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Introduction to EEG

The neural activity of the human brain starts between the 17th and 23rd week of prenatal development. It is believed that from this early stage and throughout life electrical signals generated by the brain represent not only the brain function but also the status of the whole body. This assumption provides the motivation to apply advanced digital signal processing methods to the electroencephalogram (EEG) signals measured from the brain of a human subject, and thereby underpins the later chapters of the book.

Although nowhere in this book do the authors attempt to comment on the physiological aspects of brain activities there are several issues related to the nature of the original sources, their actual patterns, and the characteristics of the medium, that have to be addressed. The medium defines the path from the neurons, as so-called signal sources, to the electrodes, which are the sensors where some form of mixtures of the sources are measured.

Understanding of neuronal functions and neurophysiological properties of the brain together with the mechanisms underlying the generation of signals and their recordings is, however, vital for those who deal with these signals for detection, diagnosis, and treatment of brain disorders and the related diseases. A brief history of EEG measurements is first provided.

1.1 History

Carlo Matteucci (1811–1868) and Emil Du Bois-Reymond (1818–1896) were the first people to register the electrical signals emitted from muscle nerves using a galvanometer and established the concept of neurophysiology [1,2]. However, the concept of *action current* introduced by Hermann Von Helmholz [3] clarified and confirmed the negative variations that occur during muscle contraction.

Richard Caton (1842–1926), a scientist from Liverpool, England, used a galvanometer and placed two electrodes over the scalp of a human subject and thereby first recorded brain activity in the form of electrical signals in 1875. Since then, the concepts of electro-(referring to registration of brain electrical activities) encephalo- (referring to emitting the signals from the head), and gram (or graphy), which means drawing or writing, were combined so that the term EEG was henceforth used to denote electrical neural activity of the brain.

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Fritsch (1838–1927) and Hitzig (1838–1907) discovered that the human cerebral can be electrically stimulated. Vasili Yakovlevich Danilevsky (1852–1939) followed Caton's work and finished his PhD thesis in the investigation of the physiology of the brain in 1877 [4]. In this work, he investigated the activity of the brain following electrical stimulation as well as spontaneous electrical activity in the brain of animals.

The cerebral electrical activity observed over the visual cortex of different species of animals was reported by Ernst Fleischl von Marxow (1845–1891). Napoleon Cybulski (1854–1919) provided EEG evidence of an epileptic seizure in a dog caused by electrical stimulation.

The idea of the association of epileptic attacks with abnormal electrical discharges was expressed by Kaufman [5]. Pravidch-Neminsky (1879–1952), a Russian physiologist, recorded the EEG from the brain, termed the dura, and the intact skull of a dog in 1912. He observed a 12–14 cycle/s rhythm under normal conditions, which slowed under asphyxia and later called it the *electrocerebrogram*.

The discoverer of the existence of human EEG signals was Hans Berger (1873–1941). He began his study of human EEGs in 1920 [6]. Berger is well known by almost all electroencephalographers. He started working with a string galvanometer in 1910, then migrated to a smaller Edelmann model, and after 1924, to a larger Edelmann model. In 1926, Berger started to use the more powerful Siemens double coil galvanometer (attaining a sensitivity of 130 μV/cm) [7]. His first report of human EEG recordings of one to three minutes duration on photographic paper was in 1929. In this recording he only used a one-channel bipolar method with fronto-occipital leads. Recording of the EEG became popular in 1924. The first report of 1929 by Berger included the alpha rhythm as the major component of the EEG signals, as described later in this chapter, and the alpha blocking response.

During the 1930s the first EEG recording of sleep spindles was undertaken by Berger. He then reported the effect of hypoxia on the human brain, the nature of several diffuse and localized brain disorders, and gave an inkling of epileptic discharges [8]. During this time another group established in Berlin-Buch and led by Kornmüller, provided more precise recording of the EEG [9]. Berger was also interested in cerebral localization and particularly in the localization of brain tumours. He also found some correlation between mental activities and the changes in the EEG signals.

Toennies (1902–1970) from the group in Berlin built the first biological amplifier for the recording of brain potentials. A differential amplifier for recording EEGs was later produced by the Rockefeller foundation in 1932.

The importance of multichannel recordings and using a large number of electrodes to cover a wider brain region was recognized by Kornmüller [10]. The first EEG work focusing on epileptic manifestation and the first demonstration of epileptic spikes were presented by Fischer and Löwenbach [11–13].

In England, W. Gray Walter became the pioneer of clinical electroencephalography. He discovered the foci of slow brain activity (delta waves), which initiated enormous clinical interest in the diagnosis of brain abnormalities. In Brussels, Fredric Bremer (1892–1982) discovered the influence of afferent signals on the state of vigilance [14].

