

EEG -basics to bedside clinics



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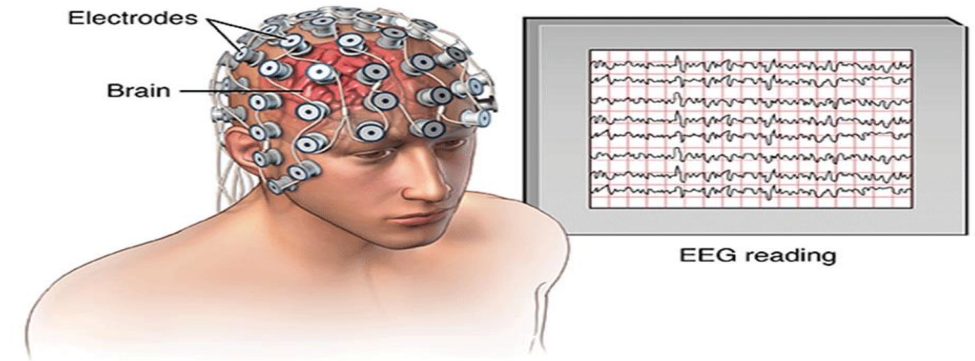
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Schema of presentation:

- What is EEG?
- What generates EEG potentials?
- EEG Vs ECG : Differences
- For which patients it is done ?
- Ideally When to do EEG after first unprovoked seizure ?
- EEG setup
- Where are the electrodes placed?
- Different montages
- Prerequisites for EEG : Patient preparation

- **Normal EEG wave forms**
- **Epileptiform abnormalities**
- **Wave forms during sleep**
- **Neonatal EEG**
- **Activation procedures**
- **Benign epileptiform variants**
- **Artifacts**
- **Complications**
- **Contraindications**
- **Limitations of scalp EEG**
- **Various types of EEG**

What is EEG????????



- Electro encephalogram : is **recording of electrical activity of the brain** with the help of electrodes placed over the scalp.
- First recordings were made by **Hans Berger**, German neuropsychiatrist, in 1924.
- In India, first EEG was performed by **Professor Baldev Singh**, at Christian Medical College, Vellore.

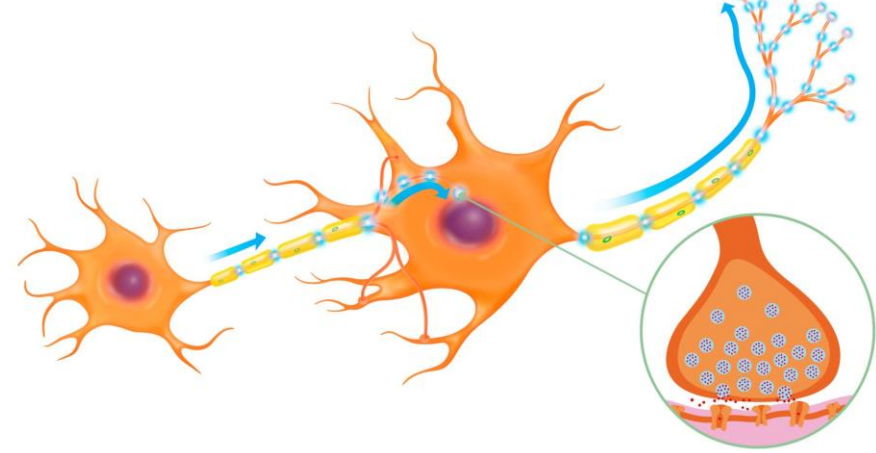


Figure 1.3: Baldev Singh (1904-1998)

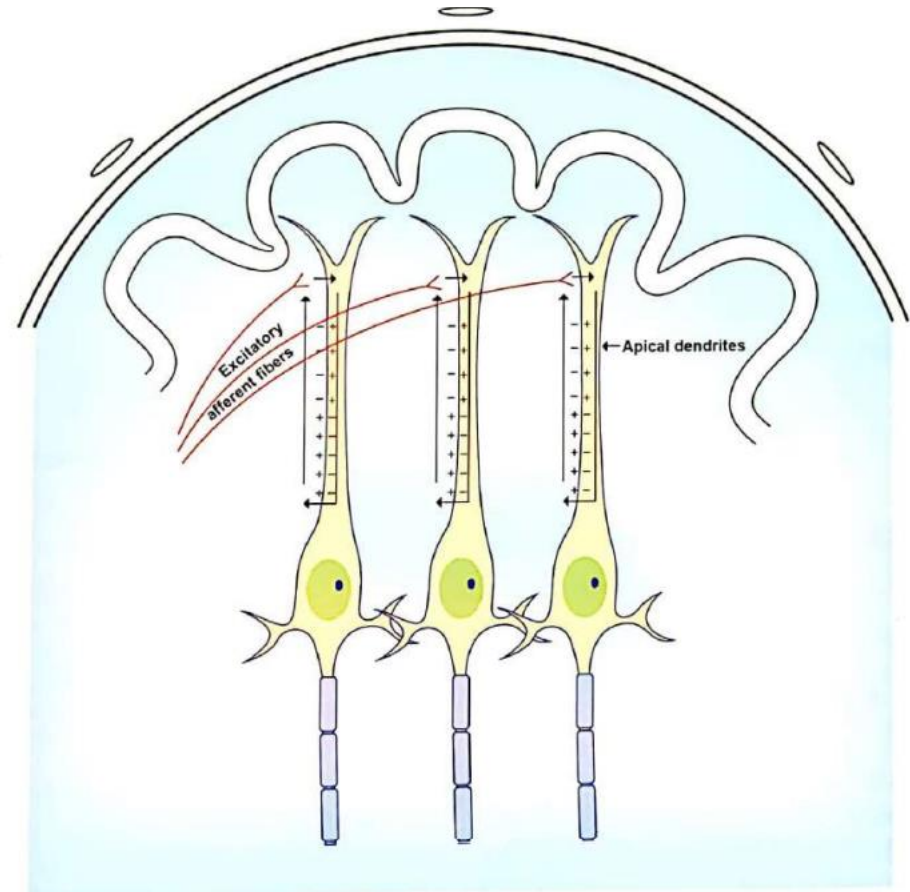


Figure 1.2: Hans Berger (1873-1941)

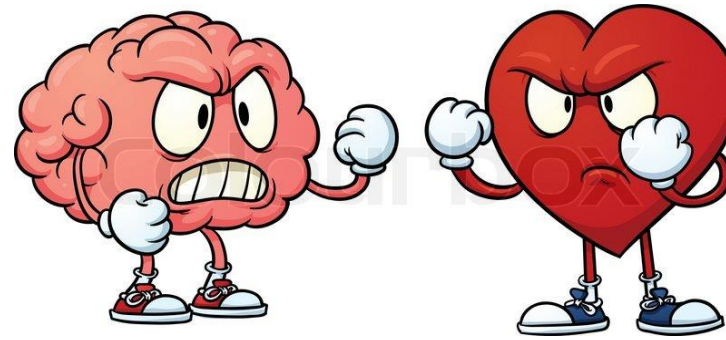
What generates EEG potentials?



- **action potentials** are responsible for the generation of EEG waves.
- Each pyramidal neuron consists of a **cell body, axon, a single large apical dendrite and multiple basal dendrites**.
- **Electrical signals** from one cell travel through its axon to the dendrite of the next cell in the form of action potentials.



EEG Vs ECG :



1) Electrodes are attached to the *scalp*, in *EEG*.

2) EEG is used for diagnosing *seizure disorders, infections, tumors, degenerative disorders and metabolic disturbances affecting the brain.*

3) EEG comes with *certain adverse effects*

4) *“EEG is not done routinely unless there is a clear indication (already clinically diagnosed by neurologist),*

“ Epileptiform abnormalities are found in 1% of normal subjects also ,so WE WILL TREAT SUBJECT , NOT EEG.”

- Attached to the *chest, legs, arms and neck*, in *ECG*.

- ECG determines rate of heartbeats, heart chamber positions and if there is any damage to heart.

- ECG involves no risks or pain

- *ECG can be done for any person routinely.”*

For which patients it is done???????????

- *For every patient with seizure????????????????*



Abnormal jerky movements



Loss of consciousness



Frothing from the mouth



Involuntary urination

case 1:

- 49 year old male, known diabetic , hypertensive was brought to emergency in altered sensorium with family members witnessing 1 episode of seizure with tonic, clonic movements of upper limbs, lower limbs with tongue bite and frothing from mouth. On examination, his GCS was E2V2M4 ,with cool extremities BP: 150/90 mm Hg , PR= 98/min, CBG=40 mg/dl , sensorium improved to E4V5M6 with emergency management.
- MRI brain : no abnormality
- *EEG advised for this patient or not ????????*

Case 1

- 49 year old male, known diabetic , hypertensive was brought to emergency in altered sensorium with family members witnessing 1 episode of seizure with tonic, clonic movements of upper limbs , lower limbs with tongue bite and frothing from mouth. On examination, his GCS was E2V2M4 ,with cool extremities BP: 150/90 mm Hg , PR= 98/min, CBG=40 mg/dl , sensorium improved to E4V5M6 with emergency management.
- MRI brain : no abnormality
- ***EEG advised for this patient : no***
- ***Here the seizure is acute symptomatic : due to hypoglycemia; which is reversible and transient .***
- ***Treatment with Antiepileptics also not needed.***

Case 2:

- 24 old male with history of 1 episode of GTCS .On examination, BP: 130/90 mm Hg, PR= 98/min, CBG=130 mg/dl.Neurological examination : normal. Developmental & perinatal history: normal,no childhood seizures, no family history of seizures.
- MRI brain : normal
- *EEG advised for this patient or not ????????*

Case 2.....

- 24 old male with history of 1 episode of GTCS .On examination, BP: 130/90 mm Hg, PR= 98/min, CBG=130 mg/dl. Neurological examination : normal. Developmental & perinatal history: normal , no childhood seizures, no family history of seizures.
- MRI brain : normal
- ***EEG advised for this patient or not : yes***

Done, to decide ,whether to start antiepileptics or not

- If EEG : abnormal: start him on antiepileptics
- If EEG : normal : wait and watch, reassure.

Case 3:

- 42 year old male, daily alcoholic, stopped alcohol since 4 days was brought to emergency with altered sensorium, irrelevant talk, shouting, 2 episodes of tonic, clonic movements of upperlimbs, lowerlimbs and frothing from mouth. On examination, his GCS: E3 V3 M5 BP: 140/80mmHg, PR= 78/min, CBG=164mg/dl,
- CT brain : no abnormality
- ***EEG should be done???????????***

Case 3.....

- 42 year old male, daily alcoholic, stopped alcohol since 4 days was brought to emergency with altered sensorium, irrelevant talk ,shouting ,2 episodes of tonic, clonic movements of upperlimbs,lowerlimbs and frothing from mouth.On examination, his GCS:E3 V3 M5 BP: 140/80mmHg,PR= 78/min, CBG=164mg/dl,
- CT brain : no abnormality
- ***EEG should be done or not : no***
- ***Here the etiology of seizures is evident : acute symptomatic : due to alcohol withdrawal***

Case 4:

- 16 year old female with history of seizures since childhood, with aura, behavioural arrest, focal seizures, sometimes GTCS, recurrent seizures inspite of good drug compliance, old records not available .On examination, BP: 150/90 mm Hg, PR= 98/min, CBG=130 mg/dl,
- MRI brain : left hippocampal sclerosis
- ***EEG advised for this patient or not ???????***

Case 4.....

- 16 year old female with history of seizures since childhood, with aura, behavioural arrest, focal seizures, sometimes GTCS, recurrent seizures inspite of good drug compliance. old records not available .On examination, BP: 150/90 mm Hg, PR= 98/min, CBG=130 mg/dl,
- MRI brain : left hippocampal sclerosis
- ***EEG advised for this patient or not : yes***
- ***For clinical-radiological-electrophysiological correlation***
- ***For identifying epileptogenic zone, for appropriate surgery.***

Case 5 :

- 49 year old man with no previous comorbidities was brought to emergency in altered sensorium with family members witnessing 6 episode of seizures with tonic, clonic movements of upperlimbs, lowerlimbs with tongue bite and frothing from mouth. H/O multiple episodes of vomitings and loose stools since 2 days .On examination, his GCS was low, BP: 100/70 mm Hg, PR= 96/min, CBG=126mg/dl, S.Na = 110 mg/dl ,S.K = 4.2, S.Creatinine = 1.1 mg/dl
- MRI brain : no abnormality
- ***EEG advised for this patient or not ????????***

Case 5

- 49 year old man with no previous comorbidities was brought to emergency in altered sensorium with family members witnessing 6 episode of seizures with tonic, clonic movements of upperlimbs, lowerlimbs with tongue bite and frothing from mouth. H/O multiple episodes of vomitings and loose stools since 2 days. On examination, his GCS was low, BP: 100/70 mm Hg, PR= 96/min, CBG=126mg/dl, S.Na = 110 mg/dl, S.K = 4.2, S.Creatinine = 1.1 mg/dl
- MRI brain : no abnormality
- ***EEG advised for this patient or not : no***
- ***Here the etiology is evident. History, examination, lab findings are clearly suggestive of hyponatremia: which is transient, metabolic and reversible***

Case 6:

- 16 year old female with history of seizures since 2 years on good drug compliance and no seizure recurrence, normal developmental history, no family history. Last seizure : 2 years back. Came for review ,as she wants to stop AED s. On examination, BP: 130/90 mm Hg, PR= 78/min, CBG=160 mg/dl. Neurological examination: normal
- MRI brain : normal
- Previous EEG: epileptiform abnormalities in bilateral frontal regions
- ***EEG advised for this patient or not ????????***

Case 6.....

