The source of the EEG

SUMMARY

(1.1) The EEG is generated by cortical nerve cell inhibitory and excitatory postsynaptic potentials. These postsynaptic potentials summate in the cortex and extend to the scalp surface where they are recorded as the EEG. In addition to postsynaptic potentials, intrinsic cell currents produced by activation of ionic channels probably also contribute to the EEG, although their role has not been clearly established. Although nerve cell action potentials were originally thought to be the source of the EEG, they have a much smaller potential field distribution (less penetration into the extracellular space) and are much shorter in duration than postsynaptic potentials (about 1 ms compared to postsynaptic potentials of 15 to more than 200 ms). Action potentials, therefore, do not contribute significantly to either scalp or clinical intracranial EEG recordings.

(1.2) Rhythmic cortical EEG activity arises from an interaction between the thalamus and cortex. Many thalamic, thalamicocortical and cortical neurons have intrinsic oscillatory firing properties that allow them to participate in cellular networks that generate rhythmic EEG activity. Sleep spindles are currently among the best understood of the rhythmic EEG activities. During sleep thalamic pacemaker cells in the nucleus reticularis of the thalamus stimulate thalamocortical cells which send excitatory impulses to the cortex. When the pacemaker cells within the nucleus reticularis are surgically or pharmacologically isolated their spindle activity continues whereas sleep spindle activity in the cortex and other structures ceases.

Desynchronization is the loss of rhythmic activity. It results from: (1) alterations in subcortical pacemaker activity (e.g., pacemaker cells of the nucleus reticularis in the thalamus fire tonically during desynchronization and in rhythmic bursts when producing sleep spindles), (2) alterations in systems that project diffusely to the cortex from the brainstem and basal forebrain areas (e.g., activation by ascending cholinergic systems), or (3) direct suppression or injury to any of the components of rhythmic cellular networks (i.e., damage to the subcortical pacemaker, thalamocortical, corticocortical, or cortical networks).

(1.3) Scalp electrodes record mainly the summed postsynaptic potentials of neurons in the underlying cortex, favoring slow, simultaneous, potential changes generated in large cortical areas of pyramidal cells oriented in parallel at 90° to the plane of the scalp surface. The tissue lying between the generating cells and the recording electrode through which electrical current must flow (e.g., brain, CSF, skull, and scalp) forms an electrical volume conductor. The volume conductor greatly modifies the amplitude and morphology (shape) of the cortical signal before it reaches the recording electrodes. The amplitude of potentials recorded directly from the cortex is typically 2 to 58 times greater than that seen in scalp electrodes depending on: (1) the degree of postsynaptic potential synchronization, (2) the orientation of the pyramidal cells to the scalp surface, and (3) the size of the area of participating cortex. The larger the area of cortex generating the potential, the less attenuation there is at the scalp.

The EEG is mainly generated by pyramidal cell postsynaptic potentials that form an extracellular cortical dipole layer. This dipole layer parallels the surface of the cortex projecting opposite electrical polarities towards the cortical surface compared to the innermost layers of the cortex. The scalp electrodes see only those potentials that are aimed at them and conducted from the cortical surface to the scalp.

The scalp electrode that is closest to the cortical generator does not always record the maximal potential. This is explained by the solid angle theorem of volume conduction.

1.1 THE GENERATOR OF THE EEG

The EEG is represented as a graph of voltage versus time where the y (vertical) axis is voltage and the x (horizontal) axis is time. Voltage at any given instant is always (for purely technical reasons to be discussed later) obtained as the difference in voltage between at least 2 electrode sites on the body, at least one of which is placed on the scalp. Thus, the operational definition of the EEG is that the EEG is the difference in voltage between two different recording locations plotted over time. This simple definition is actually seen to apply to all clinical biocerebroelectrical recording techniques (ECG, EMG, ENG, etc.). The interpretational definition of the EEG is that it consists of inhibitory and excitatory postsynaptic potentials of pyramidal cells generated in the cortex of the brain.

