

Chapter 2

Preliminary Concepts

In this chapter of the book, we illustrate the brain structures, properties of the central nervous system, and generation of the electroencephalogram (EEG) signal. In particular, from the organization of the central nervous system and its peripheral organs, we describe the neuroanatomy of the brain cortex, and how neurons generate the electric potentials and create the synapses to consolidate the memory and send messages to the different parts of the brain and of the body. In fact, neurons are the basic units of the nervous system and are specialized to generate electrical signals, which are then used to encode and convey information: these signals are expressed by alterations of the resting membrane potential. The synchronization of neurons activity within specific frequency ranges define the characteristic rhythms (or bands) of the EEG. The scientific literature widely demonstrated how such EEG rhythms are correlated to specific cognitive processes, and brain lobes to specific functions and behavior, such as auditory rather than visual stimuli processing, long-term memory, and language. Therefore, the analysis of the EEG rhythms across the brain and how they communicate allow to investigate the different user's cognitive functions and mental states.

2.1 Nervous System

Gross Anatomy of the Brain

The nervous system essentially exhibits a bilateral symmetry, with structural features and pathways located on one side of the midline (Noback et al. 2005). It is subdivided anatomically into the *Central Nervous System* (CNS) and the *Peripheral Nervous System* (PNS), while functionally into the *Somatic Nervous System* (SNS) and the *Autonomic* (visceral) *Nervous System* (ANS). The CNS comprises the brain and the spinal cord. The brain is encapsulated within the skull, while the spinal cord is located at the center of the vertebral column. The PNS consists of the

nerves emerging from the brain (called *cranial nerves*) and from the spinal cord (called *spinal nerves*). The peripheral nerves convey neural messages from (1) the sense organs and sensory receptors in the organism inward to the CNS, and from (2) the CNS outward to the muscles and to the glands of the body. The SNS consists of those neural structures of the CNS and PNS responsible for (1) conveying and processing conscious and unconscious sensory (*afferent*) information, vision, pain, touch, unconscious muscle sense from the head, body wall, and extremities to the CNS; (2) motor (*efferent*) control of the voluntary (striated) muscles. The ANS is composed of the neural structures responsible for (1) conveying and processing sensory input from the visceral organs (e.g. digestive system and cardiovascular system); (2) motor control of the involuntary (smooth) and cardiac musculature, and of the glands of the viscera. The sensory signals originating in the sensory receptors are transmitted through the nervous system along sensory pathways, e.g. pain and temperature pathways and visual pathways. These signals may reach consciousness or may be utilized at unconscious levels. The neural messages for the motor activity are conveyed through the nervous system to the muscles and the glands, along motor pathways. Both the sensory pathways (ascending) and the motor pathways (descending) include processing centers for each pathway (e.g. ganglia, nuclei, laminae, cortices), located at different anatomic levels of the spinal cord and of the brain. The processing centers are the computers of the complex high-speed systems within the brain. Differences in the basic sequence are present in some ascending systems. In a general way, the motor systems are organized so as to receive stimuli from the sensory systems, at all levels of the spinal cord and brain, and to convey messages via motor pathways to neuromuscular and neuroglandular, endings at muscle and gland cells in the head, body, and extremities. The motor pathways comprise sequences of processing centers, and their fibers conveying neural influences to other processing centers within the CNS, and the final linkages extending from the CNS via motor nerves of the PNS to muscles and glands. The CNS comprises *gray matter* and *white matter* (Fig. 2.2). The gray matter consists of neuronal cell bodies, dendrites, axon terminals, synapses, and glial cells, and is highly vascular. The white matter—whose color is imparted by the myelin—consists instead of bundles of axons (many of which are myelinated and oligodendrocytes); it lacks of neuronal cell bodies and is less vascular than the gray matter. Groupings of neuronal cell bodies within the gray matter are variously known as nucleus, ganglion, lamina, body, cortex, center, formation, and horn. A cortex is a layer of gray matter on the surface of the brain. Two major cortices are recognized: *cerebral* and *cerebellar* cortices. The superior and inferior colliculi of the midbrain and the hippocampal formation also form cortex-like structures.

The *cerebrum* includes the paired cerebral hemispheres, a small median segment (derived from the telencephalon), and the diencephalon. The *cerebral hemispheres* consist of the cerebral cortex (gray matter) that underlies the white matter, the corpus striatum and corpus callosum, the anterior commissure, the hippocampal formation, and the amygdala (Fig. 2.1). The brain hemispheres are marked on the surface by slit-like incisures called *sulci*, and the raised ridge between the two sulci