- 16 year old female with history of seizures since 2 years on good drug compliance and no seizure recurrence, normal developmental history, no family history. Last seizure : 2 years back. Came for review ,as she wants to stop AED s. On examination, BP: 130/90 mm Hg, PR= 78/min, CBG=160 mg/dl. Neurological examination: normal
- MRI brain : normal
- Previous EEG: epileptiform abnormalities in bilateral frontal regions
- ***EEG advised for this patient or not : YES***
- ***To taper AEDs, epileptiform abnormalities in previous EEG record should return to normal baseline.***
- ***If persisting , she has to continue AED s***

Case 7:

- 55 year old female, known diabetic , hypertensive was brought to emergency with right hemiparesis and tonic, clonic movements of right upperlimb,lowerlimb with tongue bite and frothing from mouth.On examination, her GCS:E3 VA M5 BP: 180/100 mmHg,PR= 98/min, CBG=424mg/dl,
- MRI brain :large infarcts in left fronto parietal area with haemorrhagic transformation
- ***EEG should be done??????????***

Case 7

- 55 year old female, known diabetic , hypertensive was brought to emergency in right hemiparesis and tonic, clonic movements of right upperlimb,lowerlimb with tongue bite and frothing from mouth.On examination, her GCS:E3 VA M5 BP: 180/100 mmHg,PR= 98/min, CBG=424mg/dl,
- MRI brain :large infarcts in left fronto parietal area with haemorrhagic transformation
- ***EEG should be done or not : no***
- ***As the etiology is evident : acute symptomatic seizure due to infarct***

Case 8:

- 48 year old male, known diabetic , hypertensive was brought to emergency, by family members finding him in unresponsive state on the floor in the morning. On examination, his GCS :E1V1M4 BP: 130/90 mm Hg, PR= 78/min, CBG=130 mg/dl, S.Na =138 ,S.creatinine= 0.8 , other lab work ups: with in normal range
- MRI brain :normal
- *EEG advised for this patient or not ????????*

Case 8.....

- 48 year old male, known diabetic , hypertensive was brought to emergency, by family members finding him in unresponsive state on the floor in the morning. On examination, his GCS :E1V1M4BP: 130/90 mm Hg,PR= 78/min, CBG=130 mg/dl,S.Na =138 ,S.creatinine= 0.8 , other lab work ups: with in normal range
- MRI brain :normal
- ***EEG advised for this patient or not: yes***
- ***As it is a state of unexplained coma: to know the etiology: encephalopathy/ encephalitis /drug intoxication/non convulsive status epilepticus***

Case 9:

- 26 year old female, G2P1L1 with 8 months of gestation ,with gestational hypertension on treatment, was brought to emergency by family members witnessing 1 episode of seizure with tonic,clonic movements of upper limbs , lower limbs with tongue bite and frothing from mouth . On examination, her GCS: E3-4 V5 M6, BP: 170/90 mm Hg , PR= 86/min, CBG=146mg/dl.
- MRI brain : cortical cerebral edema.
- *EEG advised for this patient or not ????????*

Case 9.....

- 26 year old female, G2P1L1 with 8 months of gestation ,with gestational hypertension on treatment, was brought to emergency with family members witnessing 1 episode of seizure with tonic, clonic movements of upper limbs , lower limbs with tongue bite and frothing from mouth. On examination, her GCS: E3-4 V5 M6, BP: 170/90 mm Hg , PR= 86/min, CBG=146mg/dl.
 - MRI brain : cortical cerebral edema.
 - ***EEG advised for this patient or not : no***
-
- ***As the etiology of seizure is clear: due to eclampsia , MRI Brain also no significant abnormality other than cerebral edema secondary to eccampsia***

Case 10:

- 56year old male, victim of road traffic accident with head injury ,was brought to emergency. he had 2 episodes of Seizures during transit to ER. On examination, his GCS was low, BP: 160/90 mm Hg , PR= 68/min, CBG=180 mg/dl , S.Na+ = 142, S.K+ = 4.1, S.creatinine = 0.9
- CT brain : fracture of left parietal bone with contusion and subarachnoid haemorrhage .
- *EEG advised for this patient or not ????????*

Case 10

- 56year old male, victim of road traffic accident with head injury ,was brought to emergency. he had 2 episodes of Seizures during transit to ER. On examination, his GCS was low, BP: 160/90 mm Hg,PR= 68/min, CBG=180 mg/dl,S.Na =142,S.K =4.1, S.creatinine= 0.9
- CT brain : fracture of left parietal bone with contusion and subarachnoid haemorrhage.
- ***EEG advised for this patient or not : no***
- ***As it is evident that seizure is acute symptomatic , due to subarachnoid haemorrhage.***

Case 11:

- 22 year old married female, with marital conflicts brought to OPD by family members, witnessing 4 episodes of jerky wavy movements of upper and lower limbs with eyes closed, pelvic thrusting lasting 10 minutes , no frothing/tongue bite/involuntary micturition . On examination, BP: 120/80 mm Hg , PR= 78/min, CBG=152mg/dl,
- MRI brain : normal
- *EEG advised for this patient or not ????????*

Case 11.....

- 22 year old married female, with marital conflicts brought to OPD by family members, witnessing 4 episodes of jerky wavy movements of upper and lower limbs with eyes closed, pelvic thrusting lasting 10 minutes, no frothing/tongue bite/involuntary micturition. On examination, BP: 120/80 mm Hg, PR= 78/min, CBG=152mg/dl,
- MRI brain : normal
- ***EEG advised for this patient or not : yes***
- ***Clinically diagnosed as psychogenic non epileptiform seizure (PNES), EEG is advised to confirm the diagnosis and reassure and avoid unnecessary antiepileptics***

Case 12 :

- 46year old male, known diabetic was brought to emergency 1 episode of seizure with tonic, clonic movements of upper limbs, lower limbs with tongue bite and frothing from mouth . On examination, his GCS :E4V5M6 BP: 150/90 mm Hg , PR= 98/min, CBG=130 mg/dl,
- MRI brain : multiple ring enhancing lesions in right fronto parietal areas ? tuberculomas
- *EEG advised for this patient or not ???????*

Case 12

- 46year old male, known diabetic , hypertensive was brought to emergency 1 episode of seizure with tonic, clonic movements of upper limbs, lower limbs with tongue bite and frothing from mouth.On examination, his GCS :E4V5M6 BP: 150/90 mm Hg,PR= 98/min, CBG=130 mg/dl,
- MRI brain : multiple ring enhancing lesions in right fronto parietal areas ? ? Tuberculomas.
- ***EEG advised for this patient or not : no***
- ***Etiology is clearly evident : structural lesion causing seizures***

Case 13:

- 67year old male, known diabetic, cardiovascular disease with stenting done few years back, had head injury due to RTA 10 years back, came to OPD with 1 episode of loss of consciousness with mild jerking of limbs, yesterday morning. On examination, BP: 100/70 mm Hg, PR= 52/min (bradycardia +), CBG=180 mg/dl, S.Na =142, S.K =4.1, S.creatinine= 0.9
- CT brain : left frontal gliosis
- EEG : done outside: showing pathological slowing in left frontal region
- *Will you start him on antiepileptics???????*

Case 13.....

- 67year old male, known diabetic, cardiovascular disease with stenting done few years back , had head injury 10 years back due to RTA, came to OPD with 1 episode of loss of consciousness with mild jerking of limbs, yesterday morning. On examination, BP: 100/70 mm Hg , PR= 52/min (bradycardia +),CBG=180 mg/dl, S.Na =142, S.K =4.1, S.creatinine = 0.9
 - CT brain : left frontal gliosis
 - EEG : done outside: showing pathological slowing in left frontal region
 - ***Will you start him on antiepileptics?????? : no***
-
- ***Loss of consciousness is due to syncopal attack , as evidenced by bradycardia(cardiac etiology)***
 - ***Here,EEG abnormality is incidental finding .***
 - ***So, don't treat EEG.....***

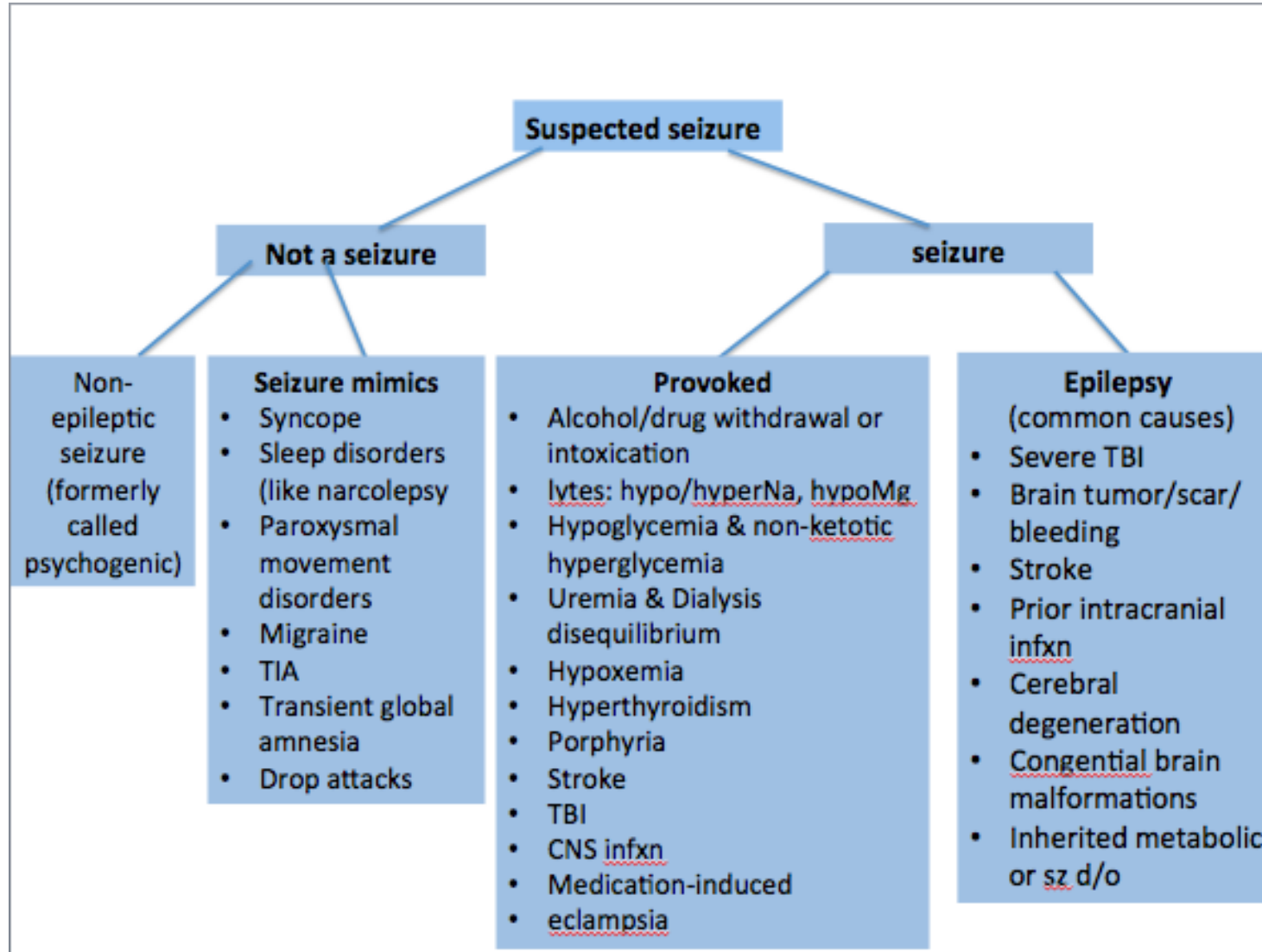
So, in which patients EEG is done..???????

“In patients with clinical suspicion of epilepsy, not for every subject with seizures”

“Not done for symptomatic seizures/provoked seizures”

- **Seizure:** transient events that include symptoms and/signs of abnormal excessive hypersynchronous activity in the brain
- **Epilepsy:**
- ILAE 2017 definition: **a disease of brain defined by**
 - 1) **at least 2 unprovoked seizures occurring > 24 hours apart/**
 - 2) **or 1 unprovoked seizure and > 60 % recurrence risk**
 - 3) **or diagnosis of an epilepsy syndrome**

So, EEG is done for epilepsy subjects...not every seizure



Symptomatic, provoked, unprovoked definitions :

- *acute symptomatic seizures /Provoked/reactive/situation related* : seizures, occurring due to acute CNS insult, which may be metabolic, toxic, structural, infectious, or due to inflammation.
- *unprovoked seizures/epilepsy* : seizures occurring in the absence of a potentially responsible clinical condition or beyond the interval estimated for the occurrence of acute symptomatic seizures.

For which patients it is done??????????

In patients with clinical suspicion of epilepsy:

- To **support the diagnosis**, in first seizure.
- To *identify epilepsy syndrome* , in known epilepsy patients.
- To *monitor response* to antiepileptics
- To *identify seizure triggers*, in reflex epilepsies.
- In focal seizures, *to identify epileptogenic zone, for surgery posted subjects*
- To identify ideal candidates for **AED withdrawal**, after achieving seizure remission.

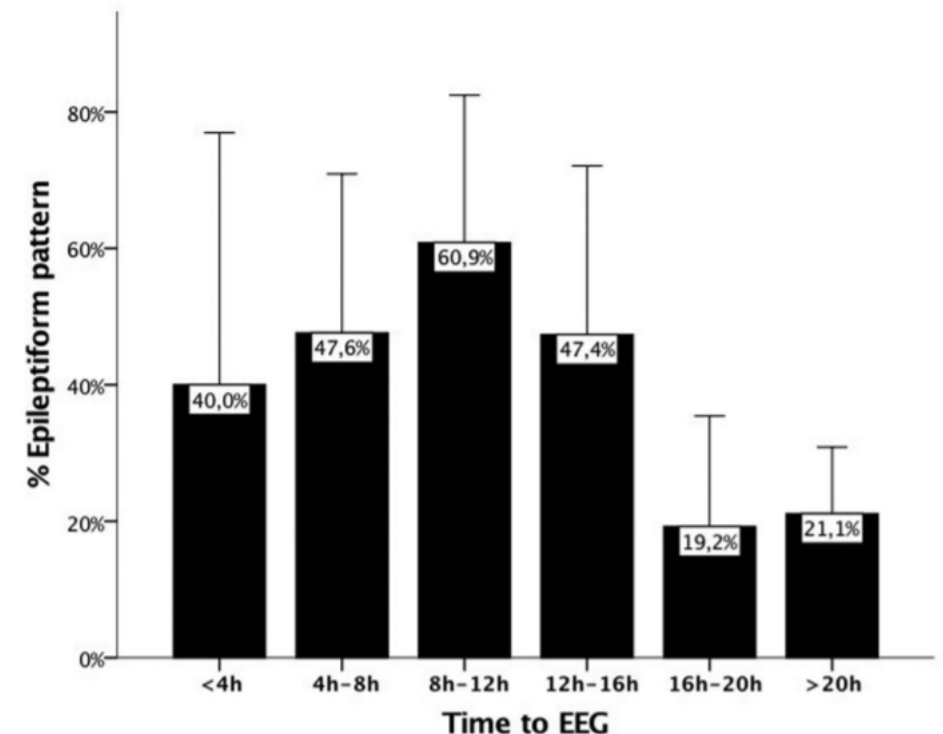
For which patients it is done??????????

