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

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

POST GRADUATE DIPLOMA IN NEURO TECHNOLOGY

LOG BOOK

Submitted by,

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CERTIFICATE

I **Mr. Jishad K T** hereby declare that I have performed all the procedures listed carried out the project, under report.

Signature: .....

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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); THIRUVANANTHAPURAM, KERALA, INDIA.

◄◄Hospital wing

BMT wing►►

◄◄Achutha Menon Centre

The Sree Chitra Tirunal Institute for Medical Sciences and 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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PG Diploma in Neuro Technology

PG Diploma in Neurotechnology (PGDNT) 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 visual acuity testing and visual 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 and doing Intra operative monitoring, neuro navigation 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
EEG

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 has been carried out in animals as early as 1870. The wave forms recorded are thought to reflect the activity of the surface of the brain, the cortex. The activity is influenced by the electrical activity form the brain structures underneath the cortex.

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

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

Basic physiology of cerebral potentials

The origin of cerebral potentials is based upon the intrinsic electrophysiological properties of the nervous system. Identifying the generator source(s) and electrical field(s) of propagation are the basis for recognizing electrographic patterns that underlay 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 (uV)) 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 electro-chemical 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 through 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 post-synaptic potentials (EPPs) flow inwardly (extracellular to intracellular) to other parts of the cell (sinks) via sodium or calcium ions. Inhibitory post-synaptic potentials (IPPs) flow outwardly (intracellular to extracellular) in the opposite direction (source), and involve chloride or potassium ions.

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

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

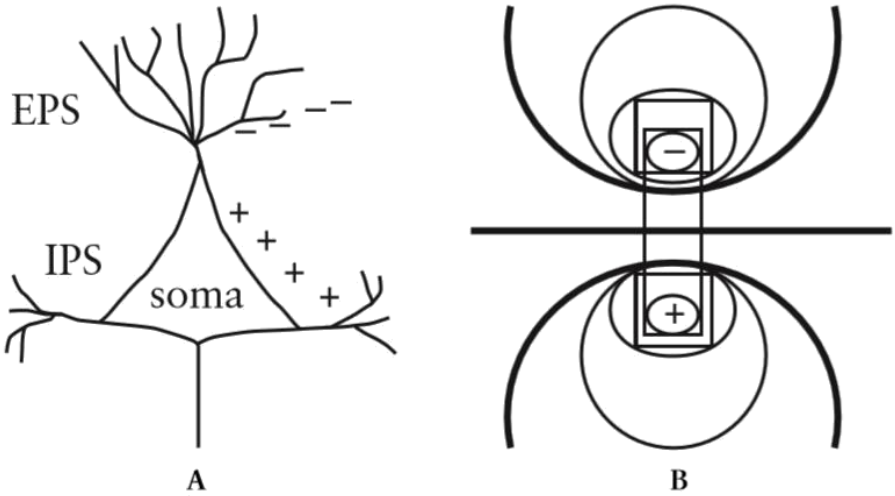
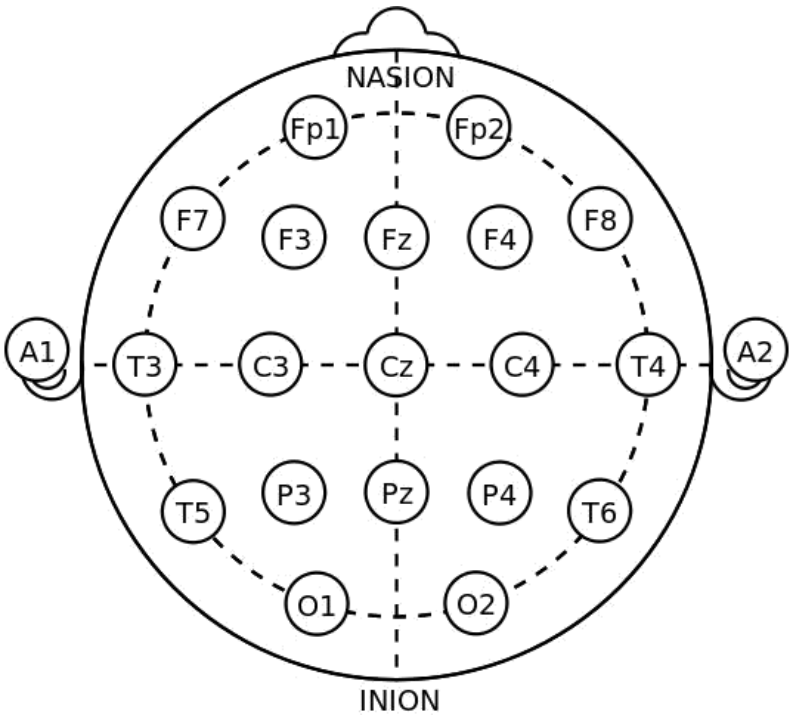


FIGURE 1.1. (A) A pyramidal cell with excitatory postsynaptic potentials and inhibitory postsynaptic potentials. (B) Dipole depicting a field of charge separation.

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

Electrodes

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



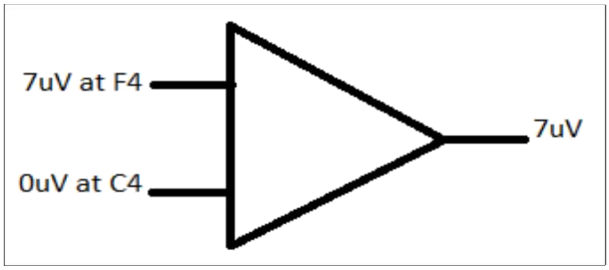
There are a great variety of electrodes that can be used. The majority are small discs of stainless steel, tin, gold or silver covered with a silver chloride coating. These normally have a lead attached. Alternative methods consist of a cap in which the electrodes are already embedded.

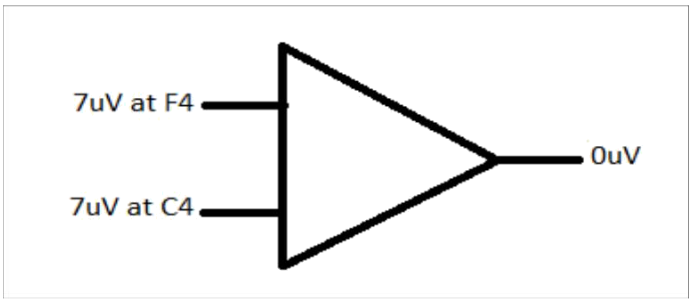
Montages

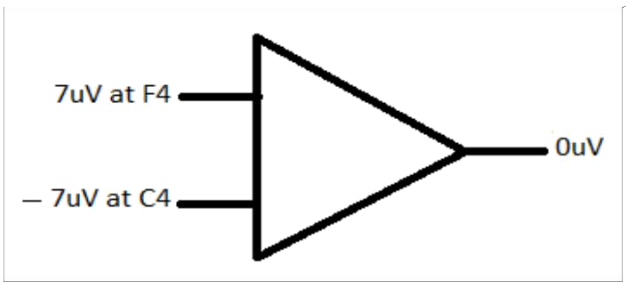
EEG machines use a differential amplifier to produce each channel or trace of activity. Each amplifier has two inputs. An electrode is connected to each of the inputs.

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.



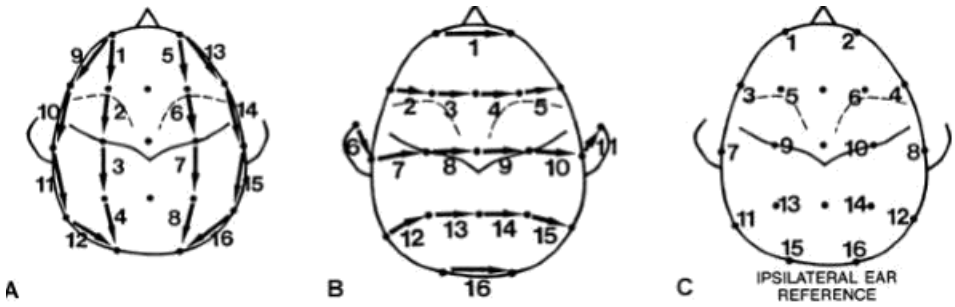




The manner in which the pair of electrodes are connected to each amplifier of the EEG machine is called a montage.

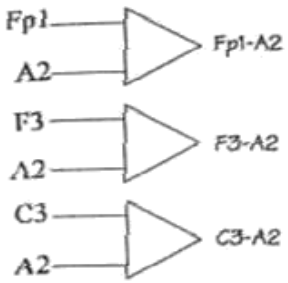
If one is mainly interested in a striking pattern, such as a spike-and-wave complex, then the display of such a pattern in a way that makes it stand out at a fairly large amplitude is desirable. For such a pattern, which is generally predominantly parasagittal, the use of the earlobe, whether ipsilateral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

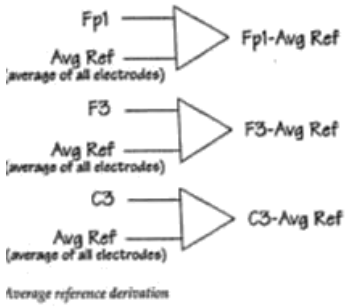


A: A representation of bipolar (scalp-to-scalp) montage in a longitudinal or anteroposterior (AP) direction. This montage is designed for easy comparison of left-right differences in the parasagittal or temporal area, but a montage with strict left-to-right sequence of sets of four could provide the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

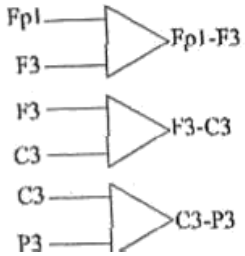


Average reference derivation: Activity from all the electrodes are measured summed together and averaged before being passed through a high value resistor. The resulting signal is then used as a reference electrode and connected to input 2 of each amplifier and is essentially inactive. All EEG systems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



Analogue EEG instruments

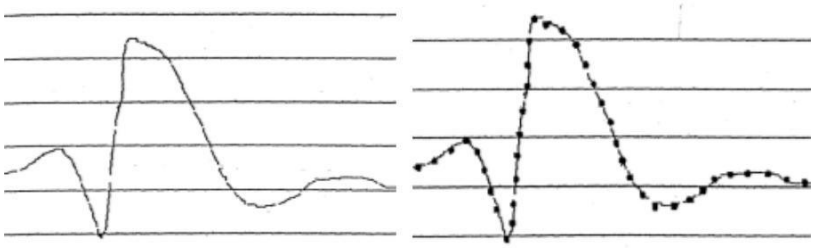
Conventional analogue instruments consist of an amplifier, a galvanometer and a writing device. A galvanometer is a coil of wire inside a magnetic field. The output signal from the amplifier passes through the wire causing the coil to oscillate. A pen mounted on the galvanometer moves up and down each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

High and low frequency filters and sensitivity 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 micro (u)/mm means that a signal with amplitude of 100 micro(u)V will produce a 1 cm vertical deflection.

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

Digital EEG instruments

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



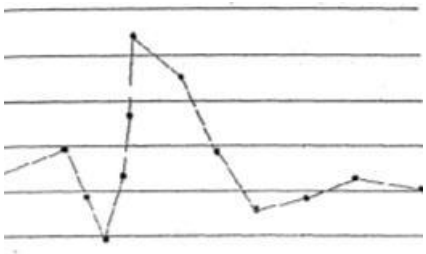
Analogue waveform sampling rate of 240 Hz

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

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

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

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



Sampling rate 50 Hz

A second factor that affects the accuracy of the waveform is *sampling skew*. Sampling skew occurs when all channels are not sampled simultaneously. Many digital EEG systems sample channel first. Then sample channel2, then channel3 etc. The time lag between sampling of each channel is known as sampling skew.

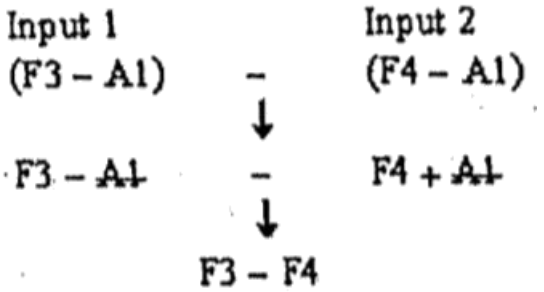
To reduce the sampling skew, some digital systems use ***burst mode sampling***. This increases the speed between successive channels sampling in order to reduce the amount of sampling skew.

A third factor that affects the accuracy of digital EEG waveforms is the display. The accuracy of a monitor display depends on the number of points or pixels that are available. The number of pixels available is referred to as the screen resolution. Screen resolution is described in numbers that represent the pixels available in the horizontal and vertical axis.

A VGA display has a resolution of 640 x 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 BEG 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 e1ectrode. Regardless of the montage used to display the data while it is being recorded, data is stored into the computer memory in common reference mode. This allows the data to be displayed using different montages at a later time. Since digital systems store the analogue signal as numerical values, remontaging is a simple subtraction process, which results in cancellation of the common reference.

An example is shown in the next figure. The reference electrode Al is common to both channels on input 2. It has the identical value in each channel. 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).



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)

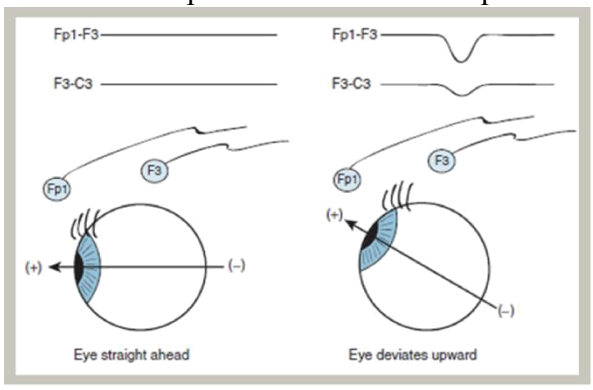
Artifacts originate from a variety of sources, and their recognition, identification, and possible elimination are a primary responsibility of the electroencephalographic (EEG) technologist. If unrecognized, can lead to misinterpretation of the recordings.

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

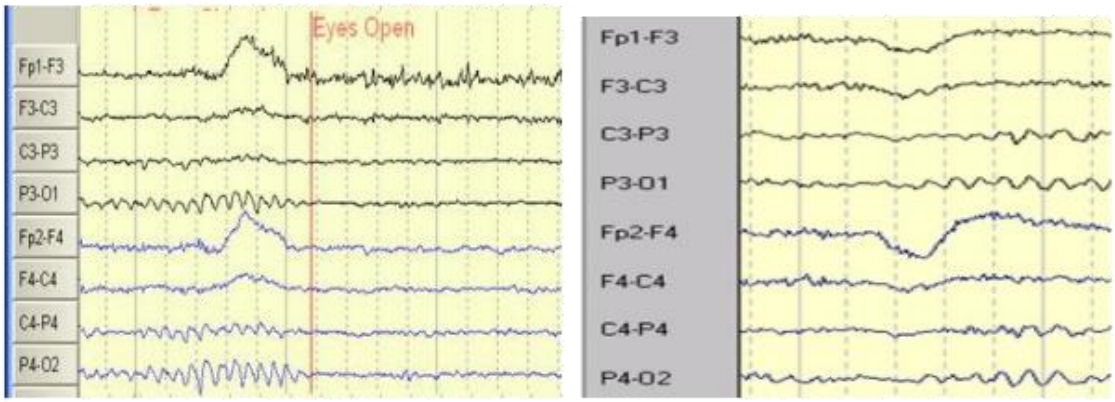
Physiological artifacts

Eye Movements

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



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

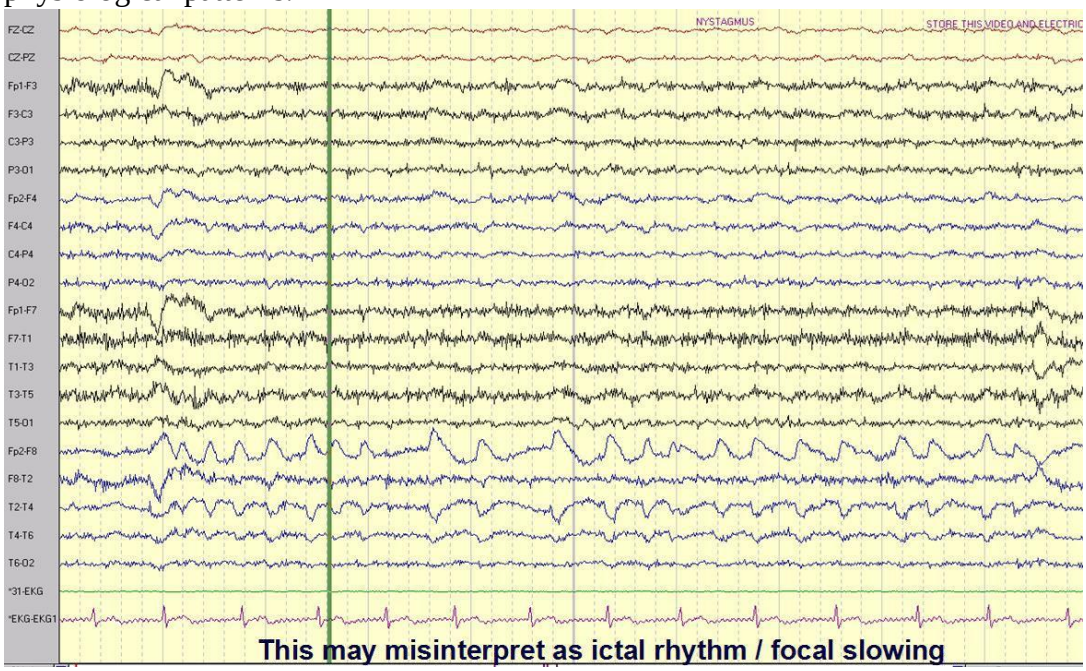


Eyelid flutter

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

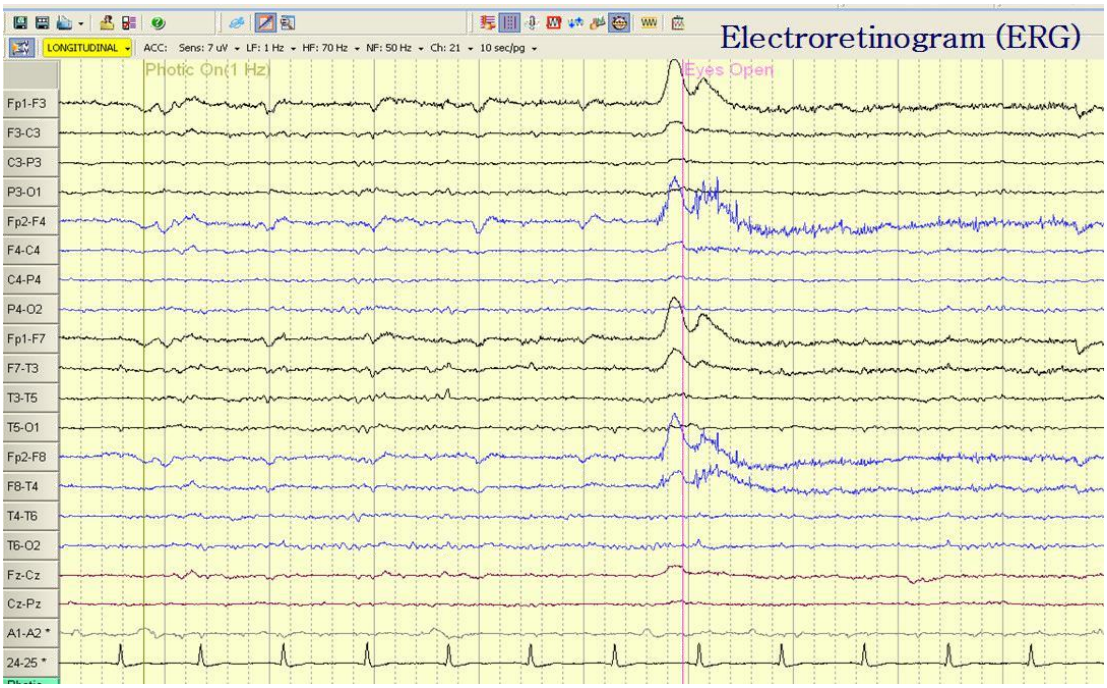
Nystagmus

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



Electroretinogram (ERG)

Low-voltage (<50microV) response to light stimulus of the retina. Appears in the anterior head regions. Especially during recording of electro-cerebral 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

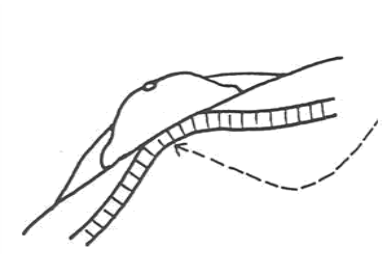
Whenever there is decreased movement of 1 eye, Asymmetrical placement of electrodes, Unilateral enucleation and prosthetic eye replacement, External ophthalmoplegia , Bell's palsy, Skull defect(eye movement artifact will be lower in amplitude ipsilateral to skull defect.) we can see asymmetric eye movement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

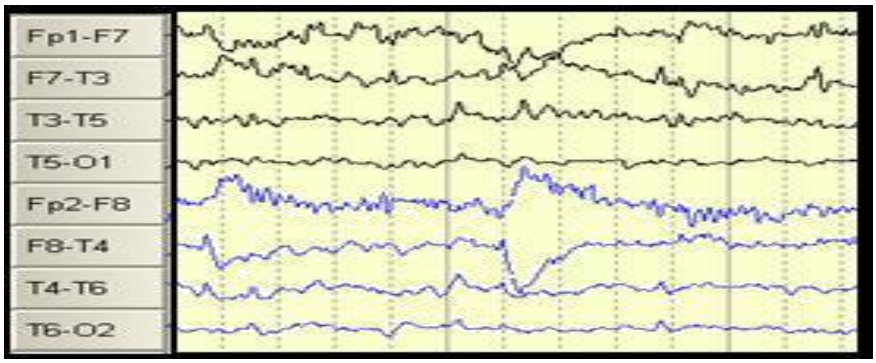
Cardiac arrhythmias

Cardiac arrhythmias can be misinterpreted as ictal events because – it's 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 identify 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 photo-myoclonic response is recorded. It may obscure EEG activity and even be confused with a photo-convulsive response

Temporalis EMG

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

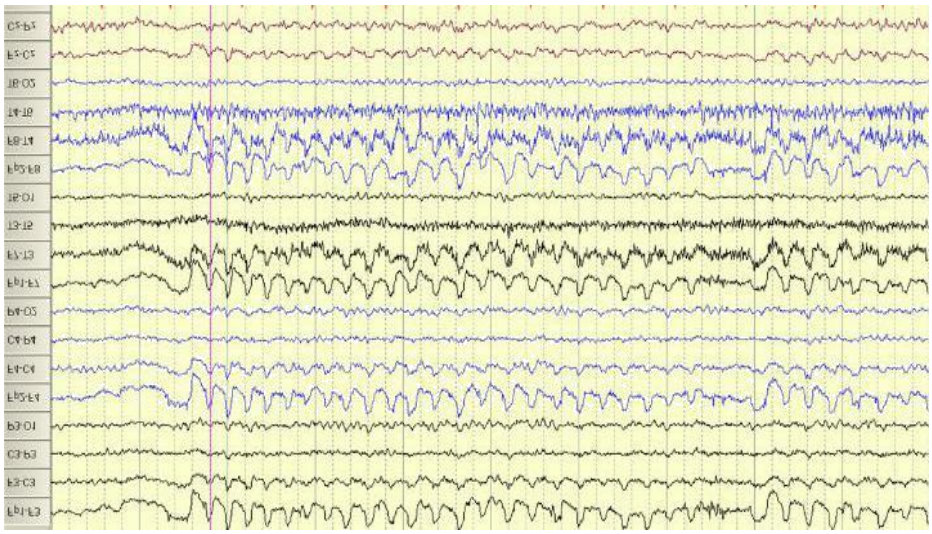
Note: EMG activity recorded over the frontal and occipital areas are common in tense individuals.

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

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

Glossokinetic Artifact

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



Galvanic skin response

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

Physiological Movements

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

NON-PHYSIOLOGICAL ARTIFACTS

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

Instrumental and Environmental

Electronic noise:

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

50-cycle interference recorded in an EEG:

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

Capacitive, inductive, magnetic, and electrostatic artifacts:

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

Electrostatic artifact:

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

Drip artifact:

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

Ventilator Artifact:

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

Muscle artifacts vs 50 Hz artifact

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

Environment for artifact free recording:

Quiet atmosphere, Comfortable bed, No synthetic carpeting, convenient control of lightening, proper shielding, Comfortable temperature and humidity levels to avoid 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 equipment's. 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 center or front of the head. Some drugs will increase the amount of beta activity in the EEG.

Alpha activity:

Alpha is 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:

Theta 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:

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

ATYPICAL BUT NORMAL WAVEFORMS DURING AWAKE

Lambda:

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

MU activity:

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

Posterior slow waves of youth:

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

THE FEATURES OF EEG DURING DROWSINESS AND SLEEP

The transition from awake to the drowsiness or stage I sleep, is marked by some profound changes in background activity of the BEG. The transition may be gradual or abrupt. The most prominent change is disappearance of the posterior dominant (alpha) rhythm. With the alpha rhythm gone, the background becomes dominated by theta activity, which occurs in generalized distribution, but is 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 ate picked up in the C3 and C4 electrodes. Their fields frequently spread to fronto-central regions and sometimes even extended to parietal regions. V waves usually are biphasic, but occasionally may be triphasic as well.

F waves or frontal waves

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

K complex

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

Sleep spindles

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

POSTS

The positive occipital sharp transient of sleep (POSTS) are occur over the occipital regions on either side, being positive related to other areas, seen in stage II sleep. They occur singly or more 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 N sleep is characterized by a background that consists of irregular and semi-rhythmic 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 in to epileptiform abnormalities and periodic patterns.

Epileptiform Discharges

Spike discharges

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

Sharp wave discharges

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

Polyspikes or multispikes

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

Spike and wave complexes

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

Focal epileptiform abnormalities

Inter-ictal 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 patern is the features of the diagnosis of Lennox -Gastaut syndrome.

Generalized polyspike and wave discharges

Generalized polyspike or polyspike and wave complexes may occur as an Interictal phenomenon in patent with primary generalized epilepsy (often myoclonus) 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 if it. The discharges should stand out from the background.

Generalized periodic paroxysmal patterns

SSPE

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

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

Suppression Burst Pattern

This may be 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 wave 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(BIPLEDs)
- ✓ Generalized periodic epileptiform discharges (GPEDs)
- ✓ Triphasic waves

High potential for SZs & convulsive / non-convulsive 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.