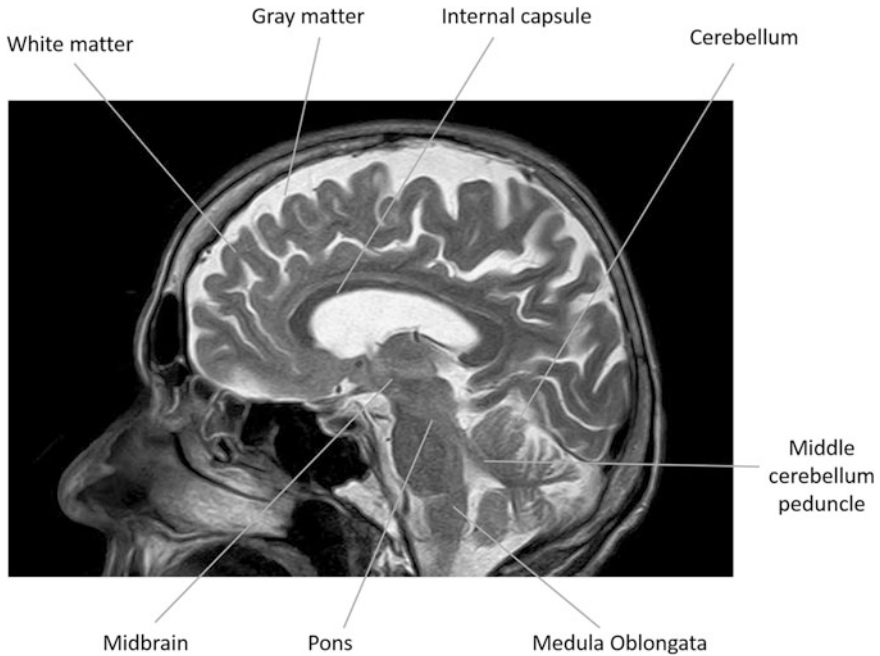


Fig. 2.1. A sagittal brain section in which some of the main structures of the brains are labeled. In particular, gray matter and white matter, cerebellum, midbrain, medulla oblongata, pons, middle cerebellar peduncle, and internal capsule

is called *gyrus*. The cortex lining a sulcus is considered part of the adjacent gyrus, while the hemispheres are separated from one another in the midline by the longitudinal *fissure* (particularly deep and constant sulcus). Each hemisphere is conventionally divided into six *lobes*: frontal, parietal, occipital, temporal, central (insula), and limbic (Fig. 2.2). The portion of the frontal, parietal, and temporal lobes that overlies the insula is called *operculum*. The lobes are delineated one from another by several major sulci, including the lateral sulcus of Sylvius, the central one of Rolando, the cingulate, and the parieto-occipital sulci. The *lateral sulcus* is a deep furrow that outspreads posteriorly from the basal surface of the brain along the lateral surface of the hemisphere, terminating usually as an upward curve within the inferior part of the parietal lobe. The *central sulcus* of Rolando, instead, extends obliquely from the region of the lateral sulcus across the dorsolateral cerebral surface and, for a short distance, onto the medial surface. The *cingulate sulcus* is a curved cleft on the medial surface, lengthening parallel to the curvature of the corpus callosum. The *parieto-occipital sulcus* is a deep cleft on the medial surface located between the central sulcus and the occipital pole (Fig. 2.3).

The lobes on the lateral cerebral surface are situated on the following boundaries: (1) the frontal lobe is in front of the central sulcus and above the lateral sulcus; (2) the occipital lobe is located behind an imaginary line parallel to the