- 1.Epilepsy
- 2.Encephalopathy : to know the severity, etiology: toxic/hepatic/anoxic
- 3.Acute encephalitis : HSV encephalitis, anti-NMDA encephalitis
- 4.Rapidly progressive neuro cognitive decline: SSPE/CJD
- 5.Recurrent amnesia episodes: epileptic amnesia/global amnesia
- 6.Altered sensorium : to diagnose Non Convulsive Status Epilepticus
- 7.Brain death: to confirm, by electrocerebral inactivity.

Timing of EEG after a single unprovoked seizure: early vs late

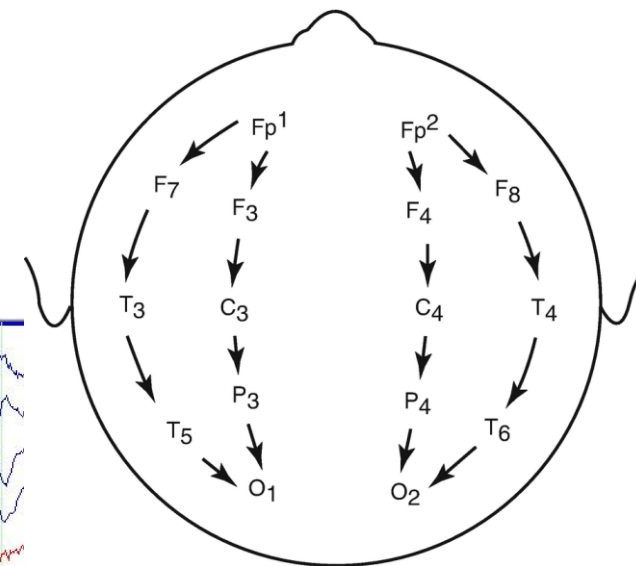
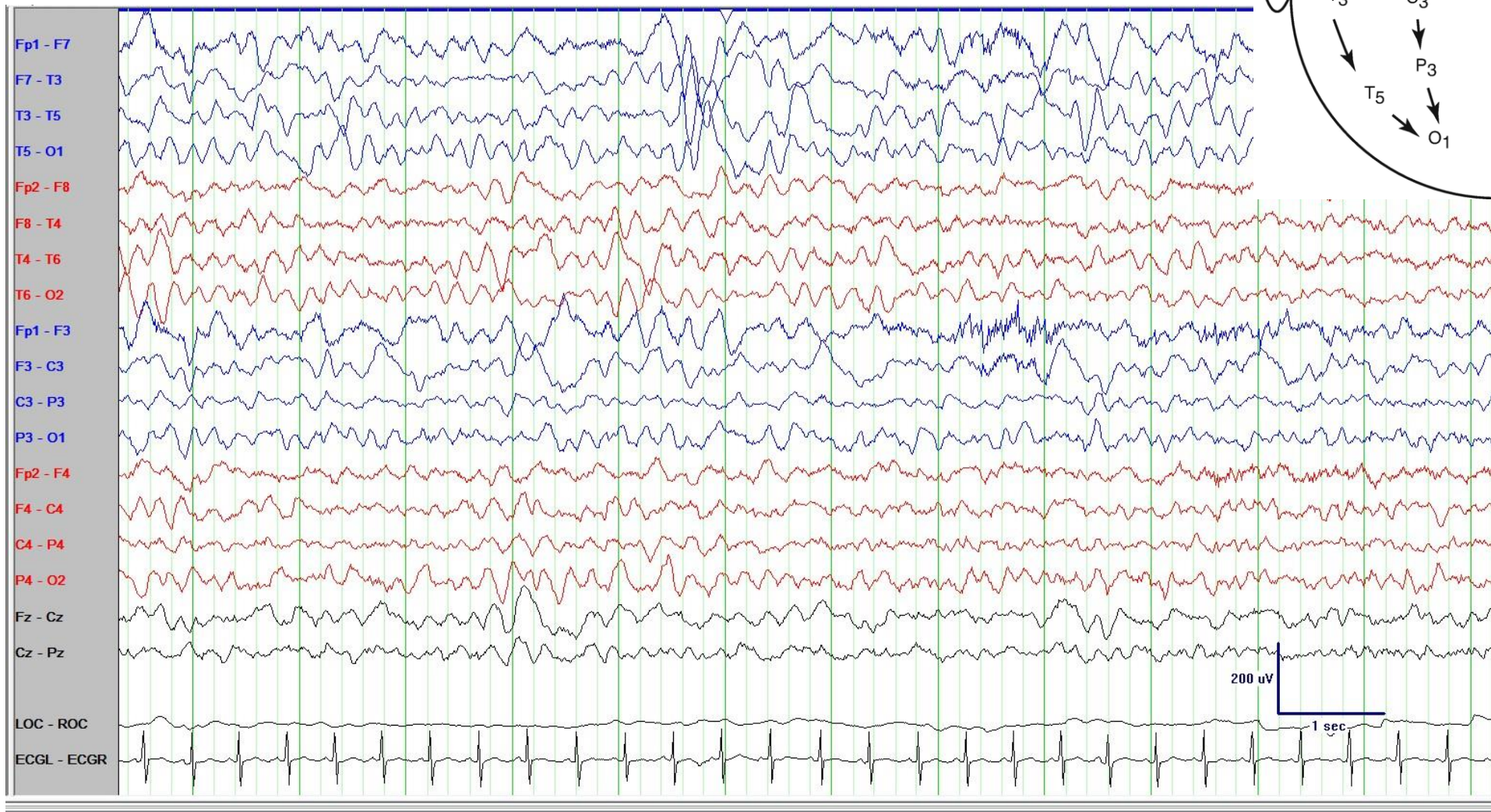
comparing early and late EEG recordings:

- immediately after a seizure, *the brain is in a state of hyperexcitability*, which reduces over time. According to this hypothesis, an early EEG (shortly after seizure occurrence) should offer a higher yield with respect to recording epileptiform abnormalities, if present.
- epileptiform abnormalities in **51%** of patients who had an EEG within 24 h of the seizure, as compared to **34%** in patients who had their initial EEG at a later point in time.



How to increase the yield of first EEG ???

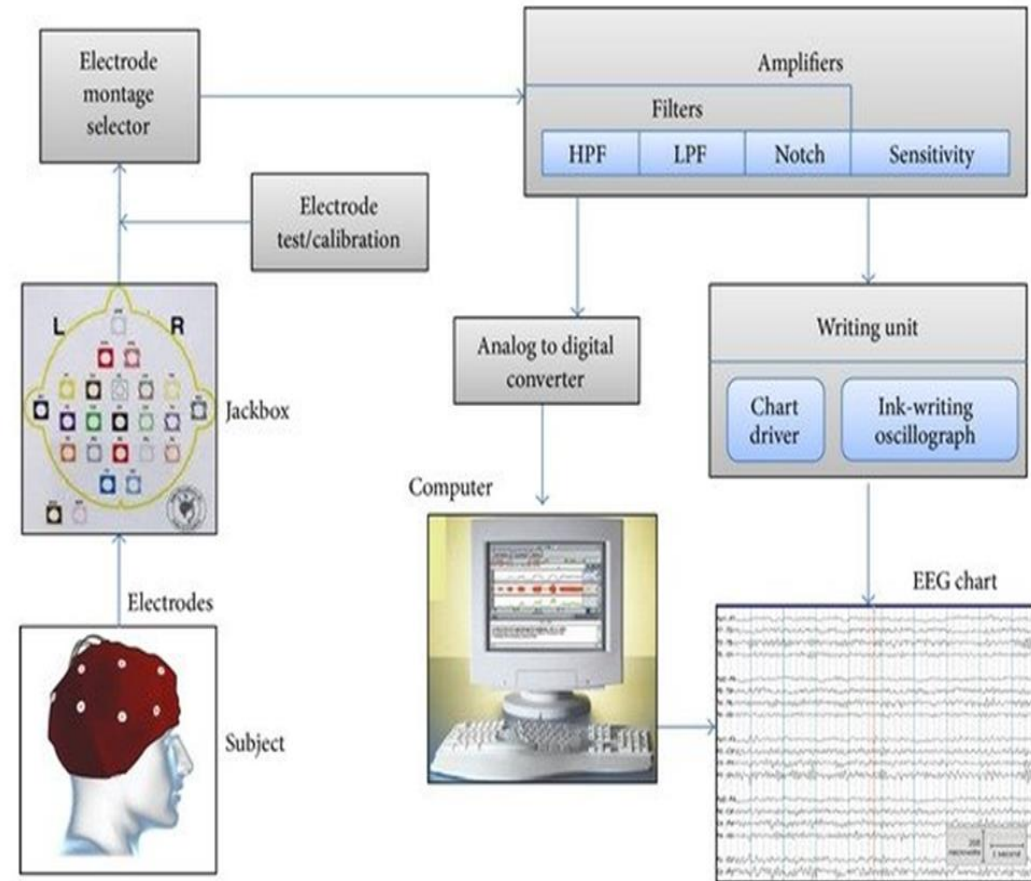
- Recording soon after a seizure
- obtain a sleep record, after keeping the patient sleep deprived .
- Activation procedures: like hyperventilation, photic stimulation, repeated eye opening & closure, using triggers in reflex epilepsies
- Increasing the duration & overnight recording.
- Recording after additional electrode placement.
- Serial EEG studies
- After reducing/withdrawing AEDs



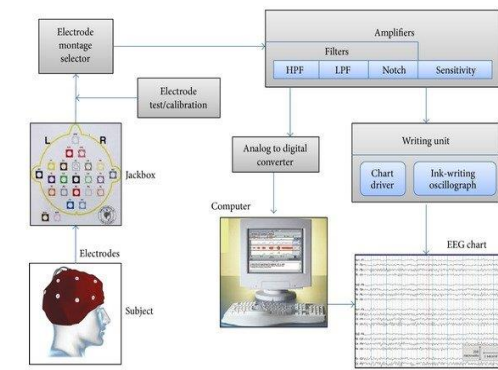
EEG set up consists of ????????

EEG machine has 4 basic components:

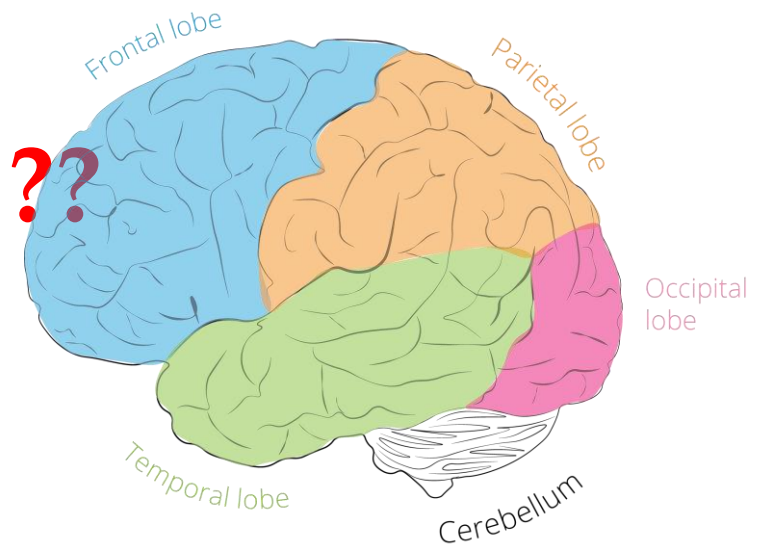
- recording **electrodes**,
- a differential **amplifier**,
- **analog to digital convertor**
- **display and storage device.**



- **Electrodes**: are placed over the scalp to record the brain signals.
- Types: disposable, reusable, needle electrodes, electrode caps.
- For routine scalp recordings, reusable disc electrodes made of silver and coated with silver chloride are most commonly used. These electrodes are attached to long flexible leads, which can be plugged into the amplifier.
- **Amplifier** : electrical potentials generated by the cortical neurons are very small and further attenuated by intervening tissues between the brain and scalp. So they require significant amplification in order to display them on the paper/computer screen. EEG amplifier is a differential amplifier , which amplifies the potential difference between 2 inputs , while removing the common signals.
- **Analog to digital convertor** : converts physical analog signals to digital signals

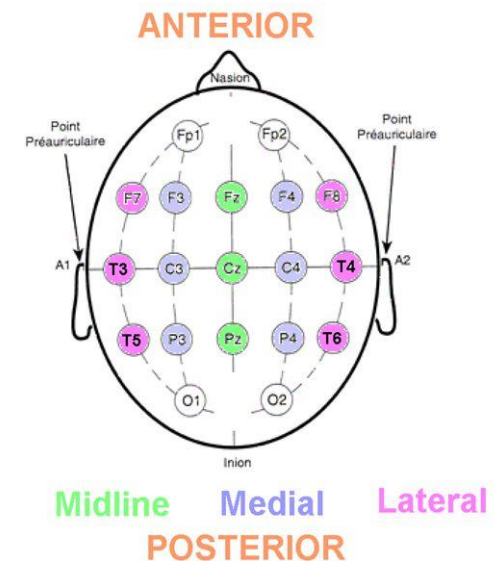


Where are the electrodes placed ??



