

EEG reviewing, BCNEPS tutorial

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www.bcneps.org

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BCNEPS 1 yr certification course 2020-21

- What is our aim?
 - A Clear idea about cl NP, EEG
 - Able to perform a routine test using 10-20 electrode placement technique.
 - Able to identify normal background activity for the age and state
 - Able to identify cerebral reactivity to external stimuli
 - Able to identify common epileptogenic discharges
 - Able to understand a report while you treat a child
- What is Expected at the end of LI ?
 - Perform a routine EEG record in children and adult independently
 - Communicate with referring doctor about the test and the patient
 - Write a descriptive review
 - Understand a report made by an expert neurophysiologist
- What next?
 - SKY IS THE LIMIT
 - LIFE TIME IS THE LIMIT

How much we achieved?

What are the Clinical Neurophysiology tests?

NeuroInvestigative tests

- **EEG:** Routine and special
- Polysomnography (PSG), Actigraphy
 - Epilepsy, Encephalopathy, sleep disorders
- EVOKED potentials: VEP, ERG, AEP, SEP
- EMG, NCV studies

EEG in Therapy: Neurofeedback, BCI

- ADHD
- ASD



What CL NP tests can do

	Confirms	Exclude	Helps	
EEG	Seizure & Epilepsy syndrome	Non-epileptic Paroxysms Epileptic cause for psychomotor deficit	Metabolic dis. Inf Battnes dis	
	Epileptic encephalopathy	Epileptic cause for Transient Behavioral disorder by special technique	Non-Ketotic hyperglycemea	
	Encephalopathy / Encephalitis	Paroxysmal event	Structural anomaly	
	Acute stroke syndrome Retts syndrome		Predict Neurodev. Outcome	
	Localized cerebral dysfunction		Underlying cause	
	SSPE at early stage	Myoclonic epilepsy syndrome		
VEP	Maturation		Neurometabolic	
ERG	Ant .Chamber defect		Neurometaboic dis	
EMG, NCV	Neurogenic	Pathology at Peripheral Nervous system	Disorder of PNS	
	Myogenic, Myopathic	Neuro-muscular junction	Muscle and	
	Neuro –muscular junction defect	Muscle	Neuromascular junction	



What does the EEG record?

Mainly NOISE!!

Volume Conduction

- The electrical activity flows through the tissue between the electrical generator and the recording electrode.
- Thus, the EEG is a 2-D representation of a 3-D reality, which poses a problem in localizing the sources of the electrical activity
- **Inverse problem:** Simulating the potentials at the electrode positions from current sources inside the brain is known as the EEG forward problem; inference of the position of the current sources from electrode potentials is known as the EEG inverse problem or **the neural source imaging problem** (Grech et al., 2008, Brannon et al., 2008)





NP Basis of EEG

The neurophysiological basis of EEG can be summarized conveniently as follows at present

- 1. Repeated waves recorded from scalp or from brain surface are summated Synaptic potential generated by the pyramidal cells
- 2. Synaptic Potentials are the response of the cortical cells to the rhythmic discharges from thalamic nuclei
- 3. Frequency and size of the thalamic discharges (hens the cortical potentials) are determined by the special arrangement of excitatory and inhibitory interconnections among the thalamic cells
- 4. During 'activation' inputs from reticular formation abolish the rhythmic discharges in the thalamic nuclei and cause the cortical potentials to become desynchronized.

Neural basis of the EEG



The principal generators of EEG fields measured at the scalp are graded synaptic potentials; i.e., EPSPs and IPSPs of the pyramidal neurons.

At the synaptic site of an EPSP there is an **active current sink (extracellular negative** field).

Positive ions migrate to the cell and depolarize the membrane. At the distal part of the cell (body and distal dendrites) a passive current source out of the cell is associated with **extracellular positive field**.

EEG fields are primarily generated by the large, vertically oriented pyramidal neurons located in cortical layers III, V, and VI.

The electrical activity from deeper generators gets dispersed and attenuated by volume conduction effects.