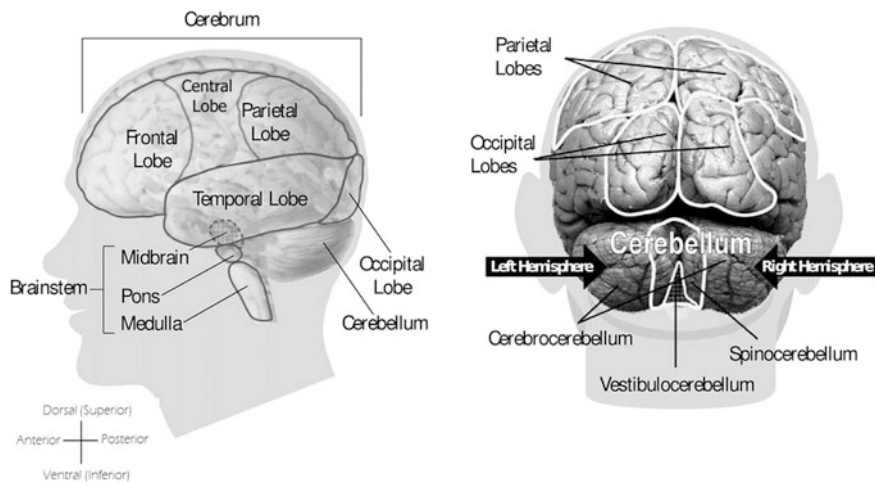


Fig. 2.2 Brain lobes and hemispheres

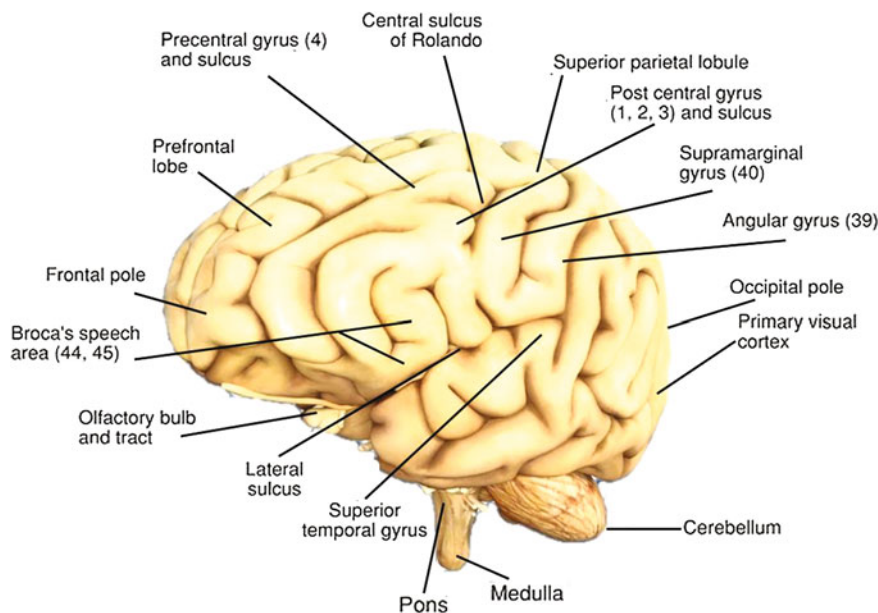


Fig. 2.3. Medial surface of the cerebral hemisphere. The limbic lobe consists of the cingulate gyrus, the isthmus and the parahippocampal gyrus. The amygdala is located within the uncus. The hippocampal formation (hippocampus and dentate gyrus) is located in the floor of the temporal horn of the lateral ventricle

parieto-occipital sulcus, which is situated on the medial surface; (3) the parietal lobe is located at the rear of the central sulcus, anteriorly to the imaginary parieto-occipital line and above the lateral sulcus; it runs out toward the occipital pole before taking an upward curve; (4) the temporal lobe is positioned below the lateral sulcus, and before the imaginary parieto-occipital line; and (5) the central lobe is placed at the bottom (medial surface) of the lateral sulcus of Sylvius, which is actually a deep fossa (depression). It is visible only when the temporal and frontal lobes are reflected away from the lateral sulcus (Fig. 2.3). Instead, the lobes on the medial cerebral surface are positioned as follows: (1) the frontal lobe is located rostral to a line formed by the central sulcus; (2) the parietal lobe is situated between the central sulcus and the parieto-occipital sulcus; (3) the temporal lobe is located lateral to the parahippocampal gyrus; (4) the occipital lobe is posterior to the parieto-occipital sulcus; and (5) the limbic lobe is a synthetic one, formed by parts of the frontal, parietal, and temporal lobes. It is located in the center of the curved line formed by the cingulate sulcus and the collateral sulcus (the latter is located lateral to the parahippocampal gyrus). The limbic lobe is the ring (limbus) of gyri bordered by this line; it includes the subcallosal area, the cingulate gyrus, the parahippocampal gyrus, the hippocampus, the dentate gyrus, and the uncus.

Basic Unit of the Nervous System: Neurons

Some 100–200 billion ($[1-2] \times 10^{11}$) *neurons*, as well as many more glial cells, are integrated into the structural and functional structures of the brain (Noback et al. 2005). They exhibit a wide diversity of shapes and sizes. The neuron is the basic unit of the nervous system and is composed of four regions, structurally defined as follows: a *cell body* (soma) that emits a single nerve process called an *axon*, which ends with the *presynaptic terminals*, and a variable number of branching processes called *dendrites* (Fig. 2.4). Each axon, including its collateral branches, usually terminates as an arbor of fine fibers; each fiber ends as an enlargement called *bouton*, which is part of a synaptic junction. At the other end of the neuron, there is a three-dimensional *dendritic field*, formed by the branching of the dendrites. The cell body is the genomic and metabolic center of the neuron. Dendrites are the main recipients of the neural signals for communication between neurons and contain critical processing complexes. The axon is the canal apt to conduct messages (action potentials) to the presynaptic terminals, where each neuron is in synaptic contact with other neurons and, thus, is part of the network that constitutes the nervous system. A neuron is designed to react to the stimuli, to rapidly transmit the resulting excitation to the other portions of the nerve cell, and to influence other neurons, muscle cells, and glandular cells. Neurons are so specialized that most are incapable of reproducing themselves, and they lose viability if denied an oxygen supply for more than a few minutes. Dendrites contain the same cytoplasmic organelles (e.g. Nissl bodies and mitochondria) as the cell body of which they are true extensions. The axon is specialized in the transmission of coded information as all-or-none action potentials. The axon arises from the *axon hillock* of the cell body,