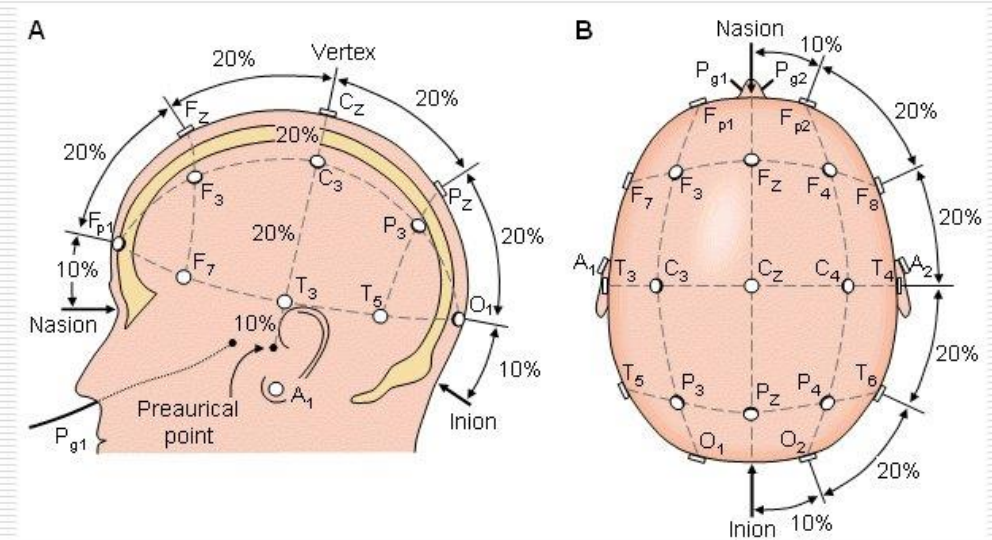
- In 1958 , International Federation of Societies for Electroencephalography and Clinical Neurophysiology , adopted a system for electrode placement called **10-20 system**.
- A Standard system of electrode placements helps in **easy communications** across different EEG centres in the world and provides for comparisons between and among patients.
- Electrodes are named as per the underlying brain areas: **F(Frontal)**, **Fp(frontopolar)**, **C(central)**, **T(temporal)**, **P(parietal)** and **O(Occipital)**

10-20 International System of Electrode Placement

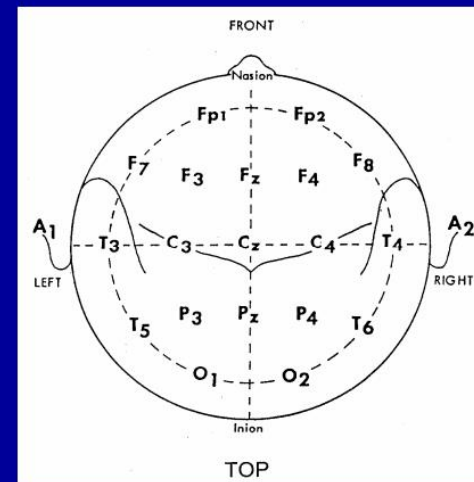


- Right sided electrodes are suffixed with **even numbers**
- Left sided electrodes are suffixed with **odd numbers**
- z is suffixed for **midline** electrodes.
- Electrodes are placed at **10 percent or 20 percent of total distance on the two lines** connecting prominent landmarks: line connecting the nasion with inion in the sagittal plane and a line connecting two preauricular points in the coronal plane

10 /20 % system of EEG electrode placement

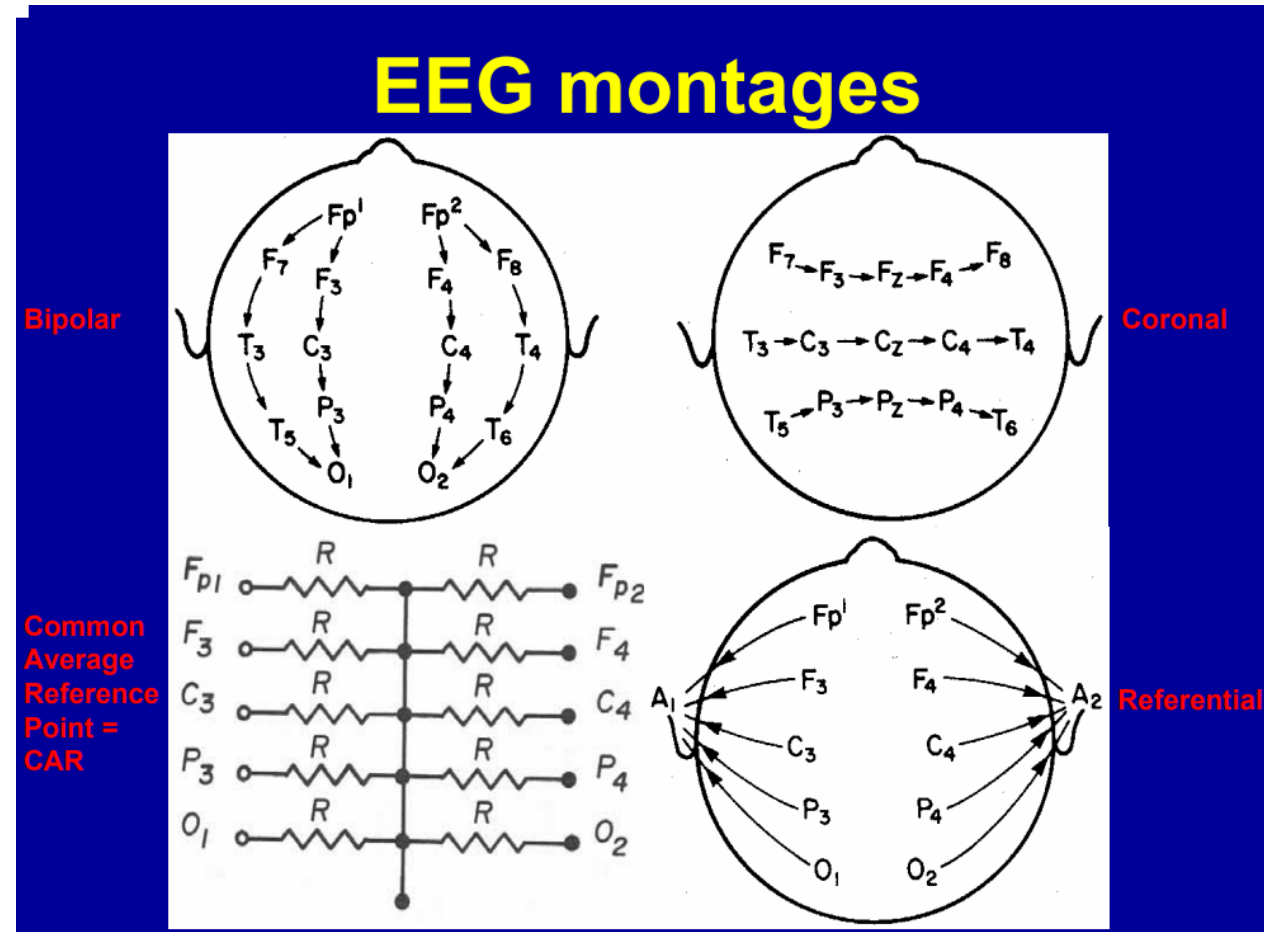


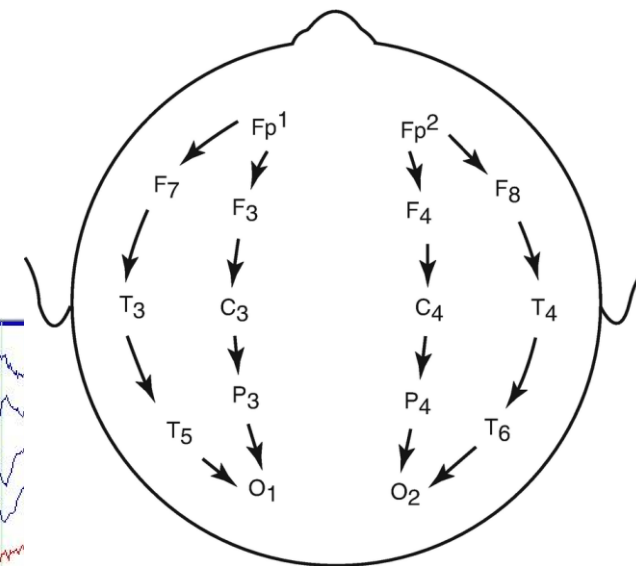
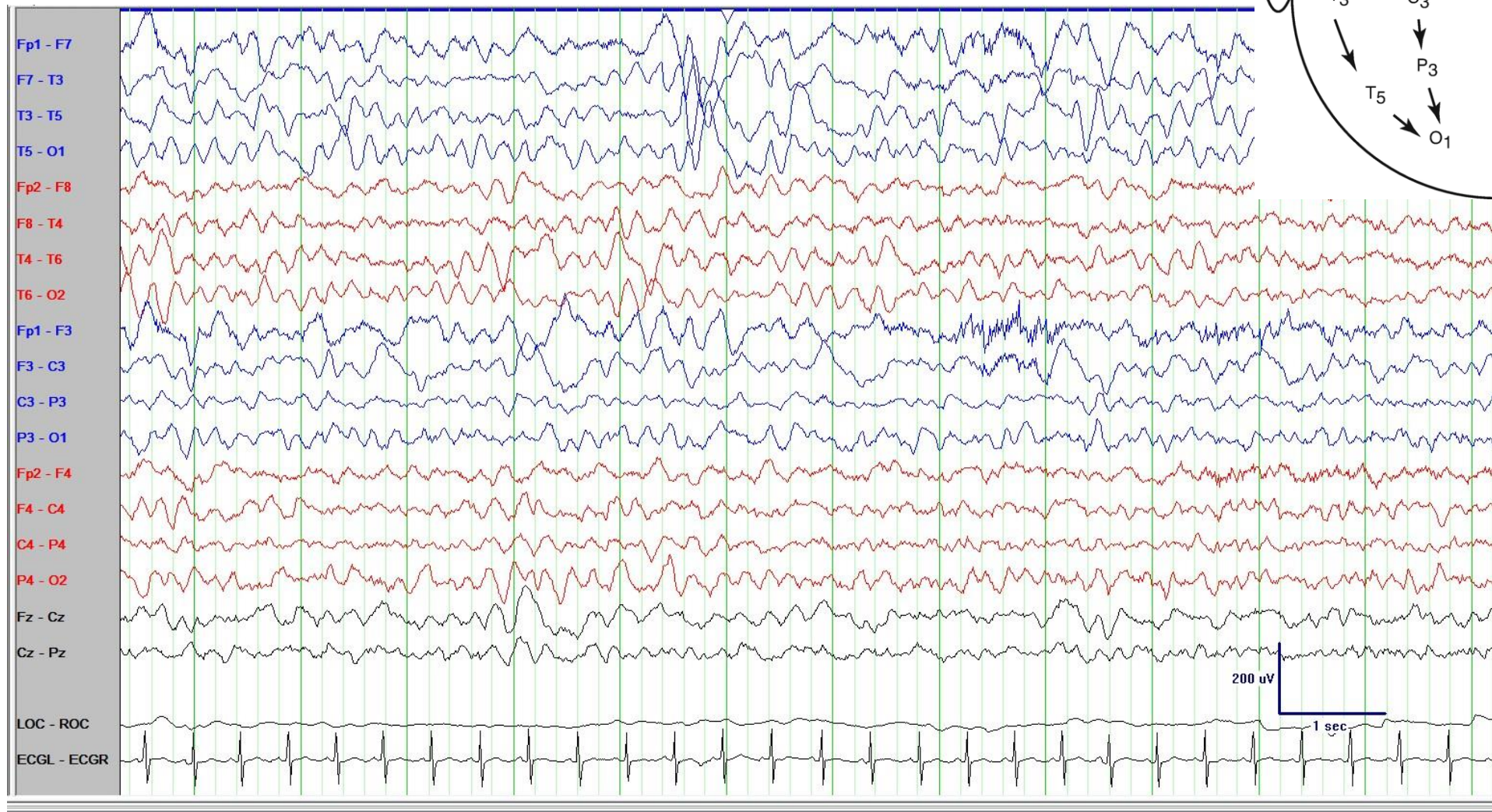
International 10-20 system of electrode placements



Montages:

- Two electrodes are connected to form a **channel**, which produces one line of EEG.
- **Sequence of electrodes' connections** is called montage





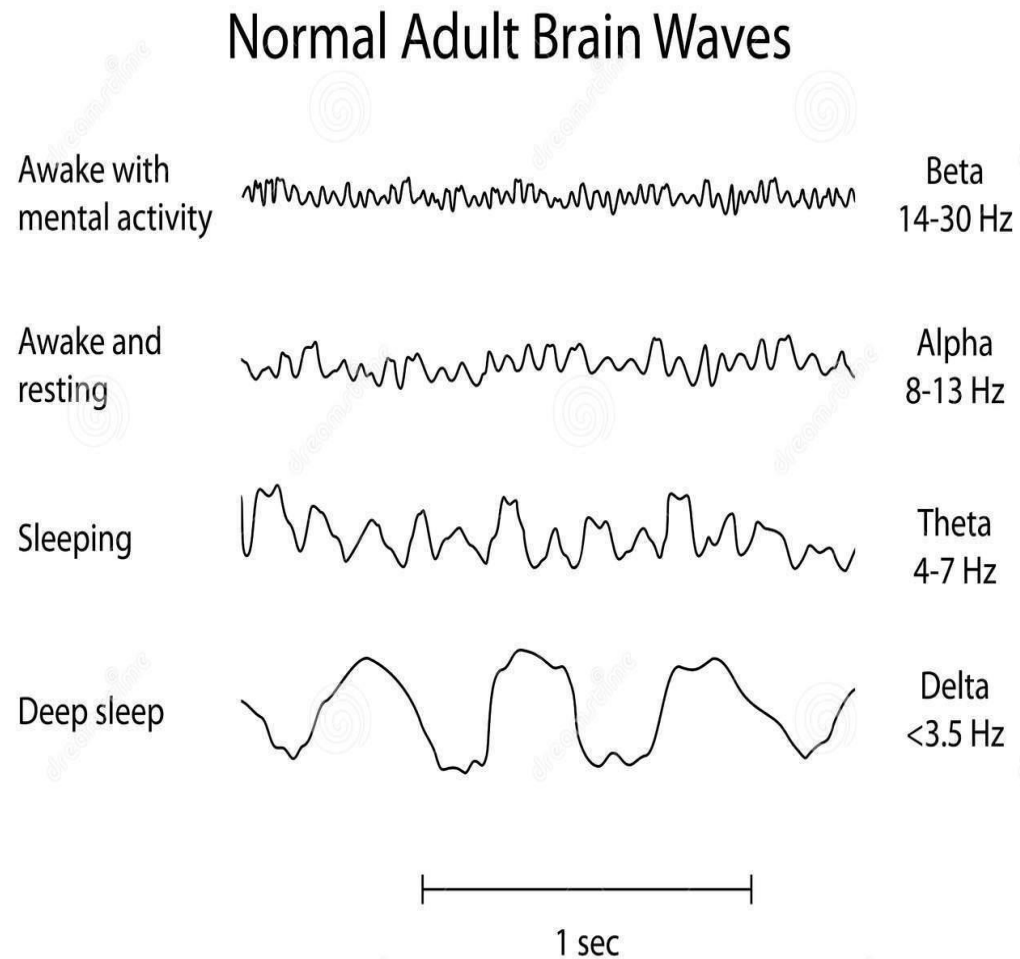
Protocol of routine EEG:

- Not more than 3-4 hours of sleep during previous night
- Patients referred with the diagnosis of seizure disorder should have awake and sleep recordings
- Try to obtain natural sleep. Use chloral hydrate, if necessary, 25 mg/kg for children and 1000 mg for adults or triclofos in the dose of 20 mg/kg
- Duration of recording : awake : 20 minutes & sleep : 20 minutes
if awake alone 30 minutes

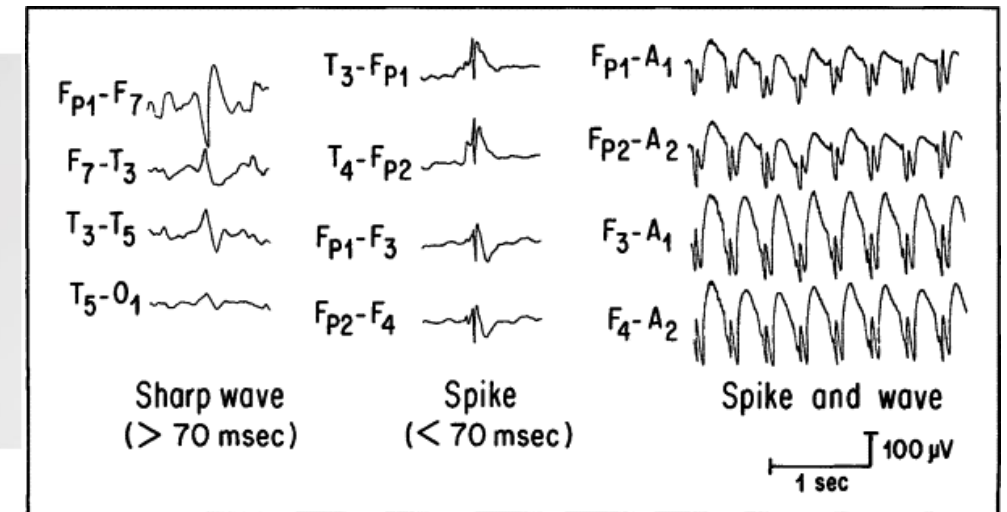
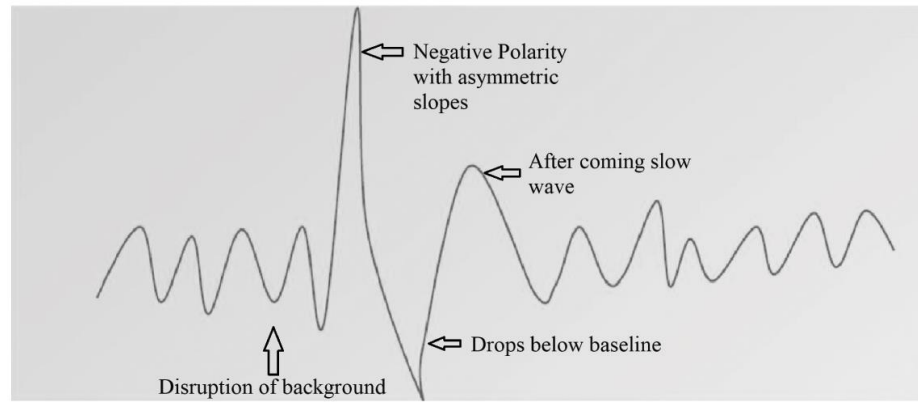
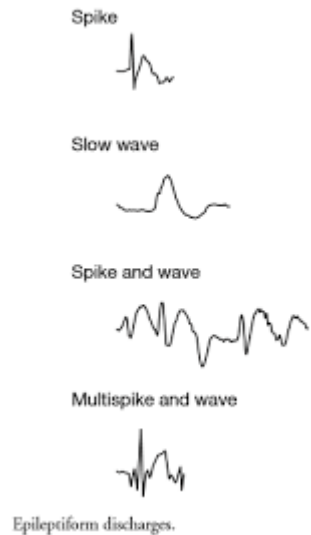
Pre requisites for EEG: Patient wise

- **clear instructions** are provided to the patients prior to their EEG appointment.
- The scalp is usually cleaned well to obtain proper recording with *low impedance*.
- They are recommended **not to use conditioner or other substances that might affect the quality of the recording (electrode impedance)**. Typically, the impedance should be lower than 5KOhm.
- For ICU patients, several measures are typically taken to **reduce the disturbances from various medical instruments, devices, and lines used**.

Normal EEG wave forms:



epileptiform abnormalities :



Spike and wave discharge abnormality :

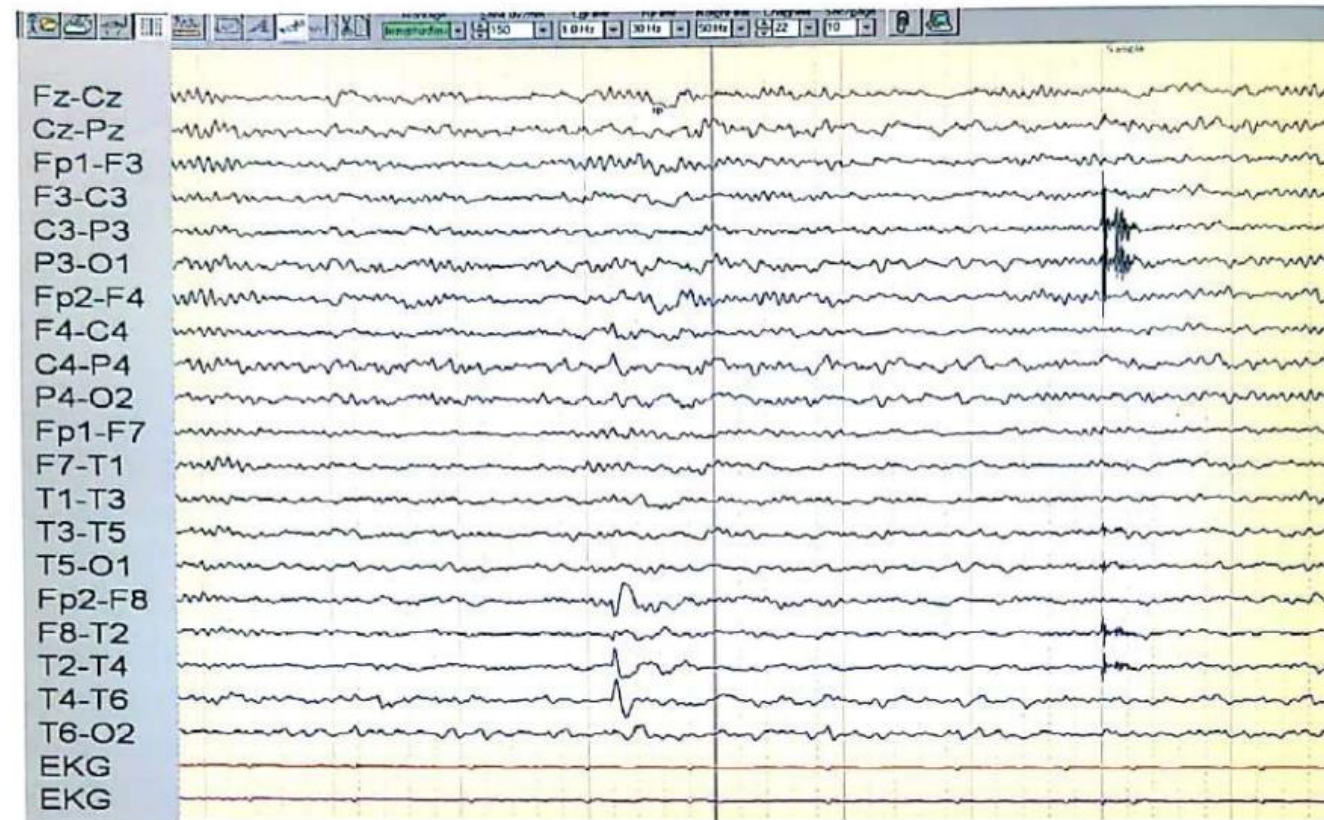


Figure 9.5: EEG seen in longitudinal bipolar montage shows right anterior temporal spike-wave discharge. F8-T2 channel shows an almost flat line with phase reversal across F8 and T2 electrodes (Sensitivity: 15 μ V/mm; LFF: 1.0 Hz; HFF: 30 Hz).

But..Every spiky morphology wave is not epileptiform ...

Table 11.1: Diagnostic characteristics of interictal epileptiform discharges

- Paroxysmal with spiky configuration standing out from the background.
- Duration of 70-200 msec for a sharp wave and 20-70 msec for a spike.
- Abrupt change in polarity from preceding wave forms.
- Surface negative polarity.
- A physiological field.

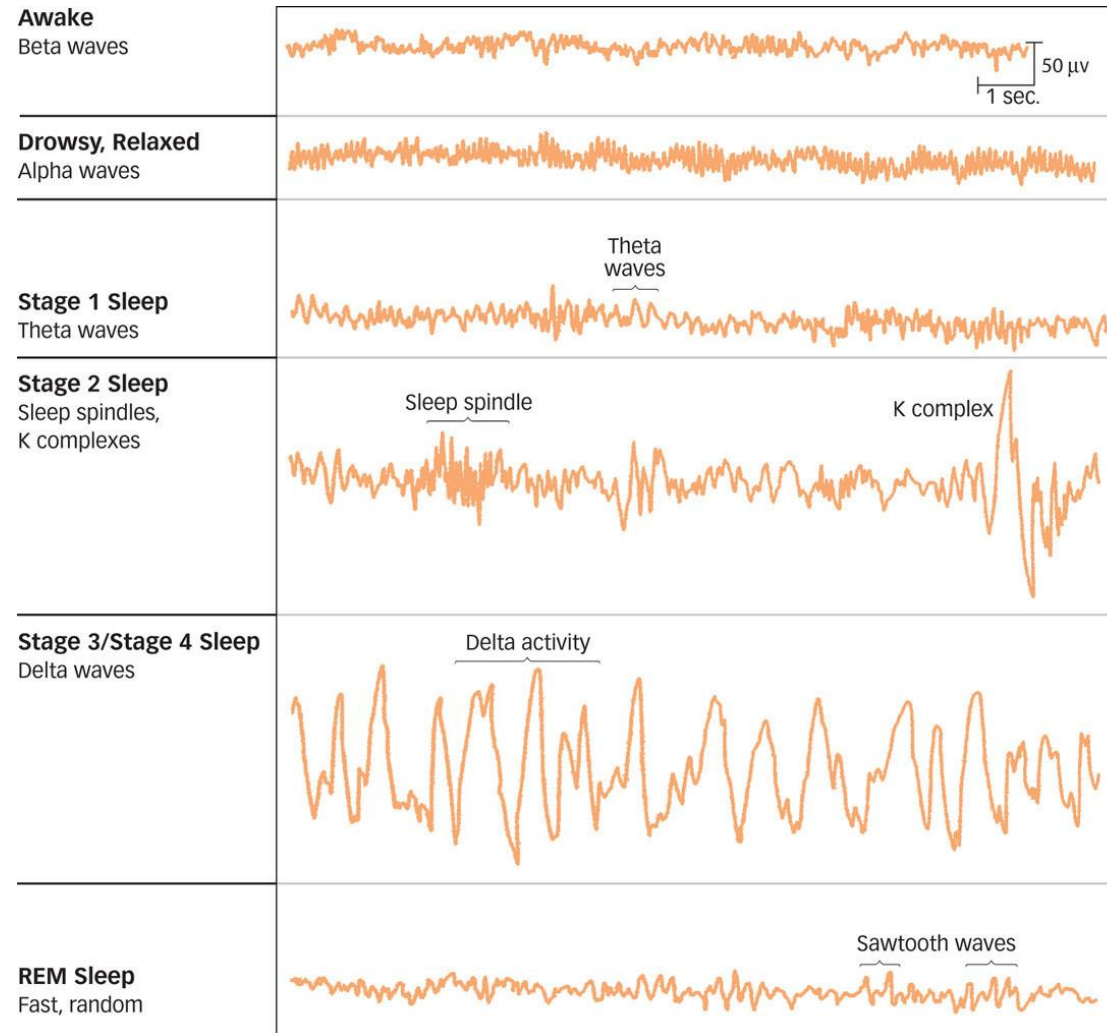
There can be

- Artifacts
- Normal spiky morphology waves during drowsiness, sleep
- Benign variants

Table 8.15: Consequences of misreading EEG

- Wrong label of diagnosis of epilepsy with its physical and psychosocial consequences.
- Unnecessary treatment with antiseizure drugs with their side effects and cost.
- Difficulty in undoing a once wrongly labeled EEG resulting in repeat EEGs and long-term video-EEG monitoring with added discomfort and cost.
- Medicolegal issues stemming from wrong diagnosis and treatment.