The EEG signal that arises from thousands of synchronized pyramidal cell postsynaptic potentials is greatly modified by the time it reaches the recording electrode on the scalp. The factors that modify the original signal are:

(a) the electrical conductive properties of whatever tissues lie between the electrical source and the recording electrodes (e.g., brain parenchyma, dura, CSF, skull, scalp),
(b) the orientation of the electrical generator to the recording electrode (i.e., to what extent the generator is 'aimed towards' the electrode), and
(c) the conductive properties of both the recording electrodes and the scalp-electrode interface (the size of the electrodes, the electrical properties of the materials the electrode is constructed from, and the resistance to current flow produced by the junction of the electrode and scalp).

The process of current flow through the tissues between the electrical generator and the recording electrode is referred to as volume conduction. It is important for the electroencephalographer to have some understanding of the effect of volume conduction on the original signal in order to create a mental picture of the original signal source. In this way the electroencephalographer can estimate the anatomical localization of a particular EEG activity. Indeed, one of the more important and challenging aspects of learning electroencephalography is not pattern recognition. It is the ability to mentally reconstruct the most plausible 3-dimensional picture of current sources from the 2-dimensional information provided by the EEG (4.8).

There are 2 important factors (one biophysical and one physiological) that limit EEG interpretation. First, for any scalp recorded EEG signal there are an infinite number of source or sources within the volume of the brain that can explain, or 'fit,' the scalp recorded signal. Therefore, one or more generators in different locations in the brain can produce the same EEG findings at the scalp. This means it is theoretically impossible to know the location of the EEG generator in the brain with only scalp recorded EEG information. This is referred to as the inverse problem. In contrast, if the
anatomical source, intensity and orientation of the electrical generator in the brain are known, then the EEG findings in scalp electrodes can be accurately predicted. This is referred to as the forward problem. However, it is the inverse problem that the electroencephalographer is confronted with in clinical practice. Because the localization of EEG sources within the brain is so important, the search for methods to help solve the inverse problem (referred to as source localization) is currently a central theme in EEG research (4.8). Fortunately, in routine EEG practice a formally trained electroencephalographer can greatly narrow the number of possible solutions to the inverse problem.

The second factor that limits interpretation is that EEG signal abnormalities do not always localize to the area of the brain where the main pathological attack is taking place. That is, an abnormal cortical signal may occasionally appear at a distance from the most prominent functional or structural damage. For example, the substance of a large structural lesion (e.g., tumor, stroke, etc.) is typically electrically silent. However, bordering tissue involved in EEG generation produces the abnormal activity seen. Indeed, in some cases EEG abnormalities recorded over the middle and anterior temporal areas may occur in the setting of a structural abnormality that more directly involves deep hemispheric structures or the frontal, parietal or posterior temporal lobes.

The cellular activity that produces the EEG consists mainly of cortical postsynaptic potential changes that alter the electrical charge across the pyramidal cell membrane (Fig. 1.1). Cortical neurons, like other nerve cells, have a resting electrical charge (membrane potential), that is the difference in the electrical potential between the interior of the cell and the extracellular space. The resting potential fluctuates as a result of impulses arriving from other neurons at synapses located on the cell body and its processes.

Such impulses generate relatively sustained local postsynaptic potentials that cause electrical current flow along the membrane of the cell body and dendrites. These changes can reduce the membrane potential to a critical level at which the membrane loses its charge completely, generating an action potential of brief duration that is propagated along the axon. The varying EEG signal is produced by the temporal and spatial summation of electrical currents that arise from postsynaptic potentials.

In addition to postsynaptic potentials there are intrinsic cellular currents mediated by ionic channels that produce high amplitude, long duration, extracellular potentials. It is highly likely that these extracellular potentials contribute to the EEG. Intracellular currents arise in neocortical cells undergoing burst firing with prolonged afterhyperpolarization potentials. Burst firing (a discharge consisting of a cluster of action potentials) with an afterhyperpolarization potential produces an event much longer in duration than a synaptic potential. Burst firing also tends to occur in synchrony with