PLEDs occur at approximately regular intervals and persist for greater than 10 min or are continuous in a specific behavioural state

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

Low amplitude rhythmic discharges (RDs) closely associated in time and in spatial distribution to inter-ictal epileptiform discharges are not seen in scalp EEGs of patients with non-periodic focal epileptiform discharges (NPEDs) but they are unexpectedly common in patients with periodic lateralized epileptiform discharges (PLEDs). A classification of PLEDs into PLEDs Proper (PLEDs without RDs), and PLEDs Plus (PLEDs with RDs) is proposed. Such a classification is useful for an easier appreciation of PLEDs' pleomorphism. It underscores the changing periodicity of PLEDs and allows for a more inclusive definition of the phenomenon than the one reported by the literature. It also emphasizes the importance of recognizing RDs in scalp EEGs as transitional anomalies intercalated between inter-ictal PLEDs and ictal seizure discharges, analogous to those observed by Ralston in animal models. Indeed, the occurrence of recorded seizures is higher in patients with PLEDs Plus than in those with PLEDs Proper or NPEDs.

Reiher's classification

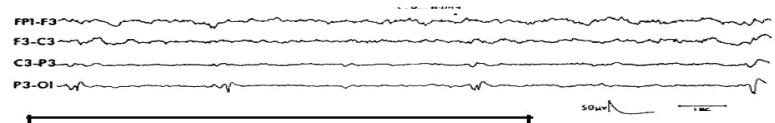
PLEDs proper (without rhythmic discharges)

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

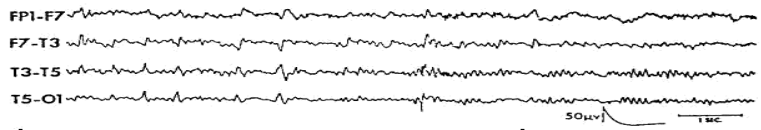
PLEDs plus (with rhythmic discharges)

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

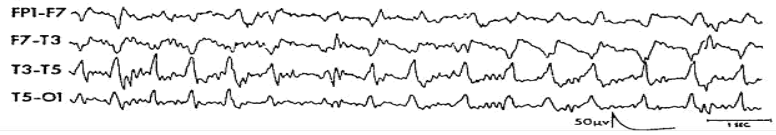
Class I - aperiodic, throughout



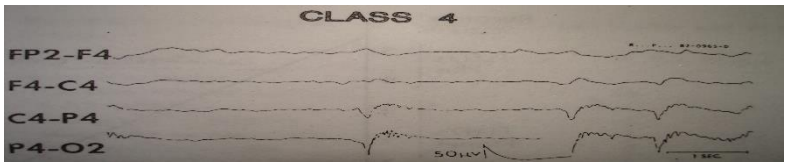
Class II metronic, intermittent



Class III - metronic ,throughtout

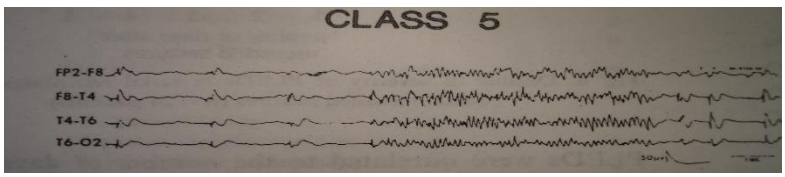


Class IV



Brief RDs < 1 sec

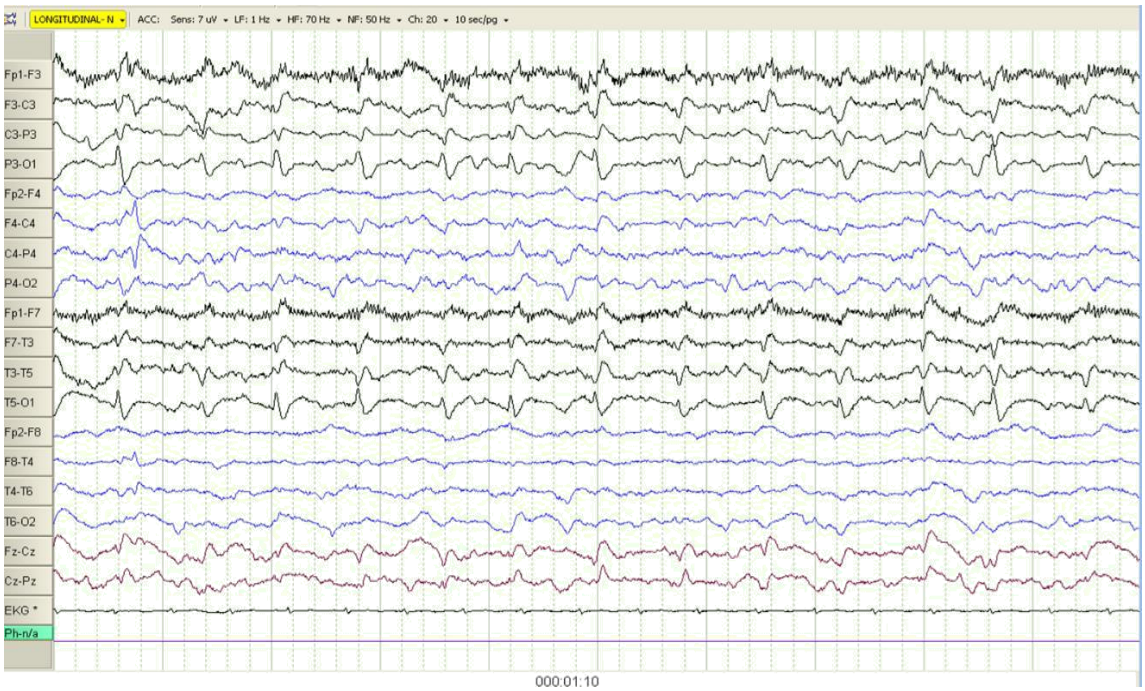
Class V



Prolonged RDs

NOTE:

EEG Typically fluctuating between 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 persists as chronic PLEDs, Recurrence of PLEDs – Recurring pathology e.g. TIA.

NOTES

- ✓ Obtundation in 95% of PTs
- ✓ Focal seizures & focal neurological signs - 80%
- ✓ Epilepsia partialis continua – 30%
- ✓ SZs are not usually generalized
- ✓ 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

BIPLEDs

- ✓ 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

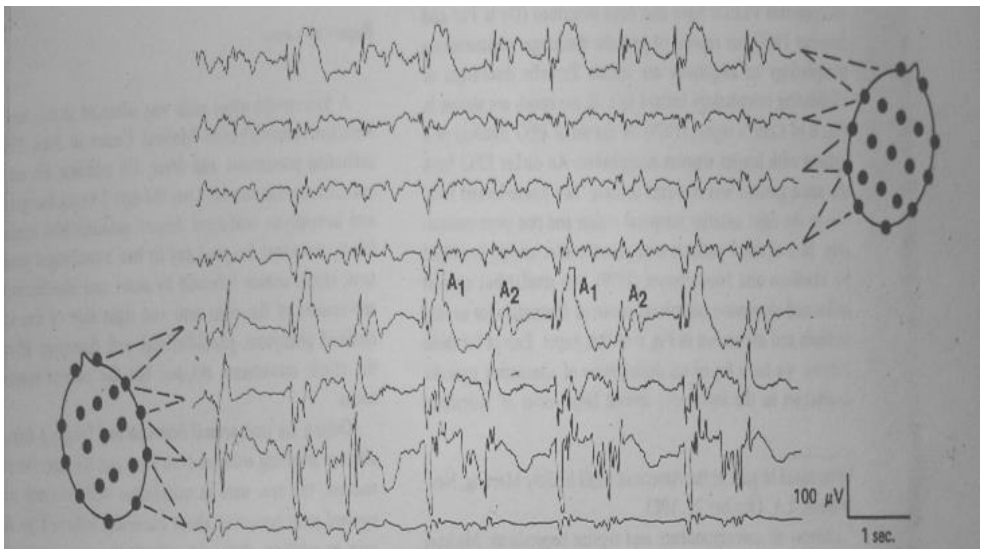


Fig. 1. Two distinct polyspike and slow-wave discharges (A_1, A_2) occur alternately at regular intervals. The A_1-A_2 interval averages 1200 msec while the A_2-A_1 interval averages 900 msec.

Multifocal PLEDs

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

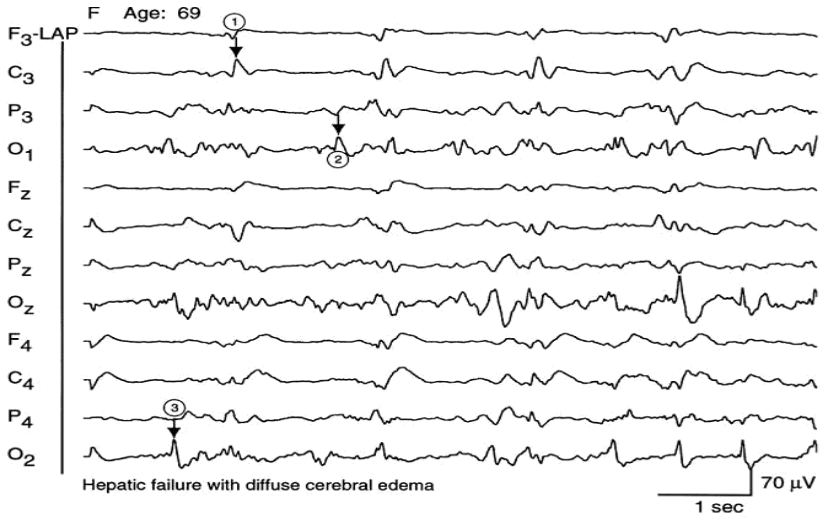
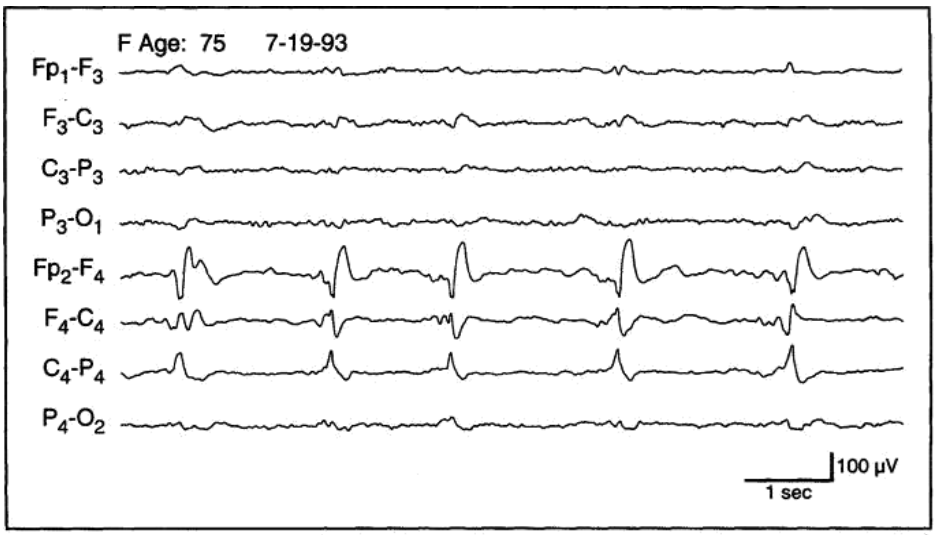


Fig. 1. Typical example of multifocal PLEDs (arrows) using the Laplacian montage, recorded from a 69-year-old woman with liver failure, sepsis, and hypoxia. Focus 1 (C_3) occurring every 1.5 s; focus 2 (O_1) occurring every 0.5–1 s; focus 3 (O_2) occurring every 0.5 s.

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

PEDIMs

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



Benign EEG variants and patterns

Benign patterns with '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 drowsiness and light sleep. They are usually of low amplitude, usually less than 50uV and of short duration, less than 50 ms. The waveform consists of single monophasic or biphasic spikes with abrupt ascending limb and steep ascending limb. The BSSS may have a single after coming 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. It look like an arch. They are not accompanied by a slow wave. They occur over the temporal areas and may be unilateral or bilateral. Wicket spikes are seen best during stage I and II sleep, although they may rarely occur on wakefulness.

14 and 6Hz positives spikes

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

6 Hz spike and waves (Phantom spikes)

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

Rhythmic patterns

Rhythmic Midtemporal Theta of Drowsiness (RMTD)

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

Subclinical Rhythmic Electroencephalographic Discharges of Adults (SREDA)

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

EEG Recording

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

Most patients will be asked to carry out a period of deep breathing for approximately 3 minutes. This may produce some abnormal activity, which would not be seen while· the patient is relaxed. The physiological effect of deep breathing is to increase the amount of carbon dioxide (Co_2) being removed from the bloodstream. This fall in Co_2 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 may turn to 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 (5-10 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 andeye 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 four main frequency bands. The resulting waveforms can be displayed as a brain map, which will show the scalp distribution of the power within each frequency band. The amplitude of the different waveforms at a single point can also be displayed in a similar format. This type of display provides a more objective analysis of the EEG activity compared to a subjective visual analysis by a physician.

ACTIVATION PROCEDURES

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

Hyperventilation

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

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

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

Intermittent Photic Stimulation

Visual stimuli are perhaps one of the most effective means of stimulation of the brain. Stroboscopes otherwise 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 (LTME), 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 brain activity over 2-l uV 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-O1 etc.

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

EEG MONITORING IN INTENSIVE CARE SETUP

EEG MONITORING IN INTENSIVE CARE SETUP

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

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

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

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

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

Prolonged Video EEG Monitoring

Introduction

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

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

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

Recording method

Facilities

A room, close friends or relatives, medical team

Duration of monitoring

3 to 7 days

Scalp electrodes

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

Ictal SPECT

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

Usually the gamma-emitting tracer used in functional brain imaging is 99mTcHMP AO (hexamethyl propylene amine oxime). 99mTc is a metastable nuclear isomer which emits gamma rays which can be detected by a gamma camera. When it is attached to HMPAO, this allows 99mTc to be taken up by brain tissue in a manner proportion 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 electro-graphical 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.

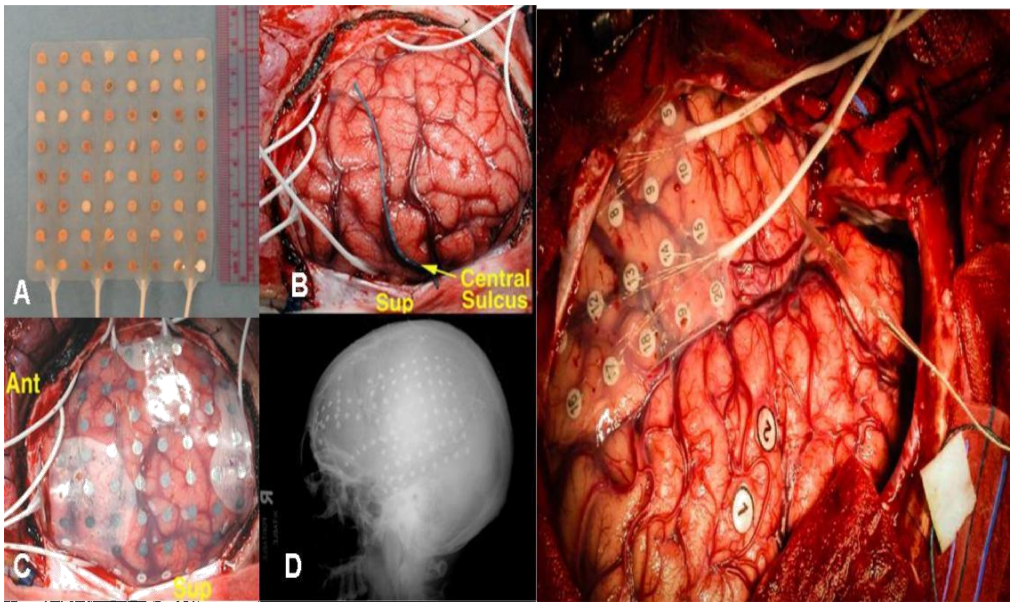
Invasive monitoring

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 inter-ictal and ictal epileptiform activity of mesial, basal-temporal origin. Several types of intracranial recording electrodes. Such as subdural strip electrode, grid electrodes, epiduralelctrodes, intra-cerebral depth electrodes or combination of each are used.

- ECOG electrodes are sterile, disposable stainless steel, carbon tip, platinum, or gold ball electrodes.
- Commonly we are using strip, grid depth electrodes.
- Each contact with 5 mm diameter and 1 or 0.5 cm inter electrode spacing.

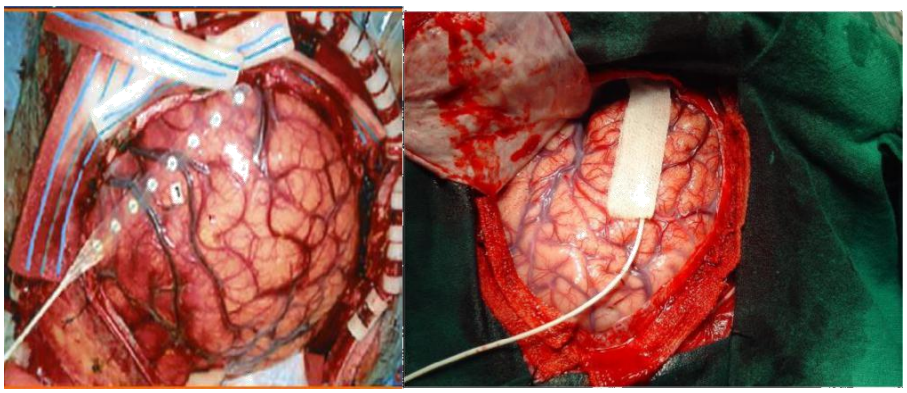
Grid electrodes

They are transparent, flexible, and numbered at each electrode contact. Standard spacing between grid electrodes is 1 cm; individual electrodes are typically 5mm in diameter.



Strip electrodes

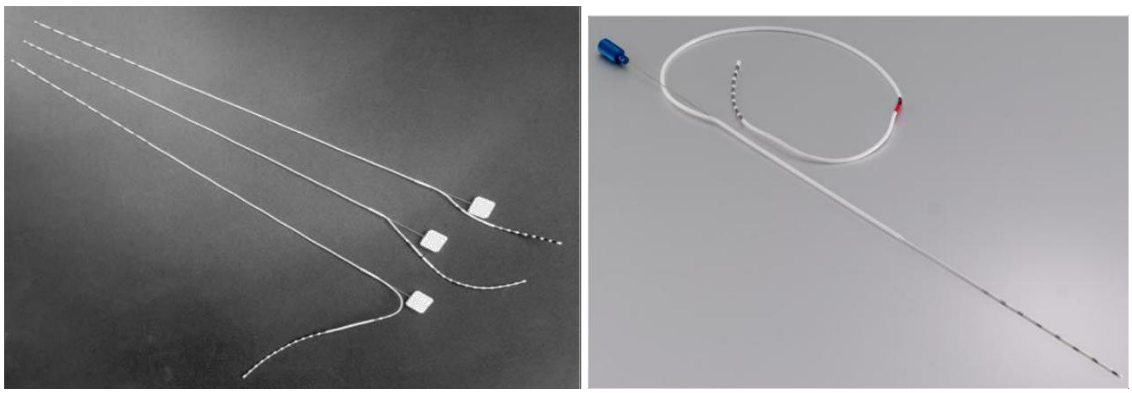
contacts are arranged in a single row.
There are 4 contact 6 contact , 8 contact and 10 contact strips



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 stereo-tactically, 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. It is used to record activity from deeper structures such as the hippocampus.



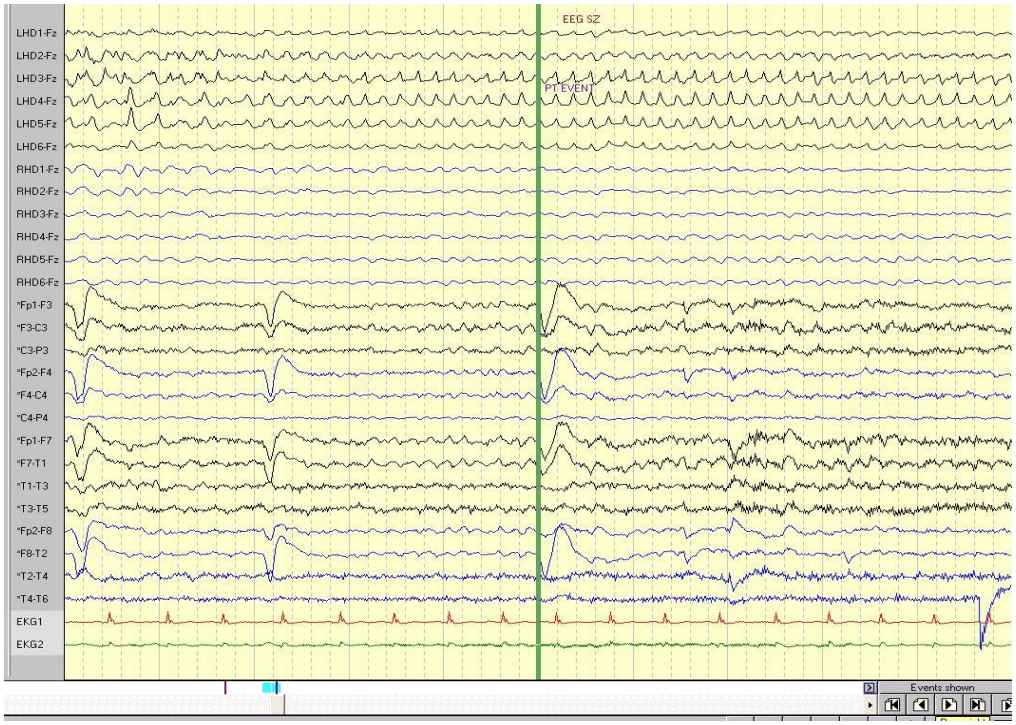
Subdural electrodes

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.

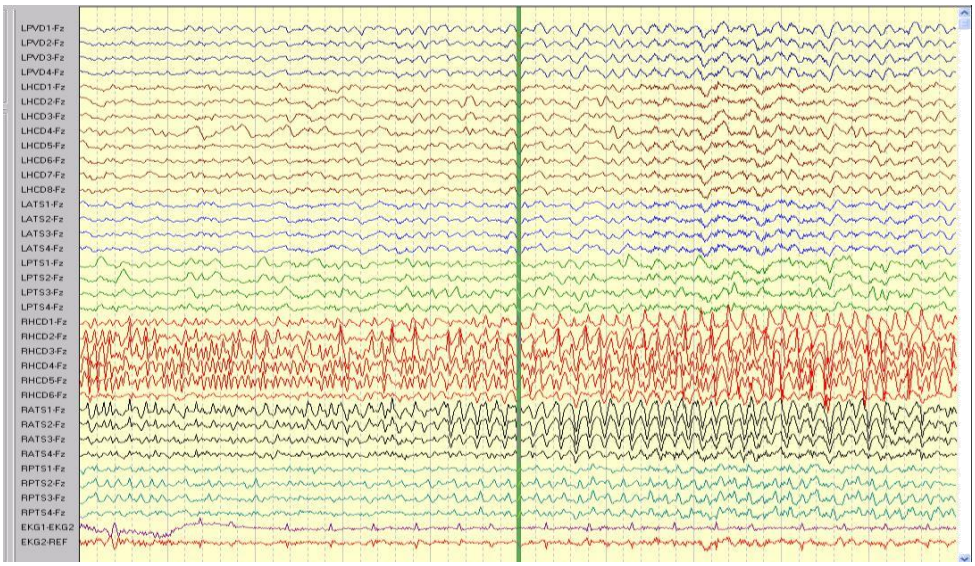
Epidural electrodes

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.

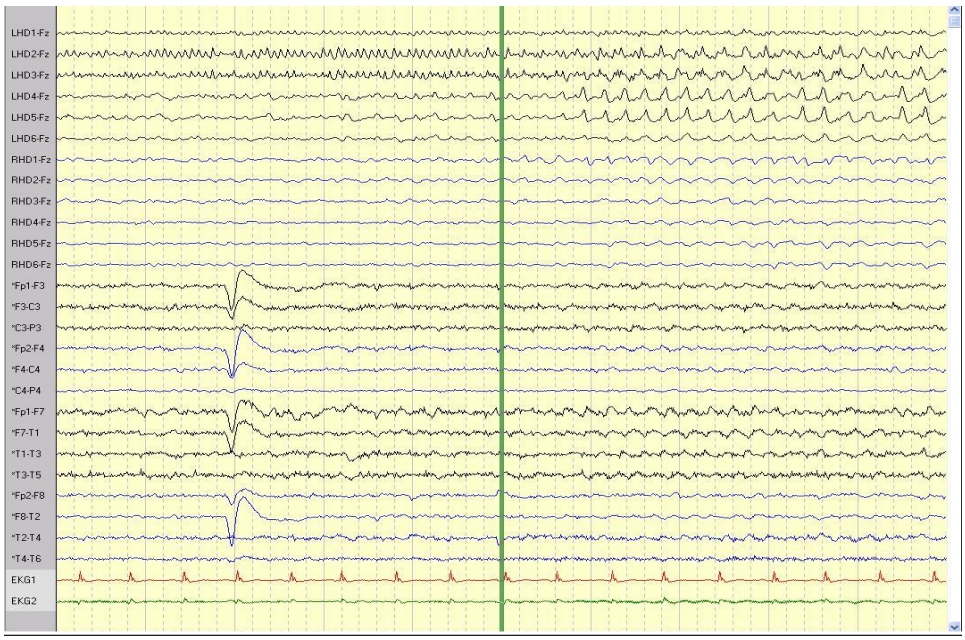
Ictal onset(Invasive monitoring using depth electrode)



EEG SZ



Ictal rhythm



Cortical stimulation mapping

Cortical stimulation mapping (often shortened to CSM) is a type of electrocorticography that involves a physically invasive procedure and aims to localize the function of specific brain regions through direct electrical stimulation of the cerebral cortex. It remains one of the earliest methods of analyzing the brain and has allowed researchers to study the relationship between cortical structure and systemic function. Cortical stimulation mapping is used for a number of clinical and therapeutic applications, and remains the preferred method for the pre-surgical mapping of the motor cortex and language areas to prevent unnecessary functional damage. There are also some clinical applications for cortical stimulation mapping, such as the treatment of epilepsy.

Procedure

Cortical stimulation mapping is an invasive procedure that has to be completed during a craniotomy. Once the dura mater is peeled back, an electrode is placed on the brain to test motor, sensory, language, or visual function at a specific brain site. The electrode delivers an electric current lasting from 2 to 10 seconds on the surface of the brain, causing a reversible lesion in a particular brain location. This lesion can prevent or produce a testable response, such as the movement of a limb or the ability to identify an object. The electric current from the electrode stimulates whatever function that site in the brain is responsible for, in essence telling the surgeon or examiner what a specific locale in the brain does.