Research activities related to EEGs started in North America in around 1934. In this year, Hallowell Davis illustrated a good alpha rhythm for himself. A cathode ray

oscilloscope was used around this date by the group in St Louis University in Washington, in the study of peripheral nerve potentials. The work on human EEGs started at Harvard in Boston and the University of Iowa in the 1930s. The study of epileptic seizure developed by Fredric Gibbs was the major work on EEGs during these years, as the realm of epileptic seizure disorders was the domain of their greatest effectiveness. Epileptology may be divided historically into two periods [15]: before and after the advent of EEG. Gibbs and Lennox applied the idea of Fischer based on his studies about picrotoxin and its effect on the cortical EEG in animals to human epileptology. Berger [16] showed a few examples of paroxysmal EEG discharges in a case of presumed petit mal attacks and during a focal motor seizure in a patient with general paresis.

As the other great pioneers of electroencephalography in North America, Hallowel and Pauline Davis were the earliest investigators of the nature of EEG during human sleep. A. L. Loomis, E. N. Harvey, and G. A. Hobart were the first who mathematically studied the human sleep EEG patterns and the stages of sleep. At McGill University, H. Jasper studied the related behavioural disorder before he found his niche in basic and clinical epileptology [17].

The American EEG Society was founded in 1947 and the First International EEG Congress was held in London, United Kingdom, around this time. While the EEG studies in Germany were still limited to Berlin, Japan gained attention by the work of Motokawa, a researcher of EEG rhythms [18]. During these years the neurophysiologists demonstrated the thalamocortical relationship through anatomical methods. This led to the development of the concept of centrencephalic epilepsy [19].

Throughout the 1950s the work on EEGs expanded in many different places. During this time surgical operation for removing the epileptic foci became popular and the book entitled *Epilepsy and the Functional Anatomy of the Human Brain* (Penfiled and Jasper) was published. During this time microelectrodes were invented. They were made of metals such as tungsten or glass, filled with electrolytes such as potassium chloride, with diameters of less than 3 μm.

Depth electroencephalography of a human was first obtained with implanted intracerebral electrodes by Mayer and Hayne (1948). Invention of intracellular microelectrode technology revolutionized this method and was used in the spinal cord by Brock *et al*. in 1952 and in the cortex by Phillips in 1961.

Analysis of EEG signals started during the early days of EEG measurement. Berger assisted by Dietch (1932) applied Fourier analysis to EEG sequences, which was rapidly developed during the 1950s. Analysis of sleep disorders with EEGs started its development in the 1950s through the work of Kleitman at the University of Chicago.

In the 1960s analysis of the EEGs of full-term and premature newborns began its development [20]. Investigation of evoked potentials (EPs), especially visual EPs, as commonly used for monitoring mental illnesses, progressed during the 1970s.

The history of EEG, however, has been a continuous process, which started from the early 1300s and has brought daily development of clinical, experimental, and computational studies for discovery, recognition, diagnosis, and treatment of a vast number of neurological and physiological abnormalities of the brain and the rest of the central nervous system (CNS) of human beings. Nowadays, EEGs are recorded invasively and noninvasively using fully computerized systems. The EEG machines are

equipped with many signal processing tools, delicate and accurate measurement electrodes, and enough memory for very long-term recordings of several hours. EEG or MEG (magnetoencephalogram) machines may be integrated with other neuroimaging systems such as functional magnetic resonance imaging (fMRI). Very delicate needle-type electrodes can also be used for recording the EEGs from over the cortex (electrocortiogram), and thereby avoid the attenuation and nonlinearity effects induced by the skull. The nature of neural activities within the human brain will be discribed next.

1.2 Neural Activities

The CNS generally consists of nerve cells and glia cells, which are located between neurons. Each nerve cell consists of axons, dendrites, and cell bodies. Nerve cells respond to stimuli and transmit information over long distances. A nerve cell body has a single nucleus and contains most of the nerve cell metabolism, especially that related to protein synthesis. The proteins created in the cell body are delivered to other parts of the nerve. An axon is a long cylinder, which transmits an electrical impulse and can be several metres long in vertebrates (giraffe axons go from the head to the tip of the spine). In humans the length can be a percentage of a millimetre to more than a metre. An axonal transport system for delivering proteins to the ends of the cell exists and the transport system has 'molecular motors', which ride upon tubulin rails.

Dendrites are connected to either the axons or dendrites of other cells and receive impulses from other nerves or relay the signals to other nerves. In the human brain each nerve is connected to approximately 10,000 other nerves, mostly through dendritic connections.