Fig. 1.1. The generation of the EEG by the cerebral cortex. 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, as it changes with time, in the form of a wave, which is indicated by the first of the two tracings at the upper right. A simultaneous recording made with a microelectrode from a single cortical neuron is indicated by the second tracing and bears no close relation to the scalp EEG. The round insets show the major ionic and electrical events at single neurons. REST: the uneven distribution of ions across the cell membrane, partly maintained by the semipermeable membrane, partly by the active extrusion of sodium ions and intrusion of potassium ions, causes a steady potential difference of about 65 mV which can be recorded with an intracellular microelectrode. IPSP: an inhibitory postsynaptic potential is caused by activation of an inhibitory synapse on the cell body, which transiently increases the permeability of the postsynaptic membrane to an influx of chloride (and in some cases also an efflux of potassium) ions and thereby increases the membrane potential, generating electrical current flow of decreasing intensity along the cell membrane. EPSP: an excitatory postsynaptic potential, caused by activation of an excitatory synapse on a dendritic process of the neuron, causes a nonselective increase of permeability to ions including sodium and thereby transiently decreases the membrane potential locally, generating current flow which tends to depolarize the membrane of the cell body. AP: an action potential is initiated at the axon hillock of the cell body by the summation of excitatory postsynaptic potentials which reduce the membrane potential by at least 10 mV to a critical level at which point the membrane suddenly becomes freely permeable to all ions so that the membrane potential momentarily collapses and reverses; local current flow depolarizes neighboring membrane parts and results in the propagation of an action potential along the membrane spreading radially from the cell body through the axon and dendritic tree.
other neurons in the same cell population. Both of these characteristics are important for generating potentials recordable at the scalp. In contrast to postsynaptic potentials and intrinsic cellular currents, isolated action potentials actually contribute little or nothing to the recorded EEG.

1.1.1 The resting potential of a neuron usually measures approximately $-65$ mV (varying from $-40$ to $-80$ mV in different nerve cells) and is negative on the inside of the cell membrane with respect to the outside. It is the result of (a) passive and (b) active properties of the cell membrane. (a) The passive properties are those which do not require metabolic energy. They result from unequal permeability of the membrane to sodium, potassium, chloride and other ions (Fig. 1.1). Diffusion and electrical gradients tend to drive ions in or out of the cell so that they distribute unevenly on both sides of the membrane. This uneven distribution contributes to a steady difference of electrical potential at rest. (b) Active properties require metabolic energy to counteract leakage of ions across the membrane; leaking ions are continuously transported against diffusional and electrical gradients back to concentrations appropriate for resting conditions. Most important is the sodium-potassium pump that actively transports sodium out of the cell and potassium into the cell. The concentration of sodium is maintained at about 10 times higher outside the cell than inside. Conditions which disrupt cerebral metabolism, such as anoxia or ischemia, may reduce or abolish this pumping action of the membrane, causing reduction of the membrane potential and increased excitability or complete collapse of neural function.

1.1.2 Postsynaptic potentials in nerve cells are caused by impulses (action potentials) arriving from other neurons via axons which terminate in a specialized contact zone, or synapse, located on the cell body or its processes. The impulse in the afferent neuron causes release of a neurotransmitter substance from its nerve terminal that diffuses across the synaptic cleft to the postsynaptic neuronal membrane patch where it interacts with a specialized receptor. The interaction produces a transient change in permeability to certain ions in the membrane portion near the synapse; this causes a local change in the resting potential or a postsynaptic potential (PSP). An excitatory postsynaptic potential (EPSP, Fig. 1.1) is a transient partial reduction in membrane potential which is usually due to an increased local permeability to sodium and potassium ions. Because sodium is a positively charged ion on the outside of the cell, its entry into the cell makes the negative intracellular resting potential less negative (i.e., partially depolarizes the cell). In contrast, an inhibitory postsynaptic potential (IPSP, Fig. 1.1) is a transient increase in intracellular negativity produced by the entry of negatively charged chloride ions into the cell or the exit of potassium ions from the cell. Even though the inside of the cell is relatively negative, the higher concentration of chloride outside the cell leads to an influx of chloride ions when the chloride channels are opened by the inhibitory postsynaptic potential. Inhibitory postsynaptic potentials also may involve an increase in permeability to potassium ions. The potentials generated at inhibitory synapses on different parts of the cell are thus summed and hyperpolarize the cell making it less likely to fire. The potential difference between the postsynaptic membrane portion and the other parts of the neuronal membrane causes an electrical current to flow along the neuronal membrane and to change the membrane potential of the cell body. Postsynaptic potentials alter the neuronal membrane potential by several millivolts and last over 100 ms.