EPSPs and IPASPs S. are response of the cortical cells to the rhythmic discharges from thalamic nuclei





 Brain is not a sphere, Its surface is not directly parallel to the overlying structure where electrodes are placed

2. Each small electrode is averaging the activity within 1inch area, limit of such region depends on many factors

3. Slight alteration of electrode placement would result in the electrode averaging the activity from a different region of the brain

4. Appropriate electrode placement and Montage setting is important for localizing the disturbed area

5. All EEG record are practically bipolar event when theoretically unipolar



How to collect and organize the data

• During recording:

- Machine calibration,
- Impedance checking,
- removing artifacts,
- patient care & activation procedure during data collection
- Montage: To organize data for reviewing

Commonly used montages

- Longitudinal OR anterior posterior (M1)
- Transvers (M2)
- Multiphotic (M3)
- Average (M4)
- Referential (M7)





- F Frontal lobe
- T Temporal lobe C Central lobe
- P parietal lobe
- O Occipital lobe
- "Z" refers to an electrode placed on the min-line



Reviewing: Montage we use





Musts: for EEG reviewing

- Patient's Age, sex, state
- Equipment Setting
- Knowledge about
 - Recording technique
 - Normal BG. activity for the age and state (normative data and their variations)
 - Cerebral reactivity to activation procedure
 - Artifacts
 - Well established/described cerebral dysfunction

Repeated reviewing use all the montage

EEG reviewing: organize your description

- Background activity
- Reactivity to eye closure
- Effect on PS
- Hyperventilation
- Drowsy state
- Sleep stages:
 - Early sleep stage/ drowsy state
 - Stages 1st, 2nd, 3rd, 4th
 - REM sleep
- On awakening
 - Arousal phenomena

WHAT TO LOOK FOR IN EEG RECORD?

Background ACTIVITY (spontaneous and reactivity to stimuli)

- Symmetry: IN voltage/ amplitude, IN frequency/ Hz / ____ c/sec
 - Mild asymmetry
 - Marked asymmetry

Reactivity

- to eye closure
- Photic stimulation
- Hyperventilation
- Activities in Drowsy state
- Sleep state
- Discharges of spike, polyspikes, sharp waves/transients, polyspike-wave complexes
 - Transient (focal, multifocal, bursts) /paroxysms/ persistent/ runs
 - Localization, describe the appearance of discharges
 - Repeated, periodic, Persistent
 - Synchrony / asynchrony
- Bursts (transient bursts/ recurrent/ periodic/ repeated
- Stretches of attenuation, periods of attenuation, Suppression
- Discontinuous/ Continuous
- Specific pattern (Hypsarrhythmic/ B-S/ PLED/CSWSS

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EEG Rhythms



Spikes range from 20 to less than <70 milliseconds (ms) High-amplitude1

Sharp waves pointed peak and duration of about 70 to 200 ms

Spike-wave complexes (SWCs) are the repetitive occurrence of a Spike followed by a Slow Wave, although a run of 3 seconds is required to classify a record as SWC.

Polyspikes are multiple repetitive spikes occurring at about 20 Hz

Hypsarrhythmia is defined as continuous (during wakefulness), highamplitude (>200 Hz), generalized polymorphic slowing with no organized background and multifocal spikes



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Photic stimulation

- A series or train of Photic Strobe 3-4 upto 20-30/sec without interruption
- IPS
- Effect?
 - Photic driving response represents repetitive visual evoked potentials produced in response to the photic flash. The response occurs in a time-locked fashion
 - A photoparoxysmal response (PPR), formerly known as photoconvulsive response, occurs when IPS generates bilaterally synchronous <u>epileptiform</u> <u>discharges</u> that may outlast the stimulus by several seconds. At times, this may precipitate a seizure (see Fig. 7.7).
 - PPRs may occur as one of two subtypes:
 - (a) limited to the stimulus train (self-limited), or
 - (b) self-sustained (nonself-limited). The latter is more likely to be associated with clinical seizures (Kasteleijn-Nolst Trenité et al., 2012).