The activities in the CNS are mainly related to the synaptic currents transferred between the junctions (called synapses) of axons and dendrites, or dendrites and dendrites of cells. A potential of 60−70 mV with negative polarity may be recorded under the membrane of the cell body. This potential changes with variations in synaptic activities. If an action potential travels along the fibre, which ends in an *excitatory* synapse, an excitatory postsynaptic potential (EPSP) occurs in the following neuron. If two action potentials travel along the same fibre over a short distance, there will be a summation of EPSPs producing an action potential on the postsynaptic neuron providing a certain threshold of membrane potential is reached. If the fibre ends in an *inhibitory* synapse, then hyperpolarization will occur, indicating an inhibitory postsynaptic potential (IPSP) [21,22]. Figure 1.1 shows the above activities schematically.

Following the generation of an IPSP, there is an overflow of cations from the nerve cell or an inflow of anions into the nerve cell. This flow ultimately causes a change in potential along the nerve cell membrane. Primary transmembranous currents generate secondary inonal currents along the cell membranes in the intra- and extracellular space. The portion of these currents that flow through the extracellular space is directly responsible for the generation of field potentials. These field potentials, usually with less than 100 Hz frequency, are called EEGs when there are no changes in the signal average and DC if there are slow drifts in the average signals, which may mask the actual EEG signals. A combination of EEG and DC potentials is often observed for some abnormalities in the brain such as seizure (induced by pentylenetetrazol), hypercapnia, and asphyxia [23]. The focus will next be on the nature of active potentials.

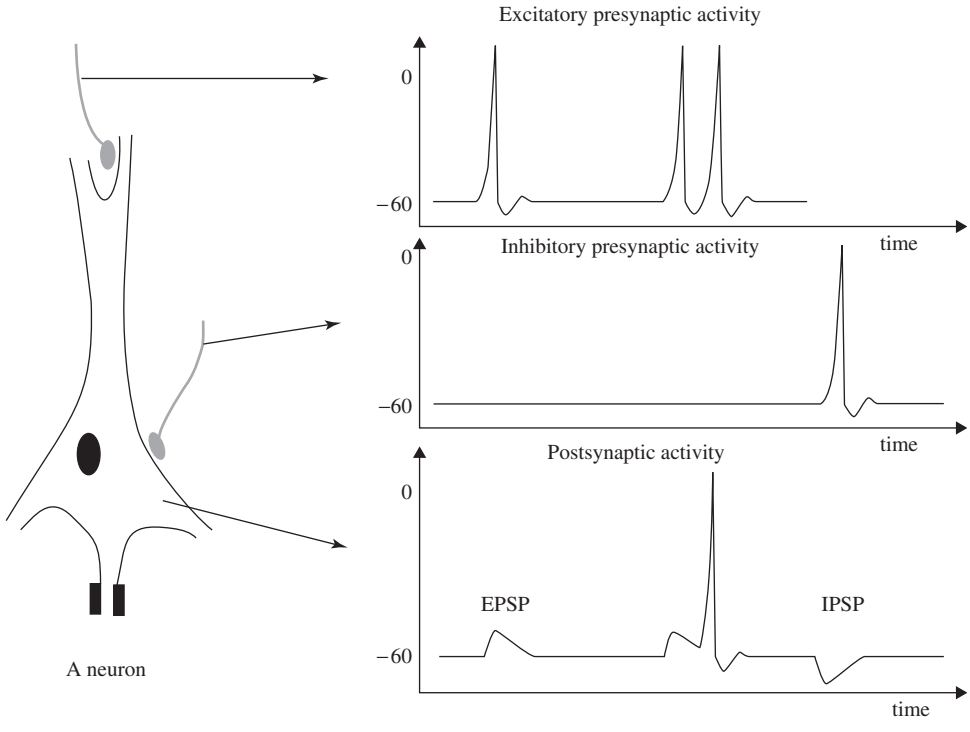

Figure 1.1 The neuron membrane potential changes and current flow during synaptic activation recorded by means of intracellular microelectrodes. Action potentials in the excitatory and inhibitory presynaptic fibre respectively lead to EPSP and IPSP in the postsynaptic neuron

1.3 Action Potentials

The information transmitted by a nerve is called an action potential (AP). APs are caused by an exchange of ions across the neuron membrane and an AP is a temporary change in the membrane potential that is transmitted along the axon. It is usually initiated in the cell body and normally travels in one direction. The membrane potential depolarizes (becomes more positive), producing a spike. After the peak of the spike the membrane repolarizes (becomes more negative). The potential becomes more negative than the resting potential and then returns to normal. The action potentials of most nerves last between 5 and 10 milliseconds. Figure 1.2 shows an example AP.

The conduction velocity of action potentials lies between 1 and 100 m/s. APs are initiated by many different types of stimuli; sensory nerves respond to many types of stimuli, such as chemical, light, electricity, pressure, touch, and stretching. On the other hand, the nerves within the CNS (brain and spinal cord) are mostly stimulated by chemical activity at synapses.