1.1.3 Action potentials occur when the neuronal membrane is depolarized beyond a critical level or threshold (Fig. 1.1). This threshold is lowest at the junction of the cell body and the axon, or the axon hillock. A depolarization of the resting potential by at least 10 mV triggers a self-limited sequence of events consisting of a brief increase of the membrane permeability to sodium and potassium ions which leads to a sudden collapse, brief reversal and quick restitution of the membrane potential. This electrical change is the action potential; it has an amplitude of about 110 mV and lasts only about 1 ms. It is referred to as an all or none phenomenon because it does not vary in amplitude. By depolarizing and inducing the same sequence of events in neighboring membrane parts, the action potential flows as a wave of excitation over the cell membrane; it travels from the axon hillock down to the dendritic terminal where the depolarization releases neurotransmitter from the cell and causes an EPSP or IPSP to occur in other neurons. The action potential itself causes only a very brief local current that does not penetrate far into the extracellular space. The amount of neurotransmitter released by the action potential depends only on the number of action potentials that are produced per second.

1.1.4 Summation of electrical potential changes in the cortex occurs mainly at the vertically oriented large pyramidal cells of the cortex. These neurons are especially suited for this role for several reasons.

(a) The dendrites of the pyramidal cells extend through nearly all layers of the cortex, guiding the flow of currents generated by postsynaptic potentials at either the cell body in the deep layers of the cortex or at dendrites in the more superficial layers through the entire thickness of the cortex.

(b) Cortical pyramidal cells are closely packed into functional vertical columns each containing several hundred cells oriented parallel to each other, facilitating spatial summation of the currents generated by each neuron towards the cortical surface.

(c) Groups of these neurons receive similar input and respond to it with potential changes that produce electrical currents of similar direction and timing. One afferent axon may contact several thousand cortical pyramidal neurons.

(d) The input to a pyramidal cell is magnified by the number
of synapses on each cell. Each pyramidal cell contains over 100,000 synapses.

The currents generated by these neurons summate in the extracellular space as indicated in Fig. 1.1. Most of the current is limited to the cortex. However, a small fraction penetrates through the meningeal coverings, spinal fluid and skull to the scalp where it causes different parts of the scalp to be at different potential levels. These potential differences, of usually only 10 to 100 μV, can be recorded between two electrodes and constitute the EEG.

Although the EEG is a result of individual neuronal potential changes, microelectrode activity from individual cortical cells correlates poorly with the ongoing EEG activity. This is partly because an extremely large number of potentials summate to produce the EEG. Furthermore the summing effects of volume conduction help to obscure the overlapping contributions (temporal dispersion) of individual neurons (see 1.3).

Interestingly, the normal geometry of synaptic distributions over the pyramidal cell makes it impossible to know whether an EEG event at the scalp is due to an inhibitory or excitatory postsynaptic potential. This is because excitatory and inhibitory synapses at opposite ends of a vertically oriented cortical pyramidal nerve cell can produce the same polarity potential changes at the cortical surface.

The biophysical explanation for this phenomenon is relatively straightforward. As mentioned in (1.1.2), the postsynaptic potential creates a cellular circuit of current. In the case of an EPSP, this current flows into the cell at the synapse as sodium ions enter the cell. The current then proceeds through the cell, emerges at a distance from the synapse, and then returns to the synapse in the extracellular space. Although EPSPs and IPSPs induce opposite polarity changes (with opposite directions of current flow), if the EPSPs and IPSPs are located at opposite ends of the vertical pyramidal cell, then the circuit of current flow seen by a surface EEG electrode will appear to have the same polarity whether it is caused by an EPSP or IPSP. Therefore, if an excitatory and an inhibitory synapse are located at opposite ends of a vertically oriented pyramidal cell, both will produce the same polarity EEG change at the cortical surface. Although there is technically no way to know if a given EEG waveform is generated by inhibitory or excitatory postsynaptic potentials (or various combinations of both), the inhibitory or excitatory generators of certain waveforms can sometimes be inferred from experimental information. For example, the spike component of the spike and slow wave pattern seen in patients with seizure disorders is known to be generated by excitation (e.g., the paroxysmal depolarizing shift) and the slow wave by inhibition.