Electrodes are usually made of stainless steel or platinum-iridium embedded in a silastic material, and are usually circular with diameters of 2 to 3 mm. Electrode positioning varies from patient to patient, and electrodes can come in rows, in a grid array, or can be individually arranged. The number of electrodes necessary and their exact spatial arrangement is often determined in the operating room. Cortical stimulation mapping allows electrodes to be placed in exact locations to test brain function and identify if stimulation of the brain location causes a functional impairment in the patient. CSM can be completed using anesthetized patients or awake patients.

Electrodes can either be placed directly on brain areas of interest or can be placed in the subdural space of the brain. Subdural electrodes can shift slightly and can be affected by cerebrospinal fluid in the subdural space, which could interfere with the current used to stimulate the brain from the electrodes and possibly cause shunting and dissipate the current, making the stimulation's effect less accurate. However, an advantage of subdural electrode grids is that they can be left in the brain for multiple days, and allow functional testing during stimulation outside the operating room.

Current levels and density are an important consideration in all cortical stimulation mapping procedures. Current density, that is the amount of current applied to a defined area of the brain, must be sufficient to stimulate neurons effectively and not die off too quickly, yet low enough to protect brain tissue from damaging currents. Currents are kept at levels that have been determined safe and are only given as short bursts,

typically bursts that slowly increase in intensity and duration until a response (such as a muscle movement) can be tested. Current intensity is usually set around bursts of 1 mA to begin and gradually increased by increments of 0.5 to 1 mA, and the current is applied for a few seconds. If the current applied causes after discharges, nerve impulses that occur after stimulation, then the levels are lowered. Studies on patients who have received cortical stimulation mapping have found no cortical damage in the tested areas.

The different types and administration techniques for anesthesia have been shown to affect cortical stimulation mapping. CSM can be done performed on awake patients, called an awake craniotomy or in patients who have been placed under general anesthesia. If the patient is under general anesthesia, the depth of the anesthesia can affect the outcome because if the levels of muscle relaxation are too high due to neuromuscular blocking drugs, then the results from the mapping can be incorrect. For the awake procedure there are different considerations for patient care that the anesthesiologist must take into account. Rather than simply ensuring that the patient is asleep, the doctor can follow what is called the asleep-awake-asleep technique. In this technique the patient is anesthetized using a general anesthesia during the opening and closing portions of the procedure, but during the interim the patient is maintained utilizing local anesthesia. The local anesthesia techniques can be either a local field block or a regional nerve block of the scalp. The more common technique for the awake craniotomy is conscious sedation. In conscious sedation, the patient is only sedated during the opening and closing process, but never fully anesthetized, eliminating the need for breathing tubes, lessening the chances of complications, and lessening the chances of problems with motor response. Patients who undergo the procedure with an awake craniotomy instead of general anesthesia have better preservation of language function, a prediction of their seizure-free outcome based on corticography, a shorter hospitalization (which corresponds to a reduced cost of care), a decreased usage of invasive monitors, and decreased number of postoperative complications due to anesthesia such as nausea and vomiting

Clinical Application in Epilepsy.

CSM is an effective treatment for focal epilepsy and bilateral or multiple seizure foci. It is an effective treatment option when resective surgery to remove the affected area is not an option, generally seen with bilateral or multiple seizure foci. CSM is routinely utilized for patients with epilepsy in order to pin point the focal point of the seizures. It is used once there is a testable hypothesis regarding brain location for the epileptogenic zone, determined through a less invasive procedure, electroencephalography. Once the focal point of the seizures is determined, this information allows aids neurosurgeons with knowing what portions of the brain could potentially be resected without any negative post-operative neurological deficits.

CSM will be considered for a patient with epilepsy when two conditions are met: the trial of anti-epileptic drugs has not controlled seizures and there is a likelihood that the surgery will benefit the patient. Due to the nature of the procedure, CSM is only utilized after noninvasive procedures have not been able to fully localize and treat the patient

The invasive electrodes are stereotaxically placed electrodes or a subdural strip or grid electrode Utilizing the information obtained through CSM, limited resection of epileptogenic brain can be performed. For focal epilepsy, resective surgery is one of the mainstay treatment options for medication resistant epilepsy. Through the technique of CSM, generally using awake craniotomies, the neurosurgeon has the ability to monitor the functioning of the patient during the resection and stimulation of the brain.

Protocol For Cortical Stimulation

- Stimulation parameters

Pulse Rate	50Hz
Pulse Duration	0.2Milliseconds (200Microseconds)
Train Duration	1-5 Seconds
Strength of current	Start at 2.0mA, Increment at 0.5-1.0 mA, upto 10mA
Channels Stimulated	Bipolar, between two neighboring channels

- Stimulation End points
  - Symptoms
    - Somatosensory suggest sensory area, but stimulate 0.5 to 1.0 mA higher to see if it is motor area, unless the sensation is intolerable or after discharge is already induced
    - Visual — Visual function
    - Aura — suggest ictal zone
    - Cognitive, Affective uncommon but may suggest cognitive or limbic areas or "psychological" reaction
- Signs
  - Motor — may appear as negative(loss of posture) or positive(twitch)
  - Speech — Suggest language functions

- After discharge or seizure — Do not stimulate at the same strength 10
- Maximum strength — mA (some centers goes up to 15mA)

- Important Pointers
  - General pointers
    - Do the study at the conclusion of video EEG monitoring and with AED coverage
    - Counsel patient before start of procedure to let you know if anything is felt, &where it is felt, including any sensation.

ELECTROCORTICOGRAPHY

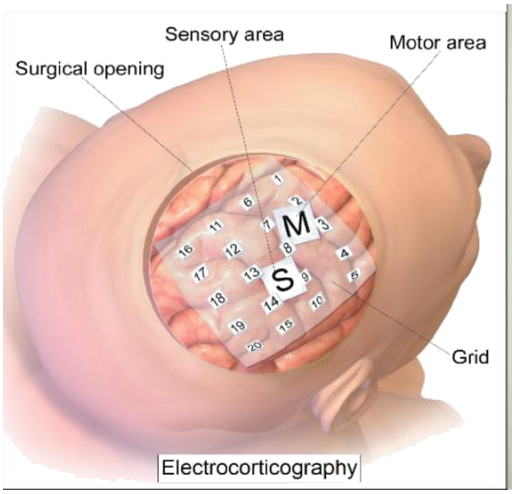
Electrocorticography

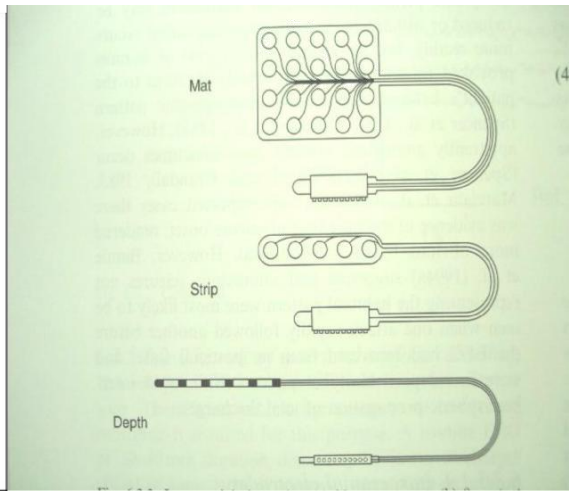
It is one of the first important applications of intraoperative recording of cerebral electrical activity, later, depth recordings were made acutely during the course of epilepsy. Electrocorticography during epilepsy surgery has been performed for several years. In particular this is for the tailored resections both temporal and extra temporal in order to identify the epileptogenic zone. Techniques for pre-operative 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.

Pre-operative 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 pre-operative 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 acoustics neurinoma. Ensure surgery will not result in disabling neuro-psychological deficits. The appearance and reproducibility of potentials recorded intraoperative is affected greatly by type & level of anesthesia, blood pressure, temperature & other physiological variables.

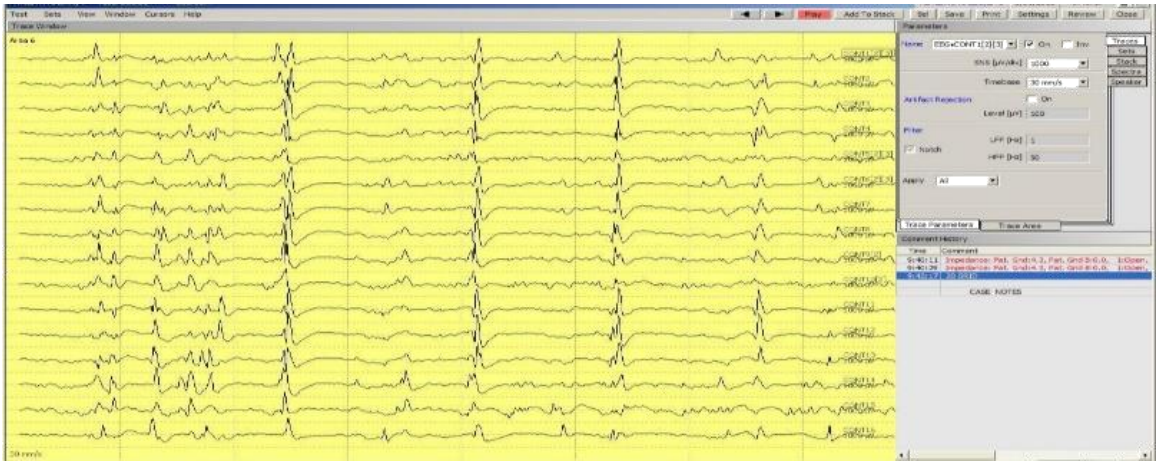
Purpose of Intraoperative monitoring

To preserve function and prevent injury to vital neuronal structure at the time when clinical examination is not possible.

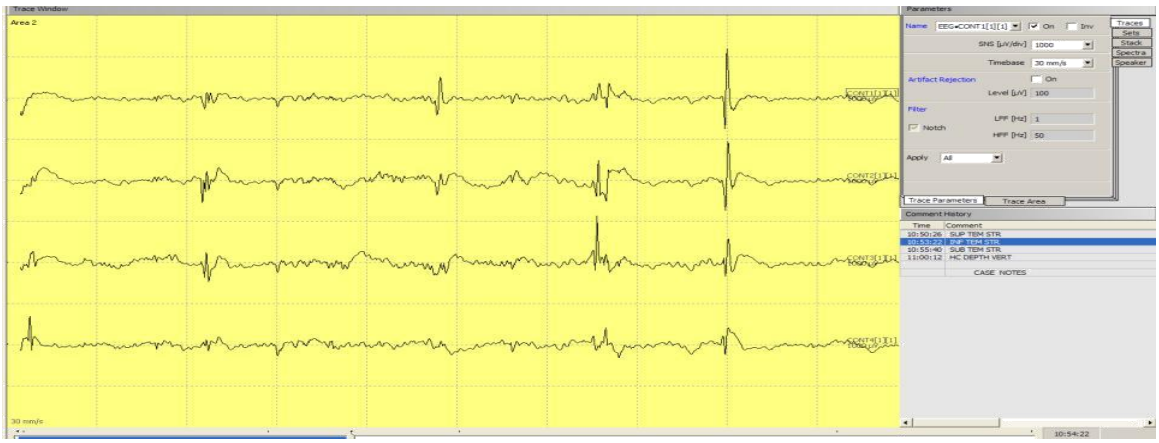




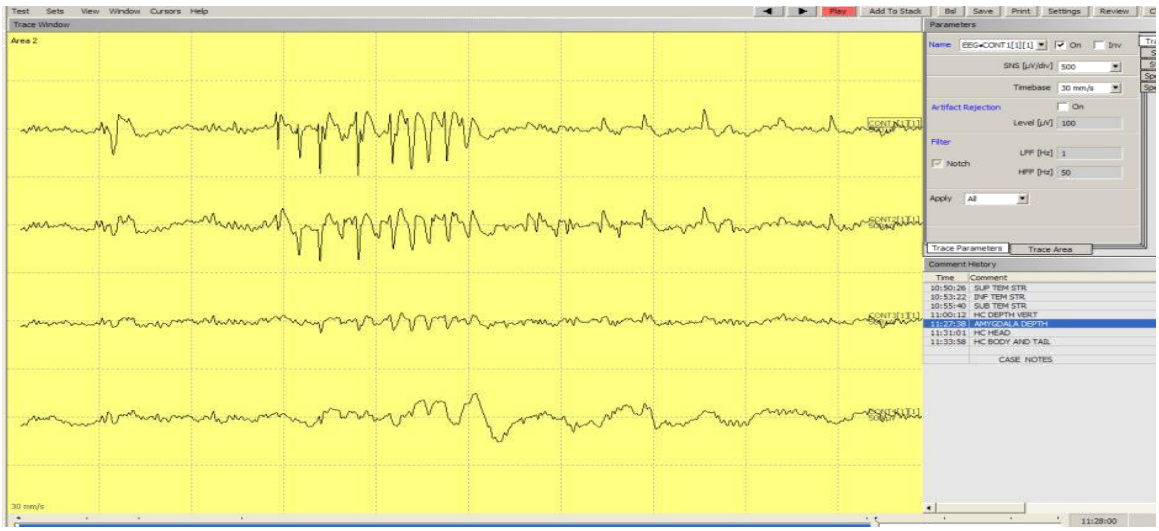
RIGHT ATL+AH (20 GRID)



4 contact Strip



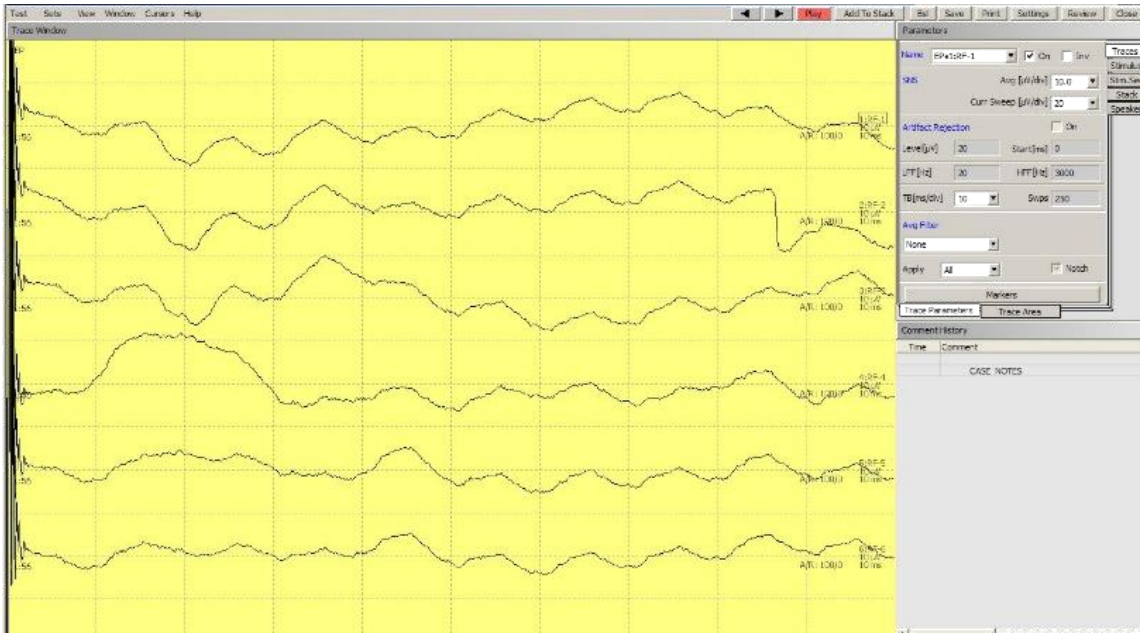
4 contact Depth(Amygdala)



Evoked potential monitoring includes somatosensory evoked potentials (SSEP), brainstem auditory evoked potentials (BAEP), motor evoked potentials (MEP), and visual evoked potentials (VEP). Electromyography (EMG) also is used extensively during operative cases. Scalp electroencephalography (EEG) provides data for analysis in SSEP, BAEP, and VEP. Scalp EEG also can be used to monitor cerebral function during carotid or other vascular surgery. In addition, EEG recorded directly from the pial surface, or electrocorticography (ECoG), is used to help determine resection margins for epilepsy surgery, and to monitor for seizures during electrical stimulation of the brain carried out while mapping cortical function.

The use of functional mapping of the white matter together with cortical mapping allowed the authors to optimize the benefit/risk ratio of surgery of low-grade glioma invading eloquent regions. Given that preoperative fiber tracking with the aid of neuroimaging is not yet validated, we used intraoperative real-time cortical and subcortical stimulations as a valuable adjunct to the other mapping methods.

Intra operative SSEP during epilepsy surgery



POLYSOMNOGRAPHY

POLYSOMNOGRAPHY

Introduction

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

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

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

• One electroencephalography (EEG) channel (central with an ear reference provides the best amplitude) to monitor sleep stage

• Two electrooculogram (EOG) channels to monitor both horizontal and vertical eye movements (electrodes are placed at U1e right and left outer canthi, 1 above and 1 below the horizontal eye axis)

• One electromyography (EMG) channel (usually chin or mentalis and/or submentalis) to record atonia of rapid eye movement (REM) sleep

Other parameters often monitored include the following:

• Additional EEG channels, particularly in patients with sleep-related epilepsy

• Additional EMG channels, particularly anterior tibialis, to detect periodic limb movements of sleep

• Airflow

• Electrocardiography

• Pulse oximetry

• Respiratory effort

• Sound recordings to measure snoring

Optional parameters include the following:

• Continuous video monitoring of body positions

• Core body temperature

• Incident light intensity

• Penile tumescence

• Pressure and pH at various esophageal levels

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

• Overnight polysomnography (PSG) is an overnight recording of the patient's sleep.

• Multiple sleep latency testing (MSLT) records multiple naps throughout a day.

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

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

Sporadic events may be missed on a 1-night PSG. External factors that disturb the subject's 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
o Bruxism
Restless legs syndrome and periodic limb movement disorder

MSLT

The Multiple Sleep Latency Test (**MSLT**) is a sleep disorder diagnostic tool. It is used to measure the time elapsed from the start of a daytime nap period to the first signs of sleep, called sleep latency. The test is based on the idea that the sleepier people are, the faster they will fall asleep.

Continuous positive airway pressure (CPAP)

Continuous positive airway pressure (**CPAP**) is a form of positive airway pressure ventilator, which applies mild air pressure on a continuous basis to keep the airways continuously open in people who are able to breathe spontaneously on their own. It is an alternative to positive end-expiratory pressure (PEEP). Both modalities stent the lungs' alveoli open and thus recruit more of the lung's surface area for ventilation. But while PEEP refers to devices that impose positive pressure only at the end of the exhalation, CPAP devices apply *continuous* positive airway pressure throughout the breathing cycle. Thus, the ventilator itself does not cycle during CPAP, no additional pressure above the level of CPAP is provided, and patients must initiate all of their breaths.

CPAP typically is used for people who have breathing problems, such as sleep apnea. CPAP also may be used to treat preterm infants whose lungs have not yet fully developed. For example, physicians may use CPAP in infants with respiratory distress syndrome. It is associated with a decrease in the incidence of bronchopulmonary dysplasia. In some preterm infants whose lungs haven't fully developed, CPAP improves survival and decreases the need for steroid treatment for their lungs.

CPAP therapy utilizes machines specifically designed to deliver a constant flow of pressure. Some CPAP machines have other features as well, such as heated humidifiers. CPAP is the most effective treatment for obstructive sleep apnea, in which the mild pressure from the CPAP prevents the airway from collapsing or becoming blocked.[1] Although delivery of CPAP through a nasal mask is the most common modality of treatment, other systems exist for interfacing with adults and children. Nasal CPAP is frequently used in infants though its use is controversial Studies have shown nasal CPAP reduces ventilator time but an increased occurrence of pneumothorax was also prevalent. Oral masks and naso-oral masks are often used when nasal congestion or obstruction is an issue. Devices that combine nasal pressure with maxillary advancement devices (MAD) also exist.

NERVE CONDUCTION STUDY

Introduction

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

Measuring the electrical activity in muscles and nerves can help detect the presence, location and extent of diseases that can damage muscle tissue (such as muscular dystrophy) or nerves (such as 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. depolarization 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 depolarization. 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. 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. This will reduce the amplitude. These two effects of the temperature on the amplitude neutralize each other. In order to standardize CV and amplitude measurements, it is recommended to keep the skin temperature at above 29° C for the dorsum of the hand and 27° C for the dorsum of the foot.

Age

Conduction velocity is age dependent. Full term infants have conduction velocities, which are approximately half of that seen in adults. Conduction velocities rapidly increase from the values recorded in infants to near adult values at around 3-5 years of age. Furthermore pre-term infants have slower values at around 14-28 m/s. In the teens conduction velocities are almost the same as those of adult values
After the second to fourth decade, conduction velocities start to decrease very slowly. cv decrease by o.5-1.8 m/s for each decade

Length of segment and height

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

Gender

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

Reference values

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

Pathophysiology

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

PROCEDURES

I- 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.

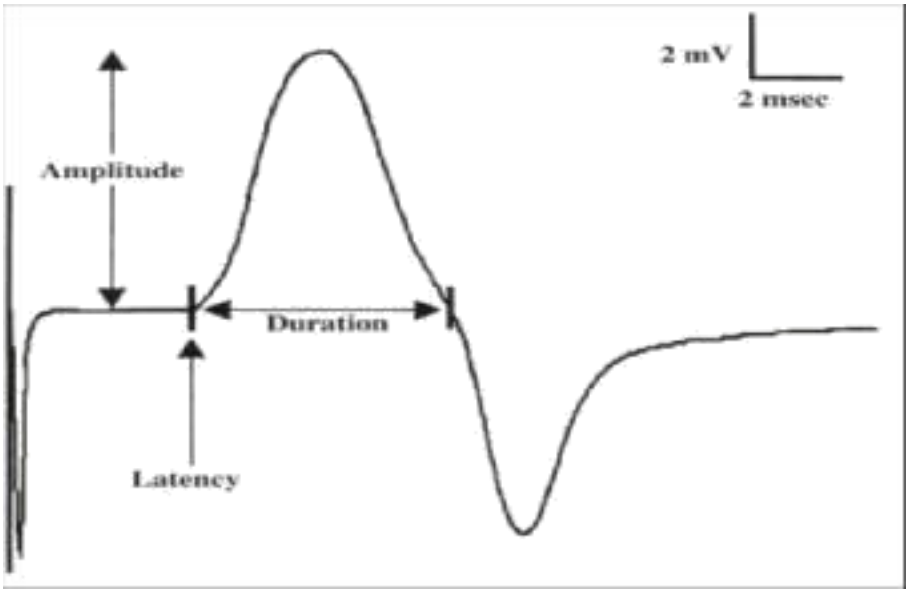
Generally surface electrodes (felt pad or steel) are used in motor nerve conduction studies. They are fixed on to a plastic bar a fixed distance apart. In a few situations it is preferable to use needle electrodes. A pair of monopolar needle electrodes is used these cases. Alternatively a surface electrode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

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.prox-Lat.distal)

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 (Dur.prox- Dur.distal) / Dur.distal

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.