Wave forms during sleep:



Benign Epileptiform variants(BEVs):

- ***EEG pattern that is morphologically epileptiform but is not associated with epilepsy.***

Significance of correctly identifying the BEVs in the EEG:

- To avoid misdiagnosis of the subjects with epilepsy based on these waveforms
- To avoid unnecessary treatments of these subjects with anti-epileptic drugs or epilepsy surgery.
- To avoid other negative impact of epilepsy on the lives of these individuals eg. driving

Sharply contoured BEVs:

- Wicket waves
- Benign sporadic sleep spikes (BSSS)

BEVs occurring in bursts or trains:

- 6 Hz spike-waves
- 14 & 6 Hz positive spikes
- Rhythmic temporal theta bursts of drowsiness (RTTD)
- Subclinical rhythmic electrographic discharge of adults (SREDA)

Benign variant :6 Hz spike and wave

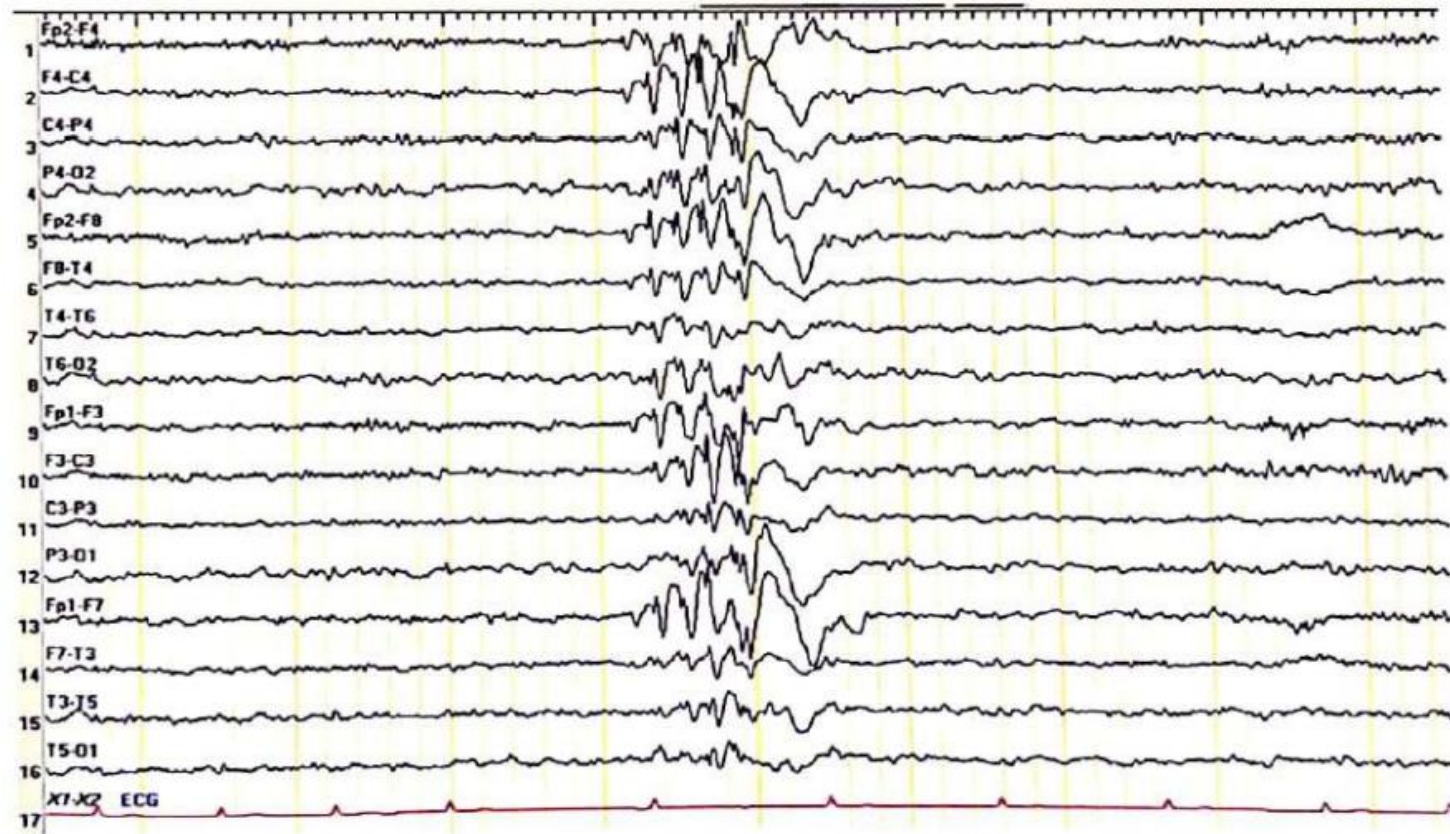
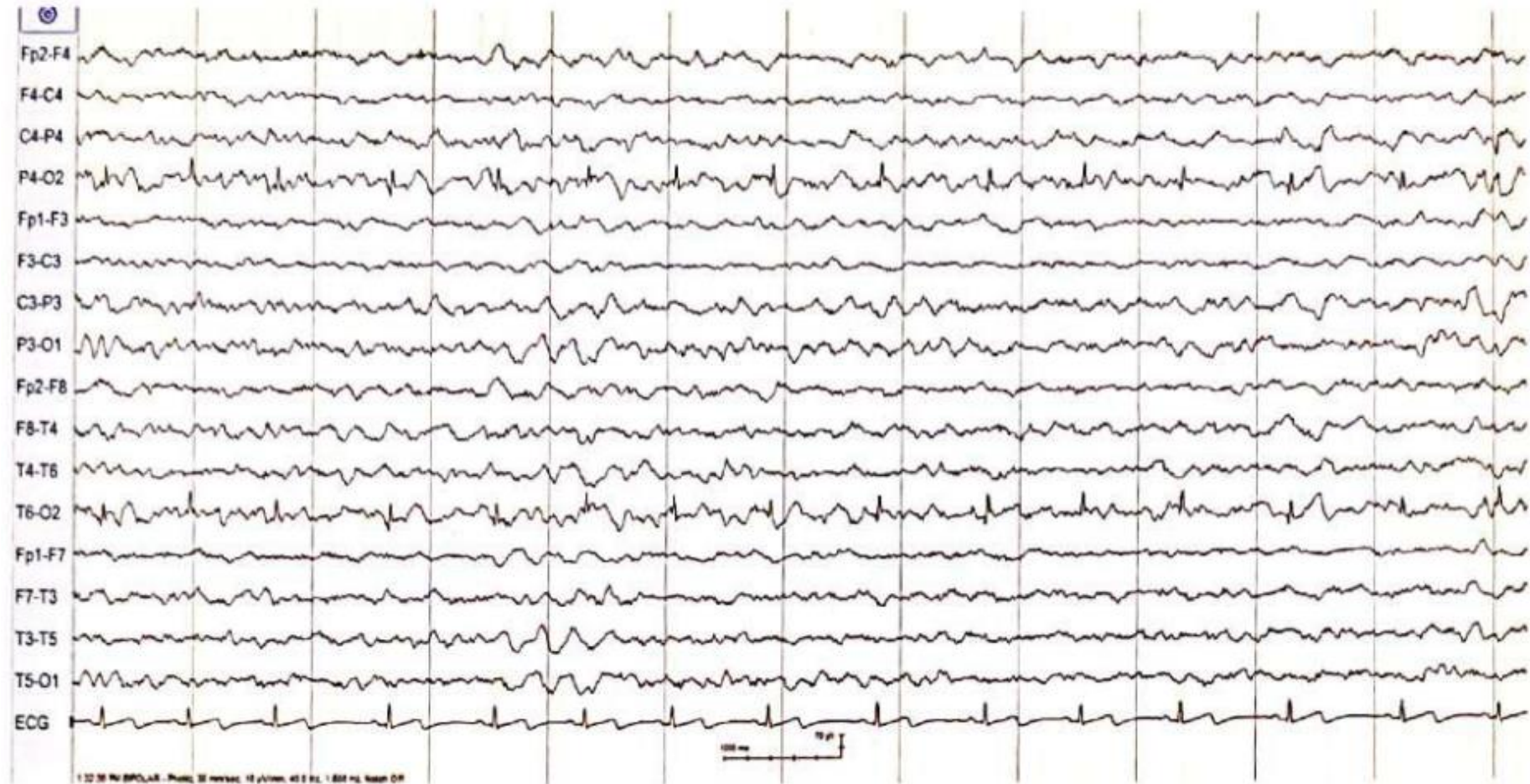


Figure 8.21: 6 Hz spike and wave discharges: A) Posterior 6 Hz spike and waves in a 12-year old girl during drowsiness. This is an example of FOLD (Female, Occipital, Low amplitude, Drowsiness); B) Anterior 6 Hz

Artifacts:

- *Unwanted electrical activity arising from different sources, other than cerebral activity.*
- EEG -highly sensitive recording device, easily interrupted by other electrical activity of very high voltages , Some readily distinguished, others closely resemble cerebral activity that their interpretation is taxing even to the most experienced eletroencephalographer.

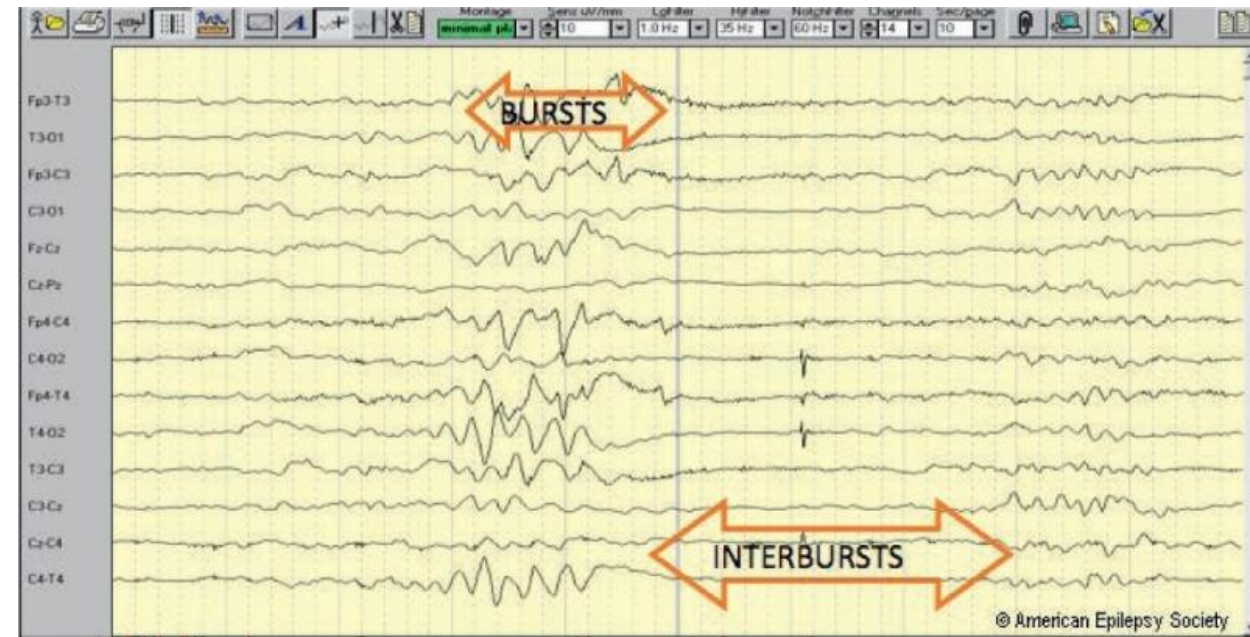
Table 2.2: Common types of artifacts	
Physiological	Non-physiological
Eye movements <ul style="list-style-type: none"> • Horizontal eye movements • Vertical eye movements • Eyelid flutter • Electropretinogram Cardiac artifacts <ul style="list-style-type: none"> • ECG artifacts • Pulse artifacts • Pacemaker artifacts • Ballistocardiographic artifacts Electromyographic artifacts <ul style="list-style-type: none"> • Lateral rectus spike • Frontalis electromyogram • Temporalis electromyogram • Occipitalis electromyogram • Swallowing and chewing • Photomyogenic (photomyoclonic) Glossokinetic artifact Galvanic skin responses <ul style="list-style-type: none"> • Salt bridge • Perspiration Physiological movement artifacts	Instrumental artifacts <ul style="list-style-type: none"> • 60Hz/50Hz artifacts • Electrostatic • Capacitive • Magnetic Electrode artifacts <ul style="list-style-type: none"> • Electrode pop • Impedance-related • Electrode lead movements • Photic cell artifact Environmental artifacts <ul style="list-style-type: none"> • Ventilator artifacts • Intravenous drip artifacts • Continuous Renal Replacement Therapy artifacts (CRRT) • Bed percussion artifacts • Extracorporeal Membrane Oxygenation artifacts (ECMO) • Cell phone artifacts



(B) ECG artifact in the occipital leads in a patient with short neck.

In neonates: normal EEG is little different.....

- EEG: *Activity is discontinuous in early life* : brief lasting high amplitude slow waves(*burst*) interspersed with long periods of electrical silence(*interburst interval*)
- *Synchrony* between two cerebral hemispheres evolves with age:
- Delta brushes
- Temporal theta bursts
- Frontal sharp waves



Activation procedures:

- *Procedures done to induce, enhance or better define abnormal EEG patterns.*

Table 3.1: Activation procedures used during EEG recording

Routinely performed activation procedures

- Eye opening and closure
- Mental alerting
- Hyperventilation
- Intermittent photic stimulation
- Sleep

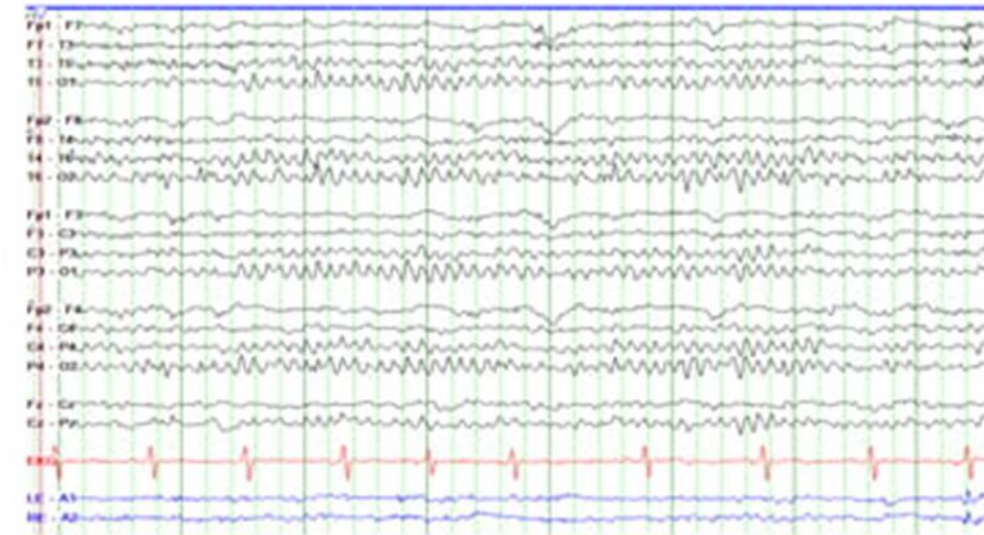
Activation procedures performed in special situations

- Scotosensitivity and fixation-off sensitivity
- Pattern stimulation
- Television and video games
- Eating
- Reading
- Cognitive tasks such as arithmetic, problem solving, chess playing, etc.
- Praxis

