1. Neurophysiological Basis of the EEG and of DC Potentials

Erwin-Josef Speckmann and Christian E. Elger

Introduction

The clinical electroencephalographer correlates central nervous system functions as well as dysfunctions and diseases with certain patterns of the electroencephalogram (EEG) on an empirical basis. Obviously, this method has been found valuable in clinical practice. Therefore, why should the clinical electroencephalographer study the basic elementary processes underlying the EEG? There is little doubt that the range of EEG interpretations can be much widened and misinterpretations avoided when the underlying elementary processes are also considered. This is true especially for convulsive disorders and cerebral metabolic disturbances. For example, an isoelectric EEG can be caused by selective pCO₂ increase while the brain is sufficiently supplied with O₂. On the other hand, in the presence of practically normal pCO₂ levels, cerebral hypoxia may be the cause. It will be pointed out below that the prognosis may be quite different in these two cases.

Elementary Processes Underlying the Generation of Extracellular Field Potentials

Here the basic mechanisms which give rise to potentials recorded outside the central nervous system (CNS) elements will be described. Such extracellular potentials are generally known as “field potentials.”

In the course of this presentation, the morphology of generator structures will be discussed briefly. Then the electrical activity demonstrable with intracellular recordings from neurons and glia cells will be described. On the basis of these data, the principles of the generation of extracellular field potentials will be outlined and the various types of field potentials will be characterized.

Generator Structures

The central nervous system essentially consists of nerve cells and glia cells. The arrangement of neurons usually shows a specific type of laminar character. Glia cells are located between neurons.

As shown in Figure 1.1, several processes emerge from the nucleus-containing cellular soma (body) of the nerve cell. These processes can be divided into two types according to their function. Most of the processes are dendrites which branch off into numerous small ramifications. Furthermore, every cell has an axon which may split up into multiple collaterals. Such an axon provides contact with other nerve cells or with other target organs. In the case of interneuronal connections, the contact consists of synapses which cover the dendrites, the soma, and the axon hillock in large numbers. Thus, nerve cells are usually covered with several thousand synapses (Palay and Chan-Palay, 1977).

The glia cells are imbedded between nerve cell somata, dendrites, and axons. They usually have several processes which make contact with somata and processes of nerve cells; they may also make contact with vessels. This histological arrangement results in a cerebral extracellular space consisting of very narrow intercellular clefts (Derobertis and Carreira, 1965).

Neuronal Activity Recorded Intracellularly

Next, those essential potentials which can be demonstrated with intracellular recordings will be characterized briefly. When the membrane of the nerve cell body is penetrated by a microelectrode, a potential of about 60 to 70 mV with negative polarity in the intracellular space can be recorded.
2 Neurophysiological Basis of the EEG and of DC Potentials

This membrane potential is subject to various fluctuations which are elicited chiefly by synaptic activities. Their mechanisms are shown in greater detail in Figure 1.2. As can be derived from this schematic illustration, the neuron from which the soma membrane potential is recorded has synaptic connections. The corresponding presynaptic structures are also explored with microelectrodes. If an action potential travels along the fiber which ends in an excitatory synapse, an excitatory post-synaptic potential (EPSP) occurs in the following neuron (Fig. 1.2, A). If two action potentials travel along the same fiber with a short interval, there will be a summation of EPSP triggering an action potential on the postsynaptic neuron after reaching the membrane threshold. If an action potential travels along a fiber ending in an inhibitory synapse, then hyperpolarization will occur representing an inhibitory post-synaptic potential (IPSP) (Eccles, 1964; Hubbard et al., 1969; Shepherd, 1974).

