Electroencephalogram (EEG)

**Types of Recording**
- Scalp electrodes (monopolar needles or small surface discs) \( \Rightarrow \) EEG
- Electrodes on the exposed surface (cortex) of the brain \( \Rightarrow \) ECOG
- Electrodes inside the brain result in a depth recording (very little damage results)

**Electrode Locations**

- Standardized locations referenced to anatomical landmarks on the skull (nasion, bregma,inion, frontal to occipital); (auditory meatus (ear hole)) left to right side.

Called 10-20 electrode system because it is located 10% of circumference (nasium to inion) from nasium followed by 20% increments as shown.

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Figure 4.28 The 10-20 electrode system This system is recommended by the International Federation of EEG Societies. (From H. H. Jasper, *The Ten-Twenty Electrode System of the International Federation in Electroencephalography**
The Normal EEG

Normal relaxed adult human
Constantly fluctuating signal with a dominant frequency of 10 Hz and amplitude range 20-200μV. This is called alpha rhythm.
Table 11-3 EEG Waveform Terminology

<table>
<thead>
<tr>
<th>Waveform</th>
<th>Frequency (Hz)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha rhythm</td>
<td>8-12</td>
<td>Parietal-occipital; associated with the awake and relaxed subject; prominent with eyes closed</td>
</tr>
<tr>
<td>Beta rhythm</td>
<td>low valley 15-30</td>
<td>More evident in frontal-parietal leads; seen best when alpha is blocked</td>
</tr>
<tr>
<td>Delta</td>
<td>1-3.5</td>
<td>Associated with normal sleep and present in children less than 1 year old; also seen in organic brain diseases</td>
</tr>
<tr>
<td>Theta</td>
<td>4-7</td>
<td>Parietal-temporal; prominent in children 2 to 5 years old</td>
</tr>
</tbody>
</table>

Sleep Stages

EEG recorded during sleep to identify different stages of sleep and also to identify brain pathology. EEG (electroencephalogram - eye movement), EOG, EMG, and respiratory are also recorded during sleep studies. Apnea is highly altered the EEG resulting in a flat trace to low voltage high frequency. Patterns with hernia, anesthetics, deep completely, slow waves result, very deep anesthesia may result in with no discernible slow activity (delta waves) or complete absence of activity (absence).
Clinical Value of EEG

In neurology it is used as a screening test for intracranial pathology. Clinical correlation of EEG patterns with disease states is much ahead of rigorous physiological explanation of these phenomena. Interpretation of EEG's therefore relies on pattern recognition of patterns of frequency, voltage, and waveform.

Epilepsy:
- Groups of neurons in the brains become hyperexcitable and can produce sensory, motor, and autonomic manifestations.
- Scalp EEG's exhibit characteristic spikes (short duration waves, sometimes coupled with theta waves) during an epileptic attack. In petit mal epilepsy, the short duration attacks may not be apparent visually but will show up as brief spike bursts in the EEG.

Space occupying lesions (tumors, subarachnoid hematomas, abscesses)
- result in slow (delta wave) and depression of normal rhythms in the EEG, which allows localization and an estimate of the extent of the lesion. Usually used by bipolar recording techniques. If one of the electrodes is over the slow wave focus and the electrode is used for two different channels, there is a phase reversal in the recordings of these two channels.

Brain injury (result of high, accelerating, and deceleration forces)

Generally, 3 depression in cerebral activity and low frequency activity accompany such injuries. Serial EEG's taken over a prolonged period of time provide useful prognostic information.

NB: Abnormal EEG's can be recorded from normal subjects and EEG findings must be correlated with other clinical findings.
Origin in the Brain of the Brain Waves

The discharge of a single neuron or single nerve fiber in the brain can never be recorded from the surface of the head. Instead, many thousands or even millions of neurons or fibers must fire synchronously, only then will the potentials from the individual neurons or fibers summate enough to be recorded all the way through the skull. Thus, the intensity of the brain waves from the scalp is determined primarily by the number of neurons and fibers that fire in synchrony with one another, not by the total level of electrical activity in the brain. In fact, strong asynchronous nerve signals often nullify one another in the recorded brain waves because they are of opposing polarity. This is demonstrated in Figure 59-3, which shows, when the eyes were closed, synchronous discharge of many neurons in the cerebral cortex at a frequency of about 12 per second, which gave the alpha waves. Then, when the eyes were opened, the activity of the brain increased greatly, but the synchronization of the signals became so little that the brain waves mainly nullified one another and the resultant effect was weak waves of generally higher but irregular frequency, called beta waves.

