Understanding Electroencephalography

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The Source of EEG

EEG = a measure of cerebral electrical activity

- The generator sources for EEG waves are within the cerebral cortex
- Electrical activity recorded on the scalp is produced by extracellular current flow associated with summated excitatory and inhibitory postsynaptic potentials (EPSPs and IPSPs)
- Individual action potentials do not contribute directly to EEG activity
Synaptic Potentials: The Basis of EEG Activity

- Synaptic potentials are of much lower voltage than action potentials, but the produced current has a much larger distribution.
- PSPs have a longer duration and involve a larger amount of membrane surface area than APs.
EPSPs and IPSPs

EPSP – produces a change in membrane permeability within a select portion of the cell membrane resulting in a net influx of + ions that depolarizes the cell

IPSP – selective activation of either Cl⁻ or K⁺ channels resulting in a net outward ionic current with hyperpolarization of the cell
EEG: A Reflection of Current

- Spontaneous EEG activity occurs when currents flow across charged neuronal membranes.
- An EEG waveform reflects a summation of PSPs from thousands or even millions of cortical neurons.
- The EEG represents the “average” behavior of large neuronal aggregates.
- The current flow from positive to negative is arranged in a dipole.
The Dipole

Theoretically, the current flows in a 3-dimensional ellipse with the greatest current density along a straight line connecting the positive pole to the negative pole.

The complex arrangement of the brain and head, differences in cell type and function within a region, and physical differences between brain areas result in an approximate dipole that is not a perfect model.
Rhythmical vs. Arrhythmical EEG Activity

- When EEG waves are rhythmical, most of the cells within the given neuronal pool are behaving similarly.
- With arrhythmic activity, there is less correlation with individual cell behavior.
Pyamidal Cells: Principal Current Generators of EEG

- Topographical organization within the cortical mantle corresponds to a dipoles oriented perpendicular to the cortical surface
Factors Affecting EEG Waveforms

- Voltage of the cortical discharge
- Area involved in synchronous activity
- Degree of synchrony
- Location of the dipole generators in relation to the convolutions of the cortical mantle.
Factors that Attenuate Voltage

- Primarily overlying spinal fluid and dura
- Scalp to a lesser extent
- Scalp recorded activity represents “spatial averaging” of electrical activity from a limited area of cortex
- 20-70% of epileptiform activity may not be seen on scalp EEG
  - Involvement of small areas of tissue is associated with much greater attenuation of activity
  - Activity arising from cortex within the walls or depths of sulci may not be recorded
Origin of EEG Rhythmicity

- Rhythmical activity – sequences of regularly recurring waveforms of similar shape and duration
- Rhythmical activity may be locally generated or occur via projected synaptic inputs from deeper structures
- The thalamus, via an anatomic cellular organization, thalamocortical projections and mechanisms that are not fully understood, governs different types of rhythmical activity
  - Sleep spindles
  - Alpha rhythm in the occipital cortex
  - 3 per second spike and wave associated with absence seizures
Vertex Wave and Sleep Spindles
Posterior Alpha Rhythm

<table>
<thead>
<tr>
<th>Electrode Pairs</th>
<th>Waveform</th>
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<tbody>
<tr>
<td>Fp1-F3</td>
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<td>F3-C3</td>
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Epileptiform Discharges: Spikes

- Indicate susceptibility to seizures
- May be generalized or focal
- Focal spikes – cortical spikes are associated with synchronous paroxysmal depolarizing bursts occurring in neurons within the focus resulting in extracellular current flow that is recorded as surface spikes
The Spike and Wave Complex

- The cellular depolarizing bursts are followed by long-lasting afterhyperpolarization in cells within the focus.
- Additional inhibition occurs in surrounding cortex, thalamus and other subcortical areas.
- This results in a surface slow wave that follows the individual spike.
Left Temporal Spikes

Fp1-F7
F7-T3
T3-T5

Spike and slow wave complex
Neuronal Synchronization

The interictal spike may be initiated by a spontaneous burst in one or a few cells.

Each cell has excitatory connections to a number of other cells.

Excitatory connections will allow burst propagation if inhibition is decreased, absent or simply overcome.

Loss of effective dendritic inhibitory synapses may occur over time.
Generalized Spike-and-Wave

- A pathological exaggeration of cortical excitability is the basic disturbance and appears to initiate the process.
- Cortical spikes precede epileptiform discharges in depths.
- Thalamocortical connections are necessary for triggering and phasing the spike-and-wave bursts.
- The brainstem reticular formation appears to modulate spike-and-wave activity by modifying the level of cortical excitability.
- Substantia nigra involvement in some way is essential in the production of generalized convulsions.
EEG Frequencies

- Delta 0 - 4 Hertz
- Theta 4 - 7 Hertz
- Alpha 8 - 13 Hertz
- Beta > 13 Hertz
EEG in Neonates

Premature newborn – due to the incomplete development of neuronal connections, synapse formation, myelination, etc., EEG activity prior to 30 weeks estimated gestational age (EGA) is discontinuous and very “abnormal” appearing.