Because of the time course of the various membrane potential fluctuations, the post-synaptic potentials are thought to contribute primarily to the generation of the extracellular field potentials in question (Creutzfeldt and Houchin, 1974; Hubbard et al., 1969). For this reason, the ionic mechanisms of these potentials are discussed in greater detail. The individual events of this process will be presented with a magnified time base. With the elicitation of an EPSP, a net inflow of cations occurs across the subsynaptic membrane. This gives rise to depolarization of the subsynaptic membrane. As shown in Figure 1.2, B, a potential gradient develops along the neuronal membrane in the intra- and extracellular space. Because of this potential gradient, cations move along the nerve cell membrane through the extracellular space in the direction of the subsynaptic region. An inversely directed flow takes place in the intracellular space. With the generation of an IPSP, there is an outflow of cations from the nerve cell and/or an inflow of anions into the nerve cell. These changes first increase the membrane potential at the subsynaptic membrane in comparison with the surrounding segments of the membrane. For this reason, a potential gradient develops along the nerve cell membrane, as in the case of the EPSP genesis. This potential gradient causes, in the extracellular space, a flow of cations from the subsynaptic region to the surrounding portions of the membrane. An inverse process develops in the intracellular space (Hubbard et al., 1969).

Fig. 1.1. Schematic drawing of morphology and histology of neuronal and glial elements.

![Diagram of neuron and glial elements](image)

Fig. 1.2. Membrane potential (MP) changes and current flows during synaptic activation.

A The MP of the postsynaptic neuron and the MP of the presynaptic fiber are recorded by means of intracellular microelectrodes. Action potentials in the excitatory and inhibitory pre-synaptic fiber lead to excitatory (EPSP) and inhibitory post-synaptic potentials (IPSP), respectively, in the postsynaptic neuron. Two EPSP sum up to a super-threshold potential, triggering an action potential in the postsynaptic neuron.

B During EPSP and IPSP, ionic current flows occur through as well as along the neuronal membrane, as shown by arrows. The density of (+) and (−) signs indicate the polarization of the subsynaptic (dark area) as well as that of the postsynaptic membrane during synaptic activation.
The ion fluxes in the extracellular space are of paramount significance in the generation of field potentials. Therefore, these processes are further discussed in the following chapters.

Glia Activity Recorded Intracellularly

In addition to the neurons, glial cells may also play a role in the generation of extracellular field potentials (Kuffler and Nicholls, 1966; Somjen and Trachtenberg, 1979). Therefore, the bioelectric properties of glial cells will be summarized.

If a glia cell is penetrated with a microelectrode, a membrane potential can be recorded with a polarity similar to that of the nerve cells. The size of this membrane potential approximates the potassium equilibrium potential and hence somewhat exceeds the membrane potential of nerve cells. In contrast with neurons, glial cells fail to show any action potentials and there are also no post-synaptic potentials. Thus, in contrast to neurons, glia cells do not show characteristic potentials which distinguish them unmistakably from other cells. The glial membrane potential, however, is also not constant. An augmentation of the extracellular potassium concentration (potassium activity) causes depolarization of glial cells (Fig. 1.3, A). Concentration changes of other ions cause only negligible alterations of the glial cell membrane potential. The glial cell is hence comparable with a potassium electrode (Kuffler and Nicholls, 1966; Kuffler et al., 1966).

The dependency of the glial membrane potential on the extracellular potassium concentration is the reason for a functional linkage with adjacent neuronal structures. Neuronal activity is associated with outflow of potassium ions. As shown schematically in Figure 1.3, B, repetitive firing of neurons gives rise to increased extracellular potassium concentration and hence to glial cell depolarization (Ornstein et al., 1966). If the potassium concentration does not affect the entire glial cell membrane and remains increased only locally, then potential gradients build up along the glial cell giving rise to intra- and extracellular current flows similar to the ones described in reference to neuronal synaptic transmissions. Glial cells frequently have widespread processes and, furthermore, may have close connections with each other. For this reason, potential fields of considerable spatial extension may develop on the basis of the aforementioned mechanisms (Somjen and Trachtenberg, 1979). In view of the above described functional interconnections, it is quite likely that, in the genesis of extracellular field potentials, an amplifying effect can be attributed to the glial cells.

Generation of Extracellular Field Potentials

It has been shown in the preceding chapter that primary transmembranous currents generate secondary ional currents along the cell membranes in the intra- and extracellular space. The portion of these currents which flows through the extracellular space is directly responsible for the generation of field potentials. As mentioned above, particular significance must be ascribed to the synaptic processes as causing events for the field potentials in question, especially for their time course. In accordance with these statements, the generation of extracellular field potentials will be discussed exemplified by extracellular fields accompanying synaptic activity (Hubbard et al., 1969; Rall, 1977). The discussion of these events will again make use of a very protracted time axis. The explanation of the events will be given in reference to the schematic view in Figure 1.4. This figure shows a widely stretched neuronal element, with one end segment lying close to the surface of a central nervous structure. At both ends of this neuronal unit, the microelectrodes ME, and ME; are inserted.

