EEG Course

A Systematic Approach to the Electroencephalogram & Activation Methods

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OUTLINE

1. Step of EEG approach
2. Activation Methods
   2.1 Reactivity
   2.2 Hyperventilation
   2.3 Photic stimulation
   2.4 Pattern stimulation
   2.5 Other stimuli
   2.6 Sleep & sleep deprivation
   2.7 Pharmacologic stimulation
Algorithm displaying an approach to the orderly visual analysis of EEG activity

**Algorithm:**
- **EXAMINE EEG**
  - **BACKGROUND**
    - NORMAL FOR AGE/STATE
    - ABNORMAL
      - GENERALIZED
      - FOCAL
  - TRANSIENTS
    - CEREBRAL
      - NORMAL FOR AGE/STATE
      - NONSPECIFIC
    - NONCEREBRAL
      - NORMAL FOR AGE/STATE
      - NONSPECIFIC
  - EPILEPTIFORM
    - SIGNIFICANT
    - BENIGN VARIANTS
      - FOCAL
      - GENERALIZED

Epilepsia 2002;43 (suppl 3): 17-26

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**Activation Methods**
EEG study in children:

Sedation is not routine!

Excessive beta activity due to chloral hydrate
2. Activation Methods

2.1 Reactivity (eye opening & closing)
2.2 Hyperventilation
2.3 Photic stimulation
2.4 Pattern stimulation
2.5 Other stimuli
2.6 Sleep & sleep deprivation
2.7 Pharmacological activation

2.1 Reactivity

- Children: the occipital alpha rhythm may totally block with the eye open.
- Adults: 24%, no alpha blocking

_Bancaud’s phenomenon_
- unilateral failure to attenuate with eye opening
- indicates abnormality of the same hemisphere that fails to attenuate
2.2 Hyperventilation (HV)

- Alternation of PCO₂ is the most important factor in producing the EEG response to HV.

- **Procedure**: over-breathe for at least 3 min.
  (children: cry or sob during the recording)

- The magnitude of HV response depends on
  - Effort
  - Age
  - Posture
  - Blood sugar (< 80 mg/100mL)

- The generation of epileptiform discharges during HV:
  - 80% for idiopathic generalized epilepsies
  - 50% for symptomatic generalized epilepsies
  - <10% for localization-related epilepsy
2.2 Hyperventilation (HV)

**Contraindications**
- Severe cardiac disease
- Recent myocardial infarction
- Active or recent asthma
- Recent stroke or TIA
- Intracerebral hemorrhage
- Severe carotid stenosis
- Moya-moya disease
- Hyperviscosity state
- Sickle cell anemia
- Uncontrolled hypertension

**Relative contraindications**
- Not cooperate patient
- A child whose EEG has already contained frequent generalized spike and wave

2.1 Hyperventilation (HV)

- **Normal response:**
  - buildup of medium to high amplitude, bisynchronous delta and theta waves.
  - Adults: 10% response; anterior dominant
  - Children: 70% response; ant/post dominant
    - (85% occurred between 8 and 12 years of age)
  - return to baseline within 60 seconds after stop HV
  - often includes FIRDA, or OIRDA in children
Hyperventilation

7-year-old girl

Hyperventilation

16-year-old girl
2.1 Hyperventilation (HV)

Abnormal response:

1. Lateralized or localized slowing

2. Delayed symmetrical or lateralized slowing
   • Moyamoya disease: a buildup of slowing several minutes after HV ends (~ 5 min. after HV ends)

3. Asymmetry of background activity
   • Usually the abnormality is on the side of higher amplitude response

4. Epileptiform patterns
   • >80% of untreated children with absence seizures
   • typical anterior-dominant 3-Hz spike-and-wave

*Pseudo-absence seizures*: impaired responsiveness during HV +
generalized high amplitude 2-to 3-Hz activity
Hyperventilation (case 2)

Baseline

Reactivity
Start HV for 20 seconds

sensitivity 7 uV/mm

Start HV for 20 seconds

sensitivity 30 uV/mm
Hyperventilation (case 3)
Brain Dev. 2007 Oct;29(9):603-6

140th second of HV

Re-build up phenomenon @ 60th second after stop HV
case 3: Moyamoya

Note
2.3 Photic Stimulation (PS)

