Neurophysiologic Basis of EEG

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Summary: This review article introduces the reader to the very basics of electroencephalography (EEG). It tries to explain in simple terms the physiologic principles of EEG generation and organization at the cellular, cortical and subcortical levels. It also introduces the basic EEG terminology (see the key words).

Key Words: EEG, Postsynaptic potentials, Volume conduction, Propagation, Synchronization, Desynchronization.

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EEG DEFINITION

What is EEG?

Electroencephalography is a graphic representation of the difference in voltage between two different cerebral locations plotted over time. The scalp EEG signal generated by cerebral neurons is modified by electrical conductive properties of the tissues between the electrical source and the recording electrode on the scalp, conductive properties of the electrode itself, as well as the orientation of the cortical generator to the recording electrode (Fig. 1).

VOLUME CONDUCTION

The EEG can be obtained because of the process of current flow through the tissues between the electrical generator and the recording electrode, which is called volume conduction. EEG provides a two-dimensional projection of three-dimensional reality, which means that theoretically it is impossible to determine the location of the EEG generator based on scalp-recorded EEG information alone. This is referred to as the inverse problem.

SYNAPTIC SOURCES OF EEG

To visualize and monitor minute (in the microvolt range) cerebral electrical activity, it must be of sufficient duration and sustained strength. To put it facetiously, one has to find a platform on which both the examiner and the examined brain find themselves in the same time-space continuum. Only synaptic activity readily fulfills those criteria and is most significant source of EEG potentials. Each synapse acts like a battery driving current in a small loop. Both excitatory postsynaptic potentials (EPSPs) and inhibitory postsynaptic potentials (IPSPs) contribute to the synaptic activity recorded as EEG (Fig. 2).

The summation of extracellular currents is slow enough to be able to generate EEG potentials. The current flowing across the external resistance of the cortex sums with the loop currents of the neighboring neurons to constitute a local mean field (Fig. 3). Viewed from outside the cells, membrane areas where current flows in or out of the cells are called respectively sinks and sources. Excitatory currents, involving Na^+ or Ca^{2+} ions, flow inward toward an excitatory synapse and outward away from it. The outward current is referred as a passive return current (from intracellular to extracellular space). Inhibitory loop currents, involving Cl^+ and K^+ ions, flow in the opposite direction.

Scalp electrodes record potential differences that are caused by postsynaptic potentials in the cell membrane of cortical neurons. The closed loops of the lighter dashed lines represent the summation of extracellular currents produced by the postsynaptic potentials; the open segments of heavier dashed lines connect all points having the same voltage level. The two scalp electrodes are at different voltage levels and record this difference (Fig. 4).

NONSYNAPTIC CONTRIBUTIONS TO EEG

The nonsynaptic activity is a less significant source of extracellular current flow that produces EEG potentials. Intrinsic neuronal activity such as fast action potentials usually lasts too short to affect EEG. Nonsynaptic intercellular interactions may potentially contribute to EEG (Buzsaki, Traub and Pedley, 2003)

INTRINSIC NEURONAL SOURCES OF EEG

Short-lasting ($< 2\ \mu s$) high-amplitude individual fast (Na^+) action potentials do not contribute to scalp recorded potentials except during synchronous events—both physiologic such as sleep transients and pathologic such as epileptic activity. Calcium-mediated action potentials (calcium spikes) are voltage generated and occur synchronously with dendritic EPSPs. They can contribute to the creation of the dendritic field sinks, especially during epileptiform activity

INTRINSIC SPIKE AFTERHYPERPOLARIZATION

Intrinsic spike afterhyperpolarization (AHP) following dendritic Ca^{2+} spikes results in suppression of fast spikes and