![Fig. 1.3. Membrane potential (MP) changes of glia cells induced by an increase in the extracellular K⁺ concentration (arrows in the schematic drawings). A Potassium is applied extracellularly to the glia cell. B The potassium concentration is increased due to an activation of a neighboring neuron. (Drawings after original tracings from Kuffler et al., 1966.)](image-url)
At the same time, the extracellular electrodes $E_1$ and $E_2$ are located at the surface and at the deeper end of the neuronal element. The potentials picked up from the intra- and extracellular electrodes are shown in the vicinity of each electrode. The potential recorded from the surface of the nervous structure is accentuated by thicker lines. Figure 1.4 shows active excitatory and inhibitory synapses, either close to the surface or located in the depth. As described elsewhere, the activation of an excitatory synapse leads to a net inward flow of cations. If this statement is applied to part A1 of Figure 1.4, then it becomes evident that the upper end of the neuronal element will be depolarized in comparison with other segments of the same cell. Accordingly, the synaptic current flow causes an EPSP at the microelectrode $ME_1$. This local depolarization then gives rise to further intra- and extracellular ionic currents along the nerve cell membrane. Because of the intracellular movements of positive charges, depolarization in the area of microelectrode $ME_1$ will also take place. This depolarization, however, is less steep and of smaller amplitude. At the superficially located extracellular electrode $E_1$, the inflow of positive charges into the neuronal element causes a negative field potential. The extracellular electrode $E_2$, is, metaphorically speaking, approached by positive charges so that a positive field potential will develop in this area. The point of reversal of the field potentials is localized between electrodes $E_1$ and $E_2$. The exact position of the point of reversal depends on the distribution of extracellular impedances.

Current flows of reversed direction (in reference to the recording electrodes) will occur if the active excitatory synapse is located at the deeper end of the neuronal element (Fig. 1.4, A2). In this case, positive charges approach the superficially located electrode ($E_1$) (again speaking metaphorically) and move themselves from the deeply located electrode ($E_2$). This arrangement of the active synaptic structures causes a positive field potential at the surface and a negative one at the deep electrode. The current flows accompanying the activation of inhibitory synapses located in deeper and in more superficial areas, respectively, are shown in part B of Figure 1.4. As can be derived from this illustration, the activation of a deep inhibitory synapse (Fig. 1.4, B1) produces a current flow which is largely similar to the one generated by the activation of a superficial excitatory synapse (Fig. 1.4, A1). In the same manner, there are also similar current flows in the extracellular space when a superficial inhibitory synapse (Fig. 1.4, B2) or a deeply located excitatory synapse is activated (Fig. 1.4, A2). Accordingly, a negative field potential will develop at the surface of a central nervous structure (in the schematic view of Fig. 1.4) whenever a superficial excitatory or a more deeply located inhibitory synapse is activated. The corresponding principle applies to generation of the superficial field potentials of positive polarity.

### Types of Field Potentials

The field potentials, the generation of which has been described, can be subdivided into different types. If field
potentials are recorded against an inactive reference point with an upper frequency limit of about 100 Hz, then two types of field potentials can be distinguished, depending on the time constant of the amplifying recording device. In the case of a time constant of 1 sec or less, the extracellular field potentials correspond with that which is commonly known as the electroencephalogram (EEG). If the recording is carried out with an infinite time constant, i.e., with a DC amplifier, then slower potentials can also be picked up. Potentials recorded with this technique are generally known as DC potentials (Caspers, 1974). Thus, DC potentials comprise slow as well as fast field potentials. The fast components correspond with the potential fluctuations of the EEG. Due to different time constants, however, the faster potential components may differ from each other as far as their time course is concerned when recordings are done either with conventional EEG amplifiers or with DC amplifiers.

Thus far, technical problems have made it difficult to carry out DC recordings from the scalp. Except for special areas of application, DC recordings are usually performed in animal experiments. DC potentials directly reflect the state of activity of central nervous cells and therefore contribute to the explanation of the mechanisms of genesis of cerebral field potentials (Speckmann and Caspers, 1979). For this reason, DC potentials will be discussed jointly with EEG waves.