1.2 RHYTHMICAL EEG ACTIVITY

Although one might expect that the complex neuronal activities of the brain would result in irregular EEG waves, the human EEG, recorded during wakefulness or sleep, commonly contains rhythmical activity (e.g., the alpha rhythm, mu rhythm, sleep spindles). Current understanding of the mechanisms responsible for the production of rhythmical EEG activity is based largely on animal experimentation. These experiments have shown that:

(a) slow frequency repetitive stimulation of nonspecific nuclei of the medial and intralaminar thalamus produces rhythmical cortical activity in a widespread distribution that resembles barbiturate induced rhythmical spindle shaped activity (the so-called recruiting response);
(b) repetitive stimulation of specific nuclei of the lateral thalamus produces rhythmical cortical activity over more localized areas (the so-called augmenting response) corresponding to the thalamic activity.

Fig. 1.2. Steps in the production of rhythmical EEG activity in the cortex (C) as proposed by the original facilitative pacemaker theory. (a) A thalamocortical relay neuron (TCR) in the thalamus (T) is excited by an afferent neuron and sends an impulse simultaneously to cortical neurons and to an inhibitory thalamic interneuron. Postsynaptic potentials of cortical neurons generate the first deflection in the cortical EEG. (b) The output of the inhibitory thalamic interneuron suppresses the activity of several TCR neurons. The resultant disappearance of postsynaptic cortical activity represents the end of the first deflection of the cortical EEG. (c) Several TCR interneurons, simultaneously released from inhibition by interneurons, send synchronous impulses to cortical neurons, causing another larger deflection of the cortical EEG. Simultaneous impulses to larger numbers of inhibitory thalamic interneurons initiate the next cycle of rhythmical activity.
responding in distribution to the thalamocortical pathways for primary sensory information;

(c) transection of the brainstem below the level of the thalamus has little influence on sleep spindles or barbiturate induced spindle-like activity, but destruction of the thalamus obliterates such activity;

(d) following surgical isolation of the cortex from the thalamus rhythmic activity persists in the thalamus whereas little evidence of such activity is recorded at the cortex;

(e) thalamic neurons in the nucleus reticularis continue to demonstrate rhythmic bursting activity (7-14 Hz) after they have been surgically or pharmacologically isolated (by synaptic blockade, in vitro), whereas similar rhythmic activity in the cortex and in target structures of the nucleus reticularis ceases;

(f) the similarity between 2 EEG alpha rhythms (i.e., the coherence) is greater between nearby cortical areas than between thalamic and cortical areas; but in experiments in dogs, for example, the pulvinar nucleus of the thalamus shows greater coherence with cortical activity than other thalamic areas;

(g) delta (< 4 Hz) waveforms in sleep occur with greatest amplitude in cortical layer V and correlate with periods of reduced pyramidal cell neuron activity.

These observations have led to the prevailing view that, in most cases, EEG rhythmicity begins with cortical activation or pacing by thalamic pacemaker cells. However, the cellular mechanism that is responsible for the pacing activity itself remains unknown.

Two leading models of how the thalamus interacts with the cortex to induce rhythmicity have been proposed. The first, and oldest, proposed by Andersen and Andersson (1968) is referred to as the facultative pacemaker theory.

According to this theory, thalamocortical cells send fibers to the cortex as well as giving off branches that turn back and end on thalamic inhibitory interneurons (Fig. 1.2a). The firing of one or a few thalamocortical neurons, in addition to affecting a few cortical neurons, excites thalamic inhibitory interneurons via recurrent collateral fibers (Fig. 1.2a). The output of the interneurons inhibits a large number of thalamocortical cells (Fig. 1.2b). At the end of the period of inhibition, the pool of thalamocortical neurons overshoots into excitation, giving off a synchronized volley which is again distributed both to cortical neurons and to inhibitory thalamic interneurons (Fig. 1.2c). The interneurons inhibit an even larger number of thalamocortical neurons, thus generating another cycle of rhythmic discharges. Theoretically, interneurons that have an inhibitory action lasting a tenth of a second could cause periodic synchronous inhibition and rebound excitation of thalamocortical neurons at about 10 times a second (in the frequency range of alpha activity). The projection of these impulses to the cortex at the same rate would thus induce presynaptic potentials and a 10 Hz rhythmic EEG activity.