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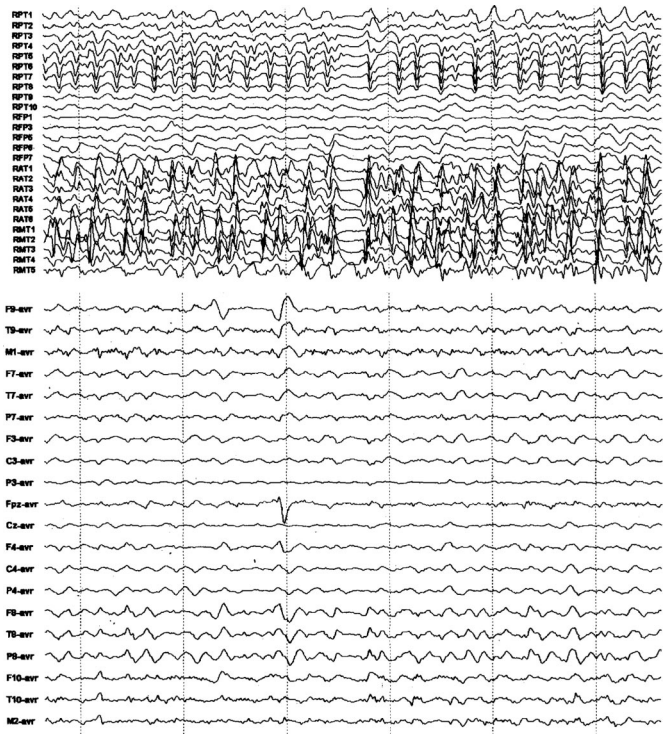

FIGURE 1. Simultaneous intracranial and scalp EEG recording of a temporal lobe seizure. The channels in the upper third of the picture represent intracranial contacts, whereas the ones in the lower part represent the scalp contacts. The intracranial recording appears to be generally more regular and of higher amplitude then the scalp one, which appears to be attenuated (from Ebersole, 2003)

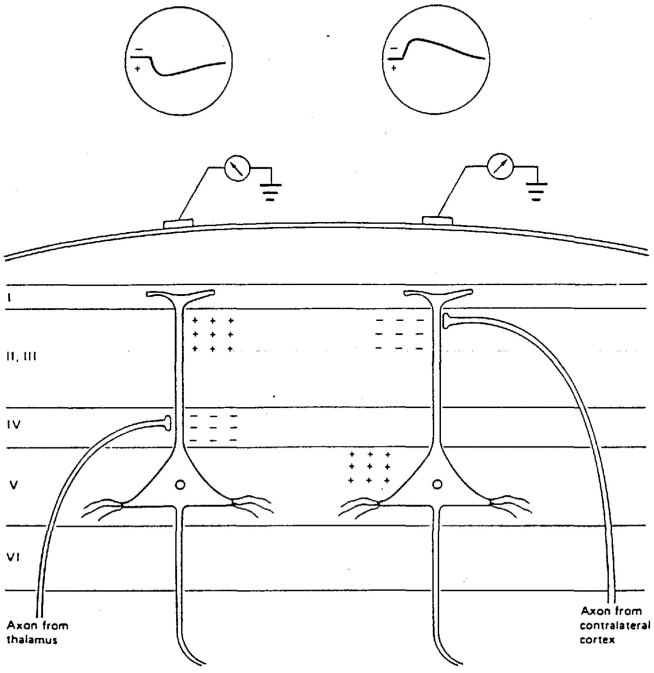

FIGURE 2. Generation of extracellular voltage fields from graded synaptic activity (from Martin, 1991). Relationship between polarity of surface potentials and site of dendritic postsynaptic potentials.

hyperpolarization of the membrane caused by activation of the Ca^{2+}-mediated K^+ conductance. These AHPs are comparable in amplitude and duration to the synaptic events, and as such, may contribute to extracellularly recorded EEG potentials. An example of AHPs may be the generation of delta waves in sleep. In the awake brain, subcortical neurotransmitters such as acetylcholine, catecholamines and histamine reduce the calcium-mediated potassium conductance, blocking the AHP-related delta waves.

NONSYNAPTIC CELLULAR INTERACTIONS

Evoked "ephaptic" effects may change transmembrane potentials creating extracellular current loops that can recruit neurons to fire even with insufficient activation by synaptic inputs. Ultrafast cortical rhythms result from short-lived interactions between interneurons and pyramidal cells that may produce a short-lived oscillatory field potential (ripple) in both hippocampus and neocortex. Although not recorded by standard EEG equipment, they may be diagnostically significant harbingers of seizure onset and offset.