For the sake of comparison, Figure 1.5 shows the EEG and the DC potentials during convulsive activity, hypercapnia, and asphyxia. As shown in this illustration, a tonic-clonic convulsion is associated with a negative DC shift (Caspers and Speckmann, 1969; Gunnit et al., 1970). Furthermore, it can be seen that the hypercapnia-induced disappearance of the EEG is associated with a monophasic positive DC shift. In the case of EEG extinction due to primary asphyxia, however, there are characteristic patterns of DC fluctuations. Hence, similar findings in the conventional EEG may be associated with different DC shifts.

Wave Generation

In the preceding sections, the generation of single field potentials was described. In the following, the principles of the generation of wave-like potential fluctuations will be outlined. This will be followed by the discussion of the laminar distribution of such potentials in the cerebral cortex.

Principal Mechanisms

In order to present the generation of wave-like potential fluctuations on the surface of a central nervous structure, a simple model as shown in Figure 1.6 will be used. This model consists of two extended pyramidal neurons of vertical orientation. Terminals of afferent fibers make contact with the superficial dendrites of both neurons via excitatory synapses. The bioelectrical activity of these structures is recorded with intracellular microelectrodes. The microelectrodes $E_1$ and $E_2$ are located in the ascending fibers and the microelectrodes $E_3$ and $E_4$ are in the superficial dendrites of the postsynaptic neurons. In order to pick up the extracellular field potentials, the electrode $E_5$ lies on the surface of the central nervous structure.

As shown in tracings 1 and 2, action potentials occur synchronously in the afferent fibers. There are grouped discharges which are temporarily supplanted by tonic activity. The ascending action potentials elicit individual EPSP in the upper dendrites of the neurons; these EPSP are subsequently summated into major depolarizations in accordance with the discharge frequency. As shown in tracings 3 and 4, amplitude and duration of the depolarizations depend on the discharge pattern of the afferent fibers. The synaptic activity at the superficial structures gives rise to extracellular current flows resulting in superficial field potentials. With the use of DC recording techniques, the superficial field potentials reflect the potential fluctuations of the dendritic membrane. If, however, the superficial field potentials are recorded with a time constant of 1 sec and less, then only the fast fluctuations of the superficial field potentials are demonstrable.
Thus far, the principle of genesis of EEG and DC waves have been shown in the schematic view of Figure 1.6. Accordingly, the generation of physiological EEG waves may be explained as follows. If a grouped and synchronous influx takes place in afferent fiber systems toward the superficial generator structures, then EEG waves evolve which are of high amplitude and distinctly separated from each other. In case of a periodic sequence of the afferent bursts, the recording of the field potentials shows sinusoidal potential fluctuations. This mechanism has been presumed by several groups of investigators as the principle of the generation of the alpha rhythm and slower periodic EEG waves. According to these workers, thalamo-cortical feedback waves are believed to play a significant role in the generation of the alpha rhythm (Andersen and Andersson, 1968).
If the afferent influx of impulses occurs at a high frequency for a longer period and/or asynchronously, then negative field potentials with small fluctuations will result from the extracellular current flows. Accordingly, the EEG recording will pick up only waves of smaller amplitude and mostly higher frequency. In the DC recording, however, the prolonged depolarization of the superficial structures caused by the afferent high frequency influx will express itself by a negative DC potential shift (Caspers, 1963; Goldring, 1974). There is a close correlation between the amplitude of the negative DC shift and average discharge frequency in the afferent fiber systems. This mechanism may principally apply to the generation of beta activity and other EEG waves of higher frequencies. A decrease of the amplitudes of the EEG waves can also occur when the afferent activity is diminished. In this case, however, the depression of EEG waves is accompanied by a positive DC shift (Caspers and Speckmann, 1974; also see Fig. 1.13).