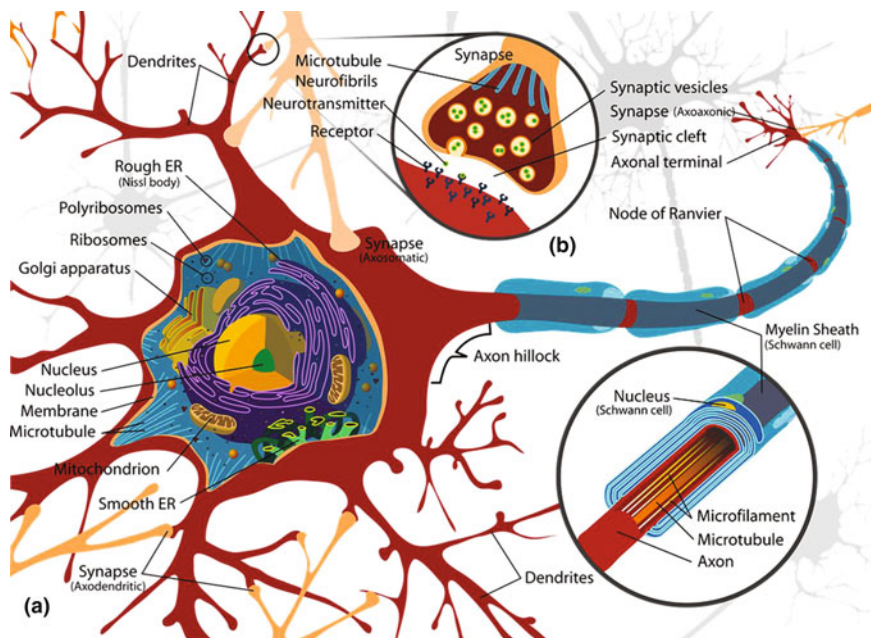


Fig. 2.4. **a** Diagram of a neuron located wholly within the central nervous system. The **b** represents a synapse between two neurons. The myelin sheath of neuron **a** is entirely the product of a glial cell, and the one of neurons **b** is produced by a glial cell inside the central nervous system and by a Schwann (neurolemma) cell in the PNS

at a site called the *initial segment*, and extends for a distance of less than 1 (mm) to as much as 1 (m) before arborizing into *terminal branches* (Fig. 2.4). The axon hillock, initial segment, and the axon lack Nissl bodies. The branches of an axon can have, potentially, two types of *bouton*. Each branch ends as a *terminal bouton* that forms a synapse with the dendrite, cell body, or axon of another neuron. In addition, along some branches, there are thickenings called *boutons en passage*, which form synapses with another neuron or smooth muscle fiber. The dendrites of many neurons are studded with tiny protuberances called *spines* (e.g. pyramidal neurons of the cerebral cortex): these dendritic spines increase the surface area of the membrane of the receptive segment of the neuron. Located on them, there are over 90% of all the excitatory synapses present in the central nervous system (CNS). Because of their widespread occurrence on neurons of the cortical areas of the cerebrum, they are thought to be involved in learning and memory (see Para 3.3).

The synapse is the contact point between the neurons. A submicroscopic space, the synaptic cleft, which is about 200 (Å), exists between the bouton of one neuron and the cell body of another neuron (*axosomatic synapse*), between a bouton and a dendrite (*axodendritic synapse*), and between a bouton and an axon (*axoaxonic synapse*). In addition, also *dendrodendritic synapses* (between two dendrites).

The axon of one neuron might terminate in just a few synapses or up to many thousands of synapses. The dendrite–cell body complex might receive synaptic contacts from many different neurons (up to over 15,000 synapses). The termination of a nerve fiber in a muscle cell (neuromuscular junction) or a glandular cell (neuroglandular junction) is basically similar to the synapse between two neurons. The synapse of each axon terminal of a motoneuron on a voluntary muscle cell is called a *motor end plate* (Fig. 2.4). The cell membrane of the axon at the synapse is the *presynaptic membrane*, and the cell membrane of dendrite–cell body complex, muscle, or glandular cell is the *postsynaptic membrane*. The *subsynaptic membrane* is that region of the postsynaptic membrane that is juxtaposed against the presynaptic membrane at the synapse. A concentration of mitochondria and *presynaptic vesicles* is present in the cytoplasm of the bouton; none is present in the cytoplasm adjacent to the subsynaptic membrane. Most neurons contain at least two distinct types of vesicle: small vesicles 50 (nm) in diameter and large vesicles from 70 to 200 (nm) in diameter.

