) on 28/1/2015
12	Najmun Nahar	22/F	373807	Cps ET Right Posterior Temporal Gliosis s/p Anterior Temporal Lobectomy, Amygdala Hippocampectomy with Post Extended Hippocampectomy on 29/1/2015
13	Bhasurangi k.	57/F	370287	CPS Temporal- Left Mesial Temporal Sclerosis , s/p Left Anterior Temporal Lobectomy and Amygdalohippocampectomy on 29/1/2015
14	Dhrupad Vipin	5/M	369067	Rasmussen Encephalitis, CPS Etright Hemispheric, s/p Right ATL + AH
15	Senthil Kumari v.	31/F	382838	CPS temporal MRI - Left Mesial Temporal Sclerosis, s/p Left Anterior Temporal Lobectomy and Amygdalohippocampectomy on 29/2015

16	Silpa Sajan	18/F	368629	CPS Pseudotemporal- Right Posterior Head Region ; Mri Brain Right Occipital Gliosis, Sip Right Occipital Craniotomy And Lesionectomy On 3/9/2015
17	Dipak Vijay Bhai Bhalani	4/M	382943	Right Fronto-Temporo-Insular Cortical Dysplasia, s/p Frontotemporo-Insular Resection (Anterior Quadrantectomy) On 16/9/2015
18	Dhruv.b.Patel	15/M	258942	Symptomatic Localisation Related Epilepsy , Right Frontal Focal Cortical Dysplasia , s/p Right Frontal Lobectomy ( 17/9/2015)
19	Saranya k	27/F	373519	Left Mesial Temporal Lobe Epilepsy Left MTS, s/p Left Anterior Temporal Lobectomy with Amygdalohippocampectomy Done On 23/9/1
20	Rajiv	23/M	311305	CPS-Right Frontal with Right Focal Cortical Dysplasia(Type I)-Operated on 15/09/2011

### INTRA OPERATIVE SSEP

Sl.No.	Name	Age/Sex	Hospital No.	Diagnosis & Operation Procedure
1	Sarang Krishna	10/M	381605	Right frontal Lesionectomy
2	Shilpi Kumari	14/F	393206	Occipital-parietal-temporal gliosis, s/p right posterior quadrantectomy on 19/6/2015
3	Dhruv B Patel	15/M	258942	Symptomatic Localisation Related Epilepsy , Right Frontal Focal Cortical Dysplasia , s/p Right Frontal Lobectomy ( 17/9/2015)

### f-MRI

Sl. No	Name	Age/Sex	Hospital No	Diagnosis
1	Rahul R L	30/M	9505284	CPS ET left hemispheric ; Rasmussen encephalitis sequelae
2	Aysha U	12/F	365847	CPS ET Left parietal DNET
3	Aniket Sarkar	14/M	400422	Left temporal AVM, SM grade III
4	Abhilash Sarkar	14/M	277217	CPS left PHR, left parieto temporal gliosis
5	Jeyasurya M	13/M	373800	CPS ET; Left hemispheric

### POLYSOMNOGRAPHY, CPAP & MSLT

Sl. No	Name	Age/Sex	Hospital No	Study Done	Report
1	Ambikumar N	52/F	394925	PSG	Severe degree of Obstructive sleep apnea with an Apnea/Hypopnea index of 73.2 event per hour
2	Anadavally Amma	72/F	7195	PSG	Severe degree of Obstructive sleep apnea with an Apnea/Hypopnea index of 61.0 event per hour
3	Binod Kediya	53/F	394777	PSG	Mild to moderate degree of Obstructive sleep apnea with an Apnea/Hypopnea index of 17.2 event per hour with significant oxygen desaturation to a nadir of 93.4 - 87.0 %.
4	Esther Bai	82/F	392860	PSG	Severe degree of Obstructive sleep apnea with an Apnea/Hypopnea index of 67.6 event per hour with significant oxygen desaturation to a nadir of 85.7 - 48.0 %.
5	Poushi Kannu n	62/f	208006	PSG	Mild Degree Of Obstructive Sleep Apnea With An Apnea/Hypopnea Index Of 8.4 Event Per Hour With Significant Oxygen Desaturation To a Nadir Of 72.2 - 52.0 %.
6	Rajesh V S	48/M	383226	PSG	Severe degree of Obstructive sleep apnea with an Apnea/Hypopnea index of 56.4 event per hour with significant oxygen desaturation to a nadir of 89.1 - 42.0 %.