Spatial Distribution Within the Cortex

The principles of generation of individual and wave-like field potentials at the surface of central nervous structures such as the cerebral cortex have been described. If the wave-like potential fluctuations are recorded not only from the cortical surface but also from different cortical layers, then it can be shown that potential fluctuations in the latter recordings may differ considerably from those at the surface. These differences imply polarity, frequency, and amplitude (Petsche et al., 1978). Such a recording from the cortex of the rat is shown in Figure 1.7. According to this illustration, field potentials reverse their polarity between electrode 1 (on the surface) and electrode 2 (located 300 μm beneath the cortical surface). Two and sometimes more of such phase reversals may be observed in deeper recording sites depending on the experimental conditions. The vertical distribution type of field potential will be discussed in greater detail in connection with the generation of cortical field potentials during convulsive activity.

Cortical Field Potentials During Epileptiform Activity

In the course of the discussion of cerebral field potentials, it was pointed out that particular significance must be attributed to synaptic activity. A view at the laminar distribution of neurons in the cortex and the dense coverage of these unitary structures with synapses makes it clear that different patterns of potentials must necessarily occur in different layers when populations of synapses are activated in a different manner. This should be clarified by the schematic drawing in Figure 1.7.

Spatial Distribution Within the Cortex

The principles of generation of individual and wave-like field potentials at the surface of central nervous structures such as the cerebral cortex have been described. If the wave-like potential fluctuations are recorded not only from the cortical surface but also from different cortical layers, then it can be shown that potential fluctuations in the latter recordings may differ considerably from those at the surface. These differences imply polarity, frequency, and amplitude (Petsche et al., 1978). Such a recording from the cortex of the rat is shown in Figure 1.7. According to this illustration, field potentials reverse their polarity between electrode 1 (on the surface) and electrode 2 (located 300 μm beneath the cortical surface). Two and sometimes more of such phase reversals may be observed in deeper recording sites depending on the experimental conditions. The vertical distribution type of field potential will be discussed in greater detail in connection with the generation of cortical field potentials during convulsive activity.

In the following sections, the generation of cortical field potentials during convulsive activity will be discussed. The first section deals with focal, and the second one with generalized, tonic-clonic convulsive activity. For methodical reasons, we will refer to data derived from experimental work in the animal.

Focal Activity

If a convulsive substance such as penicillin is applied to the surface of the cerebral cortex, steep negative potentials of high amplitude can be picked up from the area of application after a short latency period. These discharges repeat themselves in stereotyped form and periodicity (Purpura et al., 1972; also see Fig. 1.8 A). If the membrane potential of a cortical neuron is simultaneously recorded with a microelectrode while a second microelectrode picks up the corresponding field potentials, then potential fluctuations will occur as shown in Figure 1.8 B. It can be derived from this illustration that the monotonously recurrent negative field potentials are associated with equally stereotyped membrane potential fluctuations.
These oscillations of the membrane commence with a steep depolarization which, having exceeded the membrane threshold, will trigger a series of action potentials. This is followed by a plateau which, after 80 to 100 msec, changes into a steep repolarization and frequently also into a hyperpolarization. These membrane potential fluctuations have proved to be characteristic in the epileptiform activity of individual neurons. They are generally known as paroxysmal depolarization shifts (PDS) (Jasper et al., 1969).

Investigation of potential distribution within the cerebral cortex following the local application of penicillin will yield a variety of findings. An appropriate model is shown in Figure 1.9. In this experiment, recordings of interictal field potentials were carried out from the cortical surface, from inside the cortex, and from the spinal cord. The spinal field potentials permit the observation of electrical activity descending from the cortex to the spinal cord. In part A of Figure 1.9, negative field potentials are recorded from the cortical surface and from the two upper intracortical contacts following the application of penicillin together with penicillin-metabolizing enzyme penicillinase. There are, however, field potentials with predominantly positive components in the deeper contacts 4-6. If penicillin is applied to the surface without penicillinase, then negative field potential will also develop in deeper cortical layers. If it is assumed that the negative field potentials mirror the direct epileptiform activity of neuronal structures (also see Fig. 1.3), then it must also be assumed that deeper cortical elements are involved in convulsive activity in part B of Figure 1.9 in contrast with part A. This is further supported by the observation that neuronal activity descending to the spinal cord and producing characteristic spinal field potentials occurs only under the experimental conditions shown in part B. If one compares the recordings in parts A and B, it will become clear that, with a monotonous epileptiform potential at the cortical surface, the intracortical potential distribution and the occurrence of descending activity may differ considerably (Elger and Speckmann, 1980; Elger et al., 1981; also see Gumnit, 1974; Petsche et al., 1981).