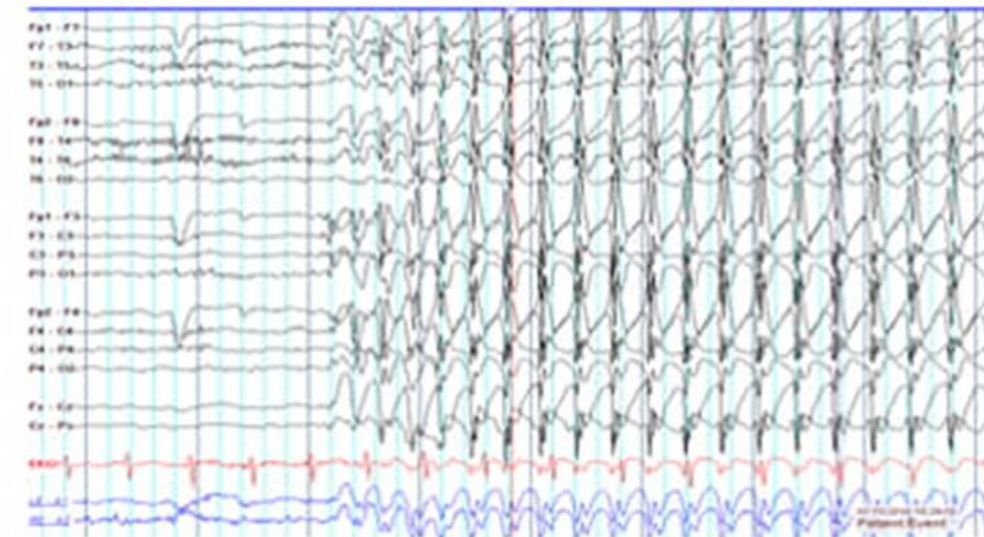
Hyperventilation:

- Hyperventilation (HV), is breathing deeply to exhale all the “dead space” air at a rate of 18 to 24 breaths per minute for 3 to 5 minutes.
- HV causes a decrease in the carbon dioxide in the blood (**hypocapnia**). Hypocapnia causes a constriction of the arteries, including the **cerebral arteries**
- **Contraindications** : subarachnoid hemorrhage, sickle cell anemia, recent cerebrovascular accident or myocardial infarction, significant cardiopulmonary disease, active asthma, known aneurysm, known moyamoya disease, and advanced pregnancy
- **HV is very effective in activating generalized 3 Hz spike and wave .**
- **Generalized slow spike and wave at 2 to 2.5 Hz and focal interictal spikes and sharp waves can be activated by HV**

Normal EEG Awake

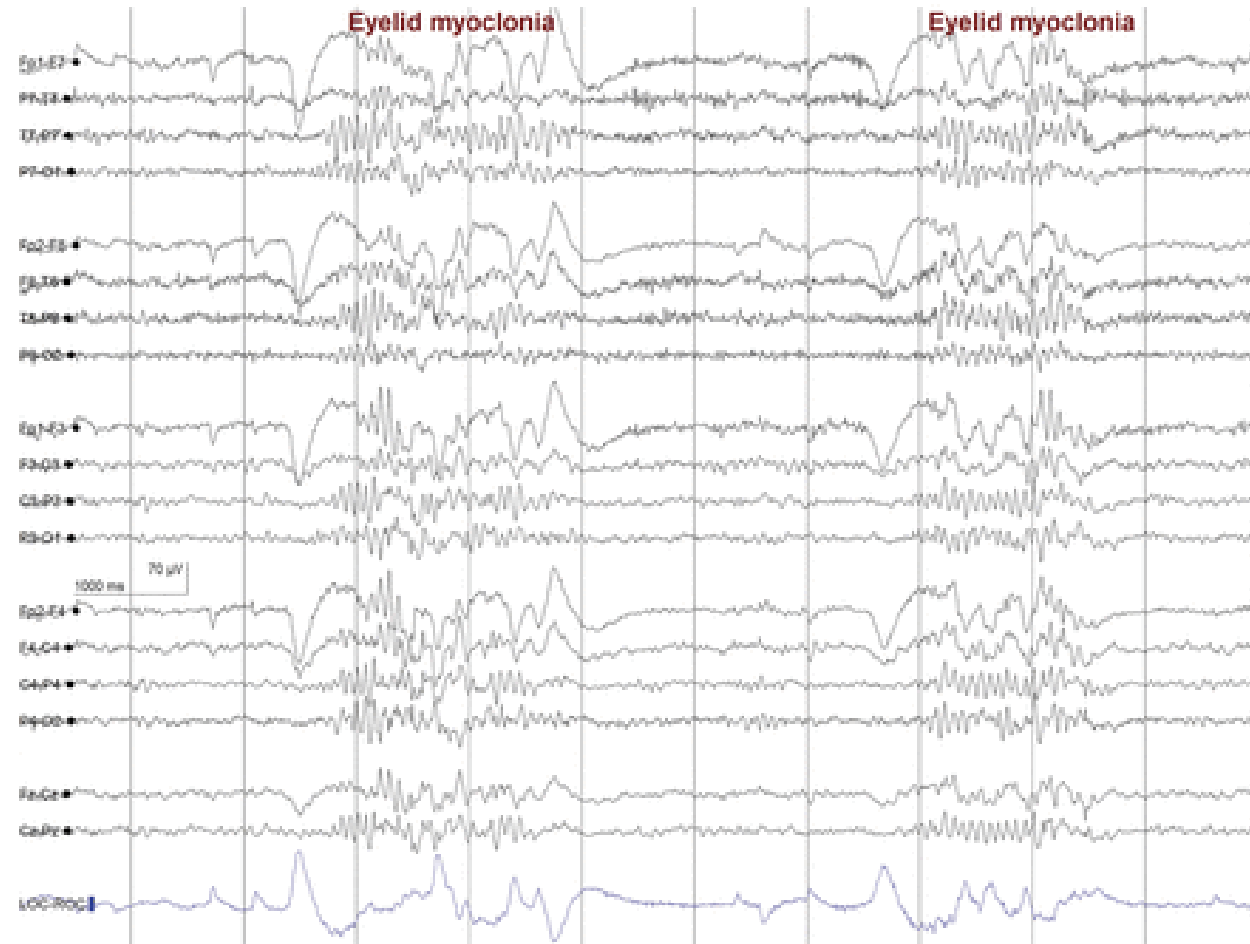


Absence Seizure

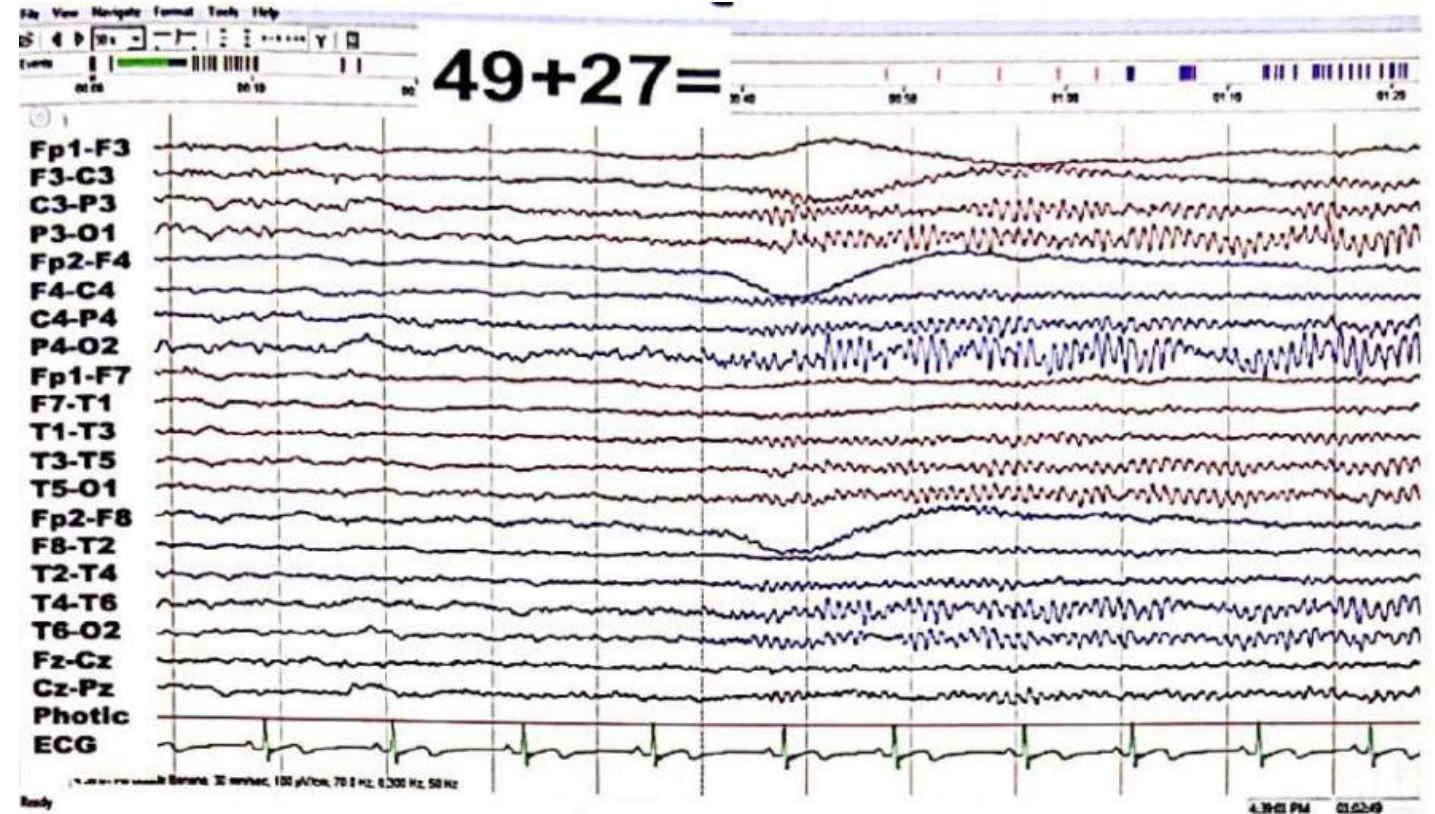


- **Eye opening and closing:**

Spikes/spike and waves occur immediately after eye closure, in some syndromes: *eyelid myoclonia with absence seizures (jeavons syndrome), idiopathic occipital epilepsy, JAE, JME, idiopathic generalized epilepsies.*



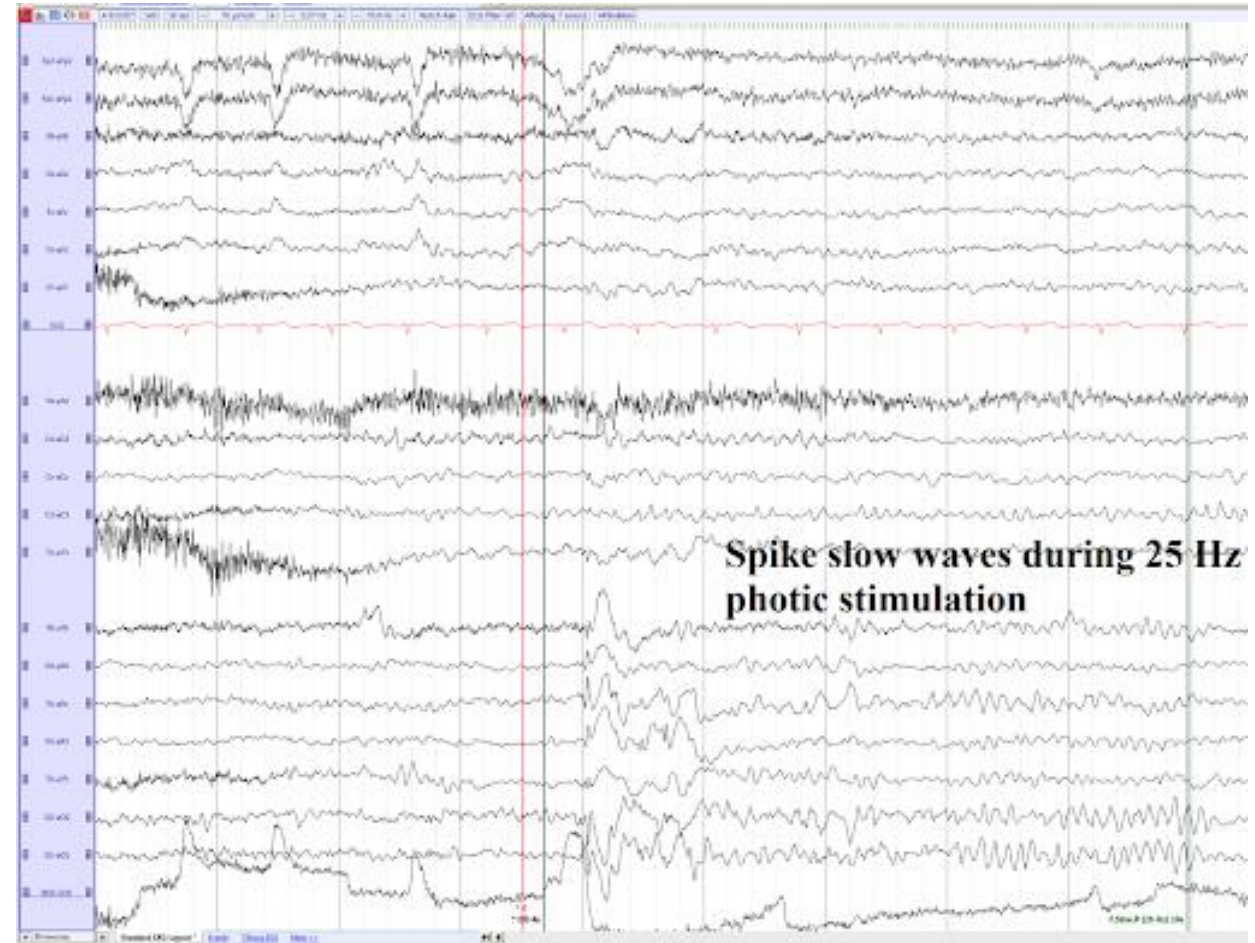
- **Mental alerting:** with mental arithmetics (additions, subtraction etc.), *if no increase in the frequency of background, slowing is pathological.*



(C) Return of posterior alpha when the task is given during the drowsy state (HFF=70 Hz, LFF=0.3 Hz, and sensitivity of 1mm=70 μ V).