A stimulus must be above a threshold level to set off an AP. Very weak stimuli cause a small local electrical disturbance, but do not produce a transmitted AP. As soon as the stimulus strength goes above the threshold, an action potential appears and travels down the nerve.

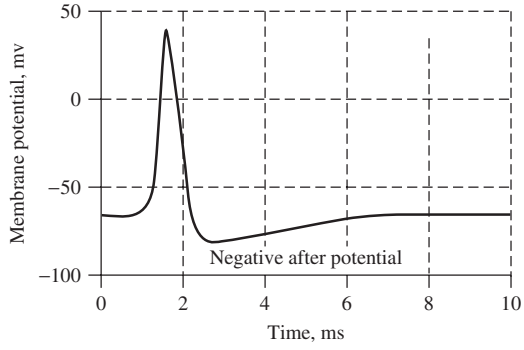

Figure 1.2 An example action potential

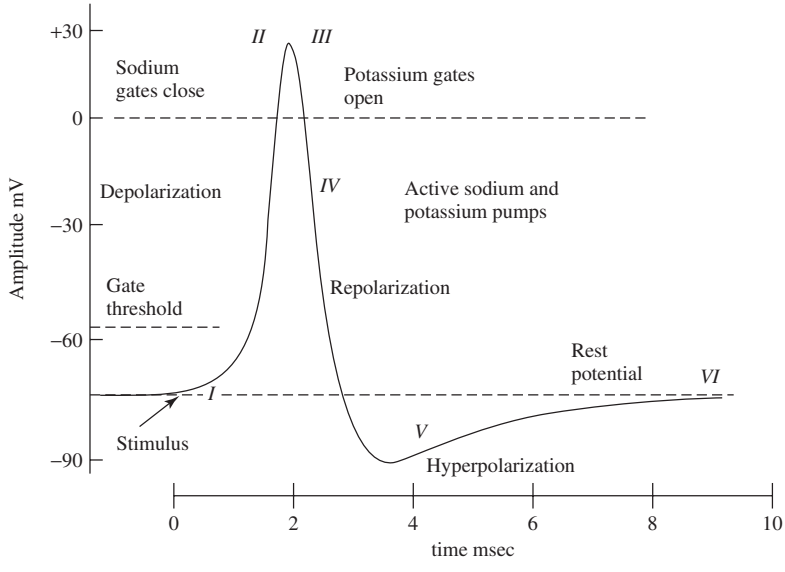

Figure 1.3 Changing the membrane potential for a giant squid by closing the Na channels and opening K channels (adopted from Ka Xiong Charand [24])

The spike of the AP is mainly caused by opening of Na (sodium) channels. The Na pump produces gradients of both Na and K (potassium) ions. Both are used to produce the action potential; Na is high outside the cell and low inside. Excitable cells have special Na and K channels with gates that open and close in response to the membrane voltage (voltage-gated channels). Opening the gates of Na channels allows Na to rush into the cell, carrying positive charge. This makes the membrane potential positive (depolarization), producing the spike. Figure 1.3 shows the stages of the process during evolution of an action potential for a giant squid. For a human being the amplitude of the AP ranges between approximately −60 mV and 10 mV. During this process [24]:

I. When the dendrites of a nerve cell receive the stimulus the Na^+ channels will open. If the opening is sufficient to drive the interior potential from -70 mV up to -55 mV, the process continues.
II. As soon as the action threshold is reached, additional Na^+ channels (sometimes called voltage-gated channels) open. The Na^+ influx drives the interior of the cell membrane up to approximately $+30$ mV. The process to this point is called depolarization.
III. Then Na^+ channels close and the K^+ channels open. Since the K^+ channels are much slower to open, the depolarization has time to be completed. Having both Na^+ and K^+ channels open at the same time would drive the system towards neutrality and prevent the creation of the action potential.
IV. Having the K^+ channels open, the membrane begins to repolarize back towards its rest potential.
V. The repolarization typically overshoots the rest potential to a level of approximately -90 mV. This is called hyperpolarization and would seem to be counterproductive, but it is actually important in the transmission of information. Hyperpolarization prevents the neuron from receiving another stimulus during this time, or at least raises the threshold for any new stimulus. Part of the importance of hyperpolarization is in preventing any stimulus already sent up an axon from triggering another action potential in the opposite direction. In other words, hyperpolarization ensures that the signal is proceeding in one direction.
VI. After hyperpolarization, the Na^+/K^+ pumps eventually bring the membrane back to its resting state of -70 mV.

The nerve requires approximately two milliseconds before another stimulus is presented. During this time no AP can be generated. This is called the refractory period. The generation of EEG signals is next described.