2.1.1 Basic Neurophysiology

Every neuron is said to have “in small-scale, the integrative capacity of the entire nervous system.” In fact neurons can transform information and transmit it to other neurons. In most, the dendrite–cell body unit is specialized as a receptor and integrator of synaptic input from other neurons, and the axon is specialized to convey coded information from the dendrite–cell body unit to the synaptic junctions, where transformation functions take place with other neurons or effectors (muscles and glands). To serve these tasks, the neuron is thus organized into a receptive segment (dendrites and cell body), a conductile segment (axon), and an effector segment (synapse) (Fig. 2.5). Neurons are specialized to generate electrical signals, which are then used to encode and convey information: these signals are expressed by alterations in the resting membrane potential. Voltage changes that are restricted to the sites where neurons are stimulated—or that are close to them—are called *graded potentials*. These ones can lead to the production of *action potentials* (*nerve impulses or spikes*), which transmit information for substantial distances along an axon. Two forms of graded potential are *generator (receptor potentials)* and *synaptic potentials*. Generator potentials are evoked by sensory stimuli from the environment (both inside and outside the body). Information that passes from one neuron to another at synapses produces *synaptic potentials* in the postsynaptic neuron. The activity of either generator or synaptic potentials can elicit action potentials, which, in turn, produce synaptic potentials in the next neuron. Synaptic potentials elicited in effectors (skeletal muscle and glands) at synapses can result in the contraction of the muscle or emission of secretory product from a gland.

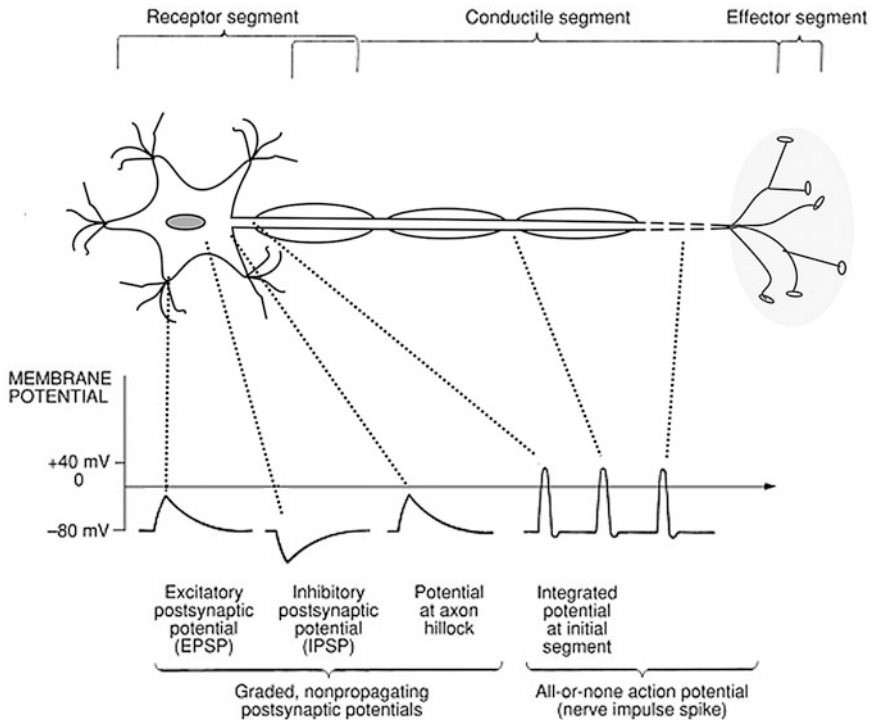


Fig. 2.5. On the surface of the dendrites and cell body are excitatory and inhibitory synapses, which, when stimulated, produce local, graded, non-propagating potentials. These are exhibited as an excitatory or depolarizing postsynaptic potential (EPSP) and as an inhibitory or hyperpolarizing postsynaptic potential (IPSP). These local potentials are summated at the axon hillock and, if adequate, could trigger an integrated potential at the initial segment and an “all-or-none” action potential, which is conducted along the axon to the motor end plate

Resting Potential of the Neuron

The resting neuron is a charged cell that is not conducting a nerve impulse. The plasma membrane, which acts as a thin boundary between the extracellular (interstitial) fluid that is located outside the neuron and the intracellular fluid (neuroplasm) that is instead inside the neuron, is critical for maintaining this charged state or resting potential. The electric charge across the plasma membrane results from a thin film of positive and negative ions, unequally distributed across the membrane. These are sodium (Na^+) and chloride (Cl^-) ions (which are in higher concentration in the interstitial fluid), and potassium (K^+) and protein (organic) ions that are in higher concentration in the neuroplasm. A tendency exists for the Na^+ , K^+ , and Cl^- ions to diffuse across the membrane from regions of high to low concentration (along concentration gradients), through Na^+ , K^+ , and Cl^- channels, respectively. The passage of ions across the membrane is known as *conductance*. Thus, the semipermeable plasma membrane is selectively permeable through