Photic stimulator

- Photic stimulator characteristics
  - Max. intensity > 100 Nit-s per flash
  - Circular field diameter of 13 cm
  - Granular diffuser producing light diffusion similar to that of the Grass stimulator
  - Central fixation point on diffuser
  - Attachment of patterns available
  - Single flashes or trains that can be delivered with constant intensity from 1 to 60 Hz
2.3 Photic Stimulation (PS)

• **Procedure**
  – IPS should not be performed during or within 3 min of HV
  – Nasion-to-lamp distance of 30 cm
  – Longitudinal bipolar or common reference montage
  – Flash trains of 10s with at least 7-s intervals
  – Eyes open for first 5s of IPS and then closed
  – Eyes fixated on center of stimulator
  – IPS frequencies: 1,2,3,4,6,8,10,12,14,16,18,20,60,50,40,30,25
  – IPS is stopped abruptly if a PPR appears

*IPS = intermittent PS
*PPR = photoparoxysmal response

2.3 Photic Stimulation (PS)

• **Normal response:**
  – Rhythmic, occipital-dominant waveforms
  – Harmonic (an integer multiple) or subharmonic (an integer dividend) of flash frequency
  – Onset: 70- to 150-millisecond delay
  – At slower flash rates (<5Hz), the photic response consists of a diffuse light evoked potential
  – Photomyogenic responses
  – Unilateral driving may be seen. Interpretation as abnormal usually requires other abnormal features.
photic driving (5Hz)

Asymmetrical photic driving response
Photomyogenic (photomyoclonic) response

- First described by Gastuat and Remond
- Prominent in 1% of individuals
- Brief, repetitive muscle spikes in the anterior head region
- Electromyographic potentials time locked to the flash frequency, anterior-dominant.
- Prominent with emotional tension or metabolic/toxic states
- Distinguish from PPR by immediate cessation of the response at the end of stimulation and prominent EMG activity
- Unknown clinical significance
2.3 Photic Stimulation (PS)

- Abnormal responses:
  1. Photoparoxysmal response (photoepileptiform, photoconvulsive)
  2. Abnormal response in specific cerebral disorders

1. Photoparoxysmal response (PPR) (Photoepileptiform response, PER)

- Generalized spike-and-slow wave and polyspike-and-slow wave complexes
- ~4% of patients with epilepsy have a PPR
- 70% - 77% of patients with PPR have epilepsy
- Maximal incidence: 6-15 years of age
- Clinical correlation:
  1. GTC
  2. Myoclonic (JME ~38%)
  3. Absence (~24%)
PPR

- Two types of PPRs:
  1. Prolonged or self sustaining
     - outlasts at least 100 ms, suggests probable epilepsy (93%)
     - generalized spike-and-wave response shows a strong association with epilepsy
  2. Self-limited
     - ceases before or when the flash stops
     - not diagnostic for epilepsy

PER

- The most suggestive features of posterior-dominant PER are:
  1. Medium- to high-amplitude spikes or sharp waves persists well beyond (>200 msec.) the termination of the flash stimulus.
  2. Association with clinical convulsive or nonconvulsive seizure activity.
2.3 Photic Stimulation (PS)

- **Pitfalls:**
  - PS is less effective when performed during sleep
  - Unilateral monocular stimulation or stimulation during conjugate ocular deviation away from the stimulus is less effective than binocular gaze-directed stimulation
  - Repeat the same stimulus train to verify that PPR is related to the flash stimulus
    - Don’t repeat immediately (habituation with blocking of the response will occur)
    - Repeat same stimulus train after > 30 seconds later
2. Abnormal responses in specific cerebral disorders

- ↑ amplitude of photic driving found in
  - cortical epileptogenic lesions
  - skull defects

- ↓ amplitude of photic driving found in
  - destructive brain lesion

- Photosensitivity
  - partial epilepsy ~ 2.8%
  - generalized epilepsy (idiopathic) ~21%
2. Abnormal responses in specific cerebral disorders

Q: What is the pathognomonic EEG findings in patient with late infantile NCL
   (Bielschowsky-Jansky form of Batten’s disease)
2.4 Pattern-activated PPR

- The first report of a patient with pattern sensitivity appeared in 1953 by Bickford et al.
- Virtually all patients with pattern sensitivity also show sensitivity to PS
- However, very few patients with sensitivity to PS also have pattern sensitivity
- Distribution of elicited epileptiform discharges:
  : generalized in two thirds of patients,
  : restricted to the posterior head region in one-third.