If penicillin is applied to deeper cortical laminae (Fig. 1.9, C), then negative field potentials will be confined to that region. These potentials are consistently accompanied by descending activity to the spinal cord. Under these conditions, there is frequently nothing but a positive potential fluctuation of minor amplitude at the cortical surface (Elger et al., 1981).

In summary, it can be derived from the described experimental models that, in focal convulsive activity limited to the cortex, the surface potential does not necessarily reflect the bioelectrical events in deeper cortical layers.

**Generalized Tonic-clonic Activity**

In the following, possible mechanisms involved in the generation of cortical field potentials during tonic-clonic convulsive activity will be described. Again, data are based on experimental observations in the animal. Tonic-clonic convulsive activity was triggered by repeated injections of pentylentetrazol (also see Purpura et al., 1972).

Figure 1.10, A, shows a tonic-clonic convulsion recorded with a conventional EEG amplifier, as well as with a DC amplifier. As mentioned, there is a negative DC shift from the baseline during a convulsive seizure. This negative DC shift gradually recedes during the termination of the convulsions and frequently changes into a transient positive after-shift (Caspers and Speckmann, 1969; Gumnit, 1974; Speckmann and Caspers, 1979).

When the membrane potential of a pyramidal tract neuron of lamina V is recorded during a convulsive seizure, it can be shown that under these conditions typical par-
oxysmal depolarization shifts (PDS) become manifest (Fig. 1.10, B). If these PDS are correlated with the potential fluctuations in the DC recording, it can be noticed that the PDS in pyramidal tract neurons are coupled at the beginning of the convulsive seizure with superficial negative potential fluctuations and at the end of the convulsive seizure with surface positive potential fluctuations (Fig. 1.10, B) (Speckmann et al., 1978; Speckmann and Caspers, 1979).

In addition to the field potentials of the cortical surface and the membrane potentials of the pyramidal tract cells, field potentials were also recorded in the fifth lamina. Under these conditions, it can be shown that every PDS is associated with a negative monophasic field potential in the depth (Fig. 1.11, A). These stereotyped potential fluctuations in deep cortical layers correspond with field potentials at the cortical surface with either monophasic negative or positive (Fig. 1.11, A1 and 3) or with polyphasic configuration (Fig. 1.11, A2). This statement does not merely apply to individual ictal potentials but is also true for prolonged trains of potentials during the convulsion. As Figure 1.11. B, shows, paroxysmal depolarizations of pyramidal tract cells may be accompanied by a sequence of either negative or positive potentials on the cortical surface. If one correlates these various field potentials on the cortical surface with the slow DC shifts occurring during the convulsion (also see Fig. 1.10, A), then it can be demonstrated that the surface-negative field potentials are associated primarily with a slight DC shift and that surface-positive field potentials will appear when the negative DC shift at the cortical surface reaches and exceeds a critical value (Speckmann et al., 1972 and 1978; Speckmann and Caspers, 1979).

These data are interpreted with flow charts in Figure 1.12. As was pointed out, the amplitude of the negative DC shift at the cortical surface depends greatly on the

---

**Fig. 1.10.** Simultaneous recordings of EEG and DC/EEG (A) and of DC/EEG and membrane potential (MP) of a pyramidal tract neuron (B) during generalized tonic-clonic seizures elicited by pentylentetrazol. (Drawings after original tracings from experiments in the cat's motor cortex. The sweep speed in B is 10 times that in A.)
Fig. 1.12. Flow charts of neuronal processes possibly responsible for the generation of DC EEG waves of opposite polarity during a generalized tonic-clonic seizure. Hatched arrows, symbols for continuous asynchronous input to the cortex; heavy lines, symbols for phasic volleys giving rise to single convulsive discharges: PTC, pyramidal tract cell; IN, interneuron; MP, membrane potential; UA, extracellularly recorded unit activity. A) During a moderate asynchronous input to the cortex (small hatched arrow), a burst of UA triggers a paroxysmal depolarization shift in a PTC. Simultaneously, it leads to a depolarization of superficial neuronal structures and therewith to a negative fluctuation in the DC EEG recording at the cortical surface. B) With an increased asynchronous input to the cortex (wide hatched arrow), the DC potential shifts to a more negative level than in A (1). When in these conditions a phasic volley reaches the cortex, paroxysmal depolarization shifts are also triggered in PTC whereas the enhanced asynchronous UA is interrupted mainly due to inactivation. The latter process results in a disfacilitation of the upper neuronal structures and therewith to a positive fluctuation of the superficial DC/EEG potential (2). (Drawings of original tracings from Speckmann et al., 1978.)