Other responses (physiological) to PS

- that resemble pathological findings may be identified during IPS.
- A photomyogenic response consists of repetitive contractions of the <u>frontalis muscle</u> synchronized to the light flash at a delay of 50–60 ms.
- A photoelectric response occurs as the result of a photochemical response generated by the electrodes, and can coexist with other waveforms, such as eye movement artifact, to mimic a PPR.
- A physiologic electroretinal response generated by retinal ganglion cells may occur in the frontal leads, mimicking an abnormality. Psychogenic nonepileptic attacks may at times be induced by HV and IPS together (Benbadis et al., 2000; Bodde et al., 2009).

Diagnostic importance of PS

- Diagnostic
 - Photo-sensitive epilepsy
 - IGE
 - Certain metabolic and degenerative diseases
- Prognostic evaluation
- Management
 - Appropriate AED selection
 - Use dark glass

More clinical importance: Unverricht–Lundborg's Disease

- A. Magaudda, in Encyclopedia of Movement Disorders, 2010
- EEG
- At the disease onset, background activity (BA) is normal or mildly slow (BA at 6–7 Hz).
- Superimposed slower activity at 4–5 Hz can be present, resulting in an irregular appearance of BA. BA keeps stable during the course of the disease.

• During the initial years of the disease, EEGs show spontaneous brief bursts of GSWD, characterized by very rapid spikes (**Figure 1**). Focal epileptiform abnormalities can be observed over the central and posterior regions of the scalp. Intermittent photic stimulation (IPS) provokes the appearance of GSWD and increases myoclonus, both generalized and focal.

Train of photic strobe without interruption



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Photic stimulation, intermittent PS

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Fig. Photoparoxysmal response (PPR) during 30-Hz photic stimulation in an 18-year-old girl with photosensitive genetic generalized epilepsy. Note the anterior-dominant spike and slow wave discharge outlasted the stimulation (nonself-limited PPR).



Figure EEG in a 15-year-old ULD patient, showing normal background activity and brief bursts of irregular generalized spike – wave discharges, characterized by very rapid spikes.

During sleep, a normal representation of physiological sleep patterns and a reduction of GSWD during nREM and <u>REM sleep</u>, along with the presence of fast spikes and polyspikes over the central and the vertex regions during REM sleep, were observed.

Long-term evolution of EEG in ULD is characterized by no relevant deterioration of BA, a gradual reduction of GSWD and PPR, correlating with good seizure outcome, and a progressive disappearance of physiological sleep patterns 10–20 years after the disease onset.

Hyperventilation

- Purpose?
- Process?
- Response?



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Sleep

- Normal sleep stages
 - Non Rapid Eye Movement sleep (NREM)
 - Rapid Eye movement Sleep (REM)
- Clinical importance
- Normal variants (age dependent)
- Misconception results in wrong interpretation

Normal human sleep cycle, 1 cycle = 90 min.



Sleep Cycles

Stage 1 lightest (1-7 min)

-Heartbeat slows down -Breathing slows down -Eye movements slow down -Muscles relax, and might occasionally twitch -Brain waves begin to slow down

Stage 2 light (10-25 min)

-Heartbeat and breathing slow down even more -Muscles relax even more -Body temperature drops -Eye movements stop -Brain wave activity slows

Stage 3

deep sleep (20-40 min)

-Heartbeat and breathing slow to the lowest levels they will reach during sleep -Muscles stay relaxed -Brain waves slow down even more

Stage 4

REM (20-40 min)

-Behind your eyelids, your eyes move rapidly from side to side -Breathing speeds up and can become irregular -Heart rate increases -Blood pressure increases

EEG activities in different stages

Alpha rhythm on EC disappears, Central theta W, Hypnogogic hypersynchrony appears

Vertex Sharp Trans K complexes and sleep spindles appear successively

Sleep spindles starts reducing, background slowing starts V. Deep sleep= slow background

Mixed activity in the BG almost like awake stage



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rtex sharp transients/K complexes: normal sleep stage 1 and 2 (*drowsy and early sleep*), mistaken for epileptiform discharges

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sleep stage2,Sen-14, Hf-30Hz, Tc- 0.03s



Mu rhythms in alert state, lateralized, discharges over left side

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Importance of sleep and wake recording, 4 mo F, MC jerks since birth,