What is the most effective activating pattern?

A
!

B
!

C
!

D
!

E
!

F
2.5 Other stimuli activation

- may be used in cases where episodic symptoms or signs suggest a convulsive disorder triggered by known stimuli and where a diagnosis is wanting.

- These procedures should be used with caution and with the intention of inducing EEG abnormalities while avoiding precipitation of seizure.

- The benefits of the diagnostic information obtainable by activation of EEG discharges must be weighed against the minor risk of inducing a seizure.

1. **Pattern sensitivity**
   - Virtually all patients with pattern sensitivity also show sensitivity to PS
   - However, very few patients with sensitivity to PS also have pattern sensitivity

2. **Video game**
   - Hormes et al., 1995: 40 patients with PPR to stroboscopic PS, 30% of these patients also had sensitivity to video games.

3. **Auditory stimuli**
   - Sudden loud noise (reflex epilepsy)
   - Specific musical piece (musicogenic epilepsy)
2.5 Other stimuli activation

4. Reading

**Primary reading epilepsy** (intrinsic or perceptive):
- epileptiform bursts occur after a period of reading
- max. in the parieto-occipital regions
- assoc. with clinical jaw jerking or ‘clicking’ while reading

**Secondary reading epilepsy** (extrinsic or sensorial):
- epileptiform discharges appear not only with reading but also under other conditions
- assoc. with pattern sensitivity
Bursts of low voltage monomorphic slow activity during reading aloud
2.5 Other stimuli Activation

5. Mental concentration
   - mental calculation with eye closure
   - rarely, mental calculation will precipitate a seizure

6. Tactile stimulation
   - touching certain parts of body may induce or abolish epileptiform activity and seizures in some patients
   - Somatosensory epilepsy
     - interictal medium to high amplitude spikes over the perisylvian or central parasagittal head regions
     - evoked by tapping on the distal contralateral limbs
2.6 Sleep & Sleep Deprivation

**2.6 Sleep Activation**

During sleep
- increase of epileptic discharges rates from 77 to 98% in absences and GTC combined with absences.

During sleep deprivation
- permit only about 4 hours of sleep
- increase of epileptiform discharges rates from 50 to 80%.
2.6 Sleep Activation

Epileptic syndromes activated by sleep:

- CSWS
- LGS
- Benign JME
- Benign Rolandoic epilepsy (BECTS)
- Frontal lobe epilepsy
- ADNFLE
- Benign occipital epilepsy in infancy
- Nocturnal epileptic myoclonus
- Epilepsy with generalized tonic-clonic seizures on awakening
2.7 Pharmacological activation

• Discontinuing AEDs for video-EEG recording
Drug Effects on EEG

**Background slower** (theta and delta)
- “Older” AEDs e.g. PB, PHT, CBZ, VPA
- Neuropsychiatric drugs e.g. clozapine, TCA, lithium

**Excess beta**
- Barbiturates
- Cocaine
- Methylphenidate
- Withdrawal from alcohol and barbiturates
- Benzodiazepines
- Amphetamine
- Tricyclic antidepressants

Triphasic waves
- Drug intoxication e.g. VPA-associated hyperammonemic encephalopathy
- Other drugs e.g. baclofen, lithium, L-dopa, pentobarbital, SSRI
Drug Effects on EEG

Epileptiform activity

a) Bursts of bisynchronous spikes or polyspikes
   - high doses of clozapine, lithium, phenothiazines, SSRI, TCA
   - acute withdrawal of alcohol or barbiturates
b) Augmentation of epileptiform discharges
   - reduction of AEDs
   - morphine in neonates

Coma pattern

- Drug intoxication e.g. pentobarbital, BZD

Clozapine. Bisynchronous spikes and excess theta may be produced by clozapine, as this segment shows.
Take home messages

- Step of EEG approach should be always kept in mind before start to interpret EEG recording.

- Good understanding of EEG waveforms, and specific pattern recognition will increase accuracy of EEG interpretation.

- Skill of EEG interpretation is crucial and need to be increased by regular practice.