Ampl.Decay =100 X (Amp distal- Amp proximal) / Ampl distal

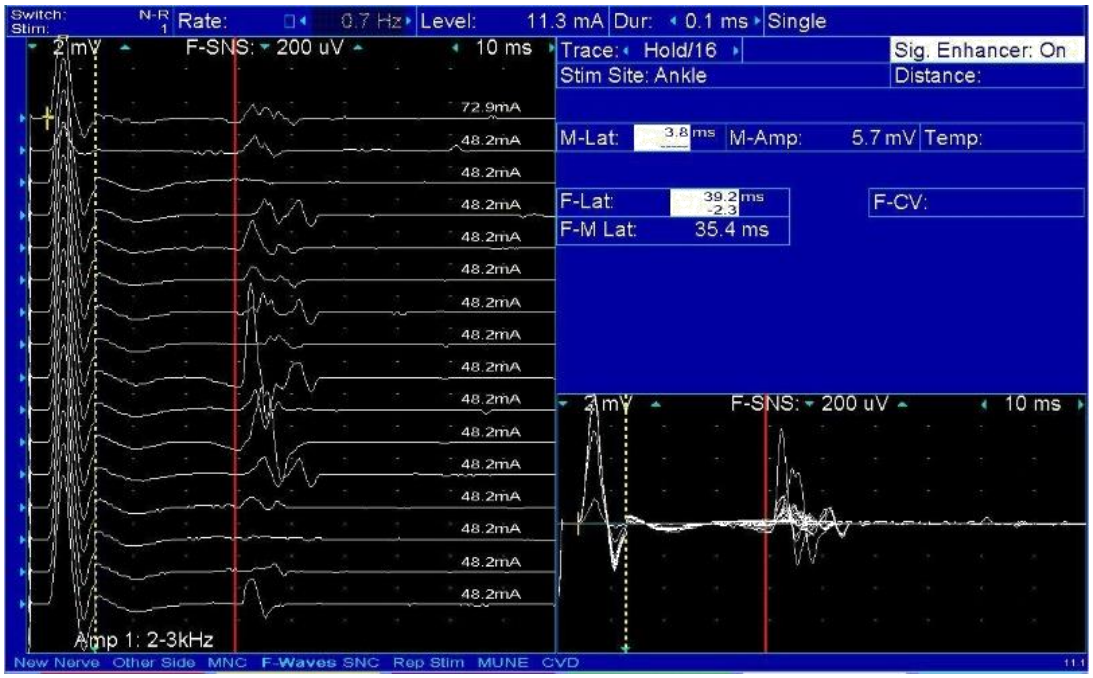
Area.Decay=100 X (Area distal- Area proximal) /Area distal

In healthy subjects, the mean value of the *Ampl.Decay* is 4.5-6.2% in the ulnar nerve [and 5.6-7.7% in the median nerve]. The *Ampl.Decay* 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 Ampl.Decay. On the other hand the *Area.Decay* is smaller than the *Ampl.Decay* in peroneal nerves. F-waves

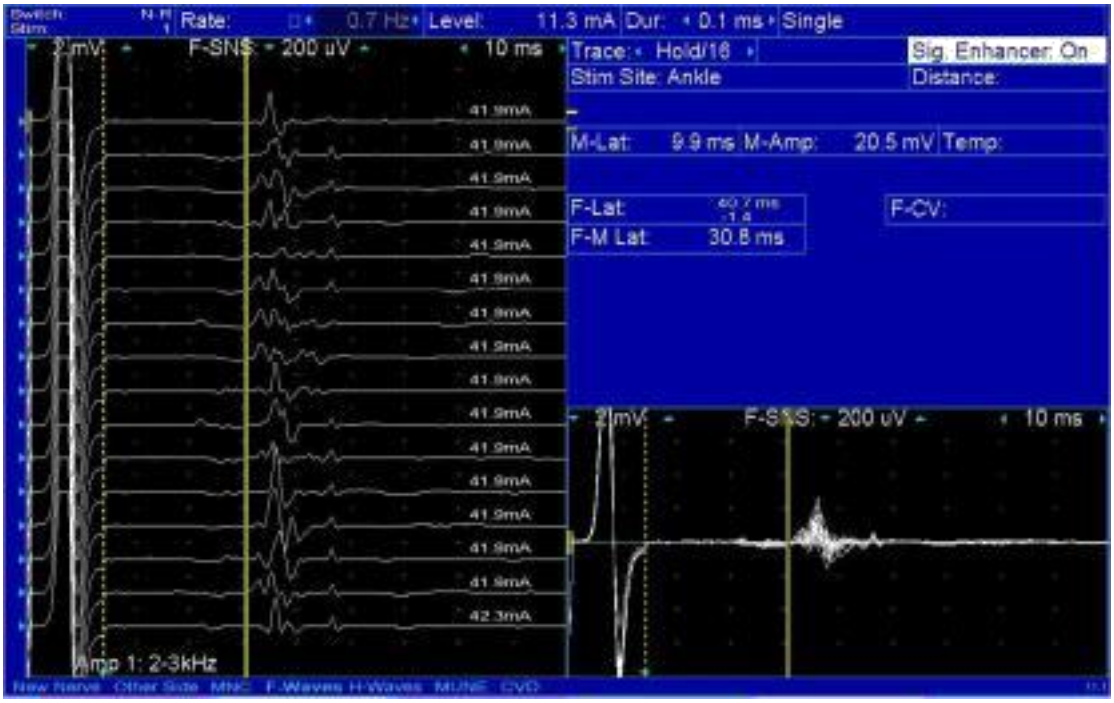
II- F WAVES

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 depolarization 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 depolarization/repolarization allows this to happen. Normally a recurrent response is evoked in 0.5-5% of the stimulations with some differences between nerves.

i) F wave from peroneal nerve



ii) F waves from tibial nerve



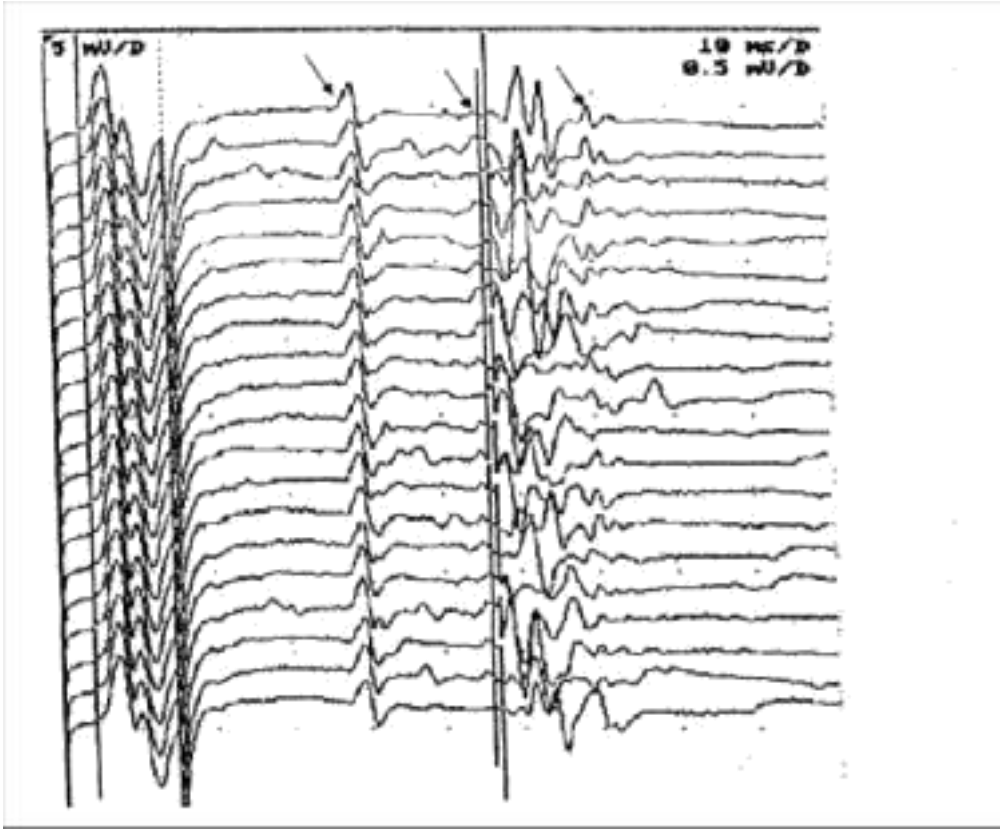
Studies of F-waves in normal peroneal (i) and tibial nerves (ii). Note the different occurrence of frequency of the F-waves in these two nerves.

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).

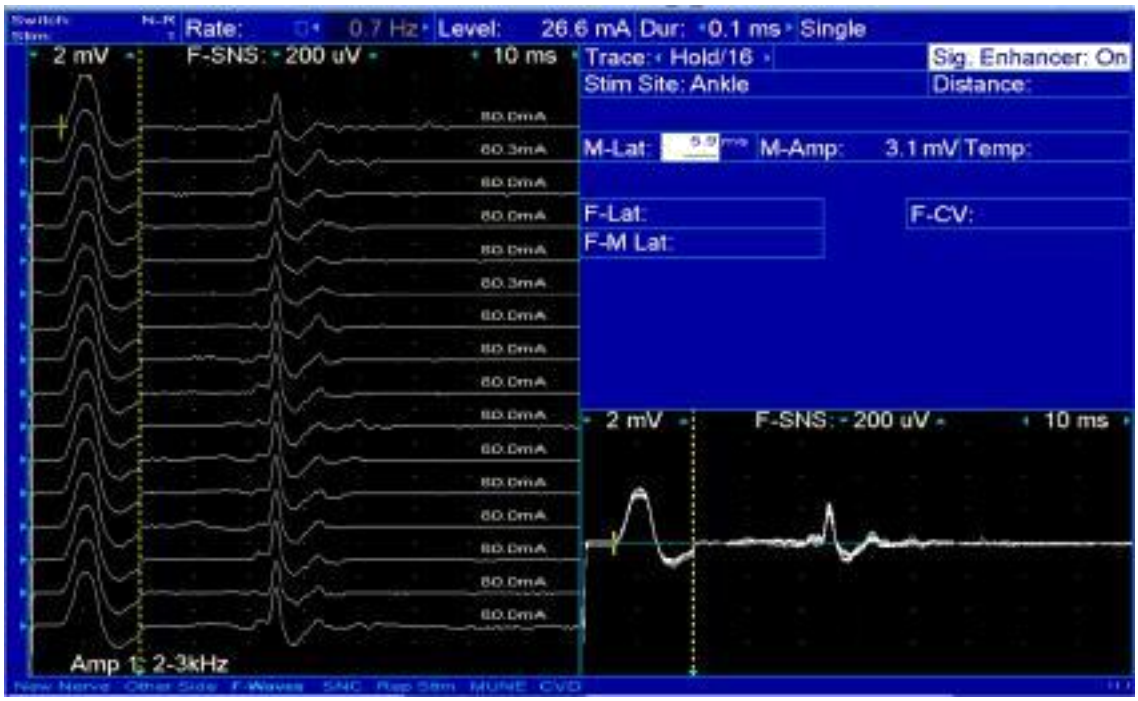
M-satellites (often called A-waves)

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 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.



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.

(i) Axon reflex from peroneal nerve

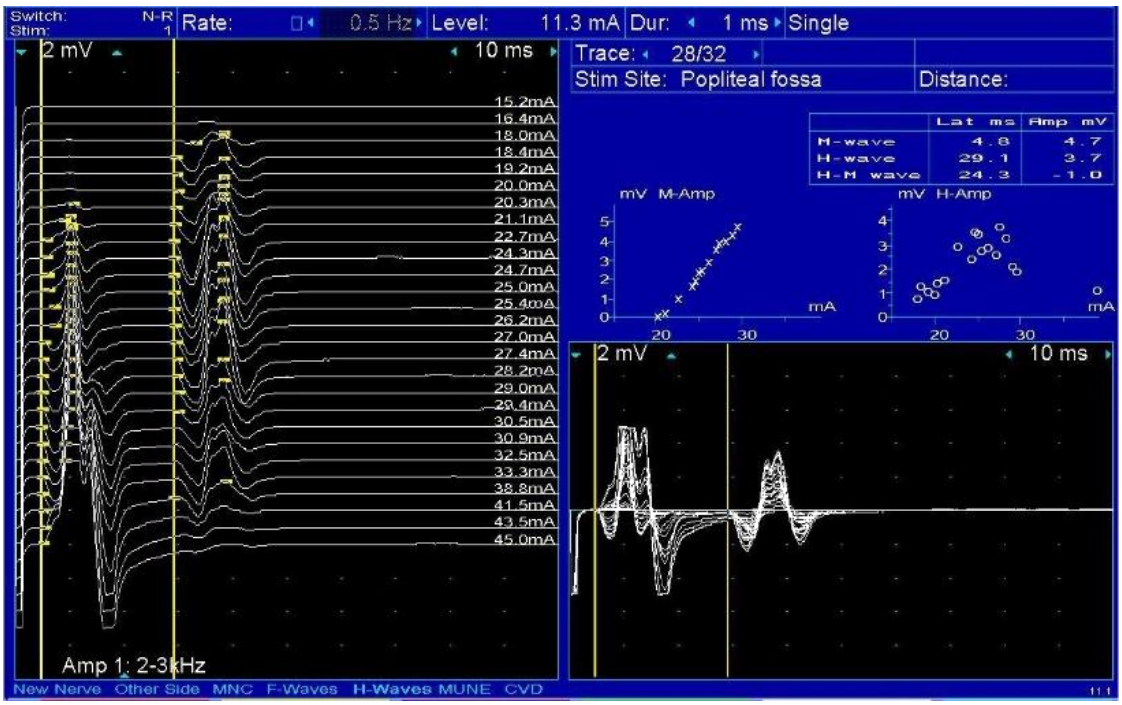


III- H WAVE

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 Jendrassic's maneuver will enhance the H-reflex amplitude and shorten the latency.



Summary of parameters

The most common motor neurography parameters are summarized in Table

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

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

Analysis

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

Motor conduction block

One of the parameters in nerve conduction studies concerns the presence of impulse conduction blocks, CB. A Conduction block is the failure of an action potential to propagate throughout the length of a structurally intact axon. This may be seen at the site of a local nerve entrapment and is typical of autoimmune neuropathies (Guillain- Barre, GBS; chronic inflammatory demyelinating 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 NCS, 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% *Ampl.Decay* or *Area.Decay* and <5% Dispersion or if there is >50% *Ampl.Decay* or *Area.Decay*, independent of Dispersion.

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

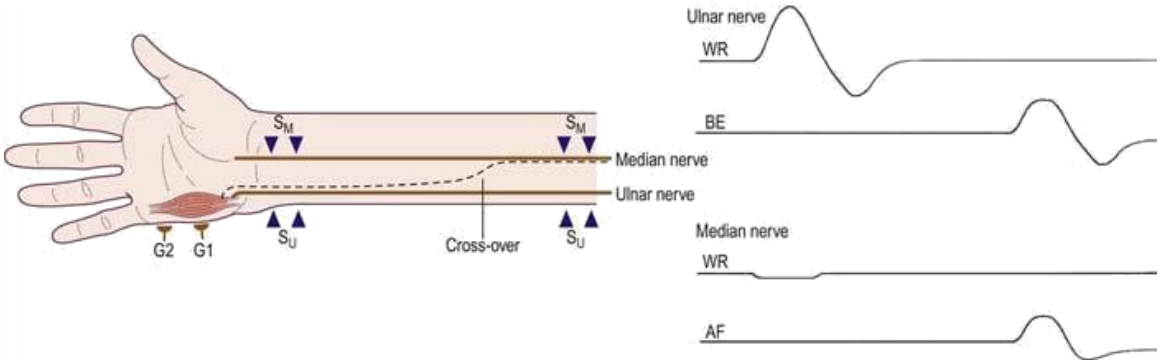
Our own (SCTIMST) modifications of these rules are:

>25% (arm) or >40% (leg) *Ampl.Decay* and <15% *Dispersion* or if there is >50% *Ampl.Decay*, independent of *Dispersion* (in this case there is a combination of CB and demyelination)

The other parameter, which indicates Conduction Block, is the frequency of occurrence of F- waves. In cases of proximal Conduction Block, there may be no drop in amplitude in the distal part of the nerve, e.g:- in early GBS. In this case, the Conduction Block 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, peroneal10, and tibial 18. As can be seen, it is impossible to detect a slight reduction in the number of F-waves in the ulnar and peroneal nerve. Lack of response 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% [17]. The fibers innervating intrinsic muscles of the hand cross from the median nerve to the ulnar nerve. Sensory fibers are not involved. Martin-Gruber anastomosis can 15e divided into three types according for 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 CAMP obtained at the proximal site also has an initial small positive deflection because of the volume of conduction from the deep ulnar innervated muscles. In type I the amplitude of the M wave from the ulnar nerve stimulation shows a reverse discrepancy; lower with elbow stimulation and higher with wrist stimulation.



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 distal latencies but a "normal" Proximal latencies. 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..

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; ˉ = decreased; n= normal

IV- 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 orthodromic and antidromic 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 inter electrode 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 inter electrode distance has to be kept constant within the laboratory.

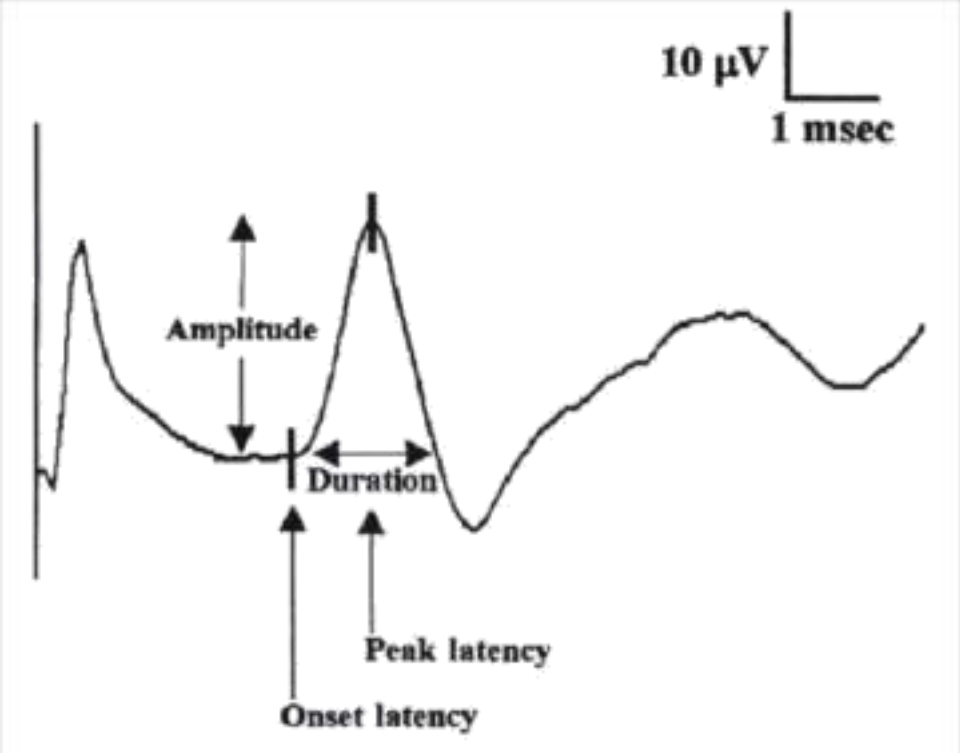
Reference data must be collected using the same technique that is used in routine studies

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 2mA). 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



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 conduction velocities.

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 mm or more.

Area

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

Duration

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

Conduction velocity

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

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

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

Temporal dispersion and decay

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

Table 4.

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

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

Analysis

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

V- AUTONOMIC NERVE TESTING

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

Autonomic tests

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

Sympathetic Skin Response

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

Procedure

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

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

SSR has two types of responses according to the polarity of the waveform with the maximum amplitude

P type & N type

P type- maximum positive deflection

N type-maximum negative deflection

P type is more frequent in healthy subjects

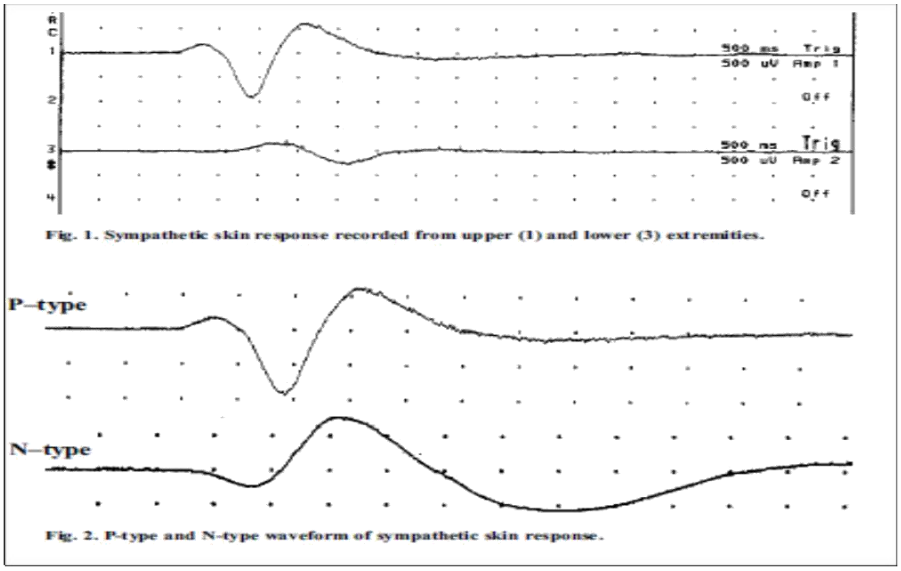
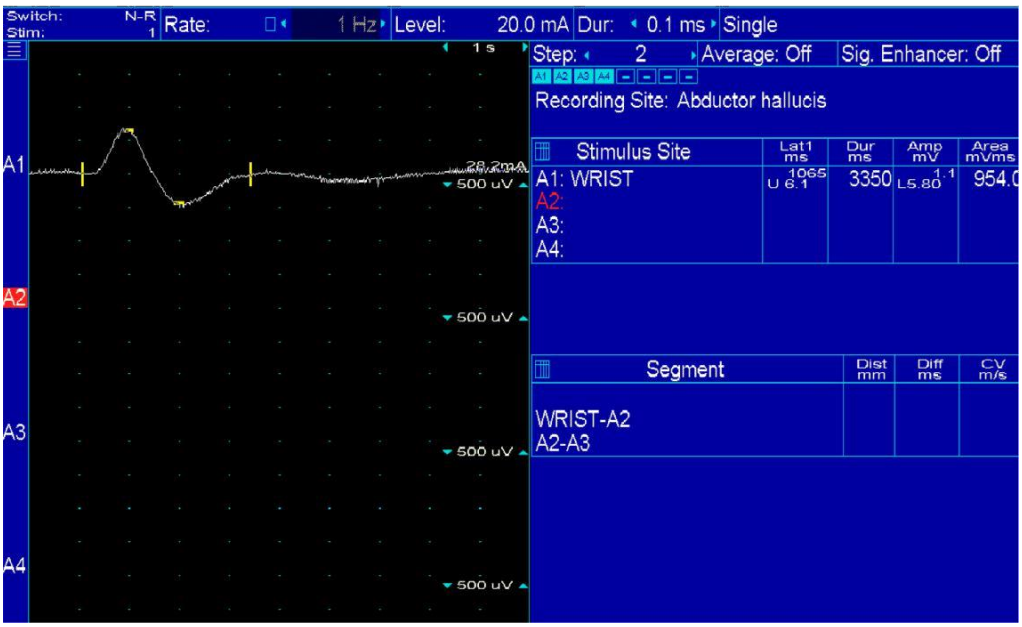


Fig. 1. Sympathetic skin response recorded from upper (1) and lower (3) extremities.

Fig. 2. P-type and N-type waveform of sympathetic skin response.



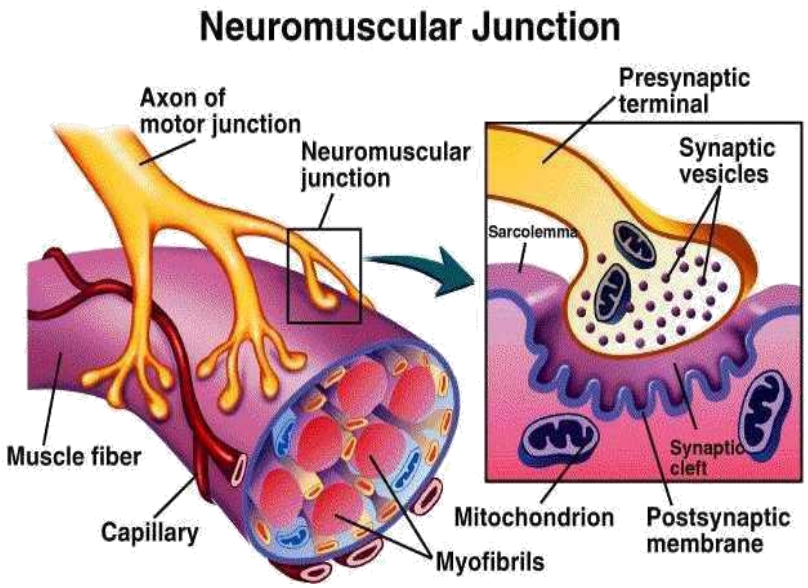
VI - REPETITIVE NERVE STIMULATION

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 of amplitudes and/or areas measured. This may be carried out at low (3-4Hz) or high frequency stimulation (20-50 Hz). In the later case the train is prolonged to allow 2-10 seconds of continuous data to be measured. Both distal and proximal muscles/nerves should be studied in every patient suspected of an NMTD as the sensitivity of the test is greatly increased by this means.

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

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

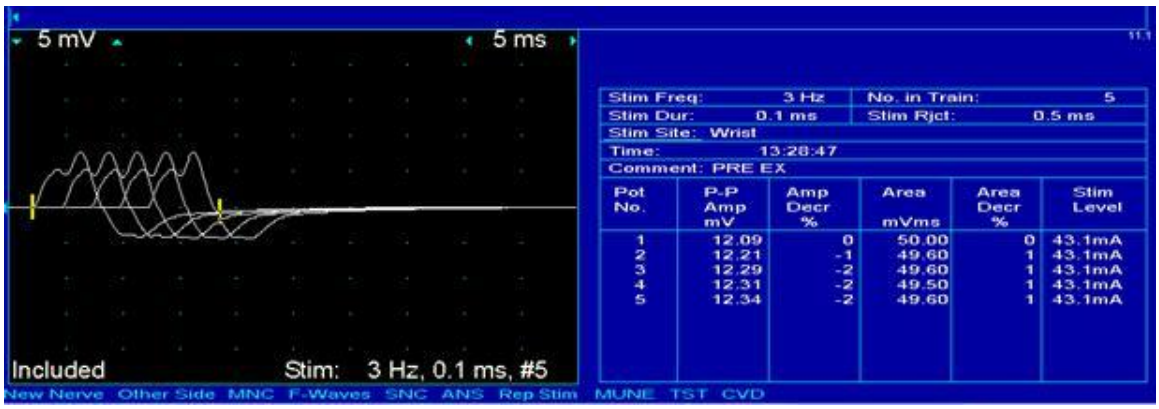
Physiological basis for the RNS



The neuromuscular junction consists of the motor axon terminal, the synaptic cleft, and the post-synaptic muscle membrane. As the motor axon potential depolarizes the nerve terminal, voltage gated calcium channels open increasing the concentration of calcium in the pre-synaptic nerve terminal. This in turn facilitates the release of quanta of acetylcholine (ACh) from the nerve terminal into the ,synaptic deft. ACh binds to receptors on the post-synaptic membrane causing depolarisation (end plate potential). The size of the end plate potential is dependent on the amount of ACh released and its binding to receptors. In the healthy state, the end plate potential reaches a threshold level and causes an action potential to be propagated along a muscle fibre resulting in muscle contraction. Normally there is a large safety factor for neuromuscular transmission with the amount of ACh released per impulse several times that required to generate a threshold level end plate potential.

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

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



Normal response of a slow RNS from ADM with right ulnar nerve stimulation

VII - BLINK REFLEX

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

Facial (VII)

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

Stimulation site:

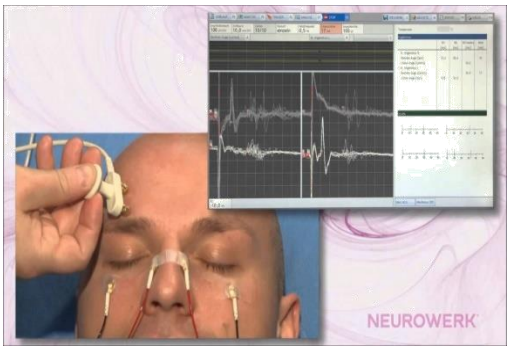
Place the electrodes behind the angle of the jaw, with the cathode posterior to the earlobe and the anode behind. This placement stimulates the nerve just before its enters the parotid gland. Alternatively, you may place the cathode over the sternocleidomastoid and anode over mastoid.

Ground:

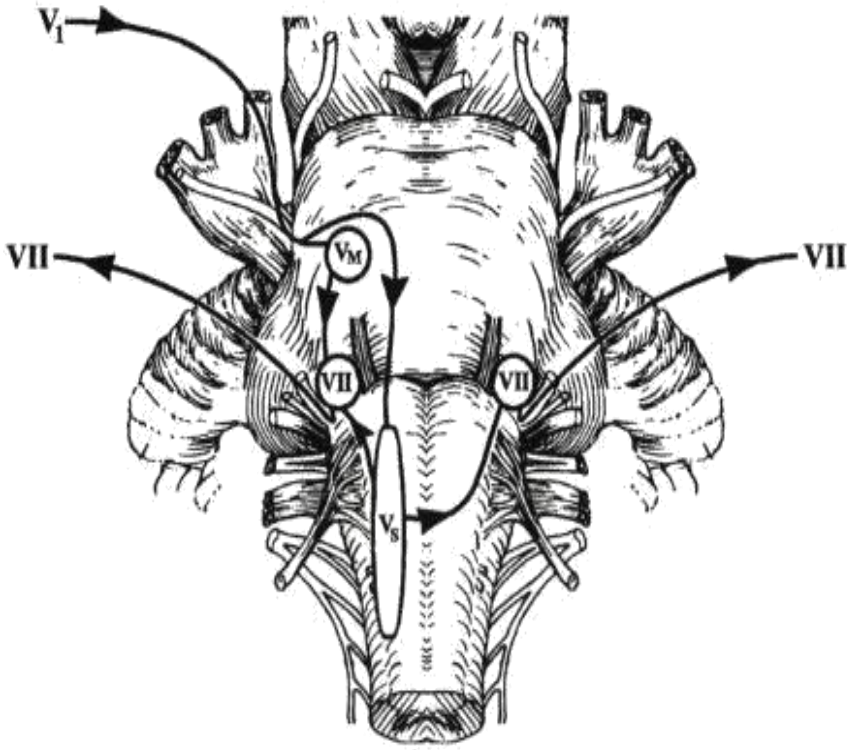
Usually the ground is placed over the parotid area, but you may place it on the chin or forehead also.