EEG Viewer - Juthi, FirstName - 3/15/2021 Б \times Navigate Format Tools Help File View >-- >+- Y 🛛 🗒 ē 10 x Events 00:00:00 00:02:00 00:04:00 00:06:00 00:08:00 00:10:00 00:12:00 00:14:00 00:16:00 00:18:00 00:22:00 00:24:00 00:28:00 00:20:00 00:26:00 0 Fp1-Ref Fp2-Ref F7-Ref• F3-Ref• Fz-Ref• F4-Ref+ F8-Ref• A1-Ref• T3-Ref• C3-Ref• Cz-Ref• C4-Ref• T4-Ref+ A2-Ref• T5-Ref• P3-Ref• Pz-Ref+ P4-Ref T6-Ref• 01-Ref+ 02-Ref+ Oz-Ref• ECG-Bip EMG 12:35:24 PM As Recorded, 20 mm/sec, 100 µV/cm, 40.0 Hz, 1.600 Hz, 60 Hz Ready 12:35:24 PM 00:26:42 2-16 D

4yr, F, ?Absent minded for a few seconds daily for last 2 months, h/o 8 episodes of Febrile szs during 3mo till 12 month age, FT delivery by CS, NC Suggestive of generalized epilepsy with absence seizures.

8yr, F, c/o sudden screaming- gen sz for >60 minutes, ended with vomiting, headache+ vomiting for 3 yrs, student.

8 Yrs boy, c/o GTCSz on the previous day, has eye blinking, transient discrete jerks for last 6 months, no recognizable comorbidity. What is missing here?, Technician's job?

Ready

7:12:13 PM 00:04:43

Artifact?

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Localized dysfunction? Describe this

 \times

Description of epileptiform discharges

➢Morphology

Size & shape/ amplitude, frequency, distribution, duration

≻How they are arising

- >In relation to time, state of the patient
- Spontaneous or Provoked by activation process
- Synchrony of firing
- ➢Spatial, Temporal distribution
- ≻In short or prolonged runs
- ➢Arising as periods

Type of discharges

- GENERALIZED
- FOCAL / LOCALIZED
- LATERALIZED
- BILATERAL
- SYNCHRONIZED/ ASYNCHRONIZED
- ISOLATED
- REPEATED
- PERIODIC
- Specific pattern of discharges
 - Burst suppression
 - Continuous / doscpmtomipis
 - Hypsarrhythmic

Effect of Machin setting

Off Low cut filter

Off HFF

Power line interference

High cut at 70Hz

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High cut at 15 Hz

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Sensitivity: 100μ V/cm to 70μ V/cm

Changing paper speed

Q & A

09/08/2021

PATIENT INFORMATION:	PATIENT NAME: Fatema DATE OF BIRTH:	P ID : D- 1443147 REC. DATE : 03/07/2021		
	AGE: 9m SEX: F GESTATIONAL AGE:	OFC: 41cm ENO: 31517 HAND DOMINANCE: Rt		
CLINICAL SUMMARY:	REFFERED BY: Prof. Dr. Md. Mizanu Poor eye contact, can't sit, no clinical sz, Jaundice.	r Rahman FT hospital delivery by CS, N.		
CURRENT TREATMENT:	Trihexy, Sirdalud.			
PATIENT STATE DURING RECORDING:	The child was sleeping at the beginning la	ter awake during recording		
PREVIOUS EEG	Nil			

DESCRIPTION: Irregular 4-5 c/s 30-45 μ V activities are seen over both the hemispheres mixed with slower components in the background. Repeated complexes mixed with polymorphic activity, 1-2 c/s 70-200 μ V are noted over the temporo-parietal and occipital regions predominantly involving the right hemisphere from beginning to end. No cerebral reactivity is observed over the posterior region on eye closure. Frequent sleep spindles are noted over the fronto-central area during sleep state.

COMMENT: This record shows high amplitude epileptogenic complexes mixed with polymorphic slow waves over the posterior regions. Other activities in asleep state are within normal limit for the age and state of the child. The EEG feature is suggestive of epileptogenic cerebral lesion. Please correlate.