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## The Generation of Brain Waves

Karen A. Baldi B.A. R.EEG T.

64 Danebury Downs  
Upper Saddle River, NJ 07458

Many technicians do not have a science background and find neurophysiology difficult to grasp. The purpose of this article is to give a simplified explanation of the mechanisms involved in the generation of brain waves. For more detailed background information on neuroanatomy and neurophysiology it would be useful to read Michael T. Long's article "Topics in Neurophysiology" (*Amer. J. EEG Technol.*, 6:7.9"90).

Hans Berger is credited with discovering human brain waves (1929) as well as naming the technique,- naming alpha and beta waves and describing the blocking reaction to eye opening. The real purpose behind Berger's work however, was to discover the physical counterpart of "psychical energy". Berger believed that the energy produced from metabolism within the brain was transformed into heat, electricity and psychical energy. By carefully measuring the temperature and electrical output of the brain he hoped to be able to calculate the quantity of this psychical energy. Berger's discovery of the human EEG was a by-product of his search for the energy of the psyche. It was not until the confirmation of Berger's work by Adrian and his associates (1934-35) that an interest in EEG grew (2).

Berger did not contribute much to our understanding of the mechanism behind the generation of brain waves though he appreciated that the mechanism must be largely within the cerebral cortex (2). Lipold, however, claims the source of the current for generating alpha to be the standing potential across the eye which is conducted back to the brain along the eye muscles (12). Studies on experimental animals suggest that the electrical activity recorded from the scalp is generated only by the cortical neurons. Subcortical structures contribute little if any to the scalp recorded EEG. However, they are important in maintaining the rhythmicity of the cortical neurons (1,7).

The electrical activity in neurons is generated primarily by two sources. One source is the action potential, which is the result of a change in membrane permeability. An action potential is generated when a nerve cell receives an impulse from a neighboring cell. Pores in the membrane of that cell open and allow large sodium ions to flow from outside to inside the axon of the cell, This flow of positively charged sodium ions causes a change in the voltage across the cell membrane and the membrane becomes depolarized. This process of depolarization continues along the axon towards the dendrite of the next neuron, and in this way an

impulse is transmitted along a nerve fiber.

The second source of electrical activity in neurons occurs at the synapse. This is the junction of the axon of one neuron and the dendrite of the next neuron. As the impulse arrives at the end of the axon of one cell, transmitter substances (chemicals such as acetylcholine) are released into the synaptic space and drift to the dendrite of the next cell stimulating that cell membrane to depolarize and pass along the impulse. This depolarization is called an excitatory postsynaptic potential (EPSP). An inhibitory postsynaptic potential (IPSP) results from the hyperpolarization of the cell membrane. The current flow in this case is from the inside of the cell outwards making the outside of the cell more positive preventing transmission of the impulse, opposite to that of the EPSP (7).

Available evidence suggests that cortical potentials and hence the EEG, are due to these EPSPs and IPSPs generated by the cell body and large dendrite of pyramidal neurons (4). The pyramidal cell of the cerebral cortex is a neuron named from the shape of its body. Each cell has a number of dendrites. However, there is one apical dendrite which extends from the apex of the pyramid and then branches (14). These cells are oriented vertically with respect to the cortex. The currents generated by the synchronous postsynaptic potentials (EPSPs and IPSPs) near one end of the pyramidal cell are found to be strong enough to generate EEG activity (10). The relatively slow time course of the EPSPs and IPSPs is more compatible with the generation of the EEG rather than the fast action potentials. EEG waves are the result of the summation of the EPSPs and IPSPs across the membranes of many neurons (10).

Studies have shown a correlation between EPSPs and surface negative waves, and IPSPs and surface positive waves (4). Any voltage change which appears as an EEG wave is the result of the more or less synchronized activity of a large number of neurons occupying an area of about 6 cm<sup>2</sup> (5).

The scalp itself acts as a spatial averager of electrical activity. The EEG records only those waves which are common to and synchronous over a relatively large area of the cortex (5). It is well known that decreasing the distance between scalp electrodes below a few cm decreases the amplitude of the EEG because the electrical activity recorded by the closely spaced electrodes is very similar. This is due to the averaging effect of the skull and scalp. In contrast, recordings directly from the cortex (ECoG) can show a difference from mm

to nlm. Other factors affecting the recorded EEG include the cortical area involved and the degree of synchronization of the electrical activity over this area, and possibly the orientation of the positive and negative charges (dipoles) (5).

The electrical fields produced from the perpendicular arrangement of the pyramidal neurons resembles that generated by a single dipole (9). Like a battery, there is a positive (+) charge, at one end and a negative (-) charge at the other end creating a potential difference or voltage. The EEG measures the potential within these voltage fields, generated by dipoles within the cortex, which are formed by the electrical current flowing into and out of the cortical neurons (7).

As the brain is not a long thin wire, but a three-dimensional mass, it is a volume

conductor. A volume conductor is any mass capable of carrying electrical currents. In a volume conductor any potential difference (voltage) set up between two points leads to a current flow *throughout* the entire mass of the volume conductor. Potential differences between two electrodes placed on the scalp arise due to the electrical activity in the brain areas immediately underneath these electrodes and may represent some activity generated a considerable distance from the electrode pair (consider EKG artifact) (9). Although some contend that the volume conductor, and dipole model, is not satisfactory (5) it is still a useful analogy.

Much work has been directed towards a search for the pacemaker mechanism responsible for the smooth rhythmicity of the impulses from groups of neurons. The possibility that various thalamic nuclei may exert a control upon the general electrocortical activity was first demonstrated by Morison and Dempsey in 1942 (3). The results of experimentation suggest that it is the thalamus which serves as pacemaker of certain cortical rhythms that are recorded at the scalp (1). The thalamus is not essential for the production of brain waves but plays a large part in maintaining the normal rhythms seen in the EEG (10). Rhythmic activity can arise in any or all of the thalamic nuclei, can spread from one nucleus to another and travel to the cortex via the thalamocortical projections (1). The impulses set up in the thalamic cells during an ESP will trigger an EPSP in the cortical cells to which they are linked. A large thalamic discharge will excite a correspondingly large number of cortical neurons and will elicit a good sized surface wave (11).

During activation (such as eye opening) inputs from the reticular formation in the brainstem abolish the rhythmic discharges in the thalamus and therefore cause the cortical potentials to become desynchronized and to decrease in amplitude (11).

Basically, the EEG is a recording of summated synaptic potentials which are generated by the pyramidal cells in the cortex. These in turn are modulated by rhythmic discharges from the thalamus, which is affected by input from the reticular formation.

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