Recording sites:

Place the active recording over the orbicularis oculi of the both eyes. The facial nerve may be evaluated differently - by using the blink reflex, which will be discussed with the trigeminal nerve (below).



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



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

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

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

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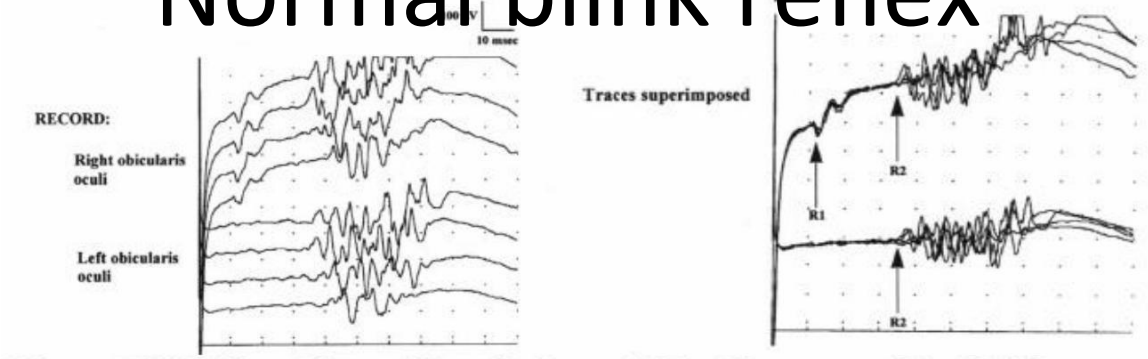
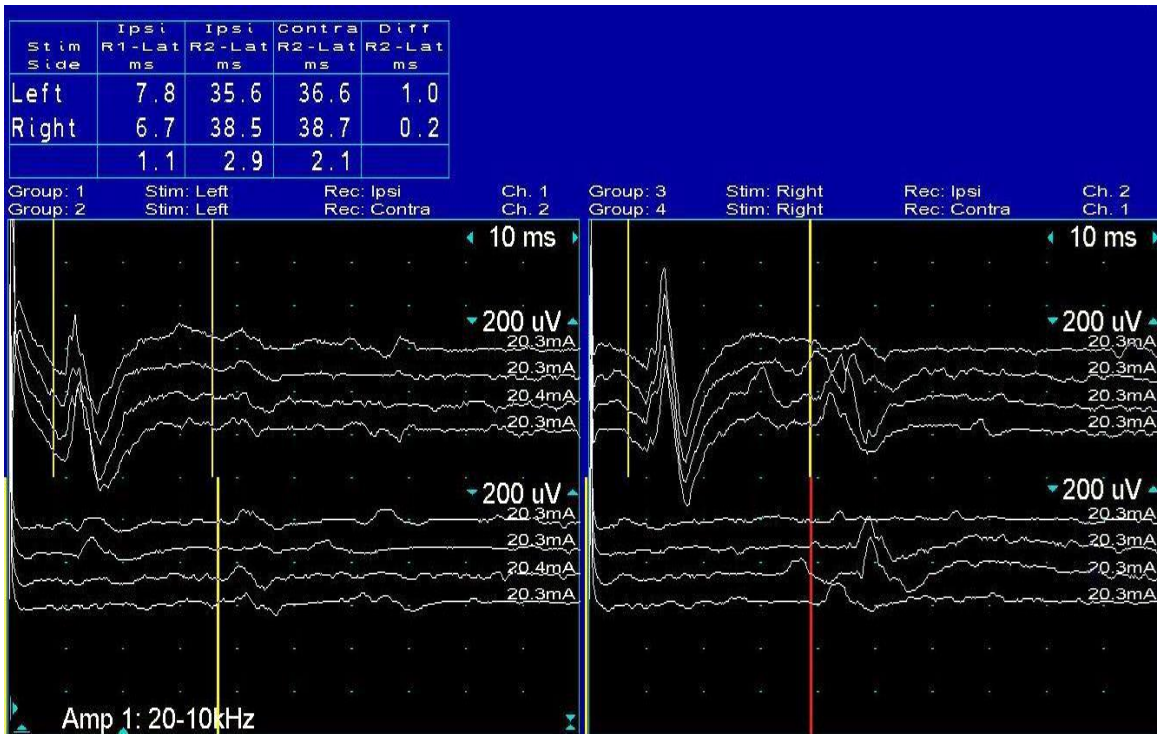
Normal blink reflex



Normal blink reflex. *Stimulating right side, recording both orbicularis oculi muscles in a normal subject. On the ipsilateral side, an early R1 potential is present at 11 ms and a late R2 potential at 34 ms. R1 usually is a biphasic or triphasic potential and stable from stimulation to stimulation. The R2 potential is variable and usually polyphasic. On the contralateral side, only a late R2 potential is seen at 35 ms. Superimposing several traces is useful to help determine the shortest R2 latencies.*

Normal Blink reflex



Blink Reflex Findings

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

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

ELECTROMYOGRAPHY

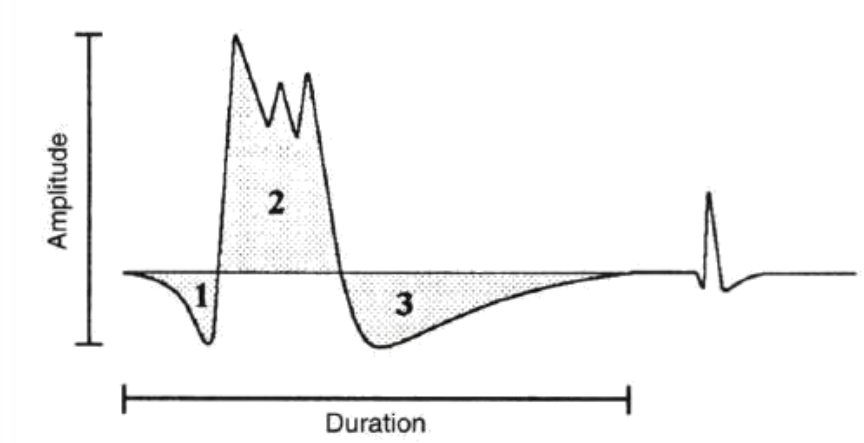
Volitional Activity in Muscle

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, and the interference pattern may be called complete or incomplete. The interference pattern can be recorded from single muscles with intramuscular monopolar needle electrodes or fine wire electrodes. Both type of electrodes are insulated except for the tip of the needle or wire. These procedures allow for an evaluation of a specific muscle with little to no interference from other muscles.

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

Evoked Activity in Muscle

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

Insertion Potentials

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

Spontaneous Activity in Muscle

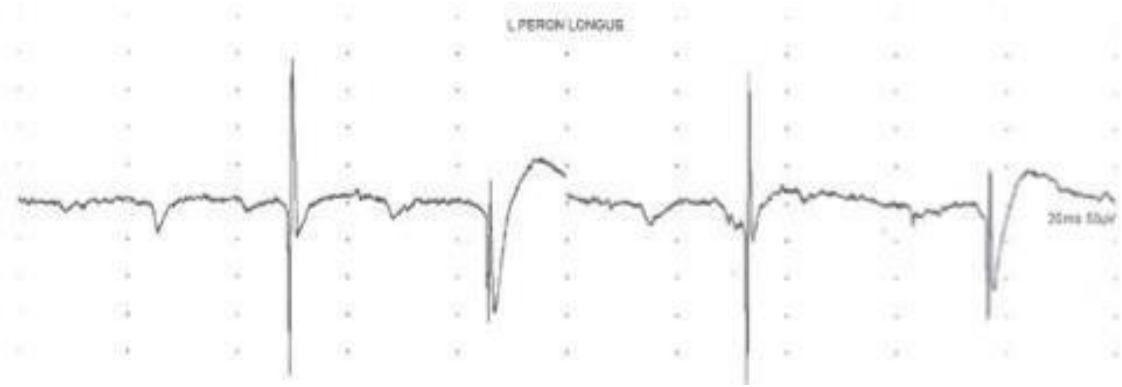
Spontaneous activity in muscle, when it occurs, may be initiated in the perikaryon of the LMN, its peripheral axon, the end plate, or the muscle membrane itself. As mentioned previously, if skeletal muscle is not volitionally or reflexively activated, it is electrically quiescent, therefore, most spontaneous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

Miniature End-Plate Potentials

Each skeletal muscle cell is 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 mV with durations of 1-2 msec. Fibs appear in denervated muscle after a latent period that is proportional to the length of remaining axons distal to the site of a nerve lesion. Their onset may be preceded by periods of increased insertional activity. Once these potentials appear in denervated muscle, their rate of occurrence increases over a period of several weeks. They will persist until muscle is reinnervated, or until no viable muscle fibers remain. Fibrillation potentials are also seen in myopathic disorders such as muscular dystrophies, polymyositis, and dermatomyositis. Their origin is related to oscillating membrane changes or irregular potentials 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.

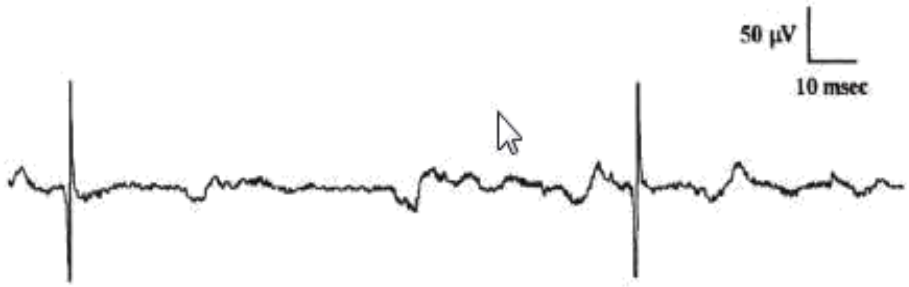
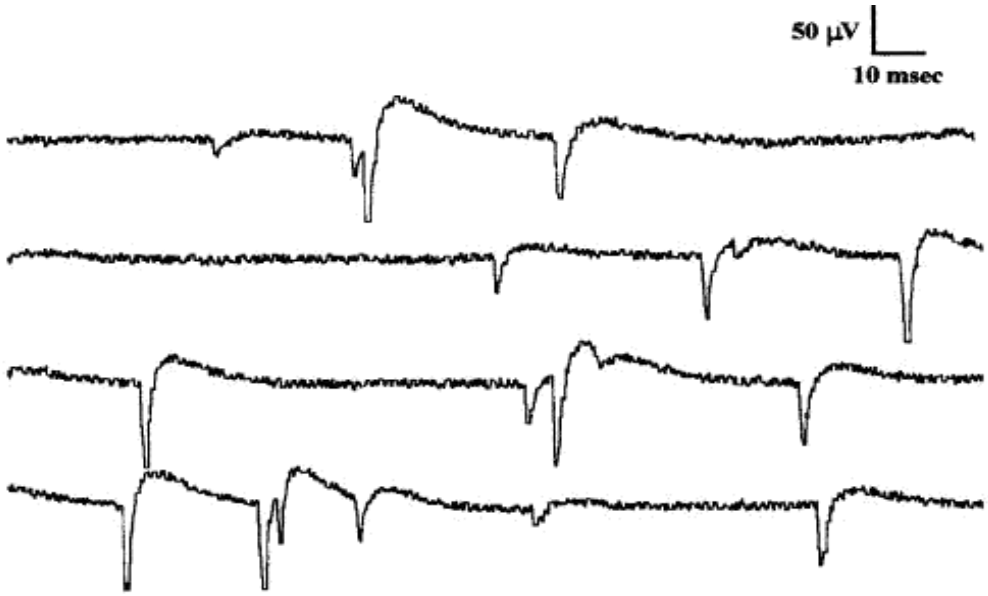


FIGURE 14–9
Fibrillation potential. *Spontaneous depolarization of a single muscle fiber. Note the initial positive deflection, brief duration, and triphasic morphology.*

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.



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 hyper adreno-corticism, and polymyositis. Some have referred to these as pseudo-myotonic 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 caption 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."

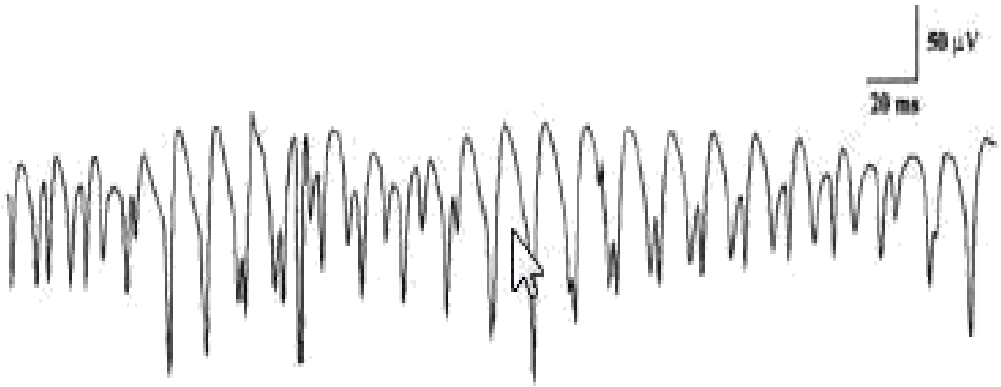


FIGURE 14–19
Myotonic discharge (spontaneous discharge). *Note the waxing and waning of both amplitude and frequency.*

Single Fiber EMG

Single-fiber electromyography (SFEMG) is a selective EMG recording technique that allows identification of action potentials (APs) from individual muscle fibers. The single-fiber needle allows extracellular recording of individual muscle fiber action potentials during voluntary contraction termed single- fiber electromyography (SFEMG), this technique has contributed substantially to the understanding of muscle physiology and pathophysiology. SFEMG supplements conventional electromyography by determining (a)fiber density—the number of single-fiber action potentials within the recording radius of the electrode and (b) electromyographic jitter—the variability of the inter potential interval between two or more single muscle fibers belonging to the same motor unit.

Principle of SFEMG

SFEMG is performed by inserting single fiber concentric needle into a muscle.

The electrode is positioned to record action potentials (APs) from 2 muscle fibers that are innervated by the same motor nerve fiber. The selectivity of the technique results from the small recording surface (25 μm in diameter), exposed at a port on the side of the electrode, which is 3 mm from the tip.

- Filter settings should be set at 500 Hz for the high pass filter, and 10–20 kHz for the low pass filter
- The selectivity of the recording is further heightened by using a high pass filter of 500 Hz.
- APs from distant muscle fibres contain relatively more low-frequency components & filtered off.

Single Fiber Needle Electrode

	Single Fibre	Concentric
Electrode diameter	25 µm	0.1 mm
Recording area	300 µm 2	1 mm2
Source	1-2 fibre	5-12 fibres
Potential shape	*Biphasic*	Bi / tri phasic
Amplitude (mV)	*1-10*	0.3 - 3
Duration (ms)	*<1*	2-15
Rise time (µs)	*100-150*	100-500

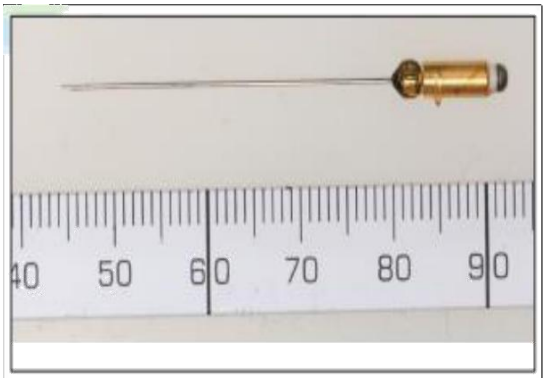


Figure 1: Single fibre needle electrode

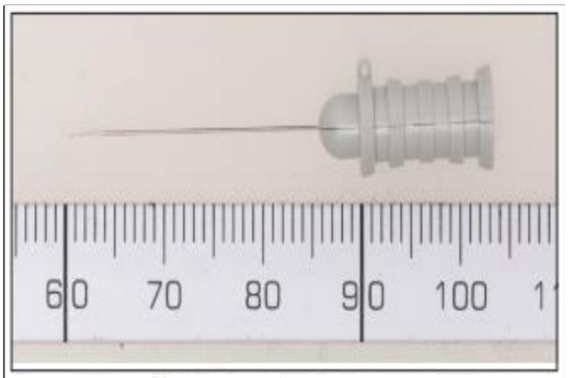


Figure 2: Concentric facial needle electrode

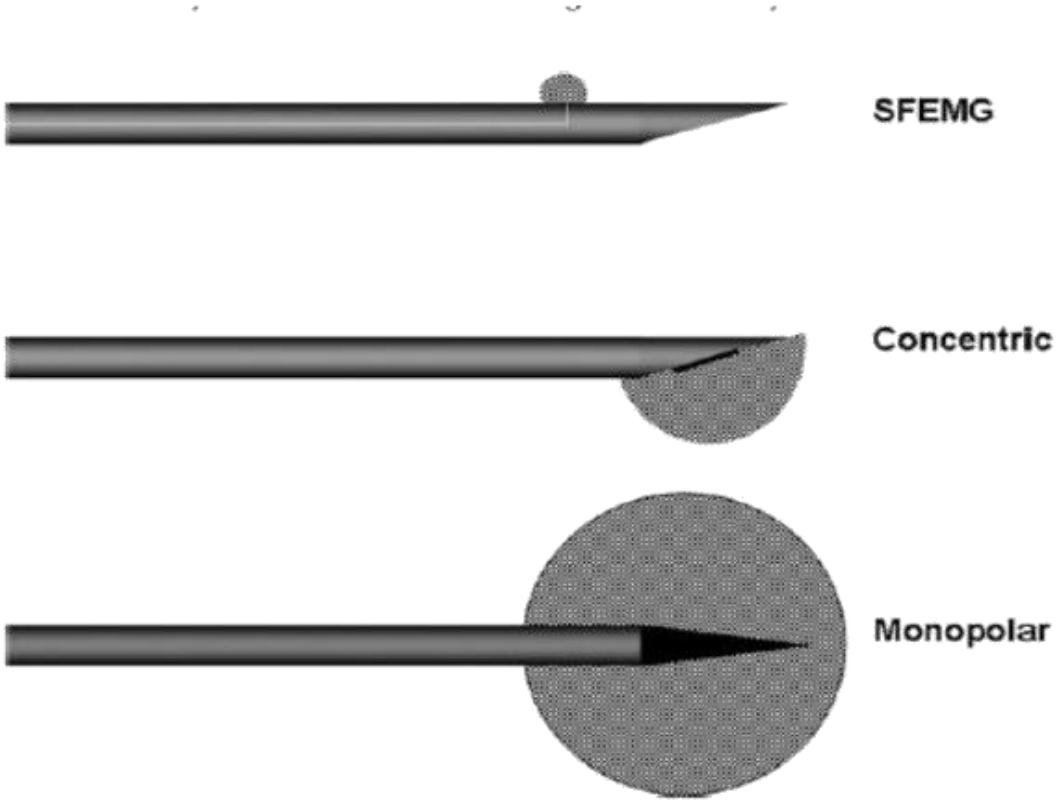


FIGURE 3. Relative recording uptake areas for the spike component of the MUP of three different electrode types. Schematic drawing based on simulation studies.[24,25] Copyright E. Stålberg, 2009.

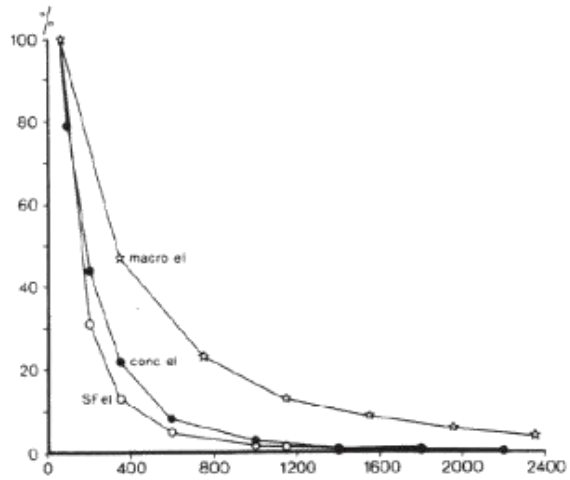


Fig. 2.2. Relative peak-to-peak amplitude of a simulated SFAP at different distances from the fiber surface as measured by a SFEMG (square), concentric needle (round) and Macro (star) EMG electrode. The SFEMG AP was filtered with a 500 Hz high-pass filter. The APs in CNEMG and Macro EMG were not filtered. (Modified from [Stålberg, 1983] with permission.)

Disadvantages of SFEMG Needle

- SFEMG electrodes are expensive,
- Develop increased impedance with use, and
- Must be sterilized for reuse, which does not entirely eliminate concern for infections main concern being prion infections.
-

What is **Jitter?**

- Neuromuscular jitter is defined as the random variability of the time interval between two MFAPs innervated by the same motor unit.it can originate from the terminal motor axon, NMJ and the segment of the muscle fiber between the NMJ and the recording electrode

Criteria for accepting an AP as being generated by a single muscle fiber

- Biphasic, smooth shape that is constant on successive firings,
- amplitude greater than 200 μV
- rise time less than 300 μs.

ACTIVATION AND RECORDING TECHNIQUE

- Voluntary Activation.
- Stimulation

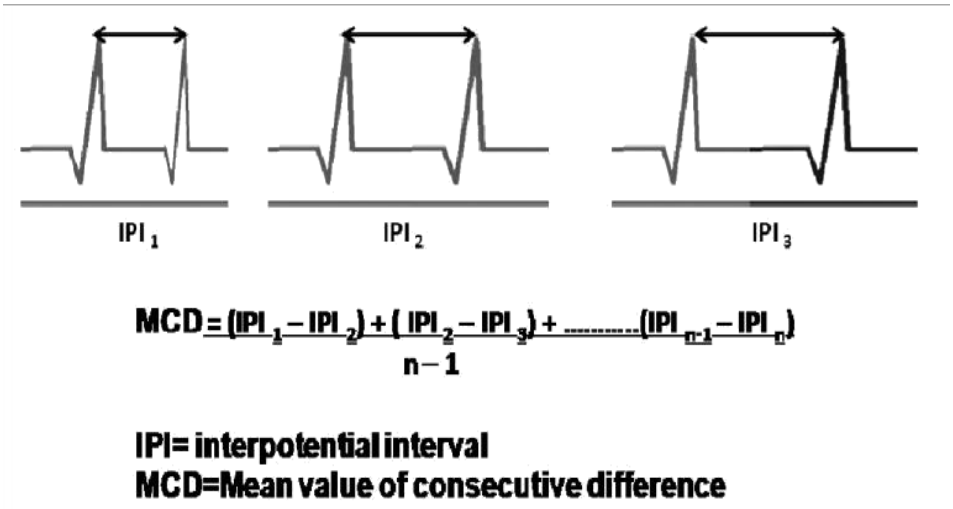
Voluntary Activation

- Usually, jitter measurements are performed during voluntary activation of the muscle
- Less subject to technical problems but it requires more patient cooperation.
- With minimal voluntary activation, the needle is positioned until two muscle potentials (a pair) from a single motor unit are recognized.
- When a muscle fiber pair is identified, one fiber triggers the oscilloscope (triggering potential) and the second precedes or follows the first (slave potential).
- With voluntary activation, fifty consecutive discharges of a single pair is recorded.

Jitter measurement techniques

- The jitter is expressed as the mean value of consecutive differences of successive inter-potential intervals (MCD)
- The normal mean MCD value varies from 10-50 μs among different muscles.
- In certain situations the inter-potential interval (IPI) may be influenced by the preceding inter-discharge interval (IDI)
- May introduce an additional variability due to changes in the velocity of AP propagation in the muscle fibers.

- The effect of preceding depolarization's becomes constant at that point, provided no impulse blocking is present to produce an irregular discharge rate
- The effect of variable firing rates (during voluntary activation) can be minimized by sorting the IPIs according to the length of the preceding IDI, then calculating the mean of the consecutive IPI differences in the new sequence.
- The result is called the mean sorted-data difference (MSD).
- If the ratio MCD: MSD exceeds 1.25, then variations in the firing rate have contributed to the jitter, and the MSD should be used to represent the neuromuscular jitter.
- The MCD is used to express the jitter if the MCD:MSD ratio is less than 1.25
- **Neuromuscular blocking** is defined as the failure of one of the muscle fibers to transmit an action potential due to the failure of EPP to reach threshold.
- This usually happens when the jitter value is markedly prolonged, usually when MCD is more than 100 microseconds.