7	Mathew . P. Rajan	52/M	390510	PSG	Severe OSA
8	Habeebulla M	59/M	391145	PSG	Severe OSA
9	Sankaranarayana Pillai N	79/M	335417	PSG	Mild apne
10	Rajendran N	68/M	229007	PSG	Moderate to severe
11	Devika . G	14/F	389677	PSG	Nil-apnea
12	Janova .P	39/F	386660	PSG	Mild apnea
13	Thankamani . P . S	69/F	389137	PSG	Mild OSA(significant during REM)
14	Amal Krishnan	12/M	388901	PSG	Apnea - Nil
15	Sanhosh K	42/M	395155	PSG	Delayed sleep onset ; mild OSA
16	Sobhana Kumari	53/F	393890	PSG	Mild apnea
17	Santhosh K	42/M	395155	PSG	Delayed sleep onset; Mild degree of Obstructive sleep apnea with an Apnea/Hypopnea index of 10.4 event per hour with significant oxygen desaturation to a nadir of 95.9 - 84.0 %.
18	Sobhanakumari	53/F	393890	PSG	Mild Obstructive sleep apnea with an Apnea/Hypopnea index of 2.2 event per hour with significant oxygen desaturation to a nadir of 96.9 - 73.0 %
19	Venugopal N	59/M	392863	PSG	Severe apnea
20	Ambikumar	53/M	394925	PSG	Severe apnea
21	Binod Kediya	53/F	394777	CPAP	CPAP pressure was found to be 16 cm H2O. This much pressure was not tolerated by the patient. As per patient request CPAP was disconnected at 3.45am; Medium size oro-nasal mask was used; The respiratory events were reduced to an AHI of 4.5 events per hour. A good home trial is advisable.
22	Gopalapanicke R	73/M	372254	CPAP	Mild degree of Obstructive sleep apnea with an Apnea/Hypopnea index of 5.9 event per hour with significant oxygen desaturation to a nadir of 96.2 - 82.0 %.; Upper Airway resistance syndrome
23	HAREESH R	15/M	392868	CPAP	Bi-level PAP was used and the pressure settings was found to be 17IPAP- cm H2O., 13 EPAP cm H2O; Patient was most comfortable with Medium size oro-nasal mask; The respiratory events were reduced to an AHI of 8.9 events per hour.
24	STELLA J	56/M	381096	CPAP	Ideal CPAP pressure was found to be 23cm H2O; Bi-level PAP was used and the pressure settings was found to be IPAP- 23cm H2O., EPAP 19cm H2O; Patient was most comfortable with Medium size oro-nasal mask; The respiratory events were reduced to an AHI of 5.3 events per hour.
25	Anadavally Amma	72/F	7195	CPAP	Ideal CPAP pressure was found to be 23 cm H2O.; Bi-level PAP was used and the pressure settings was found to be IPAP- 23cm H2O., EPAP 19cm H2O; Patient was most comfortable with Medium size oro-nasal mask; The respiratory events were reduced to an AHI of 5.8 events per hour.
26	Saji	41/M	392156	MSLT	Idiopathic hypersomnia - mean sleep latency =64min
27	Sidhique P I	50/M	321866	MSLT	MSL-1.5min; SOREM = 2
28	Jaya K S	43/F	312468	MSLT	MSL - 5.8 min
29	Sidhique	51/M	394634	MSLT	? Narcolepsy; MSL - 5.1min
30	Adith Jaya Prakash	16/M	394654	MSLT	Narcolepsy with Cataplexy ; MSL - 30 sec