non-gated open channels to Na^+ , K^+ , and Cl^- ions and impermeable to large protein ions. These channels, which are always open, are important in determining the resting potential. The ionic concentrations on either side of the membrane are produced and maintained by a system of membrane pumps called “the *sodium–potassium pump*”, which requires metabolic energy released by adenosine triphosphate (ATP). The sodium–potassium exchange pump is an integral membrane protein that utilizes ATP as an energy source for its role in *active transport*. This transport is an energy-dependent process in which the movement of Na^+ and K^+ ions is “uphill” against a concentration gradient. The activity of the pump results in the passage of three Na^+ ions out of and two K^+ ions into the neuron. This causes the restoration of a concentration of K^+ , 30 or more times higher within the neuroplasm than in the interstitial fluid, and in a concentration of Na^+ that is 10 times and Cl^- that is 14 times higher in the interstitial fluid than in the neuroplasm. Most neurons do not have a Cl^- pump; hence, Cl^- ions diffuse passively across the membrane. These are the ionic concentrations responsible for establishing an electric potential across the membrane. The transmembrane potential, known as the *resting potential*, is about -70 to -80 (mV) (millivolts) inside the neuron. The resting potential is in a steady state (*dynamic equilibrium*) requiring metabolic energy to maintain the ionic gradients across the membrane. When the neuron is “at rest,” its membrane potential is the result of a balance (involving Na^+ and K^+ ions) between the active fluxes (movements) of ions metabolically driven by *pumps* and the passive fluxes caused by *diffusion*. The active fluxes result from the pump extruding three Na^+ ions for every two K^+ ions it brings into the neuron. The passive fluxes of ions take place through non-gated channels. The outward flux of positive charges by the pump tends to hyperpolarize the membrane. The greater the hyperpolarization, the greater the inward electrochemical force driving Na^+ into the neuron, and the smaller the force driving K^+ out. The steady state for the neuron is attained when the resting potential is reached at the point when the net passive inward current (movement of electrical charge) through the ion channels exactly counterbalances the active outward current driven by the pump. The steady state is not basically the result of *passive diffusion*, which is the diffusion of a solute down a concentration gradient without the expenditure of energy.

Excitability of the Neuron

Excitability is a property that enables a neuron to respond to a stimulus and to transmit information in the form of electrical signals. The flow of information within a neuron and between neurons is conveyed by both electrical and chemical signals. The electrical signals, called *graded potentials* and *action potentials*, are all produced by temporary changes in the current flow into and out of the neuron—changes that are actually deviations from the normal value of the resting membrane potential. Ion channels within the plasma membrane control instead the inward and outward current flow. The channels have three features: (1) they conduct ions across the plasma membrane at rapid rates up to 100,000,000 ions per second; (2) they can recognize specific ions and be selective as to which can pass through; (3) they can selectively open and close, in response to specific electrical, chemical, and mechanical stimuli.

Each neuron is presumed to have over 20 different types of channel with thousands of copies of each channel. The flux (movement of ions) through the ion channels is passive, requiring no expenditure of metabolic energy. The flux direction is determined by the electrochemical driving force across the plasma membrane, and the primary role of the ion channels in the neurons is to mediate rapid signaling. These channels, called *gated channels*, have a molecular “cap” or *gate*, which briefly opens to permit anion species to pass. Gated channels open when a neurotransmitter binds to them; *voltage-gated channels* open and close in response to changes in membrane potential; *modality-gated channels* are activated by specific modalities (e.g. touch, pressure, or stretch). Gating is the process by which a channel is opened or closed during activity. Each channel consists of several plasma membrane-spanning polypeptide subunits (proteins) arranged around a central pore. Each of these classes of channel belongs to a different gene family. Each member of a family shares common structural and biochemical features, which presumably have evolved from a common ancestral gene of that family. The channels of the voltage-gated gene family are selective for Na^+ , K^+ , and Ca^{2+} ions. The channels for the transmitter-gated channels respond to acetylcholine, gamma amino butyric acid (GABA), and glycine. Most gated channels are closed with the membrane at rest: they open when activated, following the binding of a ligand (ligand gating), a change in the membrane potential (voltage gating), or the stretch of the membrane (modality gating). In the transmitter-gated channel, the transmitter binds to a specific site on the external face of a channel that activates it to open briefly. The energy to open the channels is derived from three sources: (1) from the binding of the transmitter to the receptor protein in the ligand-gated channels; (2) from the changes in the membrane voltage within the voltage-gated channels; (3) presumably, from the mechanical forces resulting from cytoskeletal interaction at the modality-gated channels. There are two types of membranes’ response: (1) *hyperpolarization* or (2) *depolarization*. During the *hyperpolarization*, the membrane becomes more negative on the inside with respect to its outside [i.e. could go from -70 (mV) to -80 (mV)]. During *depolarization*, the membrane becomes less negative inside with respect to its outside and it might even reverse polarity with its inside—becoming positive with respect to the outside. This is still called *depolarization* because the membrane potential becomes less negative than the resting potential [e.g. from -70 (mV) to 0 to -40 (mV)].