Focal sharp discharges in frontal and temporal regions are normal to some extent until about 44 weeks EGA.
Trace Discontinue in 27 week EGA Neonate
2 month old with Enterococcal meningitis and left posterior temporal focal electrographic seizure
2 month old with Enterococcal meningitis with an electrographic seizure discharge, maximal right frontal
2 month old with Enterococcal meningitis and right posterior temporal focal electrographic seizure
2 month old with Enterococcal meningitis and bilateral independent focal electrographic seizures
Progression of EEG in Childhood

Occipital rhythmical activity = Alpha rhythm

- 3-5 months: 3.5-4.5 Hertz
- 12 months: 5-6 Hertz
- 3 years: 7.5-9.5 Hertz
- 9 years: >9 Hertz
EEG During Sleep

- **Stage I sleep**
  - Dropout of alpha rhythm
  - Intermixed slowing

- **Stage II sleep**
  - Increased intermixed theta and delta slowing
  - Vertex waves, sleep spindles and K-complexes
  - Positive occipital sharp transients of sleep (POSTs)
Progression of Sleep

- Stage III sleep
  - Increased delta slowing
  - Central vertex activity diminishes
- Stage IV sleep
  - Marked delta slowing
  - Absence of vertex activity
- Stage III and IV = Slow wave sleep
Stage 3-4 Sleep
REM Sleep

- Low voltage mixed frequency activity with faster components
- Absent vertex activity
- Decreased EMG activity
Focal Epileptiform Discharges and Abnormalities

- Spikes and sharp waves
- Phase reversal localizes the focus
- Recorded seizures
- Focal slowing may indicate an underlying epileptogenic focus, structural lesion or injury, or postictal effect
Localization: Phase Reversal

- Each channel records the potential difference between two electrodes
  - G1 – G2
  - Negative is up
  - If G1 is more negative than G2, the deflection will be up
  - If G2 is more negative, the deflection will be down
Generalized EEG Abnormalities

- Generalized spike and wave
- Generalized polyspike-wave or multispike-wave
- Generalized slow spike and wave
- Generalized 3 Hertz spike-wave
- Generalized slowing
- Generalized suppression
- Generalized burst suppression
- Electrocerebral silence
Generalized Spike-Wave
Generalized Polyspike Wave
3 Hertz Spike and Wave
Generalized Delta Slowing
EEG in the Evaluation of Possible Epilepsy

- For patients with epilepsy, a single EEG will reveal epileptiform activity in:
  - 30-40% with an awake EEG only
  - 60-70% with wakefulness and sleep
- Some patients will only have an abnormality detected if an actual seizure is recorded
- A normal EEG does not rule out a diagnosis of epilepsy
EEG Monitoring in Critical Care

- Many patients with head injury, any form of encephalopathy, or severe illness are at risk for seizures.
- Patients who are mechanically ventilated are often sedated and pharmacologically paralyzed and seizures can only be diagnosed with EEG.
- The diagnosis of nonconvulsive status epilepticus can only be detected and monitored with EEG.
- Monitoring progression of coma and potentially cerebral death.
11 y/o boy with severe cardiomyopathy on ECMO following cardiac electromechanical disassociation
11 y/o boy with severe cardiomyopathy on ECMO with right temporal electrographic seizure
### Focal Status Epilepticus

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<tr>
<th>Channel</th>
<th>Graph</th>
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<td>T4-T6</td>
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<td>P4-O2</td>
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1 sec  50 µV
11 y/o boy with severe cardiomyopathy on ECMO with left hemispheric suppression due to infarction
EEG Monitoring and Epilepsy Surgery

- 20% of patients with epilepsy cannot be controlled with medications
- Focal onset seizures can sometimes be selected and treated with surgical resection of the epileptogenic focus
- Multiple methods are can be employed
Clinical Case

- 8.5 year old boy with onset of seizures at age 7
- Seizures begin with a tingling sensation in the R hand followed by extension and posturing of the R arm
- Seizures were never completely controlled with about 1 seizure per week for the first year
- December, 2001: Seizures began to dramatically increase
- January-March, 2002: Numerous seizures per day with up to 100 in a single day, and episodes of status epilepticus lasting up to 1 hour
Typical Seizure
EEG: Recorded Seizure

*Fp1-F3
*F3-C3
*C3-P3
*P3-O1
Fp2-F4
F4-C4
C4-P4
*P4-O2
3D-MRI Image
3D-MRI with Coronal Cut
Craniotomy with Lesion Localization
Grid Placement
Cortical Grid Map

R.L.
3/22/02
Craniotomy Closure
Electrocortical Seizure Recording
Functional Cortical Mapping
Cortical Stimulation: Arm
Functional Cortical Map
Cortical Resection
Second Surgery: Stereotactic Lesion Localization
Depth EEG Recording
Outcome
2 month old with Enterococcal meningitis with an electrographic seizure discharge, maximal right frontal