More recent work has demonstrated the presence of cells in
the nucleus reticularis of the thalamus that have intrinsic pacemaker properties responsible for the EEG sleep spindle pattern. The nucleus reticularis is a thin layer of neurons covering much of the anterior, ventral and lateral surfaces of the thalamus. These cells release the inhibitory transmitter GABA in rhythmic bursts of depolarizations that are directed to the neurons of the dorsal thalamus and rostral brainstem. The pacemaker cells stimulate the thalamocortical cells of the thalamus, which then produce rhythmic excitation in the cortex. The excitatory thalamocortical cells have the intrinsic property of producing bursts of depolarizations as a rebound response to each inhibitory stimulus of the pacemaker cells. The rhythmic pacing activity within the nucleus reticularis continues when the cells are surgically or pharmacologically isolated. Not only thalamocortical cells, but other thalamic, neocortical, and hippocampal cells that contain excitatory amino acids also have the intrinsic property of producing burst discharges. The burst discharges occur during periods of behavioral non-arousal whereas more continuous firing patterns occur during periods of arousal. The burst discharge consists of a sudden series of action potentials followed by a prolonged afterhyperpolarization. Burst firing is associated with rhythmic EEG patterns whereas transition to a steady firing pattern is associated with a cessation of rhythmic activity. Individual cell bursts usually occur in synchrony with bursting activity in other cells within the same neuronal network population.

Cortical rhythmicity depends, in part, on networks within the basal forebrain and brainstem and projections from the raphe nuclei and locus ceruleus. These neuronal groups receive input from practically all sensory systems and cortical areas and send their output to the entire cortex through direct connections and relays in the diencephalon (Fig. 1.3a). Rhythmic activity is thereby interrupted through a direct effect on cortical neurons and indirectly through the modulation of thalamic neurons that participate in the pacing of cortical rhythms. As mentioned above, at the cellular level desynchronization is accompanied by a transition from a burst firing pattern to a more continuous or single spike pattern. Desynchronization is enhanced by behavioral arousal and suppressed by non-REM sleep (i.e., stages I, II, III and IV sleep). Abnormal rhythmic EEG patterns, such as the alpha coma pattern, occur in the setting of widespread injury (such as anoxia or brain trauma) to the ascending neuronal systems that would otherwise produce arousal and desynchronization.

1.3 RECORDING OF ELECTRICAL POTENTIALS WITH SCALP ELECTRODES

As previously stated, electrodes on the scalp record mainly the summated electrical changes of the underlying cortex; they may also rarely record some potential changes generated in distant parts of the brain, as well as potential changes produced outside the brain (i.e., artifacts). The amplitude of the recorded potentials depends on the intensity of the electrical source, on its distance from the recording electrodes, its spatial orientation, and on the electrical resistance and capacitance of the structures between the source and the recording electrode. These factors favor the recording of potential changes which (a) occur near the recording electrodes, (b) are generated by cortical dipole layers that are oriented towards the recording electrode at a 90° angle to the scalp surface, (c) are generated in a large area of tissue, and (d) rise and fall at rapid speed.

The EEG recorded with scalp electrodes differs from that recorded simultaneously with electrodes placed directly on the underlying cortex (also referred to as the 'electrocorticogram' or 'ECoG'). The scalp EEG is of lower amplitude and is by comparison somewhat distorted in shape. Generally, faster frequencies are attenuated more than slower ones. Very fast and brief potential changes may be lost completely in scalp recordings or may be picked up only over their production site or near skull defects, whereas slower potentials tend to be conducted farther and thus recorded over greater distances.