2.1.2 EEG Generation

The EEG comes from the summation of synchronously postsynaptic potentials. The contribution to the electric field of neurons acting synchronously is approximately proportional to their number, and, for those firing nonsynchronously, is approximately proportional to square root of their number (Blinowska and Durka 2006). The problem of the origins of the EEG rhythmical activity has been studied electrophysiological field when disserting about the brain nerve cells and when

modeling of electrical activity of the neural populations (Niedermeyer et al. 2011). The question is as follows: Are the rhythms caused by single cells with pacemaker properties or by the oscillating neural networks? With regard of this, it has been shown that some thalamic neurons display oscillatory behavior, even in the absence of synaptic input (Jahnsen and Llinás 1984), since there is evidence that the intrinsic oscillatory properties of some neurons contribute to the shaping of the rhythmic behavior of networks to which they belong. However, these properties may not be sufficient to account for the network rhythmic behavior: it is generally accepted that cooperative properties of networks consisting of excitatory and inhibitory neurons connected by feedback loops play the crucial role in establishing EEG rhythms. The frequency of oscillation depends on the intrinsic membrane properties, on the individual neurons' membrane potential, and on the synaptic interactions' strength of. Bursts of oscillatory activity may constitute a mechanism by which the brain can regulate changes of state in selected neuronal networks and change the route of information (Niedermeyer et al. 2011). EEG is usually registered by means of electrodes placed on the brain scalp. They can be secured by an adhesive (like *collodion*) or embedded in a special snug cap. The resistance of the connection should be less than 10 (k Ω), so the recording site is first cleaned with diluted alcohol, and then a conductive electrode paste is applied to the electrode cup. Knowing the exact positions of the electrodes is very important for interpreting a single recording as well as comparing the results, hence the need for standardization. The traditional *10–20 electrode system* (Jasper 1958) states positions of 19 EEG electrodes (and two electrodes placed on earlobes A1/A2) related to the specific anatomic landmarks, such that 10–20% of the distance between them is used as the electrode interval. The first part of derivation name indexes the array's row—from the front of the head: Fp, F, C, P, and O. The second part is formed by numbers that are even on the left and odd on the right side, while in the center there is “z” or “0”. Progress in topographic representation of EEG recordings brought demand for a larger amount of derivations. Electrode sites halfway between those defined by the standard 10–20 system were introduced in the extended 10–20 system (Pivik et al. 1993). EEG is a measure of potential difference; in the referential (or unipolar) setup, it is measured relative to the same electrode for all derivations. This reference electrode is usually placed on the earlobe, nose, mastoid, chin, neck, or scalp center. No universal consent exists regarding the best position of the reference electrode, because currents coming from bioelectric activity of muscles, heart, or brain propagate all over the human body. In the bipolar setup (montage), each channel registers the potential difference between two particular scalp electrodes. Data recorded in a referential setup can be transformed into any bipolar montage. The common “average reference” montage can be obtained by subtracting from each channel the average activity from all the remaining derivations. The *Hjorth* transform references each electrode to the four closest neighbors, which is an approximation of the *Laplace transform* (LT). LT is calculated as a second spatial derivative of a signal, offering information about vertical current density. For the best performance, it needs an adequate spatial sampling—interelectrode distance around 20 (mm) (e.g. 128 electrodes on the scalp).

The estimates obtained by means of LT for the electrodes lying at the scalp periphery are biased and have to be excluded. Contrary to the open question of the reference, the necessity of artifact rejections is universally acknowledged: *artifacts* are recorded signals that are non-cerebral in origin. They may be divided into one of two categories, depending on their origin: *physiological artifacts* or *non-physiological artifacts*. Physiological artifacts can stem from muscles or heart activity (EMG, ECG), eye movement (EOG), external electromagnetic field, poor electrode contact, and participant's movement. Corresponding signals (EMG, EOG, ECG, and body movements), registered simultaneously with EEG, could be helpful in the visual rejection of artifact-contaminated epochs. Non-physiological artifacts arise from two main sources: external electrical interference (power lines or electrical equipment), and internal electrical malfunctioning of the recording system (electrodes, cables, amplifier).

Furthermore, artifacts may reduce the performance of machine learning techniques. Several ways of handling physiological artifacts can be found in the literature. Artifacts may be avoided, rejected, or removed from the EEG dataset. Artifacts avoidance involves asking users to avoid blinking or moving their body during the experiments (Vigário 1997). This approach is very simple, because it does not require any computation, as brain signals are assumed to have no artifacts. However, this assumption is not feasible in an operational environment, since some artifacts, eye and body movements, are not easily avoidable. Artifact rejection approaches suggest to discard the epochs contaminated by the artifacts. Manual artifact rejection is an option to remove artifacts in brain signals, and experts could identify and eliminate all artifact-contaminated EEG epochs. The main disadvantage in using manual rejection is that it requires an intensive human labor, so this approach is not suitable for real-time evaluations. In the EEG, the following frequency rhythms are considered characteristic for its analysis (Fig. 2.6): delta [0.5–4 (Hz)], theta [4–8 (Hz)], alpha [8–12 (Hz)], beta [12–30 (Hz)], and gamma [above 30 (Hz)].