NEURON–GLIA INTERACTIONS

The astrocytes are connected by gap junctions that allow spread of current and diffusion of molecular transport. This coupling allows spreading Ca^{2+} waves triggering calcium influx into the neurons. Postictally increased K^+ levels produce propagating waves across the astrocytes manifested by slowly spreading sustained potentials. Astrocytes release more K^+, that way depolarizing the neurons and blocking the afterdischarge with resulting postictal depression.

CORTICAL GENERATORS OF EEG

The main sources of EEG derive from cerebral cortex and form three-dimensional potential fields, which can be recorded as projected two-dimensional fields in the function of voltage versus time. It takes a combined synchronous electrical activity of approximately 108 neurons in a cortical area of minimum 6 cm^2 to create visible EEG. The area of cortex required for the generation of interictal spikes may be as large as 20 cm^2. As mentioned earlier, the principal generators of EEG fields measured on the surface of the brain or 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. Because of the attenuating properties of the skull, spatial (i.e., tridimensional) summation of cortical activity is critical for producing a voltage field recordable from the scalp.

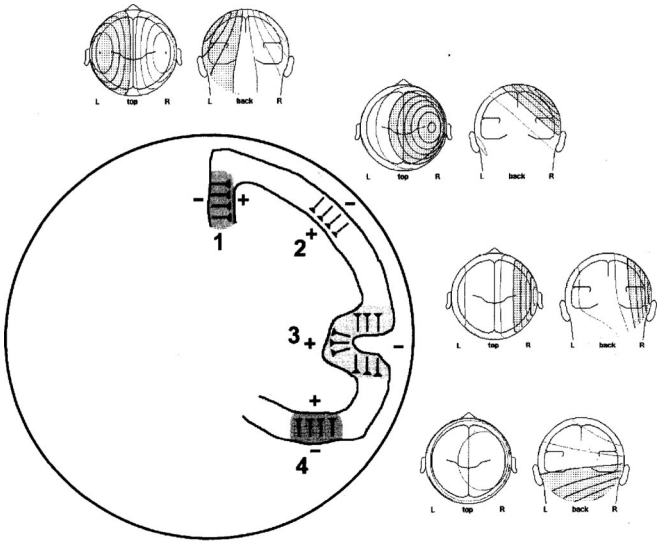

FIGURE 3. Schematic of a brain cross section, illustrating four representative cortical EEG sources (from Ebersole, 2003). Sources 2 and 3 produce radial fields, so the negative, so the negative voltage maximum is directly above them. Sources 1 and 4 produce tangential fields and both negative and positive voltage maxima are displaced to either side.

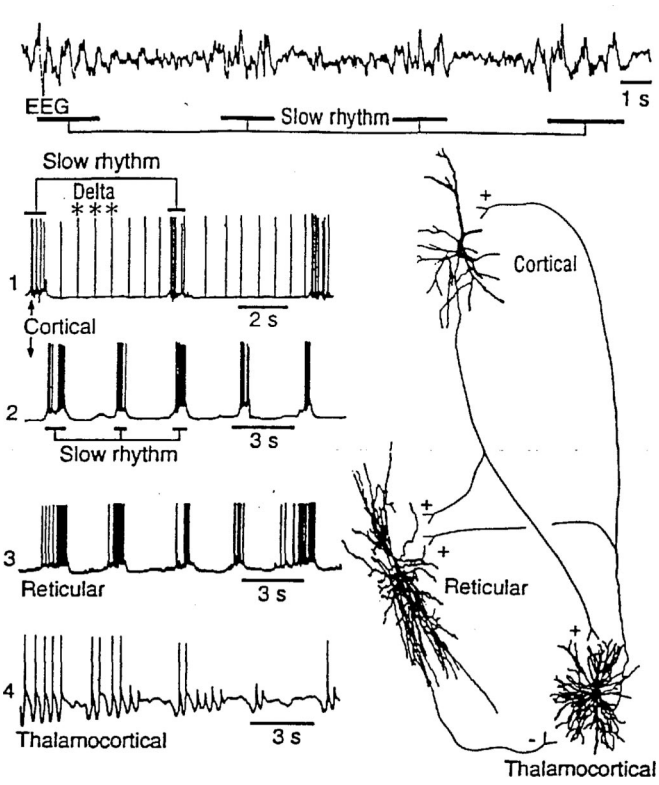

FIGURE 4. Relationship between EEG slow waves and thalamic intracellular potentials (from Steriade et al., 1993).