amount of the afferent influx of impulses to the generator structures in the superficial cortical laminae. This predominantly asynchronous afferent influx is symbolized by the width of hatched arrows in Figure 1.12. Accordingly, the afferent influx in part A of Figure 1.12 is smaller than the one in part B. Therefore, there is a smaller DC shift in part A and a prominent one in part B. In the case of part A, a synchronized influx of impulses from subcortical structures is assumed to reach the cortex (widened afferent fiber in schematic view). As a consequence, pyramidal tract cells will be stimulated to generate a PDS and structures close to the surface will be depolarized through the mediation of interneurons. Accordingly, in such a constellation of excitatory processes, the paroxysmal depolarization in the depth will be coupled with a surface-negative field potential. With augmentation of the already existing afferent influx of impulses, the interneurons involved will necessarily exhibit a heightened level of excitation (Fig. 1.12, B). If an additional highly synchronized afferent influx of impulses takes place under these conditions, then further PDS will be triggered in the pyramidal tract cells, but, in the interneurons, the previously existing high frequency activity will be temporarily interrupted, chiefly due to inactivation. This will cause a decline of the excitatory influx of impulses to the superficial cortical structures. This disfacilitation gives rise to a positive field potential at the cortical surface. In this manner, a massive afferent influx of impulses provides the basis for a correlation of positive epicortical field potentials with stereotyped paroxysmal depolarizations and monophasic negative field potentials in the depth (Speckmann et al., 1978; Speckmann and Caspers, 1979).

Fig. 1.13. Effects of an isolated hypercapnia on epicortical field potentials (EEG, DC EEG) and on membrane potential (MP). With increasing pCO₂, the EEG disappears even if the pO₂ is above normal levels. The disappearance of the EEG is associated with a positive DC shift and a hyperpolarization of most of the neurons. Simultaneously, the amplitudes of stimulus (St) evoked EPSP are markedly reduced. (Drawings after original tracings from Speckmann and Caspers, 1974.)
Cortical Field Potentials During Gas Tension Changes in Tissue

The following sections will deal with the alterations of epicortical field potentials and concomitant changes of the membrane potentials caused by deviations of the gas tension in brain tissue. Such changes of the gas tension may occur when, for instance, the pulmonary and circulatory function is disturbed or when the local cerebral blood flow is inadequate.

First, the alterations of epicortical field potentials during selective hypercapnia will be discussed and then those associated with primary asphyxia. It will be shown that EEG changes may be similar under both conditions. The cortical DC potential, however, shows typical shifts which permit inferences concerning the cause of the accompanying EEG changes. The discussion of the effects of gas tension alterations on the bioelectrical activity of the CNS will be based, again, on data derived from experimental work in the animal.

Hypercapnia

If the CO₂ tension in the brain tissue is increased in a selective manner, typical reactions of the cortical field potentials as well as of the membrane potential and the postsynaptic potentials of individual neurons are found. These findings are shown in a summarized schematic view in Figure 1.13.

The animal experiments on which Figure 1.13 is based were carried out with the use of the so-called apnea technique. With this technique interference of the effects of hypercapnia with simultaneous effects of hypoxia could be avoided. According to this technique, the experimental animal is ventilated for at least one-half hour with pure oxygen. Thereafter, artificial ventilation is discontinued while the trachea of the animal remains connected with the O₂ reservoir. Under these conditions, the CO₂ tension progressively rises in the tissue for about 15 min without a concomitant fall of the oxygen tension below the baseline level.