Comparisons of simultaneous scalp and cortical recordings of normal activity suggest that at least 6 cm² of cortex with synchronous activity is needed to create a reliably recorded scalp potential. Higher intensity, highly synchronized potentials, such as epileptiform spikes, can occasionally be recorded at the scalp with the activation of somewhat smaller areas of cortex. These filtering effects depend in part on the electrical
properties of structures between cortex and scalp electrodes. The amplitude of the scalp EEG may decrease as a result of either: (a) an increase of the overall electrical impedance between the source and the recording electrodes, for instance an increase in the thickness of the skull, which reduces the flow of currents between the source and the recording electrodes, or (b) a decrease in the impedance at different stages across the path of these currents, as may occur with a collection of subdural blood or cerebrospinal fluid that shunts (‘short-circuits’) the currents before they reach the recording electrodes.

Even though the scalp EEG reflects local potential changes, cortical potentials of very similar shape and timing over wide parts of the brain may be triggered by more localized cerebral disorders. This process has been called ‘projection’ and the resulting rhythms have been called ‘projected rhythms’ or ‘rhythmes à distance.’ Because the pacemaker for bilateral synchronous discharges was originally presumed to be located at the center of the brain, the term ‘centrencephalic’ was used. Although these rhythms may be induced by the action of distant centers, the recorded EEG is generated by cortex near the recording electrodes, not by distant or subcortical sites. Moreover, although it is clear that most widespread rhythms are secondarily generated from initiating sites in the thalamus, it is also clear that bilateral synchronous activity may arise from cortical lesions, particularly those that produce epileptiform activity.

Scalp electrodes only rarely record potentials generated at distant sites. This is illustrated by the observation that scalp electrodes over a large area of cortex with completely abolished function will generally show complete absence of electrical activity. In addition, individual epileptiform spikes generated solely by the hippocampus within the temporal lobe never appear in simultaneous scalp recordings and very rarely in direct subtemporal cortical recordings. Rarely, some types of high amplitude activity, such as slow waves or cortical spikes arising from a large cortical area, are conducted electrically through the volume of the interposed brain tissue. They then may appear in scalp electrodes at considerable distance, intermixed with the EEG representing more proximal cortical activity. Electrical conduction also accounts for the appearance of EEG potentials at scalp electrodes ipsilateral to hemispherectomies.

Aside from placing electrodes directly in the cortex, the alternatives for improving the detection of cortical potentials include: (1) changing the spatial filtering characteristics of the recording electrodes by changing their physical size, (2) changing the spatial filtering characteristics by altering the combinations of electrodes placed in any one amplifier (e.g., change of montage; Chapter 4), and (3) increasing sampling at the scalp surface by placing additional scalp electrodes, creating smaller interelectrode distances. Sampling can be increased to the point where electrodes are placed one electrode diameter apart from each other. This has led some manufacturers to develop electrode scalp nets for rapid placement of up to 128 electrodes at a time. It has also led to the relatively recent development of a newer system of electrode placement in which the nomenclature for electrode placement has been expanded to include more than double the previous number of electrodes (the 10–20 versus the modified 10–20 system; 2.3). In routine practice at least 21 scalp electrodes are used for EEG recording.

Potentials that are generated by sources other than the brain that are picked up by scalp electrodes and recorded together with the EEG are referred to as artifacts (6). For instance, scalp electrodes located over muscle frequently record muscle fiber activity. Movements of the eyes, tongue and other large and electrically charged structures also generate changing electrical fields, which are recorded by scalp electrodes. Heart muscle contraction can induce potential changes at scalp electrodes similar to the potential changes recorded in the electrocardiogram. Strong sources of alternating current near the recording site may interfere with the recording.

The transmission of a signal through a volume conductor occurs nearly at the speed of light. Therefore, similar appearing waveforms that occur in different or non-adjacent scalp locations without any difference in their time of appearance most likely arise from the same cortical generator. In contrast, those that appear with any measurable time delay between them must involve transsynaptic conduction and cannot arise from the same cortical cells. Since transsynaptic transmission may occur within milliseconds, such distinctions can only be made using digital EEG systems. Analog EEG systems do not display signals with sufficient time resolution to distinguish timing differences of several milliseconds between different recording channels.

REFERENCES