Origin of Alpha Waves. Alpha waves will not occur in the cortex without connections with the thalamus. Also, stimulation in the thalamus or in the reticular formation can cause disappearance of an alpha rhythm. The mechanism of the alpha rhythm is connected with the thalamus and the reticular formation. The thalamus is the relay center for sensory impulses and is the main source of afferent fibers that innervate the cerebral cortex. The reticular formation is a group of nuclei and fibers located in the medulla oblongata and pons that control the level of consciousness and are involved in the regulation of sleep and wakefulness.

Origin of Delta Waves. Delta waves are the slowest of the brain waves and are present in children during sleep. In adults, delta waves are usually present only during deep sleep. The origin of delta waves is not well understood, but it is believed that they may be related to the activity of the thalamus and the reticular formation.

The presence of alpha waves indicates a state of relaxation and wakefulness, while delta waves are associated with sleep and deep relaxation. Beta waves are associated with alertness and concentration.

Figure 59-2. Replacement of the alpha rhythm by an asynchronous, low-voltage beta rhythm on opening the eyes.
Delta waves also occur in deep slow-wave sleep, this suggests that the cortex then is mainly released from the activating influences of the lower centers.

Effect of Varying Degrees of Cerebral Activity on the Basic Frequency of the EEG

There is a general relation between the degree of cerebral activity and the average frequency of the EEG rhythm, the average frequency increasing progressively with higher degrees of activity. This is demonstrated in Figure 59-3, which shows the existence of delta waves in stupor, surgical anesthesia, and sleep, theta waves in psychomotor states and in infants, alpha waves during related states, and beta waves during periods of intense mental activity. During periods of mental activity, the waves usually become asynchronous rather than synchronous, so that the voltage falls considerably, despite increased cortical activity, as shown in Figure 59-2.

EEG Changes in the Different Stages of Wakefulness and Sleep

Figure 59-4 shows the EEG from a typical person in different stages of wakefulness and sleep. Alert wakefulness is characterized by high-frequency beta waves, whereas quiet wakefulness is usually associated with alpha waves, as demonstrated by the first two EEGs of the figure.

Slow-wave sleep is divided into four stages. In the first stage, a stage of very light sleep, the voltage of the EEG waves becomes very low, this is known as “sleep spindles,” that is, short spindle-shaped bursts of delta waves that occur periodically. In stages 2, 3, and 4 of slow-wave sleep, the frequency of the EEG becomes progressively slower until it reaches a frequency of only 1 to 3 waves per second in stage 4; these are typical delta waves.

Finally, the bottom record in Figure 59-4 shows the EEG during REM sleep, it is often difficult to tell a difference between this brain wave pattern and that of an alert awake person. The waves are irregular high-frequency beta waves, which are normally suggestive of excessive but desynchronized nervous activity as found in the awake state. Therefore REM sleep is frequently called desynchronized sleep because there is a lack of synchrony in the firing of the neurons, despite significant brain activity.

EPILEPSY

Epilepsy (also called “seizures”) is characterized by undirected excessive activity of either a part or all of the central nervous system. A person who is predisposed to epilepsy has attacks when the basal level of excitability of the nervous system (or of the part that is susceptible to the epileptic state) rises above a certain critical threshold. As long as the degree of excitability is held below this threshold, no attack occurs. Epilepsy can be classified into three major types: grand mal epilepsy, petit mal epilepsy, and focal epilepsy.

Grand Mal Epilepsy

Grand mal epilepsy is characterized by extreme neuronal discharges in all areas of the brain—in the cortex, in the deeper parts of the cerebellum, and even in the brain stem and thalamus. Also, discharges transmitted all the way into the spinal cord cause generalized tonic seizures of the entire body. Followed toward the end of the attack by alternating tonic and then spasmotic muscle contractions called tonic-clonic seizures. Often the person bites or “swallows” the tongue and may have difficulty breathing, sometimes to the extent of developing cyanosis. Also, signals transmitted from the brain to the viscera frequently cause urination and defecation.