With isolated increment of the CO₂ tension in the cerebral tissue by means of the apnea technique, the amplitude of the conventional EEG decreases progressively. This amplitude reduction affects first the waves of higher frequency and then those of lower frequency. Prior to the extinction of normal EEG activity, there is once again a phase characterized by high frequency EEG activity in the range of 50 to 70 Hz (Caspers et al., 1979). The extinction of the EEG is associated with a shift of the DC potential in a positive direction. If the CO₂ tension is then lowered again by reventilation, the EEG waves return in the original spectral composition after a short latency. At the same time, the positive DC shift resolves (Fig. 1.13). Experiments in animals have shown that, with reduction of the pCO₂, the EEG returns to normal activity even though the hypercapnia-induced suppression lasted for 1 hr or more.

In these cases, a positive DC deflection of monophasic character was found to occur during the whole period of apnea (Caspers and Speckmann, 1974; Caspers et al., 1979; Speckmann and Caspers, 1974).

Under the aforementioned conditions, the recording of the membrane potential of a cortical nerve cell shows a hyperpolarization while the CO₂ tension is increased. Extensive experimental studies in animals have demonstrated that such a hyperpolarization is caused primarily by a reduction of the excitatory postsynaptic potentials (Fig. 1.13; also see Speckmann and Caspers, 1974). Consideration of field potentials, of membrane potentials, and of EPSP shows that epicortical DC potentials reflect neuronal hyperpolarization. The disappearance of the EEG waves is presumed to be caused mainly by the reduction of postsynaptic activity.

---

Fig. 1.14. Alterations of EEG, DC/EEG and of neuronal membrane potential (MP) during a primary asphyxia. A) The abolition and the reappearance of EEG during a transient asphyxia goes in parallel with typical DC shifts: (1) initial negativity, (2) intermediate positivity, (3) reactive positivity. These DC fluctuations are accompanied by corresponding reactions of the MP. B) With continuing asphyxia the EEG remains abolished and the intermediate positivity (2) turns over into a terminal negativity (4). The latter DC negativity corresponds to a break down of neuronal membrane potential. (Drawings after original tracings from Speckmann and Caspers, 1974.)
Asphyxia

Primary asphyxia exemplified by respiratory arrest after air ventilation is associated with combined CNS effects of hypercapnia and hypoxia. The effects of gas tension changes on the field potentials and on the membrane potential of individual neurons are schematically shown in Figure 1.14. In the corresponding animal experiments, the artificial ventilation with air was either temporarily (part A) or persistently (part B) interrupted.

With such an interruption of artificial ventilation with air, the conventional EEG waves disappear within less than 1 min. This process is accompanied by a negative DC potential shift from the baseline, which has been characterized as “initial negativity” (see under 1 in Fig. 1.14). While the EEG shows an isoelectric line in the further course of asphyxia, additional potential shifts are detectable with DC recording technique. The initial negativity is followed by a positive DC shift termed “intermediate positivity” (see under 2 in Fig. 1.14). If reventilation is performed in this phase of asphyxia, an additional positive DC shift is observed, appropriately termed “reactive positivity” (see under 3 in Fig. 1.14). According to the analysis of the experimental work, the intermediate and the reactive types of positivity are due to an increase of CO₂ tension in the brain tissue. With the resolution of the reactive positivity, a restitution of the fast field potentials occurs which is also demonstrable with the conventional EEG. A comparison of the DC shifts and the alterations of the membrane potentials shows a parallelism of both events (Caspers and Speckmann, 1974; Caspers et al., 1979; Speckmann and Caspers, 1974).

If the interruption of the artificial ventilation is continued for a longer period of time, then the intermediate positivity converts into the so-called “terminal negativity” (see under 4 in Fig. 1.14, B). This negative DC shift correlates with the breakdown of the neuronal membrane potential. The terminal effects are due to a critical lack of oxygen. The terminal negativity may be reversible for a substantial period of time under certain experimental conditions if the artificial ventilation is resumed and the reduction of the cerebral circulation counteracted with circulation support measures (Speckmann and Caspers, 1974).

In summary, a comparison of EEG and DC potentials in selective hypercapnia and primary asphyxia shows that the recording of cortical field potentials with DC amplifiers provides a more accurate picture of the actual functional state of nerve cells.

References

References