1.4 EEG Generation

An EEG signal is a measurement of currents that flow during synaptic excitations of the dendrites of many pyramidal neurons in the cerebral cortex. When brain cells (neurons) are activated, the synaptic currents are produced within the dendrites. This current generates a magnetic field measurable by electromyogram (EMG) machines and a secondary electrical field over the scalp measurable by EEG systems.

Differences of electrical potentials are caused by summed postsynaptic graded potentials from pyramidal cells that create electrical dipoles between the soma (body of a neuron) and apical dendrites, which branch from neurons (Figure 1.4). The current in the brain is generated mostly by pumping the positive ions of sodium, Na^+, potassium, K^+, calcium, Ca^{++}, and the negative ion of chlorine, Cl^-, through the neuron membranes in the direction governed by the membrane potential [25].

The human head consists of different layers including the scalp, skull, brain (Figure 1.5), and many other thin layers in between. The skull attenuates the signals approximately one hundred times more than the soft tissue. On the other hand, most of the noise is generated either within the brain (internal noise) or over the scalp (system noise or external noise). Therefore, only large populations of active neurons can generate enough potential to be recordable using the scalp electrodes. These signals are later amplified greatly for display

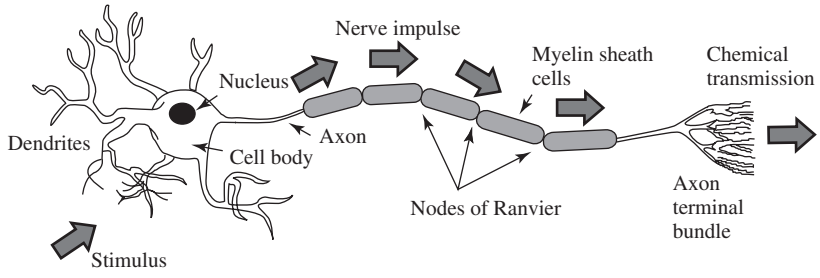

Figure 1.4 Structure of a neuron (adopted from Attwood and MacKay [25])

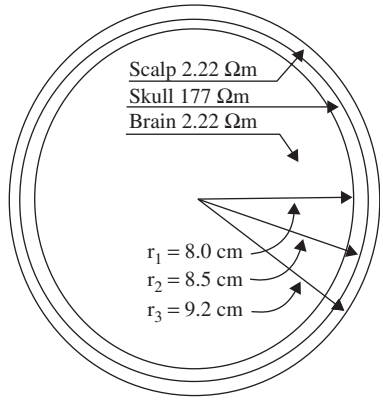

Figure 1.5 The three main layers of the brain including their approximate resistivities and thicknesses (Ω = ohm)

purposes. Approximately 10^{11} neurons are developed at birth when the central nervous system (CNS) becomes complete and functional [26]. This makes an average of 10^4 neurons per cubic mm. Neurons are interconnected into neural nets through synapses. Adults have approximately 5×10^{14} synapses. The number of synapses per neuron increases with age, whereas the number of neurons decreases with age. From an anatomical point of view the brain may be divided into three parts: the cerebrum, cerebellum, and brain stem (Figure 1.6). The cerebrum consists of both left and right lobes of the brain with highly convoluted surface layers called the cerebral cortex.

The cerebrum includes the regions for movement initiation, conscious awareness of sensation, complex analysis, and expression of emotions and behaviour. The cerebellum coordinates voluntary movements of muscles and maintains balance. The brain stem controls involuntary functions such as respiration, heart regulation, biorhythms, and neurohormone and hormone sections [27].

Based on the above section it is clear that the study of EEGs paves the way for diagnosis of many neurological disorders and other abnormalities in the human body. The acquired EEG signals from a human (and also from animals) may, for example, be used for investigation of the following clinical problems [27,28]:

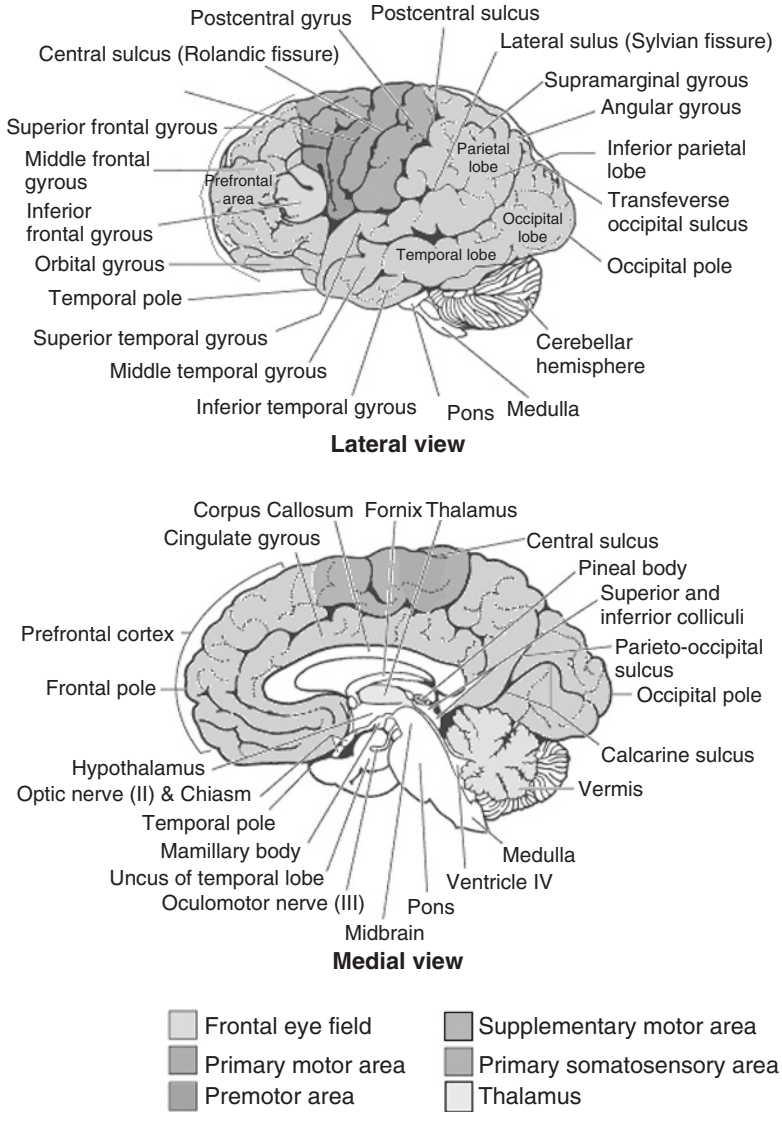

Figure 1.6 Diagrammatic representation of the major parts of the brain

(a) monitoring alertness, coma, and brain death;
(b) locating areas of damage following head injury, stroke, and tumour;
(c) testing afferent pathways (by evoked potentials);
(d) monitoring cognitive engagement (alpha rhythm);
(e) producing biofeedback situations;
(f) controlling anaesthesia depth (servo anaesthesia);
(g) investigating epilepsy and locating seizure origin;

(h) testing epilepsy drug effects;
(i) assisting in experimental cortical excision of epileptic focus;
(j) monitoring the brain development;
(k) testing drugs for convulsive effects;
(l) investigating sleep disorders and physiology;
(m) investigating mental disorders;
(n) providing a hybrid data recording system together with other imaging modalities.

This list confirms the rich potential for EEG analysis and motivates the need for advanced signal processing techniques to aid the clinician in their interpretation. The brain rhythms will next be described, which are expected to be measured within EEG signals.

1.5 Brain Rhythms

Many brain disorders are diagnosed by visual inspection of EEG signals. The clinical experts in the field are familiar with manifestation of brain rhythms in the EEG signals. In healthy adults, the amplitudes and frequencies of such signals change from one state of a human to another, such as wakefulness and sleep. The characteristics of the waves also change with age. There are five major brain waves distinguished by their different frequency ranges. These frequency bands from low to high frequencies respectively are called alpha (α), theta (θ), beta (β), delta (δ), and gamma (γ). The alpha and beta waves were introduced by Berger in 1929. Jasper and Andrews (1938) used the term 'gamma' to refer to the waves of above 30 Hz. The delta rhythm was introduced by Walter (1936) to designate all frequencies below the alpha range. He also introduced theta waves as those having frequencies within the range of 4–7.5 Hz. The notion of a theta wave was introduced by Wolter and Dovey in 1944 [29].

Delta waves lie within the range of 0.5–4 Hz. These waves are primarily associated with deep sleep and may be present in the waking state. It is very easy to confuse artefact signals caused by the large muscles of the neck and jaw with the genuine delta response. This is because the muscles are near the surface of the skin and produce large signals, whereas the signal that is of interest originates from deep within the brain and is severely attenuated in passing through the skull. Nevertheless, by applying simple signal analysis methods to the EEG, it is very easy to see when the response is caused by excessive movement.

Theta waves lie within the range of 4–7.5 Hz. The term theta might be chosen to allude to its presumed thalamic origin. Theta waves appear as consciousness slips towards drowsiness. Theta waves have been associated with access to unconscious material, creative inspiration and deep meditation. A theta wave is often accompanied by other frequencies and seems to be related to the level of arousal. It is known that healers and experienced mediators have an alpha wave that gradually lowers in frequency over long periods of time. The theta wave plays an important role in infancy and childhood. Larger contingents of theta wave activity in the waking adult are abnormal and are caused by various pathological problems. The changes in the rhythm of theta waves are examined for maturational and emotional studies [30].