The grand mal seizure lasts from a few seconds to 5 to 4 minutes. It is also characterized by postseizure depression of the entire nervous system; the person remains in stupor for 1 to many minutes after the attack and then often remains severely fatigued and asleep for many hours thereafter.
The top recording of Figure 59-5 shows a typical EEG from almost any region of the cortex during the tonic phase of a grand mal attack. This demonstrates that high-voltage, synchronous discharges occur over the entire cortex. Furthermore, the same type of discharge occurs on both sides of the brain at the same time, demonstrating that the abnormal neuronal circuitry responsible for the attack strongly involves the basolateral regions of the brain that drive the corona.

In laboratory animals or even in human beings, grand mal attacks can be initiated by administering neuronal stimulants, such as the drug Metrazol, or they can be caused by insulin hypoglycemia or the passage of alternating electrical current directly through the brain. Electrical recordings from the thalamus as well as from the reticular formation of the brainstem during the grand mal attack show typical high-voltage activity in both of these areas similar to that recorded from the cerebral cortex.

Preambulingly, therefore, a grand mal attack involves not only abnormal activation of the thalamus and cerebral cortex but also abnormal activation in the lower portions of the brain activating system itself.

What Initiates a Grand Mal Attack? Most people who have grand mal attacks have a hereditary predisposition to epilepsy, a predisposition that occurs in about 1 of every 50 to 100 persons. In such people, some of the factors that can increase excitability of the abnormal "epileptogenic" circuitry enough to precipitate an attack are (1) strong emotional stimuli, (2) alcohol caused by overindulgence, (3) drugs, (4) fever, and (5) loud noises or flashing lights. Also, even in people who are not generally predisposed, transient lesions in almost any part of the brain can cause enough excitability of local brain areas, so if discussed shortly; these, too, sometimes transmit signals into the activating systems of the brain to elicit grand mal seizures.

What Stops the Grand Mal Attack? The cause of the extreme neural excitatory during a grand mal attack is presumed to be massive activation of many reinnervating pathways throughout the brain. Presumably, also, the major factor, or at least one of the major factors, that stops the attack after a few minutes is the phenomenon of neural fatigue. A second factor is probably excitatory inhibition by inhibitory neurons that have also been activated by the attack. The stoppage and total body fatigue that occur after a grand mal seizure is over are believed to result from the intense fatigue of the neuronal synapses after their intensive activity during the grand mal attack.

Petit Mal Epilepsy
Petit mal epilepsy almost certainly involves the basolateral thalamocortical brain activating system. It is usually characterized by 3 to 50 seconds of uncontrolled or diminished consciousness during which the person has several twitch-like contractions of the muscles, usually in the head region, especially blinking of the eyes, that is followed by return of consciousness and resumption of previous activities. This total sequence is called the absence or absence epilepsy. The patient may have one such attack in many months or, in rare instances, may have a rapid series of attacks, one after the other. The usual course is for petit mal attacks to appear in late childhood and then to disappear by the age of 20. On occasion, a petit mal epileptic attack will initiate a grand mal attack.

The brain wave pattern in petit mal epilepsy is demonstrated by the middle record of Figure 59-5, which is typified by a spike and dome pattern. The spike and dome can be recorded over both or all of the cerebral cortex, showing that the seizure involves much or most of the thalamocortical activating system of the brain. In fact, animal studies suggest that it results from stimulation of a system of neurons involving thalamic reticular neurons (which are inhibitory gamma-amino-butyric acid [GABA]-producing neurons) and other excitatory thalamocortical and corticobulbar neurons.

Focal Epilepsy
Focal epilepsy can involve any part of the brain, either localized regions of the cerebral cortex or deeper structures of both the cerebellum and brain stem. Almost always, focal epilepsy results from some localized organic lesion or functional abnormality, such as a scar tissue in the brain that pulls on the adjacent neuronal tissues, or a tumor that compresses an area of the brain, or a destroyed area of brain tissue, or congenitally deformed local circuitry. Lesions such as these can produce extremely rapid discharges in the local neurons, when their discharge rates are above about 1000 per second, synchronous waves begin to spread over the adjacent cortical region. These waves presumably result from excitatory neurotransmitters that gradually expand adjacent areas of the cortex into the epileptic discharge zone. The process spreads to adjacent areas at a rate as slow as a few millimeters a minute to as fast as several centimeters per second. When r.e is wave of excitation spreads over the motor cortex, it causes a progressive "March" of muscle contractions throughout the opposite side of the body, beginning most characteristic in the mouth region and marching progressively downward to the legs but at other times marching in the opposite direction. This is called Jacksonian epilepsy.

A focal epileptic attack may remain confined to a single area of the brain, but in many instances, the strong signals from the convulsing cortex excite the