Delta activity is characterized by high amplitude and low frequency. It is usually associated with the slow wave in psychophysiology of sleep. It is suggested that it represents the onset of deep-sleep phases in healthy adults (Moser et al. 2009). Theta rhythm is generally linked to the hippocampus activity (Buzsáki 2005) as well as to the neocortex (Cantero et al. 2003). It is commonly believed to be linked to deep relaxation or meditation (Craigmyle 2013), and it has been observed during the transition between wake and sleep (Hagemann 2008). However, theta rhythms are suggested to be important for learning and memory functions (Sammer et al. 2006), encoding and retrieval (Ward 2003), which involve high concentration (Hagemann 2008). It has also been suggested that theta oscillations are associated with the attentional control mechanism in the anterior cingulate cortex (Craigmyle 2013; Smith et al. 2001), and it is often shown to increase with a higher cognitive task demand (Gundel and Wilson 1992). Alpha activity has been found in the visual cortex (occipital lobe) during periods of relaxation or idling (eyes closed but individual awake). In the continuous EEG, alpha band is characterized by high amplitude and regular oscillations, in particular over parietal and occipital areas.

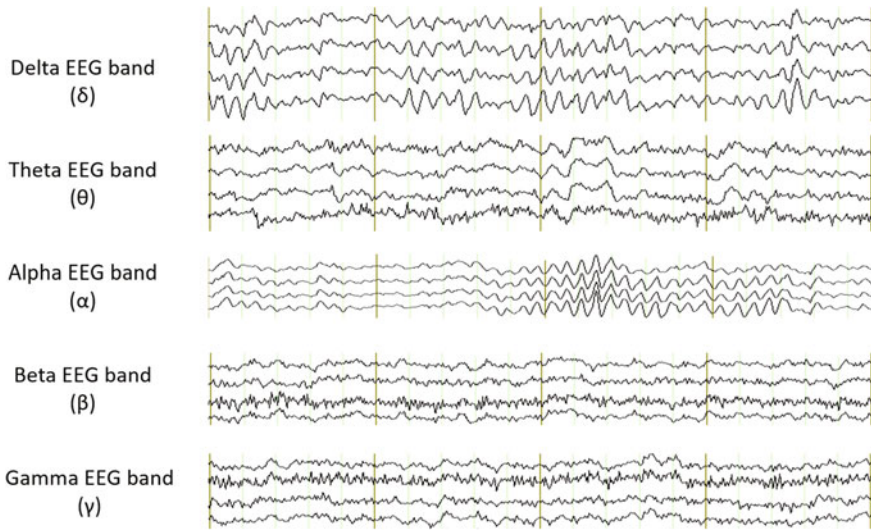


Fig. 2.6. Characteristic EEG rhythms, from the top: δ [0.5–4 (Hz)], θ [4–8 (Hz)], α [8–12 (Hz)], β [12–30 (Hz)]. The gamma band could reach 100 (Hz)

High alpha power has been assumed to reflect a state of relaxation or cortical idling; however, when the operator assigns more effort to the task, different regions of the cortex may be recruited in the transient function network leading to passive oscillation of the local alpha generators, in synchrony with a reduction in alpha power (Smith et al. 2001). Recent results have suggested that alpha is involved in auditory attention processes and in the inhibition of irrelevant tasks' areas, so as to enhance the signal-to-noise ratio (Gevins et al. 1998; Klimesch et al. 2007). Additionally, alpha activity may be further divided into subbands by means of the frequency corresponding to the user's alpha peak (Klimesch 1999), called *Individual Alpha Frequency* (IAF). For instance, *alpha 3* [$\text{IAF} \div \text{IAF} + 2$ (Hz)] reflects the semantic memory performance, while *alpha 1* and *alpha 2* [respectively, $\text{IAF} - 4 \div \text{IAF} - 2$ and $\text{IAF} - 2 \div \text{IAF}$ (Hz)] reflect general task demands and attentional processes. Beta activity is predominant in wakefulness state, especially in frontal and central areas of the brain. High power in beta band is associated with the increased mental arousal and activity. Dooley (2009) pointed out that a beta wave represents cognitive consciousness and active, business, or anxious thinking. This band can be further divided into *low beta wave* [12.5–15 (Hz)], *middle beta wave* [15–18 (Hz)], and *high beta wave* (>18 Hz). Low waves seem to be associated with inhibition of phasic movements during the sleep, and high waves with dopaminergic system (Hagemann 2008). Finally, gamma is the fastest activity in EEG and it is thought to be infrequent during waking states of consciousness (Dooley 2009). Recent studies reveal that it is linked with many cognitive functions, such as attention, learning, and memory (Jensen et al. 2007; Toppi et al. 2014). Gamma components are difficult to be recorded by the scalp electrodes,

because of their low amplitude, but with *Electrocorticography* (ECoG) components up to 100 (Hz), or even higher, it might be registered.

The contribution of different rhythms to the EEG depends on the age, on the psycho-cognitive state of the participant, and on level of alertness. Considerable intersubject differences in EEG characteristics also exist, since EEG pattern is influenced by neuropathological conditions, metabolic disorders, and drug action (Niedermeyer et al. 2011).

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Industrial Neuroscience in Aviation

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2017, XIII, 147 p. 44 illus., 39 illus. in color., Hardcover

ISBN: 978-3-319-58597-0