Intermittent photic stimulation:

- **Photoparoxysmal response:** spike/ spike and wave paroxysms, by looking at a series of flashing lights .
- Due to hyperexcitability of occipital cortex, seen in *Progressive Myoclonic epilepsies* (67 %), *JME* (22%), *idiopathic generalized epilepsy* (3%), *focal epilepsies* (15 %).



Sleep:

- Sleep is *a powerful activator of nearly all forms of epilepsy.*
- Hence sleep EEG performed for every patient with suspected epilepsy.



Complications:

- **Unexpected complications** can occur if due diligence is not performed while screening patients before performing the activation or provocative procedures like hyperventilation in certain individuals as mentioned previously.
- Long-term EEG monitoring in epilepsy monitoring units and the intensive care units are associated with *skin injury* and appropriate care needs to be provided

Contraindications:

- There are **no clear contraindications** to performing an EEG.
- However, electrode placement could be challenging following a craniotomy, and in case of breaches in the skull or open wounds.
- Activation procedures should be omitted in individuals with certain underlying conditions. For example, **hyperventilation is a relative contraindication** in patients with a history of *recent strokes, myocardial infarction, surgeries (transplants), acute respiratory distress syndrome, Asthma, Moyamoya disease, and Sickle cell anemia*.

Limitations of scalp EEG:

- **Low spatial resolution**, as scalp electrodes detect only brain activity that is synchronized in >6 cm² area of cortex, attenuated by impedance of CSF, meninges, skull & scalp.
- **Short duration of recording (30 min)**, may fail to detect interictal epileptiform abnormalities (IEDs) in > ½ of subjects with infrequent IEDs.
- **Do not cover** parts of cerebral cortex **buried in sulci & insular and basal regions**.
- **EEG reading is subjective: inter - rater variability**
- **Misinterpretation of benign epileptiform abnormalities and artifacts as truly epileptiform**: leads to misdiagnosis and unnecessary treatment with antiepileptics.
- If done without proper indication and little clinical information, interpretation is difficult, **as true IEDs are seen in 1% of normal subjects**.

“Treat the patient, not the EEG”

Various Types of EEG:

1. Standard EEG
2. Sleep EEG
3. Sleep deprived EEG
4. Ambulatory EEG
5. Video EEG
6. Invasive EEG
7. Monitoring EEG
8. Neurofeedback EEG

Ambulatory EEG:

- When a patient is experiencing *unexplained seizure episodes* at *random intervals* these episodes become very difficult to predict and *if all previous tests failed to capture a seizure episode*, an Ambulatory EEG test is prescribed.



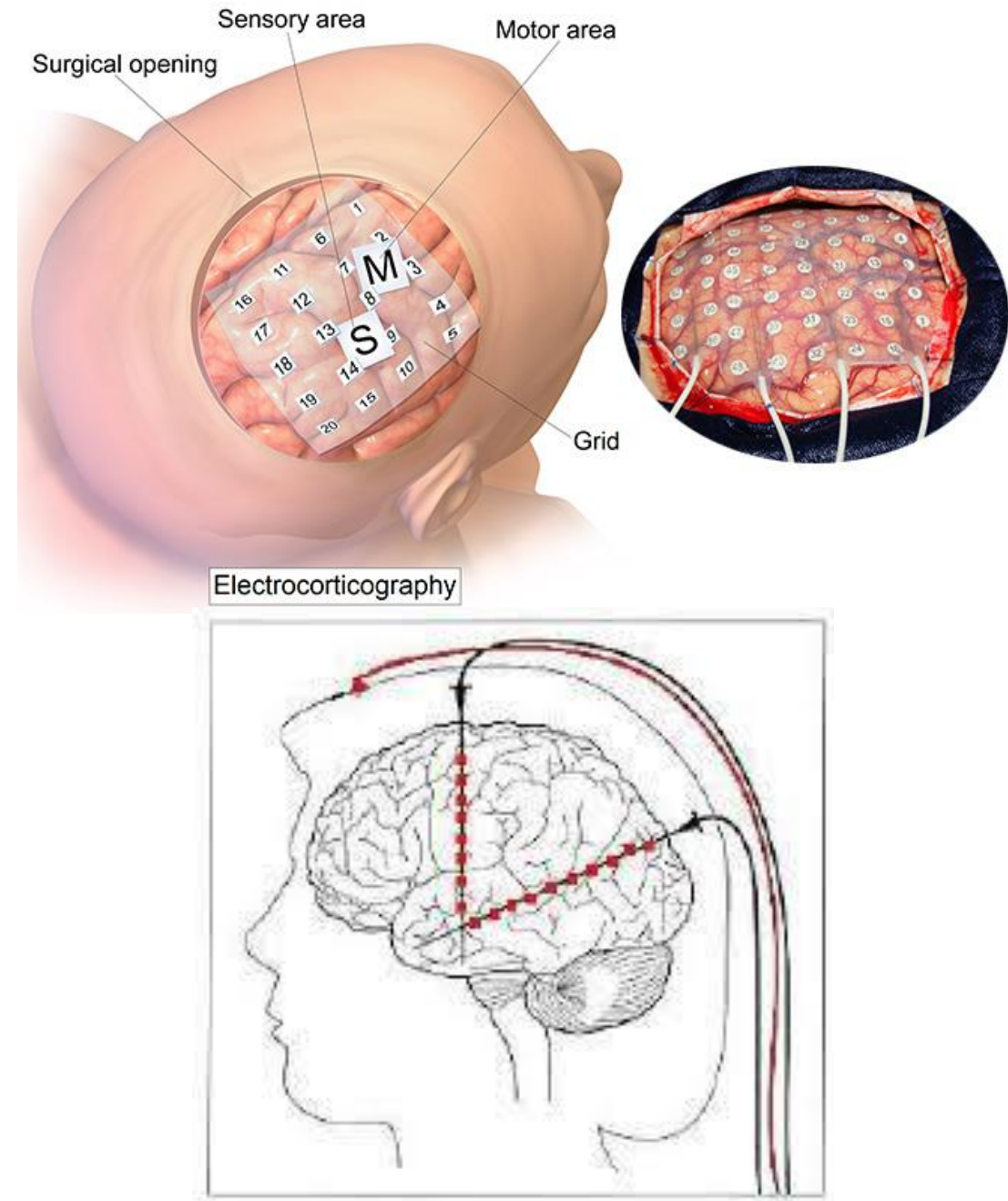
Video EEG:

- This is a test very similar to the Ambulatory EEG test with *video camera recording your test period*. Patients can chose to stay at the hospital while the test is being carried out or can chose to go home.



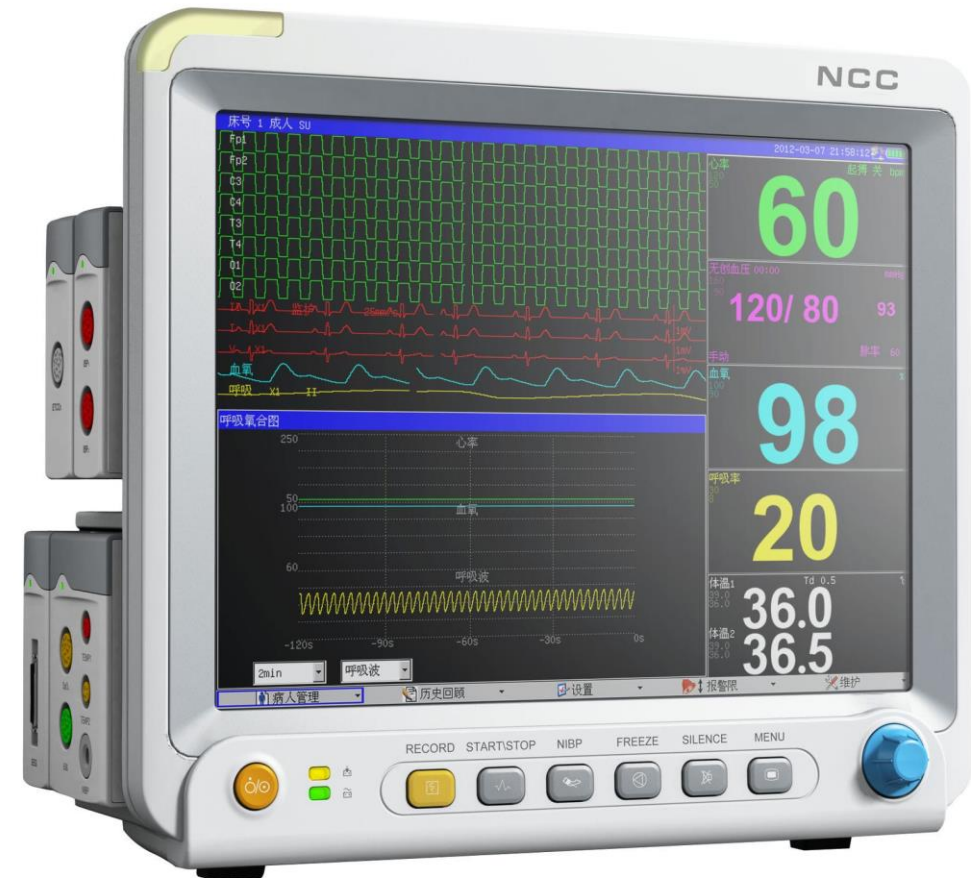
INVASIVE EEG:

- done for evaluation of patients eligible for epileptic surgery.
- 2 different techniques are used for internal electrode placement
- **Subdural EEG Electrodes:** Specialized miniature electrodes are placed on the surface of the brain with the help of surgery.
- **Depth EEG Electrodes:** The electrodes are placed deep inside the brain



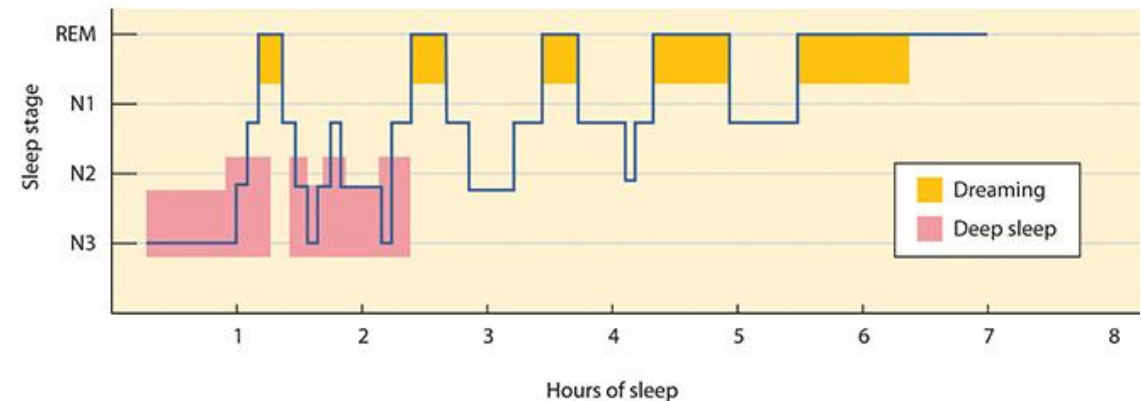
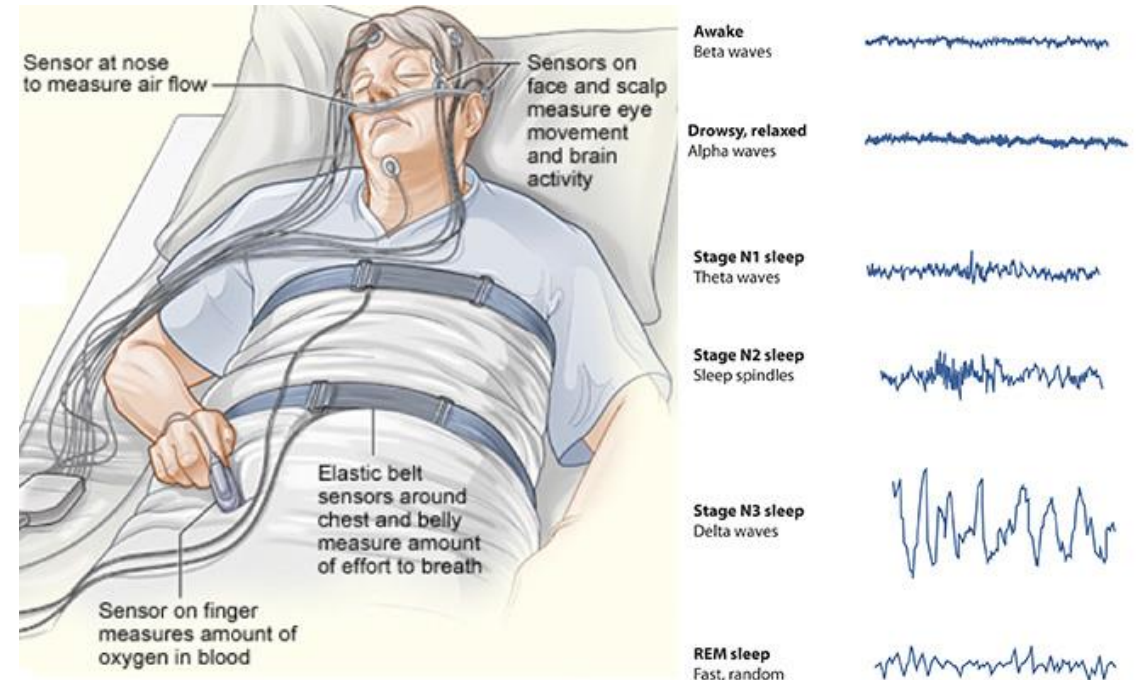
Monitor EEG:

- Patients *recovering from head trauma* or *after a brain surgery* may be monitored using an EEG monitor.
- It can also be used during surgeries as *monitoring tool* and *for delivering the optimal dosage of anesthesia* tailored for the individual patient as per the patient's reaction to the dosage.



Sleep study (polysomnography):

- It is test done for patients experiencing *sleeping disorders*. EEG monitoring is an *important component of this test*.



Neuro feedback EEG:

- It is a kind of *brain training activity* done with the help of an EEG machine.
- This is a procedure designed to *alter and improve the brainwave activity*, thereby helping the patient overcome undesirable conditions like *depression, anxiety, eating disorders ,ADHD etc.*



Thank you.....