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Basic physiology of the EEG

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Abstract

The basic physiology of the EEG and its rhythms has been the topic of intense research since its discovery in the 1920s, the subject of innumerable textbooks and articles. In this overview, we present some of the basic principles of neurophysiology, followed by a description of which elements constitute the EEG recording in general. We conclude with the underlying pathophysiology of some of the normal and abnormal EEG findings.

The typical surface EEG represents the combined electrical activity of many billions of neurons[1], sampled with relatively few large and widely-spaced electrodes. Each neuron produces electrical activity based on its own independent cellular basis, and is continually interacting with other neurons within a local network and via long range interactions. Thus, it is necessary to understand the cellular basis of neuronal electrical activity and of neuronal interactions.

The cell membrane consists of a lipid bilayer that separates the intracellular and extracellular environments, each containing different concentrations of ions. A membrane potential is therefore generated, which is related to the electrochemical drive of each ion across the membrane. For any ion X, an equilibrium potential exists at which there is no net movement across the membrane due to a balance of chemical and electrical forces. This equilibrium potential for an individual ion is given by the Nernst equation, but as a typical cell contains multiple ions, each with their own individual permeability, the Goldman equation is used to calculate the overall equilibrium potential by modifying the Nernst equation. It calculates the membrane potential by weighing each of these ions according to their permeability. In typical cellular conditions, since K^+ is the most permeable ion, the resting E_M is the most closely related to E_K (-90mV). The actual value of E_M is -70mV. Under normal cellular conditions, Na^+ would tend to flow into, and K^+ out of the cell, thus degrading the concentrations of these ions. The cell relies upon a Na^+-K^+ pump to maintain the concentration gradient at the expense of energy usage in the form of ATP.

The cell membrane also contains ion channels that open selectively with voltage changes or ligand binding, allowing signal transmission by means of the action potential. When the membrane is *depolarized*, voltage-gated Na^+ channels open, making the membrane permeable to Na^+. This in turn opens even more voltage-gated sodium channels, causing a large, but brief action potential. As the E_M approaches E_{Na}, these channels inactivate and do not reactivate until the membrane reaches back to its resting potential, thus causing a brief refractory period. Therefore, the E_M will briefly *hyperpolarize*, or fall below its resting potential.

Although action potentials are relatively large in amplitude, they are felt to contribute little to the EEG, mostly due to their very brief nature. As such, they are relatively asynchronous, as only a small percentage of neurons fire action potentials at any one instant. Additionally, they are filtered out by the capacitative lipid membrane, which serves as a low-pass filter, reducing their ability to be recorded from surface electrodes.[2]

Unlike the action potential, postsynaptic potentials (PSPs) do not exhibit the "all or none" phenomena. They come in two forms; excitatory post-synaptic potentials (EPSPs) depolarize the membrane, while inhibitory post-synaptic potentials (IPSPs) hyperpolarize the membrane. Though lower in magnitude than action potentials, they are slower, and thus more likely to overlap at any one instant and not filtered out by the lipid membrane. Moreover, lower frequency signals correspond to spatially larger transmembrane currents and can therefore propagate farther. This allows for both *temporal* and *spatial* summation.[3]

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It is for these reasons that PSPs are felt to be the main contributors to the EEG.

Gap junctions, consisting of hexameric complexes containing proteins called connexons, also play a large role in cell to cell communication via electrical transmission. As the pores are large, they allow for transmission of metabolites between cells. Passive current flow across the junctions occurs almost immediately. Thus, they may play a large role in neuronal synchronization.[4]

NEURONAL ARRANGEMENTS AND ELECTRICAL RECORDINGS

The spatial arrangement of neurons affects the ability to record extracellular potentials.[2] In an open field, the arrangement typically seen in cortex, the somata are aligned in one layer, with the dendrites in the opposite layer. As the individual fields are oriented in the same direction, the summated potential generated by this arrangement is readily recordable.

A closed field can occur when either the cells are arranged with the somata in the center and the dendrites in the periphery, or when there is a random neuronal distribution. In this situation, the individual fields cancel each other out and there is no net sum of electricity recordable externally. Closed fields can be seen in the brainstem and hippocampus.

RHYTHMS IN THE NORMAL EEG

In the awake state the most studied rhythms are the alpha and mu rhythms, and in the asleep state sleep spindles. The alpha rhythm was first described by Berger in 1929.[5] It is seen on the surface as idling visual cortical areas, and its blocking during eye opening likely represents an "event related desynchronization."[6] The underlying generator of the rhythm, though, is not fully understood. One proposition is that the alpha rhythm is driven by presynaptic input to cortical neurons from the thalamus.[7] However, it has also been modeled as a "self-excitatory oscillation."[8]

The first description of the rolandic mu rhythm was by Gastaut et al. in 1952, as the "*rhythme rolandique en arceau*."[9] They proposed that it is due to neuronal hyperexcitability in this region. Another theory based on its reactivity, similar to the alpha rhythm that blocks with visual stimuli, the mu rhythm blocks with even the thought of contralateral movement, and therefore it may represent cortical idling over rolandic cortex.[10] Nevertheless, as for alpha rhythm, there is no generally accepted theory for the neurophysiologic basis of the mu rhythm.

The mechanisms of sleep spindle generation were explored by Steriade in the 1980s.[11] Feedback loops exist between inhibitory cells in thalamic reticular nucleus and thalamocortical neurons. These thalamocortical neurons show burst firing in the spindle frequency when inhibited by the reticular nucleus. This rhythm is then in turn entrained on cortical areas, yielding the classical surface EEG manifestation of sleep spindles.