RELATIONSHIP BETWEEN POLARITY OF SURFACE POTENTIALS AND SITE OF DENDRITIC POSTSYNAPTIC POTENTIALS

Source area is maximal for a given electrode when the orientation of the active cortical region is face-on. This is the case when the voltage field is radial and electrode right above the source. A radial source produces field maximal directly above it, and another one with opposite polarity on the opposite side of the head. For a superficial source, the scalp maximum nearest it is significantly greater than the one on the opposite side of the head. Single-voltage field maximum cannot be used to define the location and/or orientation of a cortical EEG generator. Except for a purely radial source, the EEG field maxima are displaced from a position directly above it. The location of both negative and positive field maxima and their relative strengths must be taken into consideration.

PROPAGATION OF CORTICAL ACTIVATION

With propagation of electrical activity into adjacent cortical regions, the geometry of the source changes. New location and new orientation of the source take place resulting in a different voltage field. Movement of scalp field maxima over tens of milliseconds suggests propagation of source activity.

SUBCORTICAL SYNCHRONIZATION OF EEG

The dorsal thalamus is considered as the chief subcortical EEG rhythm generator synchronizing populations of neocortical neurons as voltage generators. In normal conditions both thalamic nuclei and cortical regions interact to produce the synchrony of cortical postsynaptic potentials (PSPs) during wakefulness and sleep. The facultative pacemaker theory assumes that thalamocortical relay neurons send fibers to the cortex as well as give off branches that turn back and end on thalamic inhibitory interneurons (biofeedback servomechanism). Nucleus reticularis hypothesis attributes the pacemaker properties to the nucleus reticularis thalami, whose cells release the inhibitory neurotransmitter GABA in rhythmic bursts of depolarizations directed to the neurons of the dorsal thalamus and rostral brainstem.

Alpha-frequency rhythms are generated in multiple cortical areas in addition to the occipitally dominant alpha rhythm. Local cortical connections seem to be more important in generating most alpha-frequency rhythms, although thalamocortical discharges may have limited influence on posterior dominant alpha rhythm.

Fast waking rhythms such as in beta- and gamma-frequency bands are produced by cortical generators. Pontine cholinergic input to the thalamus inhibits the generators producing sleep transients such as sleep spindles and high-amplitude delta waves.

Sleep spindles appear to be a thalamocortical phenomenon. GABAergic neurons of the nucleus reticularis thalami discharge in rhythmic cycles. They project to thalamocortical relay neurons, which in turn project to widespread cortical neurons. The relay neurons discharge after GABA-release–related inhibition subsides. That subsequently results in synchronized EPSPs in the cortex, which become visible by EEG.

Unlike sleep spindles, which require synaptic activities to establish the rhythmic oscillation, delta oscillation is an intrinsic rhythm that depends on potassium fluxes at voltage-dependent ion channels of cortical and thalamic neurons.

SUBCORTICAL EEG DESYNCHRONIZATION

Desynchronization of the EEG is the interruption of its rhythmical activity. It occurs with activation of ascending cholinergic projections of the basal forebrain and brainstem and projections from the raphe nuclei and locus ceruleus. Rhythmical activity is interrupted by both direct effects on cortical neurons and indirectly on thalamic neurons.

At the cellular level, desynchronization is accompanied by a transition from a burst firing pattern to more continuous or single spike pattern. Desynchronization is enhanced by behavioral arousal and suppressed by non-REM sleep. Certain abnormal synchronous patterns, such as alpha coma, occur in the setting of widespread injury to the ascending neuronal systems that would otherwise produce arousal and desynchronization.

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