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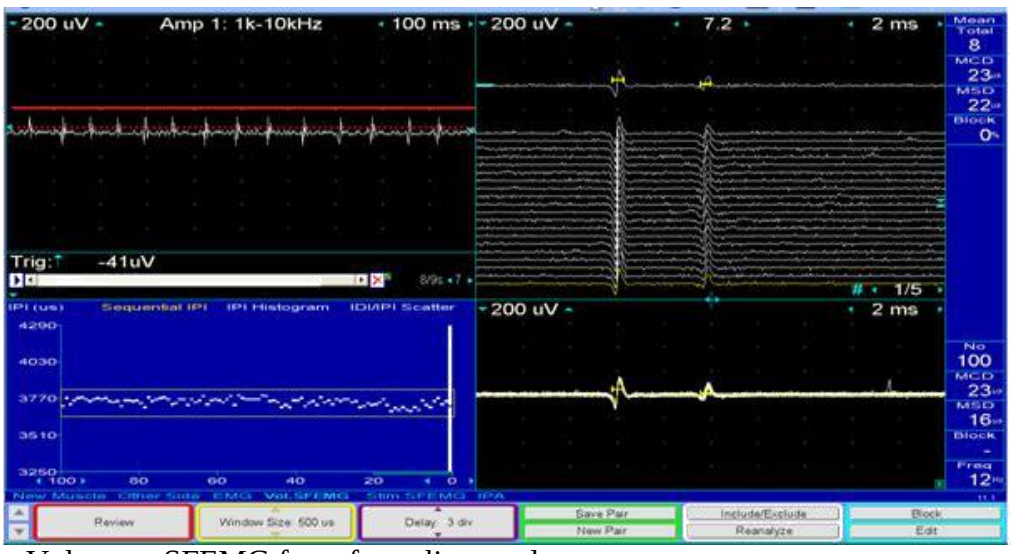


Voluntary SFEMG from frontalis muscle

EVOKED POTENTIALS

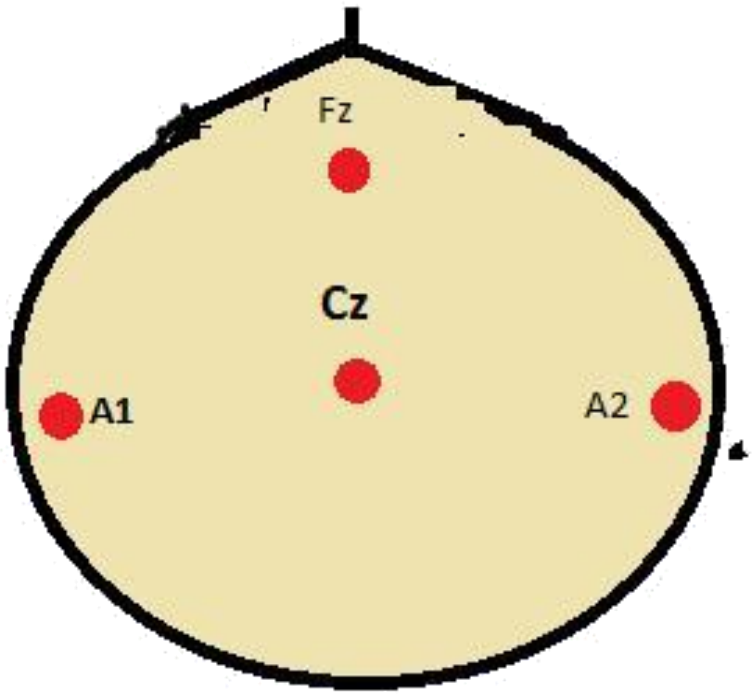
BRAINSTEM AUDITORY EVOKED POTENTIALS

Introduction

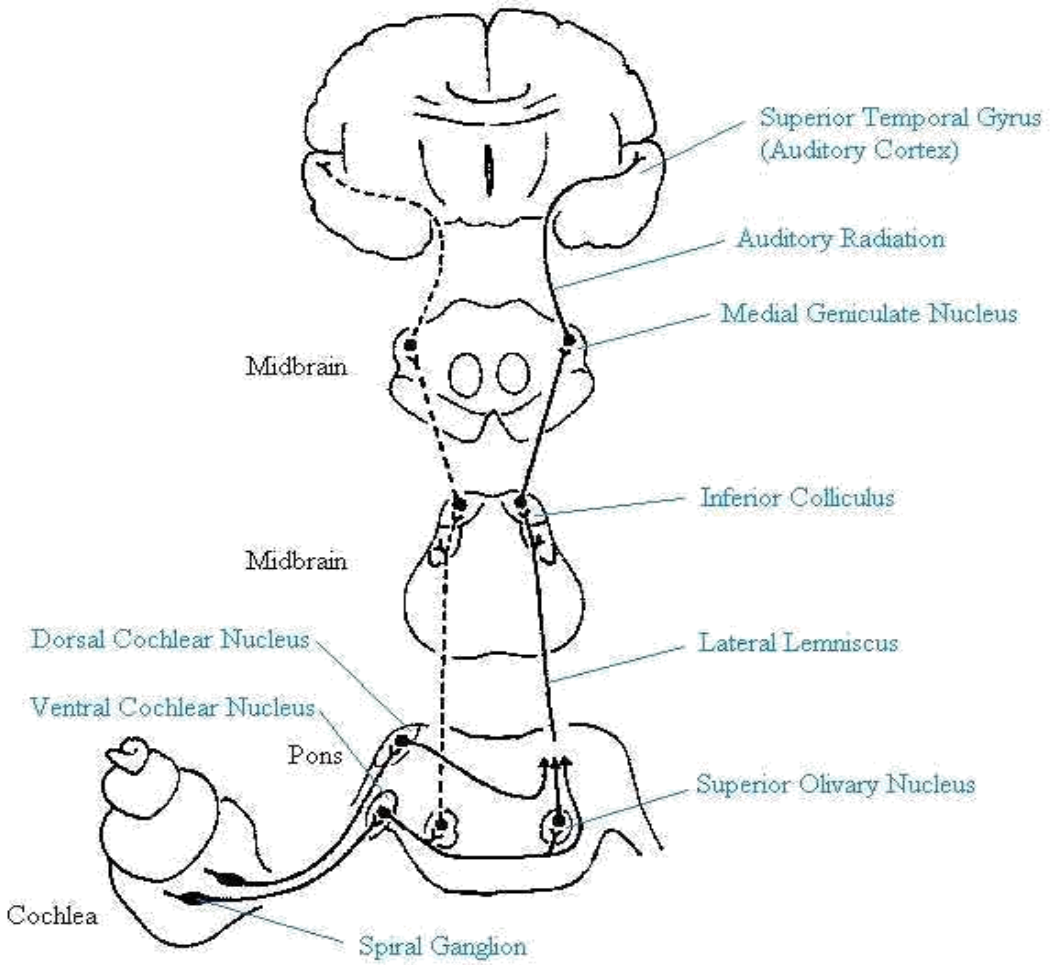
BAEP or BAER measures the function of the auditory nerve and auditory pathways in the brain stem. The short latency BAER generally is used for clinical purposes. The test can be performed under sedation or under general anesthesia. Standard broadband click stimulation is used on the ear tested, while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

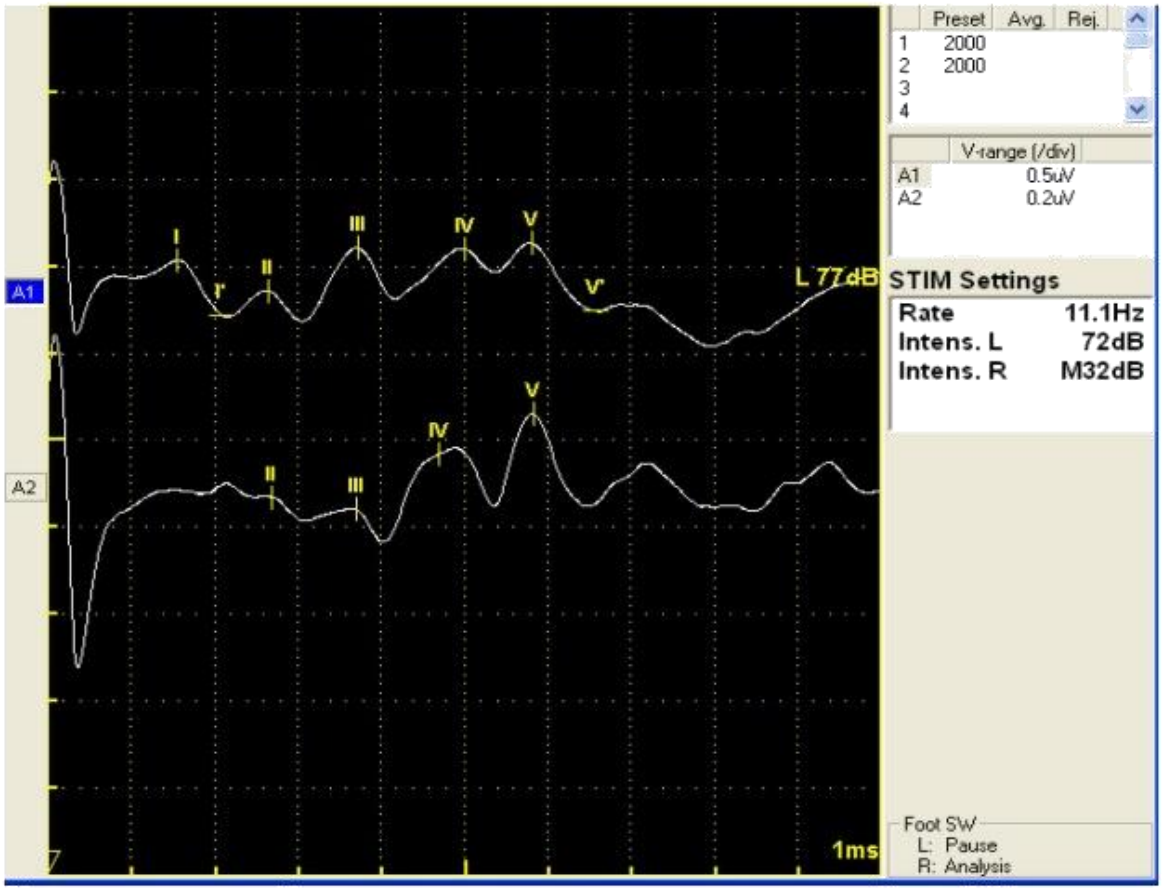
Electrode placement

An electrode is placed on each earlobe and at Cz.



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





Factors influencing peak latencies of BAERs include age, sex, auditory acuity Stimulus repetition rate, intensity, and polarity. Rarefaction (i.e., 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. 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 inter-peak 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 III and V. Peak and inter-peak 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 BAEP.

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 BAEP may be more concerned with sound localization that with hearing itself.

BAEP in Intraoperative Monitoring

USE;

BAERs are used intraoperatively to assess the functional integrity of acoustic pathway structures, particularly those located in the brainstem. Typical situations requiring BAER monitoring include surgery for acoustic tumors, and procedures involving the cerebello-pontine angle and the posterior fossa

BAEP Features

The basic BAER features used for intraoperative analysis include measurement of peak amplitudes, as well as peak and interpeak latencies [46, 20]. Occasionally, normal recordings may not contain all of the peaks. Wave V is the most reliable one and is present most of the times, along with wave I and III. Wave II is often missing, whereas wave IV may partially or completely merge with wave V

BAEP Interpretation

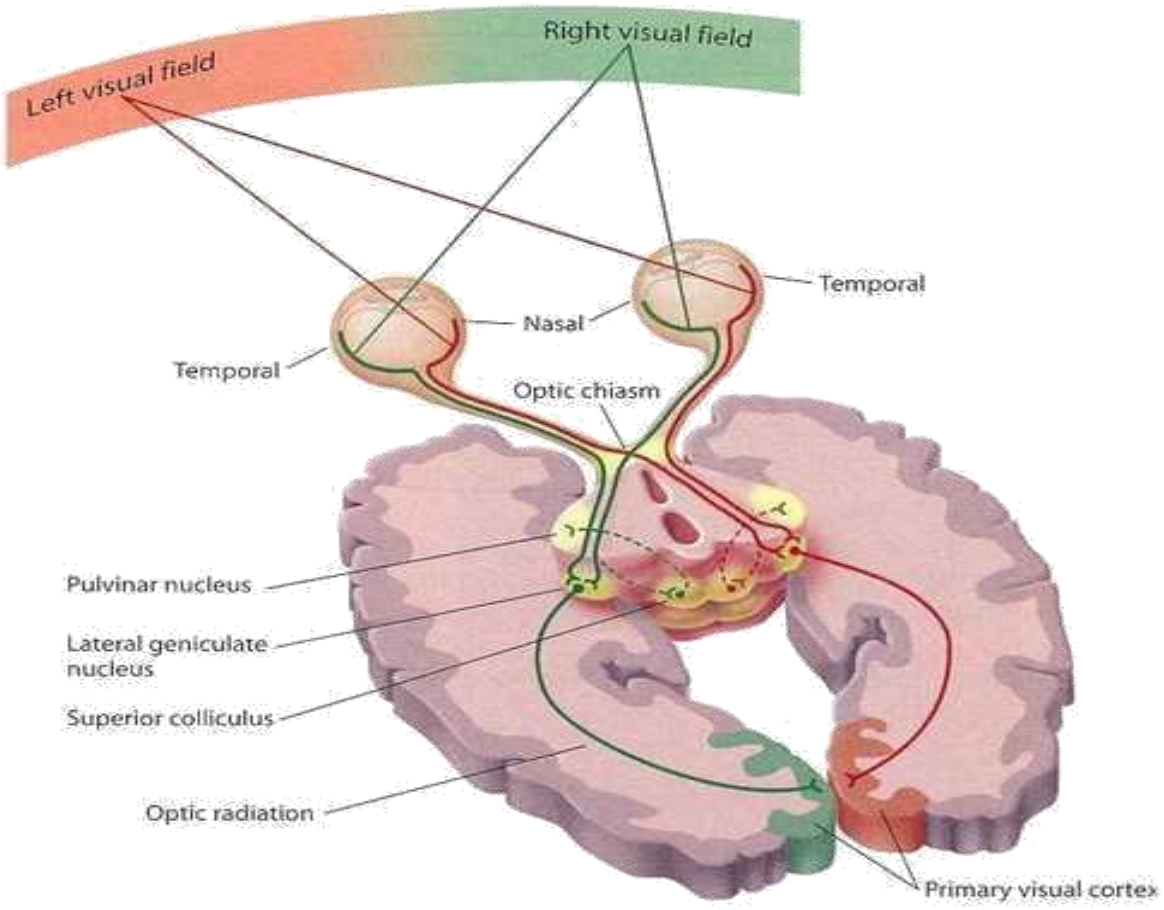
Typically, after induction and final positioning of the patient, a set of baselines is obtained which remains on the screen for comparison throughout the case. Baseline responses should contain clear and reliable components, and should also be correlated with the clinical picture of the patient. For example, peripheral hearing loss may result in unclear or absent peaks.

During surgery, BAER interpretation criteria are based on the detection of significant changes, compared to the baselines, mainly in the amplitude and latency of peaks I and V, as well as the interpeak latencies from peak I to III and from III to V. These interpeak latencies represent the peripheral and central conduction time, respectively. BAERs are subcortical in origin and, thus, little affected by anesthetics or small changes in the anesthesia regime [20]. Therefore, even small changes may be significant. Traditionally, the most important criterion involves the latency and the amplitude of peak V. A change repeated twice in a row must be reported even if the latency has increased by only 0.5 msec. A shift of 1–1.5 msec usually indicates that some action must be taken.

VISUAL EVOKED POTENTIAL

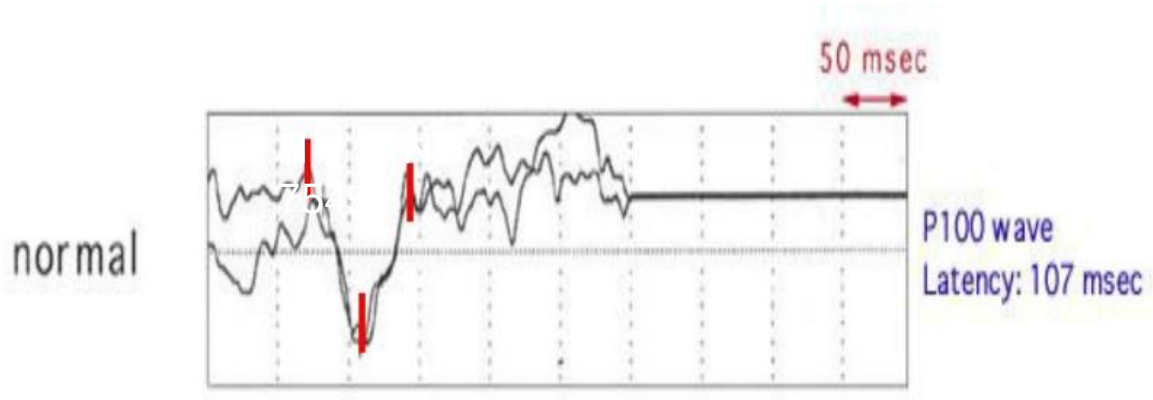
Visual Evoked Potentials

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

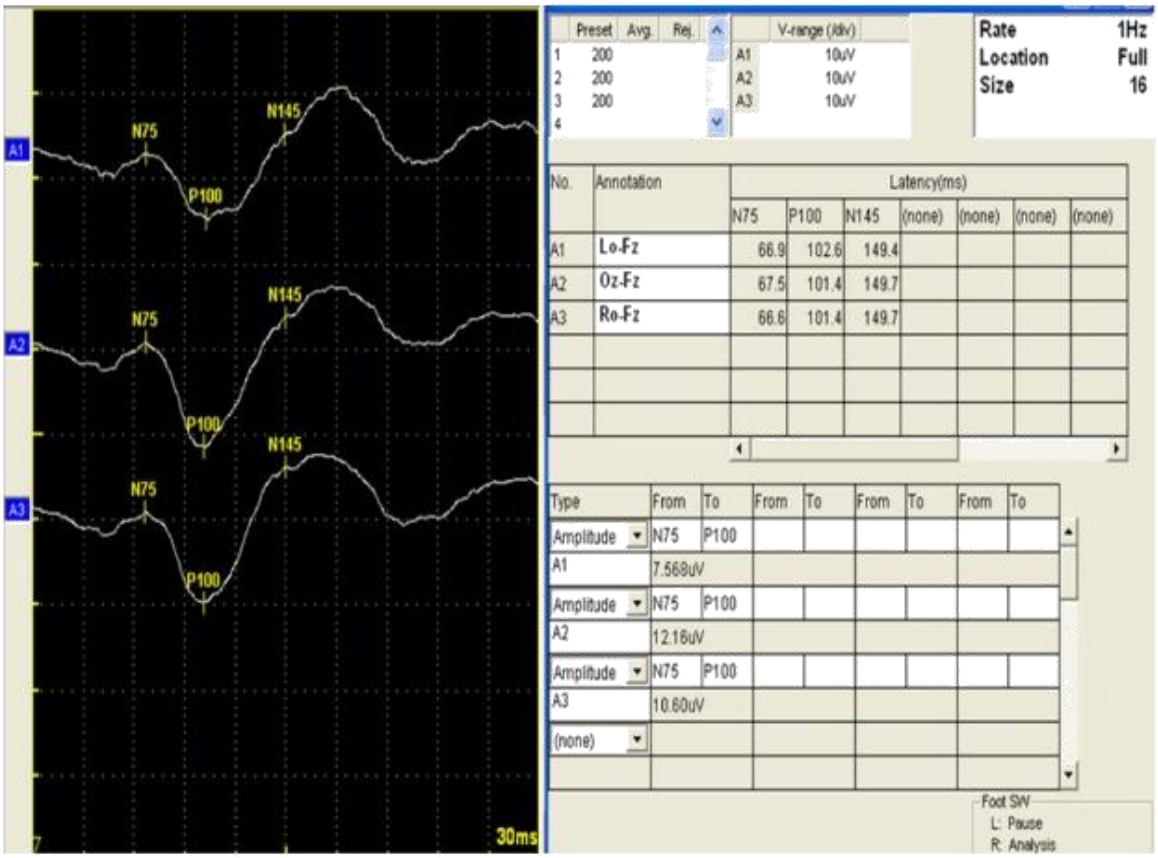


The VEP is very useful in detecting an anterior visual conduction disturbance. However, it is not specific with regard to the etiology. A tumor compressing the optic nerve, an ischemic disturbance, or a demyelinating disease may cause delaying the P100; only additional clinical history and, often, MRI are needed to uncover the etiology. The usual waveform is the initial negative peak (N1 or N75), followed by a large positive peak (P1 or P100) followed by another negative peak (N2 or N145).

Maximum value for P100 is 115 ms in patients younger than 60 years; it rises to 120ms thereafter in females and 125 ms in males. Even though, 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 be not be used, and note should be taken of medications that the patient is taking regularly. Testing circumstances should be standardized, including seating distance of 70-100 cm from the monitor screen, giving a check size of approximately 30 seconds of visual angle. The vision should be corrected to the extented 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 100ms. Normative data should be assembled on a lab-by-lab basis.

VEP In Intraoperative Monitoring.

VEPs are used intraoperatively to assess the functional integrity of the visual pathway during surgery for tumors or trauma involving the optic nerves, chiasm, optic tracts, and the occipital visual cortex. VEPs are most useful in cases involving the retro orbital and parasellar regions

VEP Features

Intraoperative analysis of VEP features involves measurement of peak amplitudes, as well as peak and interpeak latencies. Typical flash VEPs contain two major positive components, *P*1 and *P*2, found at about 100 and 170 msec after stimulus onset, respectively. Each of the components is preceded by a negative one, *N*1 and *N*2, at about 70 and 140 msec, respectively. The latter components however are less clear and stable. All components are generated by the visual cortex

Interpretation

In general, after induction and final positioning of the patient, a set of baselines is obtained which remains on the screen for comparison throughout the case. Baseline responses should contain clear and reliable components, and should also be consistent with the clinical picture of the patient.

During surgery, interpretation criteria are based on detection of reliable changes (compared to baselines) that affect the overall morphology of the response, their latency, as well as eventual asymmetry in component amplitude and latency between the left and right eyes . A change can be considered significant if (1) results in a 50% amplitude reduction or complete loss of the VEP, or (2) the maximum latency shift is more than approximately 40–50 msec . However, because of the great variability of the flash VEPs, the only reliable criterion for abnormality is the complete absence of the components resulting from monocular stimulation.
Several studies have shown the high incidence of false positives that can be as high as 95% of the cases, thus making the intraoperative use of VEPs questionable

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 lemniscuses 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, thalamo-cortical projections, or primary somatosensory cortex. Since individuals have multiple parallel afferent somatosensory pathways (e.g. anterior spino-thalamic 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 t the posterior column of the spinal cord. When a mixed peripheral nerve (with both sensory and motor components) is stimulated, both group IA muscle afferents and group II cutaneous afferents contribute to the resulting SSEP. Selective ablation of the dorsal column of the spinal cord abolishes the SSEPs generated rostral to the lesion. Diseases of the dorsal columns in which joint position sense and proprioception are impaired invariably are associated with abnormal SSEPs.

Stimulus Location

For recording median nerve SSEPs, the nerve is stimulated at the wrist. The anode is placed just proximal to the palmar crease, and the cathode is placed between the tendons of the palmaris longus muscle, 3 em proximal to the anode.

Ulnar nerve SSEPs are preferred to median nerve SSEPs for assessing the lower cervical spinal cord segment since the ulnar nerve originates from spinal roots C8-Tl, whereas the median nerve originates from C6-Tl.

For recording posterior tibial nerve SSEPs, the nerve is stimulated at the ankle, with the cathode midway between the Achilles tendon and the medial malleolus and the anode 3 cm distal to the cathode.

Peroneal nerve SSEPs, the common peroneal nerve is stimulated at the knee, with the cathode inferior to the leg crease just medial to the tendon of the biceps femoris muscle and the anode 3 cm distal to the cathode.

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

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

Stimulation

Intensity of the selected nerves are stimulated with monophasic square pulses, 100-300 microseconds in duration. Stimuli are delivered by using either a constant voltage or a constant current stimulator. The contact impedances of the stimulating electrodes should be kept low for the following reasons:

• To minimize patient discomfort
• For more effective nerve stimulation, if a constant voltage stimulator is used

To avoid electrical artifacts with constant current stimulation in the clinical setting, the stimulus intensity is set high enough to produce a consistent muscle twitch, which usually is tolerable to the patient. Because the patient is anesthetized during intraoperative SSEP monitoring, higher stimulus intensities can be used and are advisable to provide a safety margin in case the efficacy of nerve stimulation decreases during surgery.
Stimulus Rate Rapid stimulus delivery rates should be avoided, as they degrade the waveforms of SSEPs. In clinical settings, a rate of 3-6 stimuli per second usually is used. Rates that are exact sub harmonics 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,inter-peak 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.

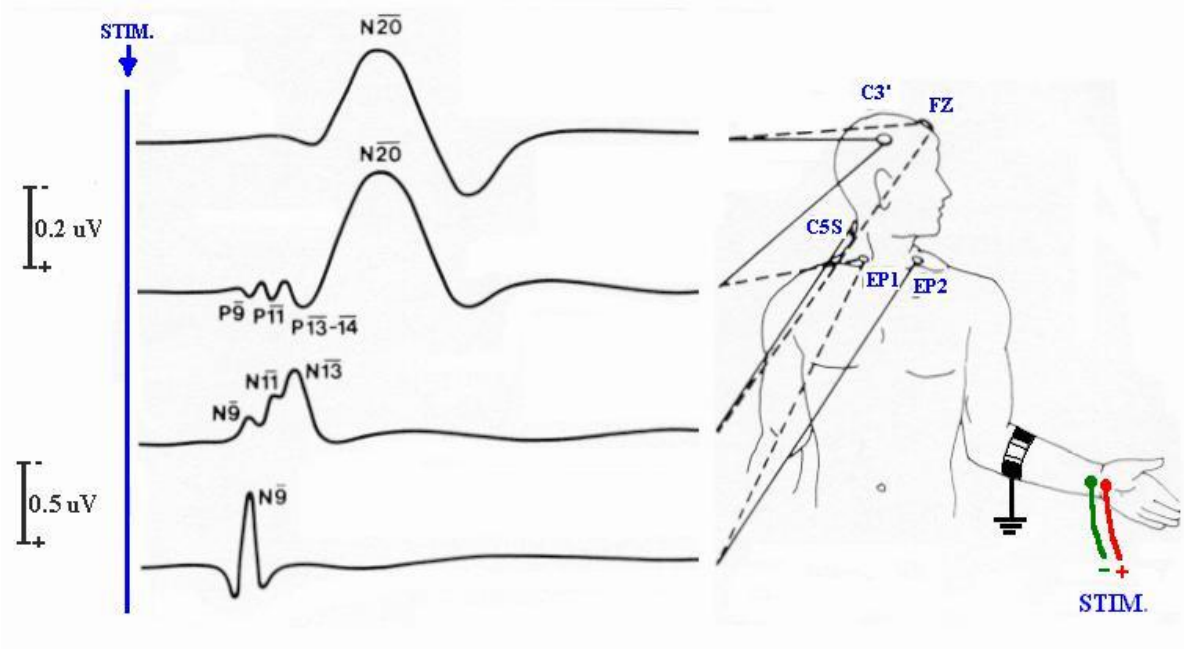
Absolute SSEP latencies vary with limb length. Inter-peak (i.e., transit) times are reliable parameters that are independent of limb length and usually independent of peripheral nerve disease. Aging is associated with some prolongation of SSEP latencies. Latencies are considered abnormal when they are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

Recording electrodes sites

Anatomical landmarks identify recording electrode sites. Those on the head are defined using the international 10-20 systems, or its extension, the 10-10 systems. Electrode CP3 is midway between C3 and P3, and electrode CP4 is midway between C4 and P4. CPi denotes either CP3 or CP4, whichever is ipsilateral to the stimulated limb; CPc is the contralateral Centro parietal scalp electrode. CPz is midway between Cz and Pz.
Recording electrodes over the spine are placed in the midline, and they are labeled with the name of the vertebral body they are placed on followed by the letter S, for example:- TlOS.

Recording montages for cortical SSEP components are either cephalic bipolar, in which both electrodes are placed over the head, and 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 Erbs point (ie:-the angle between the clavicular head of the sternocleido mastoid muscle and the clavicle), both ipsilateral and contralateral to the stimulus (labeled EPi and Epc). For lower limb SSEP studies, Ic denotes an electrode placed over iliac crest.