THE ABNORMAL EEG

When discussing the abnormal EEG, it is important to distinguish between non-epileptiform (slowing, etc.) and epileptiform (spikes, etc.) abnormalities. Generalized polymorphic delta and theta slowing result from diffuse white matter disturbances. The etiology is nonspecific, including toxic-metabolic disorders, white matter encephalopathies or bilateral lesions.[12] Continuous *focal* polymorphic delta and theta slowing are indicative of focal disturbances of the subcortical white matter. Lesions such as tumor, stroke, abscess, hematoma, and contusions can cause this.[13] Alternatively it can be seen as a postictal phenomenon or in the setting of migraine.[14]

The paroxysmal depolarizing shift (PDS) is considered the cellular substrate of epilepsy.[15] It begins with a calcium mediated depolarization, followed by opening of voltage-gated sodium channels and relatively synchronized action potentials, seen as a "spike" at the surface. As positive charge moves into the cell, a negativity is recorded at the surface. Inhibitory inputs to the axon hillock follow the spike. This results in an influx of negative charge at the axon and an outflux of negativity at surface. This is seen as a slow surface negativity, i.e. "slow-wave."

Of more recent interest has been the pathophysiology of high frequency oscillations (HFOs), which represent short-term neuronal synchronization. HFOs can be divided into ripples (80-250 Hz) and fast ripples (250-500 Hz). Whereas ripples can be recorded from normal hippocampus[16], fast ripples are typically associated with epileptic brain.[17] These pathologic fast ripples are though to represent local abnormally synchronous action potentials.[18]

In conclusion, the surface EEG gives us a valuable but incomplete view into the inner workings of the brain. This is at least partly due to the limited ability of a handful of relatively large and widely-spaced electrodes to discern the cellular and local network interactions governing the underlying rhythms. However, these technical

barriers are being breached as computing power and engineering techniques advance, allowing recordings from larger numbers of ever smaller electrodes, and in frequencies both above and below those used in typical clinical EEG.

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REFERENCES

1. Braendgaard H, Evans SM, Howard CV, Gundersen HJ. The total number of neurons in the human neocortex unbiasedly estimated using optical disectors. *J Microsc* 1990; 157(Pt 3):285-304.
2. Niedermeyer E, Lopes da Silva F, eds: Electroencephalography: Basic principles, clinical applications, and related fields. 5th ed. Lippincott Williams & Wilkins, 2004.
3. Purves D, Augustine GJ, Fitzpatrick D, *et al*., ed: Neuroscience. 2nd ed. Sinauer Associates, 2001.
4. Bennett MVL, Zukin RS. Electrical coupling and neuronal synchronization in the mammalian brain. *Neuron* 2004; 41(4):495-511.
5. Berger H. Über das Elektroenkephalogramm des Menschen. *Archiv für Psychiatrie und Nervenkrankheiten* 1929; 87:527-70.
6. Pfurtscheller G. Event-related EEG desynchronization. *Electorencephalogr. Clin. Neurophysiol* 1990; 75: S117.
7. Steriade M, Gloor P, Llinás RR, Lopes de Silva FH, Mesulam MM. Report of IFCN Committee on Basic Mechanisms. Basic mechanisms of cerebral rhythmic activities. *Electroencephalogr Clin Neurophysiol* 1990; 76(6):481–508.
8. Watanabe S. Rhythmicity of EEG and stability of alpha rhythm. *Jap J EEG-EMG* 1981; 9:99-101.
9. Gastaut, H. Etude electrocorticographique de la reativite des rhythmes rolandiques. *Rev Neurol* 1952; 87: 176-82.
10. Bostem F, Dongier M, Demaret A. Discussion on mu rhythm. *Electorencephalogr Clin Neurophysiol* 1965; 18: 721.
11. Steriade M, Llinás RR. The functional states of the thalamus and the associated neuronal interplay. *Physiol Rev* 1988; 68:649-742.
12. Gloor R, Kalabay O, Giard N. The electroencephalogram in diffuse encephalopathies: Electroencephalographic correlates of grey and white matter lesions. *Brain* 1968; 91;779-802.
13. Jasper H, Van Buren J. Interrelationship between cortex and subcortical structures: Clinical electroencephalographic studies. *Electorencephalogr Clin Neurophysiol* 1953; 4(Suppl):168-88.
14. Neufeld MY, Treves TA, Korczyn AD. EEG and topographic frequency analysis in common and classic migraine. *Headache* 1991; 31(4):232-6.
15. Ayala GF, Dichter M, Gumnit RJ, Matsumoto H, Spencer WA. Genesis of epileptic interictal spikes. New knowledge of cortical feedback systems suggests a neurophysiological explanation of brief paroxysms. *Brain Research* 1973; 52:1-17.
16. Buzsaki G, Horvath Z, Urioste R, Hetke J, Wise K. High frequency network oscillation in the hippocampus. *Science* 1992; 256:1025-27.
17. Bragin A, Engel J Jr, Wilson CL, Fried I, Buzsaki G. High frequency oscillations in human brain. *Hippocampus* 1999; 9:137-42.
18. Bragin A, Mody I, Wilson CL, Engel J Jr. Local generation of fast ripples in epileptic brain. *J Neuroscience* 2002; 22:2012-21.