Alpha waves appear in the posterior half of the head and are usually found over the occipital region of the brain. They can be detected in all parts of posterior lobes of the brain. For alpha waves the frequency lies within the range of 8–13 Hz, and commonly appears as a round or sinusoidal shaped signal. However, in rare cases it may manifest itself as sharp waves. In such cases, the negative component appears to be sharp and the positive component appears to be rounded, similar to the wave morphology of the rolandic mu (μ) rhythm. Alpha waves have been thought to indicate both a relaxed awareness without any attention or concentration. The alpha wave is the most prominent rhythm in the whole realm of brain activity and possibly covers a greater range than has been previously accepted. A peak can regularly be seen in the beta wave range in frequencies even up to 20 Hz, which has the characteristics of an alpha wave state rather than one for a beta wave. Again, very often a response is seen at 75 Hz, which appears in an alpha setting. Most subjects produce some alpha waves with their eyes closed, which is why it has been claimed that it is nothing but a waiting or scanning pattern produced by the visual regions of the brain. It is reduced or eliminated by opening the eyes, by hearing unfamiliar sounds, by anxiety, or mental concentration or attention. Albert Einstein could solve complex mathematical problems while remaining in the alpha state, although generally beta and theta waves are also present. An alpha wave has a higher amplitude over the occipital areas and has an amplitude of normally less than 50 μV. The origin and physiological significance of an alpha wave is still unknown and yet more research has to be undertaken to understand how this phenomenon originates from cortical cells [31].

A beta wave is the electrical activity of the brain varying within the range of 14–26 Hz (though in some literature no upper bound is given). A beta wave is the usual waking rhythm of the brain associated with active thinking, active attention, focus on the outside world, or solving concrete problems, and is found in normal adults. A high-level beta wave may be acquired when a human is in a panic state. Rhythmical beta activity is encountered chiefly over the frontal and central regions. Importantly, a central beta rhythm is related to the rolandic mu rhythm and can be blocked by motor activity or tactile stimulation. The amplitude of beta rhythm is normally under 30 μV. Similar to the mu rhythm, the beta wave may also be enhanced because of a bone defect [29] and also around tumoural regions.

The frequencies above 30 Hz (mainly up to 45 Hz) correspond to the gamma range (sometimes called the fast beta wave). Although the amplitudes of these rhythms are very low and their occurrence is rare, detection of these rhythms can be used for confirmation of certain brain diseases. The regions of high EEG frequencies and highest levels of cerebral blood flow (as well as oxygen and glucose uptake) are located in the frontocentral area. The gamma wave band has also been proved to be a good indication of event-related synchronization (ERS) of the brain and can be used to demonstrate the locus for right and left index finger movement, right toes, and the rather broad and bilateral area for tongue movement [32].

Waves in frequencies much higher than the normal activity range of EEG, mostly in the range of 200–300 Hz, have been found in cerebellar structures of animals, but they have not played any role in clinical neurophysiology [33,34].

Figure 1.7 shows the typical normal brain rhythms with their usual amplitude levels. In general, the EEG signals are the projection of neural activities that are attenuated by

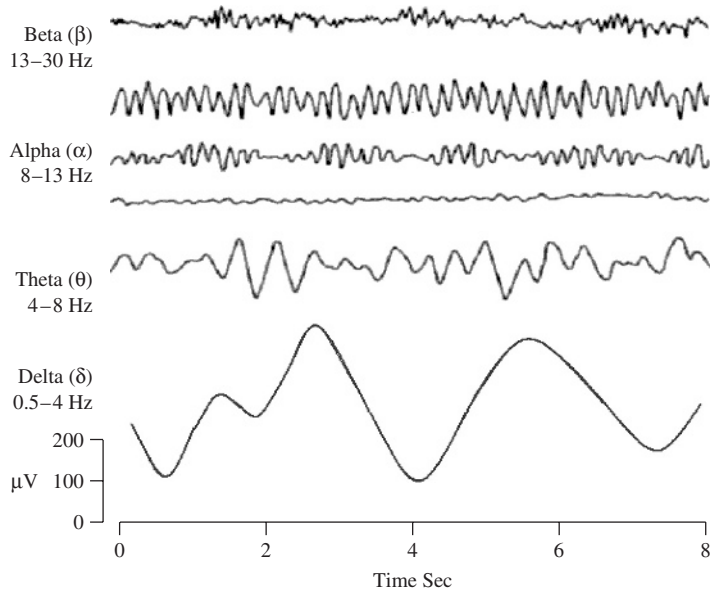

Figure 1.7 Four typical dominant brain normal rhythms, from high to low frequencies. The delta wave is observed in infants and sleeping adults, the theta wave in children and sleeping adults, the alpha wave is detected in the occipital brain region when there is no attention, and the beta wave appears frontally and parietally with low amplitude

leptomeninges, cerebrospinal fluid, dura matter, bone, galea, and the scalp. Cartographic discharges show amplitudes of 0.5–1.5 mV and up to several millivolts for spikes. However, on the scalp the amplitudes commonly lie within 10–100 μV.