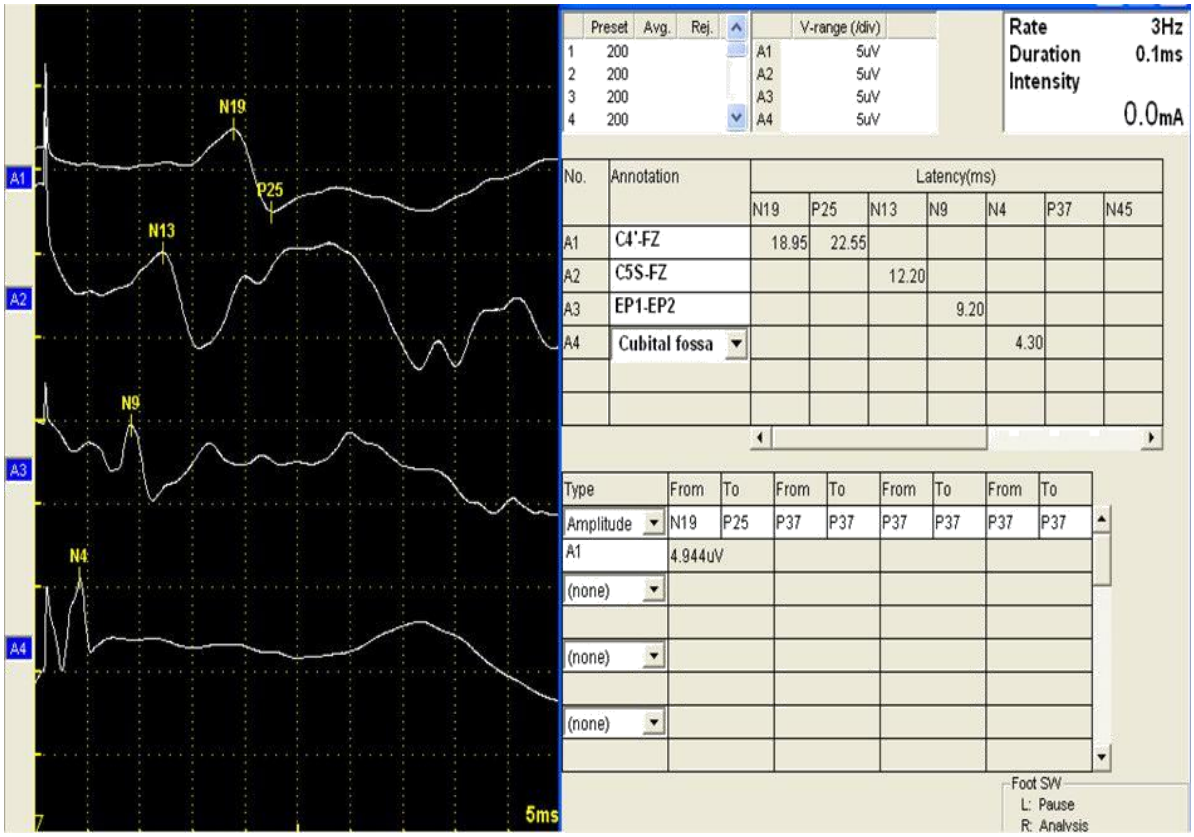
Normal values

Site	Peak latency (msec) (mean +/- I SD)	Side – to - side latency difference (rang, msec)	Peak Amplitude (micro V) (mean +/- I SD)	Side - to - side amplitude difference (range, micro V)
Elbow (N4)	4.3 +/- 0.3	0.24 – 0.99	3.4 +/- 0.7	0.11 – 2.54
Shoulder (N9)	9.9 +/- 0.6	0.44 – 1.74	2.1 +/- 0.6	0.17 – 0.94
Cervical • 1st peak(N12) • 2nd peak(N13)	12.1 +/- 0.2 13.4 +/- 0.3	0.47 – 1.89 0.43 - 1.52	1.5 +/- 0.4 1.9 +/- 0.3	0.14 – 1.31 0.16 – 1.28
Scalp • N1(N20) • P1(P25) • N1 – P1	19.2 +/- 1.1 25.2 +/- 2.1	0.72 – 3.10 1.08 – 4.05	0.6 +/- 0.2 2.1 +/- 0.9	0.19 – 1.81 0.21 – 2.31

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.



SSEP in 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) .

Uses

Somatosensory evoked potentials are used intraoperatively to:

- Monitor blood perfusion of the cortex or the spinal cord (e.g., during an aneurysm clipping).
- Monitor the structural and functional integrity of the spinal cord during orthopedic or neurological surgery (e.g., for scoliosis or a spinal tumor).
- Monitor structural and functional integrity of peripheral nerves (e.g., sciatic nerve during ascetabular fixation), spinal nerve roots (e.g., during decompression in radiculopathy), and peripheral nerve structures (e.g., the brachial plexus).

Determine *functional* identity of cortical tissue (e.g., one can separate the sensory from motor cortex by identifying the central sulcus).

SSEP Interpretation

After induction and final positioning of the patient, a set of baselines is obtained which remains on the screen for comparison throughout the case. Baseline responses should be of familiar morphology and contain clear and reliable components. The baselines should also be consistent with the clinical picture of the patient.

During surgery, interpretation criteria are based on detection of reliable and significant changes compared to the baselines established at the beginning of the case. Changes mainly involve the amplitude and latency of the SEP components recorded at different levels. A change is *reliable* if it is repeatable at least twice in a row; and it is *significant* if the amplitude has decreased by at least 50% or the latency has increased by at least 10%

Motor Evoked Potentials

Introduction

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 pre-central 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 cortico-spinal 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 radiate 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 · 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, high voltage pulses of brief duration for percutaneous stimulation. The output current range is 0-1000 mill amperes, 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.

MEP In Intraoperative Monitoring

Somatosensory EP monitoring measures the integrity of sensory tracts only. Therefore, selective damage to motor tracts may go undetected. Additionally, since the ventral and dorsal portions of the spine are supplied by different blood vessels, ischemia affecting the motor tracts only will not be detected by SEPs. Thus, MEPs are used intraoperatively to protect the structural and functional integrity of the motor tracts in the spinal cord

MEP Features

Neurogenic MEPs recorded, for example, at the popliteal fossa, in addition to the *orthodromic* motor component, contain an *antidromic* sensory component. The former results from intentional stimulation of the motor tracts in the spinal cord and travels in the direction of normal signal propagation, while the latter results from unintentional stimulation of the sensory tracts in the spinal cord and travels in the opposite direction of normal signal propagation. However, because of differences in conduction velocity in the sensory and motor tracts, the motor component leads the sensory one, allowing for proper discrimination of the two types of activity

VAGUS NERVE STIMULATION

Introduction

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

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

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

Vagus nerve

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

Aim of VNS

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

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

VNS Pulse Generator

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

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

Bipolar Lead

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

Programming Wand and Software

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

Tunneling Tool

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

Accessory Pack

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

Working of VNS during seizure

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

Side effects

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

Precautions

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

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

VISUAL ACUITY
&
VISUAL FIELDS TESTING
(OPTOMETRY)

Introduction

Optic nerve is the most important nerve which carries visual impulses from the retina to the optic chiasm and on the optic tract to the lateral geniculate body to act as the afferent pathway for the papillary light reflex by means of fibers 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.

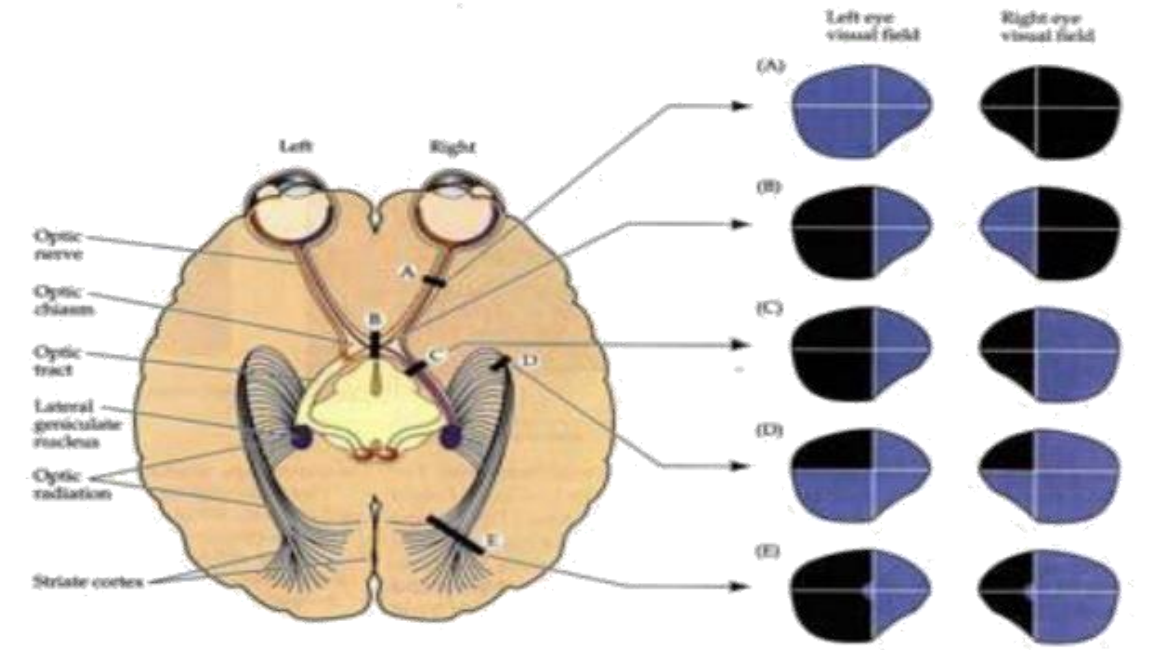
(ii) To chart the visual fields

VISUAL ACUITY

The standard Snellen's Type of charts are used for testing distant vision and the Jaegers types cards for near vision. The Snellen's type chart is placed, evenly illuminated, 6 meters (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 Jaegers 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, N24etc according to their size. The average acuity lies between N6 and N9.

VISUAL FIELD

Charting the visual field is the most important method of locating a lesion in the visual pathways or of interpreting certain fundus appearances, yet in clinical notes it is often difficult to find evidence that any attempt has been made to examine them at all. The purpose of the test is to chart the periphery of the visual fields; to determine the position, size and shape of the blind spot and any abnormal scotoma; 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.



Normal

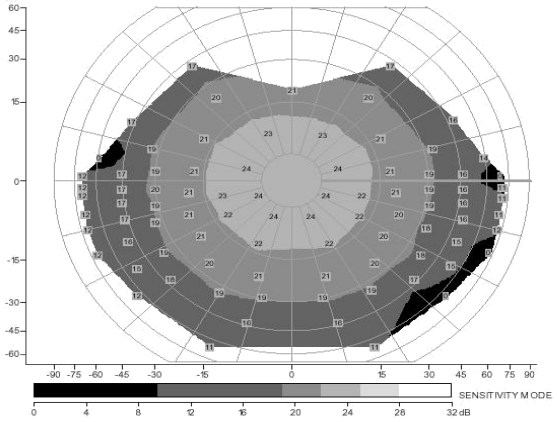
patient's ID : RARICHAN
file number : 366215
birth date : 20/05/1960

Rx :
exam. date : 20/07/2015
exam :

VISUAL FIELD EXAM

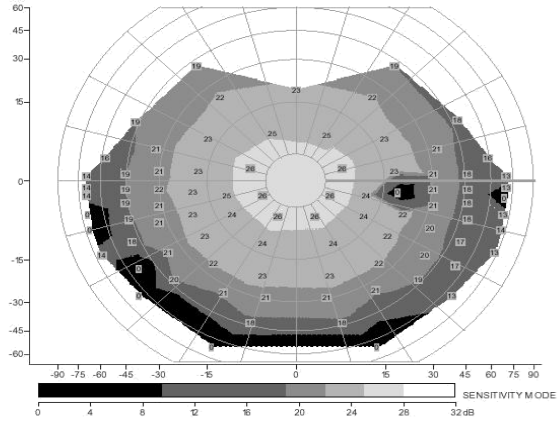
Incapacity index
LE stimulated

2D sensitivity map
MD=0.6dB CMD=0.6dB



Incapacity index
RE stimulated

2D sensitivity map
MD=-0.6dB CMD=1.4dB



BOTULINUM TOXIN INJECTION

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

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

Botulinum toxin types A and B are available and effective in treating focal or segmental dystonia's. Botulinum toxin has seven forms (serotypes): A, B, C, D, E, F, and G.

Botulinum toxin injections are not a cure, but they usually ease symptoms in the injected muscle in seven to 10 days. Patients usually receive maximum benefit one to two weeks after the injections. The effects can last several months, and the treatments can be repeated, often indefinitely.

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

Because symptoms vary during the course of dystonia, the treatment's effectiveness and duration vary from patient to patient. Subsequent injections may produce results that are less dramatic than the first, and doses may have to be adjusted. Identifying and injecting the affected muscle is not always easy.

In rare cases, a patient can develop antibodies to tl1e botulinum toxin proteins, rendering the treatment ineffective.

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

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 hemodynamic 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 hemodynamic 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

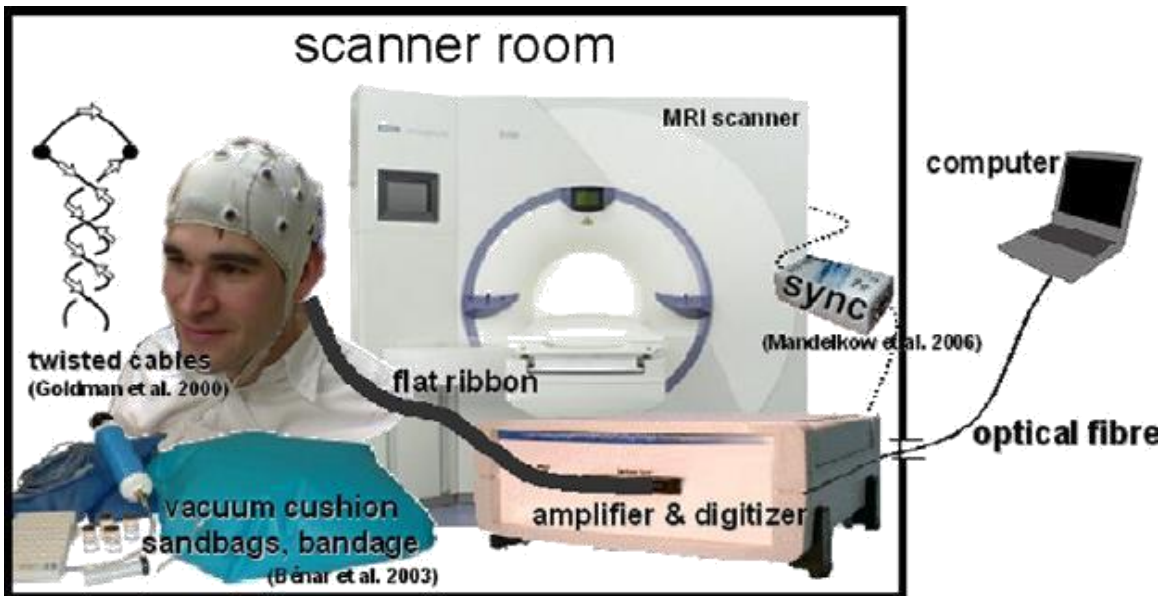
These are interleaved acquisitions, in which each acquisition modality is interrupted in turn (periodically) to allow data of adequate quality to be recorded by the other modality, continuous acquisitions, in which both modalities are able to record data of adequate quality continuously. The latter can be achieved using real-time or post processing 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 Geodesies, Inc.), but issues remain. There are significant residual artifacts in the EEG that occur with each heartbeat. The traces in the EEG that record this are often referred to as a, "Ballistocardiogram (BCG)," because of their presumed origin in the motion of the EEG leads in the magnetic field that occurs with each heartbeat. In practice, the causes of this artifact are not well proven and may be the results of factors such as induced electrical fields with the movement of blood, etc.

Applications

In principle, the technique combines the EEG's well documented ability to characterize 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 characterize 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 inter-ictal epileptiform discharges (lED, or inter-ictal 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 lED 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.



Technologists' role in patient safety

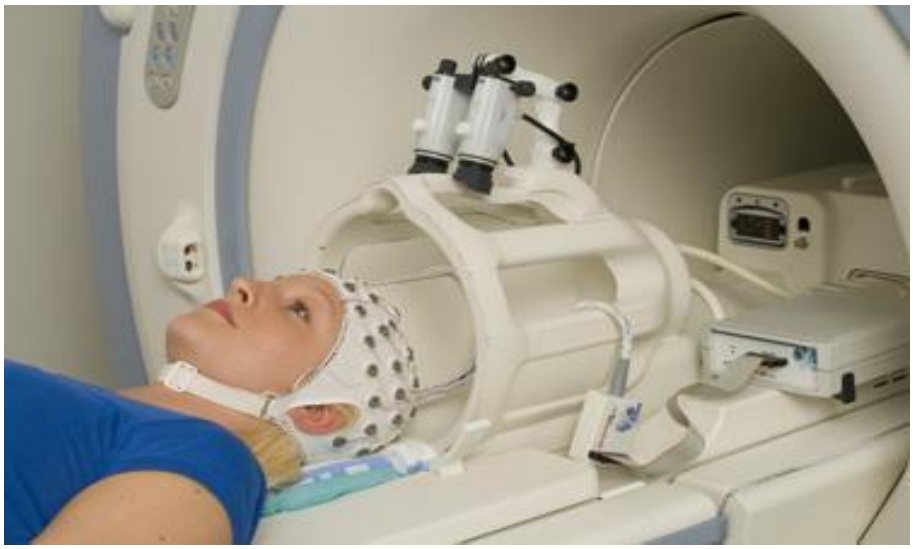
It is important to understand that there is a potential hazard during the performance of a electrophysiological procedure. The electricity can kill a patient if the equipment is not properly maintained or grounded and if adequate precaution are not taken.

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

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

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

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



LOG BOOK

Electroencephalography

Sl No	Name	Age/Sex	Hospital No	Diagnosis
1	Sheena	31/F	414893	CPS Temporal
2	Parnavi Jana	5/F	411871	Right hemispheric FCD
3	Aleena Asokan	8/F	414523	GDD
4	Anandan A	13/M	413200	PGE
5	Muhammed Ali	55/M	419421	JAE
6	Santhi B	36/F	419264	PNES
7	Akhila N	14/F	402982	Encephalitis
8	Kanak Kumar	23/M	403861	JME
9	Sanu Fasal K	12/M	418297	Symptomatic LRE
10	Liya biju	6/F	415235	Panayiotopoulos syndrome
11	Adithya Anil	6/F	411507	Symptomatic LGS
12	Sivani	2/F	406767	Dravet syndrome
13	Sreenand J D	4/M	403387	CPS ET
14	Abdulla kutty	46/M	396336	Left MTS
15	Treesa francis	17/F	413195	JAE
16	Shifa V K	13/F	415166	CPS-ET right frontal
17	Safarulla	62/M	413776	Single episode of LOC
18	Amith Krishna	5M	415509	Absence Seizure
19	Sreeraj Ramesh	14/M	415486	GDD
20	Mohammed Basith Ali	34/M	392886	PGE

Sl No	Name	Age/Sex	Hospital No	Diagnosis
21	Jadeja Kuldeep Singh	33/M	373068 R	Right posterior quadrant gliosis lesionectomy
22	Richard M Bijoy	10/M	415508	BOE
23	Ashitha D	13/F	397674	Probable PGE
24	Athira V G	20/F	402899	JME
25	Gopinathan Nair N	75/M	385016	PGE
26	Kirthikesh A	4/F	400649	Static encephalopathy
27	Biju R	32/M	402009	CPS-temporal
28	Bavisuma	1/F	385514	West syndrome
29	Arundhathi Devi	10/F	401041	BOE
30	Beethu Prince	25/F	402597	JME
31	Thankappan N	76/M	370388	Rule out NCSE
32	Vikraman nair	60/M	283913	Syncope V/S Seizure
33	Juel B J	2/F	402734	Febrile seizure
34	Anju B Nair	33/F	401484	JME
35	Aleyamma Mathew	66/F	413318	Left temporal gliosis
36	Anushka S Nair	1/F	401663	West syndrome
37	Navadeep P S	2/M	402498	Epileptic encephalopathy
38	Vineeth Menon	28/M	298625	JME
39	Shriya Mohan	2/F	413163	Typical Febrile Seizure
40	Rayan V S	1/M	414477	Hypoxic brain damage

Sl No	Name	Age/Sex	Hospital No	Diagnosis
41	Krishnendu	10/F	298079	GEFS+
42	Adithya Sagar	12/M	413368	BECTS
43	Sarabeegum	37/F	416155	Parasagital Meningioma
44	Sandeep M Lalni	18/M	268340	Hypothalamic hematoma
45	Jismi K S	18/F	387745	SGE
46	Sagar S Madhavan	18/M	397079	IGE
47	Mahila K S	56/F	399101	Non-Hodgkin lymphoma
48	Amal Krishna S	11/M	402131	Childhood LRE
49	Neel J	6/M	401926	Down's Syndrome
50	Virendra Kumar	35/M	383912	PNES
51	Vishnu	9/M	272385	West Syndrome
52	Muralidhar Tripathi	35/M	383909	JME
53	Nikhil V K	23/M	402020	PGE
54	Smitha Kumari	45/F	314319	FLE
55	Jordin Henry	2/M	374544	Dravet syndrome
56	Pranav S P	7/M	305863	PGE
57	Aravind G Nair	18/M	376479	JME
58	Rajani M S	36/F	401425	Absence seizure
59	Leyana Premson	4/F	400507	CAE
60	Lekshmi Devi	33/F	261994	TLE

Sl No	Name	Age/Sex	Hospital No	Diagnosis
61	Rabeesh Rahman	4/M	401630	SSPE
62	Murugeswari M	11/F	401624	Autoimmune encephalitis
63	Aneeja Alex	23/F	400674	JME
64	Jigar joshi	11/M	401501	SSPE
65	Ashik Mohammed	12/M	273985	BLRE
66	Reshma Rajeev	21/F	243798	TLE
67	Muhammed Shadil	16/M	400518	JME
68	Adarsh A B	11/M	398674	BRE
69	Smithi C	34/F	287845	JME
70	ManiKrishna	13/M	369320	LKS
71	Meenskshi V B	8/F	364084	CAE
72	Gangadharan	68/M	400936	FTD
73	Shubham Kesherwani	17/M	401632	Rasmussen's encephalitis
74	Rabiya Beevi	58/F	277286	Limbic encephalitis
75	Ramesh A	52/M	401212	CJD
76	Neha Bohan	16/F	336306	JAE
77	Mohammed Shifnas	1/M	401131	LRE
78	Roshan V Roy	10/M	308948	BLRE
79	Akhil V	23/M	400254	PGE vs FLE
80	David Raj M	17/M	400286	Static encephalopathy

Sl No	Name	Age/Sex	Hospital No	Diagnosis
81	Yeldos K S	8/M	401161	Static encephalopathy
82	Jullianne Nedumpara	35/F	400950	Stiff person syndrome
83	Bijoy R M	7/M	401427	Impulse control disorder
84	Vipin Jose V S	21/M	401291	CPS-ET v/s PGE
85	Fathima Liyana	4/F	351561	benign familial neonatal convulsion(BFNC)
86	Santhoshkumar v	42/M	398881	Right parietal Cavernoma
87	Ananya Ratheesh	2/F	400007	Mitochondrial encephalopathy
88	Padmini	68/F	400487	AVM
89	Nizamudeen B	28/M	421109	IGE
90	Azad Sunny	23/M	421107	Syncope
91	Shibi Chakravrthy	23/M	421035	Focal epilepsy
92	Renga Rajan	30/M	420998	OLE
93	Jacob Rajan	15/M	420997	JME
94	Mohnachandran	56/M	420994	Symptomatic LRE
95	Riya Rejin	6/F	420998	Probable BOE
96	Hareendranath	28/M	420953	SGE
97	Radhakrishnan nair	63/M	420889	CPS-ET
98	Mumthas Mariyam B	32/F	420669	PGE V/S SGE
99	Jofia Jose	33/F	420423	Left MTS
100	Vishnu T M	19/M	420469	PME V/S CPS ET

Nerve Conduction study
And Electromyography

SlNo	Name	Age/Sex	Hospital No	Diagnosis
1	Narayanan Potti	73/M	325646	C5-C6 Radiculopathy
2	Jake Joe Jinu	10/M	407611	Right Ulnar + median Neuropathy
3	Jaseed Shajahan	22/M	407233	Right Peroneal neuropathy
4	Ajayan G	51/M	292019	Rule out Myositis
5	Beenambika	49/F	406254	Meralgia paresthetica
6	Ansalna Saleem	52/F	401833	Right CTS
7	Mariya N	63/F	406264	Left Ulnar Palsy
8	George S	58/M	407362	GBS
9	Ibrahim Kutty Ravuthar	64/M	407300	Brachial Plexopathy
10	Ajitha A	12/F	407077	Leukodystrophy
11	Rajitha C	44/F	405106	MND
12	Sushama P	64/F	400973	Rule out Myopathy
13	Jalaludeen	68/M	403093	Diabetic Neuropathy
14	Bindhu	48/F	402878	L5-S1 Radiculopathy
15	Muraleedharan Nair G	56/M	402916	Right Radial Nerve Palsy
16	Ajojith	5/M	402916	GBS
17	Milan Antony Paul	21/M	403045	Brachial Plexus
18	Suseelamma J	62/F	402687	Mononeuritis Multiplex
19	Chandravalli Amma	70/F	402711	Large fiber Neuropathy
20	Muhammed Aslam	8/M	401604	Rule out Myopathy
21	Aji kumar	22/M	401763	Femoral Neuropathy

SlNo	Name	Age/Sex	Hospital No	Diagnosis
22	Sheeja	31/F	366599	Mitochondrial Myopathy
23	Sivasankaran pillai	55/M	245135	Right common Peroneal Neuropathy
24	Anil	39/M	401478	HMSN
25	Shajida Asharaf	36/F	402086	Young Onset ALS
26	Musthaq Rifas	18/M	401645	Right Erbs Palsy
27	Ramachandran	46/M	402357	Probable PLS
28	Selikath	60/F	401815	Bulbar onset ALS
29	Narayana Kurup	80/M	401817	L5-S1 Radiculopathy
30	Akash P	19/M	398589	Hirayama Disease
31	Ali K	61/M	401706	Left peroneal neuropathy
32	Thampi Thomas	55/M	401717	Peripheral Neuropathy
33	Thankamma	82/F	400416	C5-C6 Radiculopathy
34	Joy kutty V	54/M	385402	Mono neuritis Multiplex
35	Mariyamma Joseph	72/F	400625	Bulbar Onset MND
36	George	58/M	407362	AMAN V/S AIDP
37	Thankappan	62/M	406552	CIDP
38	Fathima Nabila	25/F	407532	LGMD
39	Rajeev	38/M	407329	Polyneuropathy
40	Bindhulekha	42/F	407813	Brachial Plexus
41	Raveendran S	66/M	406383	Diabetic Neuropathy
42	Sreekanth J	46/M	336543	Right C6 Radiculopathy

SlNo	Name	Age/Sex	Hospital No	Diagnosis
43	Sanoop M S	25/M	406911	Myotonic Dystrophy
44	Biju SS	40/M	406176	S1 Radiculopathy
45	Saji Mathew	58/M	408207	MND
46	Sasidharan	66/M	407282	Left C6-C7 Radiculopathy
47	Vijayan	56/M	390819	Bilateral Foot Drop with PIVD
48	Vijayakuamar P	39/M	408535	Left Radial nerve Palsy
49	Balakrishnan Pillai	76/M	408612	Lumbosacral Radiculopathy
50	Tomy Mathew	45/M	378107	Large fiber Neuropathy
51	Jasmine Baiju	38/F	388058	Autonomic Neuropathy
52	Alex P K	79/M	388572	Diabetic Neuropathy
53	Manoharan G	49/M	405987	Bilateral Radiculopathy
54	Jalaja Kumari s	45/F	274503	Left CTS
55	Sulaiman Kunj	68/M	349751	Right Ulanr neuropathy
56	Ambika N	51/F	374141	Upper trunk Plexopathy
57	Geetha P	49/F	371382	To Rule out Cervical Radiculopathy

SlNo	Name	Age/Sex	Hospital No	Diagnosis
58	Shanid C	18/M	411284	Hirayama Disease
59	Adarsh Mathew Vaidyan	16/M	413453	AIDP
60	Lini R	46/F	413251	L5-S1 Radiculopathy
61	Vimal Das	29/M	412704	Left Sural neuropathy
62	Rema devi	63/F	240686	PLS v/s ALS
63	Vijyalekshmi Kunjamma	62/F	395832	Cryptogenic peripheral neuropathy
64	Arunkumar	7/M	413397	Becker's Muscular Dystrophy
65	Sangeetha U	29/F	413491	HMSN
66	Rajendran Nair P	64/M	412786	Bilateral Meralgia Paresthesia
67	Rajendran	65/M	401398	Left Brachial plexopathy
68	Anoop Kumar Saha	59/M`	414066	Cervical or Lumbar Radiculopathy
69	Mercy	46/F	413387	SCA
70	Balakrishna Pillai P R	71/M	414154	CIDP V/S IBM
71	Shiju Surendran	36/M	414149	CPEO
72	Dawn Joseph	7/M	414109	Right Elbow sarcoma recurrence
73	Sumathy Kutty Amma	70/F	405348	L4-L5 Radiculopathy
74	Anasuya S J	8/F	413461	Leigh Disease

SlNo	Name	Age/Sex	Hospital No	Diagnosis
75	Babu TM	51/M	9802595	Myeloneuropathy
76	James Jerald	46/M	414544	L1-L2 Intradural Meudallary lesion
77	Yousuf Kunju	64/M	413806	ALS
78	Joykutty P S	58/M	414866	CIDP
79	Suppiya	42/M	413894	AIDP
80	Santha Sivakumar	55/F	9302696	Brachial Plexopathy
81	Ajitha L	39/F	417147	L5-S1 PIVD
82	Maria Nesam	75/F	417982	Bulbar onset MND
83	Kalayana Krishnan	66/M	415780	Right ulnar Neuropathy
84	Muhammed Kunju	50/M	381391	C5-C6 Radiculopathy
86	Jeena Jhon	22/F	418129	Progressive Myoclonic Ataxia
87	Vishnu C	26/M	256815	Recurrent GBS
88	Aswanth B	2/M	419397	CMT
89	Sulfath Abdulkader	16/F	417512	Small Fiber Neuropathy
89	Arjunan N	73/M	372641	Diabetic Neuropathy
90	Sukumaran Nair T	83/M	310796	Right ulnar neuropathy v/s C8-T1 Radiculopathy

SlNo	Name	Age/Sex	Hospital No	Diagnosis
91	Symala Devi B	57/F	17662	Left CTS V/S Radiculopathy
92	Jayachandran	49/M	219101	C7-C8 Radiculopathy
93	Vishnu K	16/M	419207	SCA
94	Mohanan K	65/M	419726	Deep Peroneal Nerve Palsy
95	Beena Kumari S	62/F	420213	Right
96	Suma G S	25/F	420213	HSP,Peripheral Neuropathy
97	Radha B	53/F	420410	AHC Disease
98	Sivan Chettiyar	57/M	419492	Right Lumbosacral Radiculopathy
99	Babu B	27/M	418123	Focal AHC V/S Upper Trunk Brachial Plexopthy
100	Vasudevan Pilai	57/M	301969	Bulbar Onset MND

Video EEG

SlNo	Name	Age/Sex	Hospital No	Diagnosis
1	Yashasvi S	27/F	414986	FLE V/S PGE GTCS
2	Dafne M	03m/F	415003	Cps ET(Bilateral PHR gliosis)
3	Samar Prathap S P	3/M	356760	Symptomatic LGS
4	Sree Sailesh V B	16/M	412649	CPS ET / frontal v/s PNES
5	Saketh Vidydhara	9/M	353890	Primary Startle Epilepsy
6	Manukrishna M M	15/M	391686	IPOE V/S PGE
7	Hardik H Soni	16/M	399687	Static Encephalopathy
8	Lakshitha S	9/F	413239	Benign Myoclonic Epilepsy
9	Tulsi Davi Veshnav	51/F	414334	CPS Temporal Plus
10	Devi Prasad	3/F	414274	Dravet Syndrome
11.	Nency Kishor Bhai Patel	17/F	414329	JME
12	Surya R	6/M	400339	West –LGS
13	Soumyadeep Ray	7/M	397110	Atypical BLRE
14	Swati Masukhbhai Desai	29/F	396352	CPS temporal
15	Happy Bansal	13/M	405446	IGE with Eyelid Myoclonus
16	Samanwita Maji	8/F	414839	CPS Temporal,Right MTS
17	Sarita Jain	36/M	387929	Right MTS
18	Sri Lavanya	12/M	387945	BOE
19	Kalpesh B Wala	24/M	384678	FLE
20	Atul Vijayakumar	38/M	387870	Left orbito frontal FCD

SlNo	Name	Age/Sex	Hospital No	Diagnosis
21	Nemani Aarosh	2/M	388128	? Panayiotopoulos syndrome
22	Aswin S	5/M	388375	Atonic BRE
23	Girish K	40/M	184672	CPS Temporal Plus
24	Pitla Renuka	17/F	388448	Left Frontal FCD
25	Nikhy Anna	23/F	387894	JME
26	Pratik Prakash	15/M	388442	B/L PHR Gliosis
27	Rafkhana H	8/F	388509	LGS
28	Ajit Jain	5/M	388617	Absence Seizure
29	Manit Singh	7/M	387721	BLRE vs FLE
30	Sreejith S V	15/M	388219	PNES
31	Arush Raj	1/M	388093	DNET
32	Ishan Ahirwal	8/M	399460	Post encephalitis Sequele
33	Samar Pratap	3/M	356760	B/L parieto- occipital gliosis
34	Muhammed Mahadi	1/M	389963	CPS-Temporal
35	Madhav Kumar	5/M	389523	Tassinari syndrome
36	Jefrey Jasper	4/M	352124	LGS
37	Raja M D	34/M	389976	CPS-ET
38	Anurag Betal	9/M	260395	BLRE
39	Sindhuja S	24/F	389951	CPS-ET
40	Abhiram P S	7/M	389704	Left parieto – occipital FCD
41	Syed Mahibul	9/M	372505	Neck myoclonus

SlNo	Name	Age/Sex	Hospital No	Diagnosis
42	Priya R S	20/F	388263	PNES
43	Maimoona Zaheer	16/F	389525	CPS-ET PHR
44	Jessamuel Sebastine	4/M	389519	Epileptic encephalopathy
45	Rihan P P	7/M	389326	Startle epilepsy
46	Savio M A	13/M	411060	CPS ET Right FCD
47	Divya R	22/F	379786	Left MTS neocortical
48	Daksh Kulshreestha	4/M	372703	Static encephalopathy
49	Shamood Abdallah	11/M	366284	Left frontal FCD
50	Thameem Ansari	34/M	380559	Right MTS
51	Rakesh A	10/M	366706	CPS Temporal V/S Pseudo temporal
52	Prdeep Viswamber	30/M	344764	PGE
53	Jaiswal S S	41/M	380935	SGE vs PGE
54	Kajal Patra	18/F	380943	PNES
55	Niraj Jain	18/M	381076	Right hemispheric gliosis
56	Balraj R	15/M	354473	Right parieto occipital lesion
57	Rawiya Said	16/M	381282	PME
58	Shashank Kethan	14/M	330828	Familial TLE
59	Jaganathan	11/M	381327	CPS-ET Frontal
60	Sivasankar Paul	16/M	268032	Epileptic encephalopathy
61	Amijith Ramesh	14/M	388631	Static encephalopathy
62	Naksh Hareshbhai	2/M	390364	GDD
63	Sweta G Nair	7/F	380122	BOE

SlNo	Name	Age/Sex	Hospital No	Diagnosis
64	Rishan Mohammed	1/M	381633	LRE
65	Debjit Moitra	14/M	363401	FLE
66	Prity Tripathy	17/F	381664	PNES
67	Sudha Shiju	30/F	350132	FLE
68	Rahul Kaurav	18/M	381611	CPS-ET Right frontal
70	Ahammed Navas	5/M	382635	TLE
71	Adwaith Hariprasad	5/M	381273	B/L parieto – occipital gliosis
72	Sreerag P	6/M	381803	Right temporal DNET
73	Hiranmaya Lal	4/M	316690	LGS
74	Anju Soye	15/F	296373	Right MTLE
75	Akshay N Borkar	17/M	390712	PME
76	Nirmal S	13/M	381163	Pseudo temporal vs temporal
77	Bhasurangi K`	56/F	370287	Left MTS
78	Karthik M	22/M	381911	IGE
79	Jay Rajunayyer	15/M	381901	Reflex eating epilepsy
80	Preeti C J	34/F	381971	CPS ET
81	Ayush Mahale	14/M	381901	B/L PHR gliosis
82	Subhash G	38/M	381526	FLE
83	Jagruti Mohanty	6/F	381969	BOE
84	Arjun S R	1/M	381981	Benign non epileptic myoclonus
85	Revathi S	11/F	382148	Right parietal gliosis

SlNo	Name	Age/Sex	Hospital No	Diagnosis
86	Vishal Anand	37/M	376867	Left MTS
87	Sarang Krishna	25/M	381605	PGE
88	Yash Pratap Singh	16/M	390972	PGE
89	Deepinder Singh	3/M	391061	Symptomatic LRE
90	Krish Harshbhai Vekariya	8/M	391066	CPS-Temporal
91	Harchita Sharma	20/F	420152	Post Status Epilepticus Sequlae
92	Uditi Saraf	13/F	405125	CAE
93	Komalavally	47/F	408251	CPS Temporal PlusRightMTS
94	Devika M S	18/F	421151	FLE v/s GEFS plus
95	Indhu Thakur	27/F	419557	CPS Temporal
96	Sonia Prasad	34/F	421299	CPS Temporal V/S PNES
97	Abhijith S	16/M	421429	FLE
98	Vikram Sharma	10month/M	419756	Symptomatic West
99	Rhea Shergill	22/F	412541	Right Insular FCD
100	Niyas P A	36/M	406899	CPS temporal Neocortical

Visual Acuity and Visual Field charting (OPTOMETRY)

Sl No	Name	Age/Sex	Hospital No	Diagnosis
1	Devi Krishna	23/F	385602	ADEM
2	Sethulekshmi S	17/F	242901	Pituitary adenoma
3	Ammini Kunjikutty	65/F	370368	Left vestibular schwannoma
4	Sreelekha C S	26/F	378082	Meningioma
5	Sreekumar N V	45/M	323057	Temporal lesion
6	Aswathy L	24/F	323671	NMO Spectrum disorder
7	Rajan K R	47/M	251388	Pituitary adenoma
8	Sreelatha S	44/F	363224	IIH
9	Mini George	39/F	363269	Bifrontal meningioma
10	Sarojini O V	59/F	363818	Pituitary adenoma
11	Sheeba S	43/F	363848	Optic neuritis
12	Sreedevi T	43/F	363887	Pituitary adenoma
13	Manivarnnan T	52/M	359755	Pituitary tumor
14	Sivan pillai C	66/M	363637	Anterior ischemic neuropathy
15	Paul Varghese	50/M	359820	Visual blurring
16	Anilkumar G	39/M	363935	Pituitary adenoma
17	Appu M	75/M	363996	Pituitary adenoma
18	Jyothi S	22/F	208896	RRMS
19	Josna Jose	16/F	354794	Chiasmatic glioma
20	Praveena T N	31/F	361754	Left optic neuritis

Sl No	Name	Age/Sex	Hospital No	Diagnosis
21	Shakunthala V	45/F	389172	Tubeculous sellar meningioma
22	Manesh M	30/M	389236	Pituitary adenoma
23	Leelamani Amma	57/F	389260	Occipital lesion
24	Shibin Y	18/M	266921	Cranio pharyngioma
25	Kailasanathan A	31/M	389414	Pituitary adenoma
26	Vinod Kumar T	39/M	190413	Operated case of Pituitary adenoma
27	Shaji S	43/M	329671	B/L Papilledema
28	Bin P Jeorge	12/M	353482	Right optic neuritis
29	Narthana B Suresh	16/F	379815	Idiopathic intracranial hypertension
30	Sneha S	11/F	379895	IIH
31	Abdul Khader	52/M	374835	Pituitary cist
32	Malini Kumar	32/F	242865	Recurrent lesion
33	Viswanathan P	60/M	373381	Trigeminal schwanoma
34	Preetha S	35/F	369528	Blurring of Vision
35	Sulaiman P	51/M	379168	Diplopia
36	Meera Murali	29/F	357762	? Demyelination
37	Rajakimari A	63/F	378342	Left cavernoma
38	Aneesh R	27/M	380049	Vascular head ache
39	Anil Kumar M	38/M	379510	Right insular glioma
40	Geetha S	28/F	380095	CVT

Sl No	Name	Age/Sex	Hospital No	Diagnosis
41	Raja S R	34/F	301931	IIH
42	Udya kumar P	38/M	290599	Case of HSP
43	Meera Thomas	18/F	379893	Craniopharyngioma
44	Praleema	42/F	296962	Pituitary marcoadenoma
45	Anila Kumari	44/F	286761	Headache
46	Visha Suresh	52/F	380291	IIH
47	Roshini Jayan	19/F	292125	Recurrent craniopharingioma
48	Subbaraman	55/M	356712	Pituitary marcoadenoma
49	Shibina L	21/F	380413	IIH
50	Shyny Baby	34/F	380137	Meningioma

Repetitive Nerve Stimulation

Sl No	Name	Age/Sex	Hospital No	Diagnosis
1	Mohammed Kasim	68/M	404397	Myasthenia Gravis
2	Surendran G	69/M	376391	Isolated third nerve palsy
3	Asok Kumar	56/M	377890	Myasthenia gravis
4	Sulochana D	50/F	376381	Ocular myasthenia
5	Chandra Kumar	47/M	342098	GBS
6	Mohammed Yaseen K S	9/M	414246	CPEO, To Rule out Myopathy
7	Padma Sankar	46/M	8702612	Myasthenia gravis
8	Khadeeja K P	77/F	408187	Myasthenia gravis
9	Manuel Mohan	60/M	378988	Rapidly progressive myopthy
10	Ansalin A R	16/F	367545	Myotonia congenita
11	Adhvan L S	02/M	370117	Occular Myasthenia
12	Liya Rani	29/F	380802	Bulbar and ocular weakness
13	Vipin S	25/M	381174	Case of Myasthenia
14	Laila P S	57/F	401980	Stiff Persons Syndrome
15	Al Qualiq N	10/M	380553	Ocular myasthenia
16	Mini Jude	41/F	420405	Generalized Myasthenia
17	Jayachandran P R	60/M	380343	Ocular Myasthenia
18	Gadha	26/F	408291	Myasthenia gravis
18	Muthukrishnan	70/M	382626	Myasthenia gravis
19	Akhil P	21/M	381406	Ocular myasthenia

Sl No	Name	Age/Sex	Hospital No	Diagnosis
20	Sai prabha	22/F	382107	Ocular myasthenia
21	Sindhu B	42/F	407618	Myasthenia gravis
22	Thrisha Jyaprakash	10/F	381321	Myasthenic syndrome
23	Abraham Y Babu	59/M	381184	Ocular myasthenia
24	Abdul Lathef	78/M	8707166	Bilateral Ptosis
25	Prasanna S	51/F	383630	Double vision
26	Santha Kumari B	60/F	407488	Occular myasthenia
27	Sruthi S	26/F	384245	Myasthenia gravis
28	Rahul S	12/M	407895	Congenital myopathy
29	Samson P	67/M	382849	Occular myasthenia
30	Antony Y	35/M	414119	Myasthenia gravis

Blink Reflex

Sl No	Name	Age/Sex	Hospital No	Diagnosis
1	Lekshmi Vishnu	32/F	417647	V th nerve Neuropathy
2	Yeshwini	39/F	374464	Progressive increase in size of pupil
3	Sosanna G R	59/F	380894	Right facial palsy
4	Sukumaran C	66/M	412003	Right Bells Palsy
5	Yasodha Amma S	58/F	383326	Trigeminal nerve palsy
6	Soumya P s	32/f	383296	Left trigeminal neuropathy
7	Usha U	55/F	394368	Left LMN VII th Nerve palsy
8	Viswnathan Nair	74/M	408669	Bells Palsy
9	Sajitha K R	55/F	418727	Left UMN Facial Palsy
10	Mohanan	57/M	390825	Left LMN Bell's palsy

SFEMG

SlNo	Patient Name	Age/Sex	Hospital No	Dianosis
1	Aswathy P M	19/M	413870	Right Eye Ptosis
2	Sreebha S	42/M	411346	Myasthenia gravis
3	Habeeba B	60/F	412352	Myasthenia gravis
4	Helen	47 /F	400200	To rule out Myasthenia gravis
5	Thejaswini	13/F	419724	Myasthenia gravis
6	Kavya Sankar	13/F	412447	Myasthenia gravis
7	Vishnu Priya	21/F	419999	To rule out Myasthenia
8	Laiju C Raj	46/F	332973	Ocular Myasthenia
9	Ramzan Deen Beevi	28/F	303409	Myasthenia Gravis
10	Shibi J L	31/F	414805	Myasthenia Gravis

Visual Evoked Potential

Sl No	Name	Age/Sex	Hospital No	Diagnosis
1	Pandia raj	60/M	408654	Left MCA Stroke
2	Priyanka	22/F	394889	RRMS
3	Eldho Mathew	15/M	410107	leukodystrophy
4	Arul Kumar	19/M	411178	PME
5	Nazeem A M	44/M	9009632	Dystonia
6	Jayasree	42/F	410905	Dural Arteriovenous Fistula (DAVF)
7	Akul Syam	3/M	411063	Right Acute onset esotropia
8	Ajith S	18/M	411553	Post viral optic neuritis
9	Satheesan S	47/M	405626	Neuromyelitis optica (NMO)
10	Bijumon T	36/M	410973	NMO disorder
11	Sangeetha P	32/F	397042	**Pituitary adenomas**
12	Prabhakaran Pillai	58/M	411834	Bilateral Optic Atrophy
13	Manjusha	17/F	412405	Multiple sclerosis (MS)
14	Mercy A	46/F	413384	SCA
15	Binu John	38/M	415093	Long Segment Myelitis
16	Suma Devi K	48/F	415090	LETM
17	Ambily A	43/F	414882	Right MCA Stroke /? Demyelination
18	Madaswamy M	52/M	417098	Right CP angle lesion
19	Sijimol L	35/F	420890	IIH
20	Amanulla S	22/M	421989	Marfan syndrome

Brainstem Auditory Evoked Potential

SlNo	Name	Age/Sex	Hospital No	Diagnosis
1	Santhi G	55/F	404567	Right Sensorineural hearing loss (SNHL)
2	Don Vincent	3/M	403687	Cerebellar Ataxia
3	Rani Jhon	35/F	408964	SPMS
4	Afsar A	3/M	411074	PRES v/s post anoxic sequlae
5	Vishesh Jain	10/M	400003	HIE
6	Shebas Salim	15/M	421955	ADEM
7	Sindhu	40/F	419219	Multiple Sclerosis
8	Rhythm Meena	1/F	419024	Mitochondrial Disorder
9	Sajith M	5/F	418262	SMA II
10	Ardra A B	0.5/F	417568	HIE
11	Aryan Arun	2/M	418179	ADEM
12	Gaurav Rajput	2/M	417958	Static encephalopathy
13	Krishna Sunder	38/F	415905	Demyelination
14	Asmina R	14/F	416456	SSPE
15	Mariyam L	46/F	415778	PPMS
16	Gautham	4/M	411497	Autism
17	Arul Kumar	19/M	411178	PME
18	Karthik S	3/M	411074	Mitochondrial ataxia
19	Neethu S	15/F	411058	Secondary dystonia
20	Madaswamy	52/M	417098	Right CP angle lesion

Somato Sensory Evoked Potential

SlNo	Name	Age/Sex	Hospital No	Diagnosis
1	Abin B	14/M`	405553	SSPE
2	Vishnu Dutt	14/M	378829	Cerebellar Ataxia
3	Muhammed Abdullla	22/M	395486	SCA
4	Prem Kumar	22/M	391341	Multifocal Myoclonus
5	Krishna M S	23/F	385352	ADEM
6	Shyja	26/F	399849	Myoclonic Epilepsy
7	Burzan k	35/M	414649	CNS demyelination
8	Manjusha G	17/F	412405	PME
9	Madubala	12/F	419029	Focal Cortical Myoclonus
10	Malavika M	24/F	416252	DAVF
11	Shahina	19/F	416719	Primary V/S Secondary Demyelination
12	Anvar S	37/M	414430	Multiple Sclerosis
13	Sulekha S	32/F	406992	NMo
14	Abhiram Raja	28/M	409242	Myelitis
15	Sabu D	47/M	394708	Large fiber mononeuropathy
16	Anju benny	20/F	390352	RRMS
17	Bindhu B L	38/F	388755	Young Onset Dementia
18	Asiya Beevi	42/F	406552	Focal Cervical AHC disease
19	Sandhya V komath	51/F	410225	Demyelination
20	Manoj B M	32/M	414535	Left Optic neuritis

Electrocorticography

Sl NO	Patient Name	Age/Sex	Hospital No	Procedure
1	Sushil Oli	20/M	382424	Left Temporal Grid placement (Left MTS) For Invasive Monitoring
2	Maharoofa P C	22/F	372440	Right Occipital Gliosis
3	Muhammed Nihal	19/M	376707	Right Parieto Occipital Lesionactomy
4	Akhil Cherain	24/M	376460	Bilateral Depth and Grid Electrode Placement For Invasive Monitoring
5	Jismy K B	23/F	386521	Left ATL+ AH
6	Rajgor Mahesh Kumar	31/M	377784	Right ATL+AH
7	Ram Shukla	11/M	391021	Right occipital craniotomy and lesionactomy
8	Savitha K	31/F	308687	Grid electrode Placement on frontal,
9	Rohith K p	13/M	350777	Right Parieto Occipital Lesionactomy, For Invasive Monitoring
10	Nishant Pramod Kirange	15/M	406865	Right Frontal Craniotomy and decompression
11	Kripeena K	21/F	376840	Bilateral Depth Electrode Placement for invasive Monitoring
12	Dikshit M K	12/M	398843	Left Parieto Occipital lobectomy
13	Pragya Kedia	21/F	397081	Right Temporal craniotomy and lesionactomy
14	Daiwik Babu	4/M	390262	Left Posterior quadrantectomy
15	Anees babu	22/M	410063	Left ATL+AH
16	Akash dave	32/M	376672	Right ATL+AH
17	Divya Darshini	10/F	382625	Right ATL+AH +Lesionactomy
18	Sreenanda	11/F	402426	Right occipital craniotomy and lesionactomy
19	Tista Manna	12/F	395512	Right ATL+AH
20	Bivin Scaria Ninan	28/M	355845	Grid Electrode placement on temporal, For Invasive monitoring.

Polysomnography

Sl.No	Name	Age/Sex	Hospital No	Diagnosis
1	Dr.Ram Krishnan	62/M	417323	HTN,?OSA
2	Elizebath Stephan	53/F	414058	Rule out Co existent OSA
3	Praveeen N B	37/M	369994	Papillary Ca Thyroid
4	Pradeep A	39/M	387732	OSA
5	Lal J L	20/M	384950	OSA
6	Lissy Jacob	47/F	387542	EDS
7	Reghunatha Panicker	77/M	417542	T2DM,HTN,CKD
8	Lissa mol Jijo	10/F	415823	Narcolepsy with Cataplexy
9	Mathew P Rajan	52/M	390510	OSA
10	Habeepbulla M	59/M	391145	OSA
11	John Joseph	54/M	397277	EDS
12	Unnikrishnan V	39/M	395881	OSA
13	Anamika Sabu	10/F	391717	Narcolepsy
14	Salini S	40/F	397982	OSA
15	Sreelatha J V	41/F	397278	OSA
16	Vinod C R	34/M	417870	OSA
17	Prasanth S	41/M	397280	OSA and EDS
18	Naseer Y	43/M	391459	Severe OSA
19	Santha Menon	61/M	421370	EDS with snoring
20	Shaijan A	44/M	398832	OSA

Continuous Positive Airway Pressure

Sl.No	Name	Age/Sex	Hospital No	Diagnosis
1	Ramachandran Nair	67/M	397280	OSA
2	Mohandas	62/M	391214	OSA
3	Habeepbulla	59/M	391195	OSA
4	Sasidharan B	73/M	384407	OSA
5	Sameer Mustahafa	43/M	9804254	Severe OSA
6	Binod Hediya	53/M	394777	OSA
7	Shaji M	37/M	391483	OSA and snoring
8	Shaji Kumar S	47/M	384623	EDS and OSA
9	Jayachandran Pillai	48/M	392224	Snoring
10	Gopalakrishnan	49/M	389140	OSA

Multiple Sleep Latency Test

Sl.No	Name	Age/Sex	Hospital No	Diagnosis
1	Nisamol	23/F	412059	EDS
2	Saj Murali	41/M	392156	Idiopathic hypersomnia
3	Kalesh P S	31/M	392501	Narcolepsy
4	Adith Jayaprasad	16/M	400622	Narcolepsy and cataplexy
5	Lissy jacob	47/F	387542	Narcolepsy
6	Anandu K R	7/M	381328	Hypersomnolence
7	Keerthi Devan	19/F	379775	Idiopathic hypersomnolence
8	Allen Sam Biju	14/M	416704	?Narcolepsy
9	Venugopalan K S	57/M	373393	EDS
10	Prahaladan	59/M	366541	Periodic hypersomnolence