The above rhythms may last if the state of the subject does not change and therefore they are approximately cyclic in nature. On the other hand, there are other brain waveforms, which may:

(a) Have a wide frequency range or appear as spiky-type signals, such as K-complexes, vertex waves (which happen during sleep), or a breach rhythm, which is an alpha-type rhythm due to a cranial bone defect [35], which does not respond to movement, and is found mainly over the midtemporal region (under electrodes T3 or T4), and some seizure signals.

(b) Be a transient such as an event-related potential (ERP) and contain positive occipital sharp transient (POST) signals (also called rho (ρ) waves).

(c) Originate from the defective regions of the brain such as tumoural brain lesions.

(d) Be spatially localized and considered as cyclic in nature, but can be easily blocked by physical movement such as mu rhythm. Mu denotes motor and is strongly related to the motor cortex. Rolandic (central) mu is related to posterior alpha in terms of amplitude and frequency. However, the topography and physiological significance are quite different. From the mu rhythm the cortical functioning and the changes in

brain (mostly bilateral) activities subject to physical and imaginary movements can be investigated. The mu rhythm has also been used in feedback training for several purposes such as treatment of epileptic seizure disorder [29].

There are also other rhythms introduced by researchers such as:

(e) Phi (φ) rhythm (less than 4 Hz) occurring within two seconds of eye closure. The phi rhythm was introduced by Daly [36].
(f) Kappa (κ) rhythm, which is an anterior temporal alpha-like rhythm. It is believed to be the result of discrete lateral oscillations of the eyeballs and is considered to be an artefact signal.
(g) The sleep spindles (also called the sigma (σ) activity) within the 11–15 Hz frequency range.
(h) Tau (τ) rhythm, which represents the alpha activity in the temporal region.
(i) Eyelid flutter with closed eyes, which gives rise to frontal artefacts in the alpha band.
(j) Chi (χ) rhythm is a mu-like activity believed to be a specific rolandic pattern of 11–17 Hz. This wave has been observed during the course of Hatha Yoga exercises [37].
(k) Lambda (λ) waves are most prominent in waking patients, but are not very common. They are sharp transients occurring over the occipital region of the head of walking subjects during visual exploration. They are positive and time-locked to saccadic eye movement with varying amplitude, generally below 90 μV [38].

It is often difficult to understand and detect the brain rhythms from the scalp EEGs, even with trained eyes. Application of advanced signal processing tools, however, should enable separation and analysis of the desired waveforms from within the EEGs. Therefore, a definition of foreground and background EEG is very subjective and entirely depends on the abnormalities and applications. Next to consider is the development in the recording and measurement of EEG signals.

1.6 EEG Recording and Measurement

Acquiring signals and images from the human body has become vital for early diagnosis of a variety of diseases. Such data can be in the form of electrobiological signals such as an electrocardiogram (ECG) from the heart, electromyogram (EMG) from muscles, electroencephalogram (EEG) from the brain, magnetoencephalogram (MEG) from the brain, electrogastrogram (EGG) from the stomach, and electroocclugram (or electrooptigram, EOG) from eye nerves. Measurements can also have the form of one type of ultrasound or radiograph such as sonograph (or ultrasound image), computerized tomography (CT), magnetic resonance imaging (MRI) or functional MRI (fMRI), positron emission tomography (PET), and single photon emission tomography (SPET).

Functional and physiological changes within the brain may be registered by either EEG, MEG, or fMRI. Application of fMRI is, however, very limited in comparison with EEG or MEG for a number of important reasons:

(a) The time resolution of fMRI image sequences is very low (for example approximately two frames/s), whereas the complete EEG bandwidth can be viewed using EEG or MEG signals.
(b) Many types of mental activities, brain disorders, and malfunctions of the brain cannot be registered using fMRI since their effect on the level of oxygenated blood is low.
(c) The accessibility to fMRI (and currently to MEG) systems is limited and costly.
(d) The spatial resolution of EEG, however, is limited to the number of recording electrodes (or number of coils for MEG).

The first electrical neural activities were registered using simple galvanometers. In order to magnify very fine variations of the pointer a mirror was used to reflect the light projected to the galvanometer on the wall. The d'Arsonval galvanometer later featured a mirror mounted on a movable coil and the light focused on the mirror was reflected when a current passed the coil. The capillary electrometer was introduced by Lippmann and Marey [39]. The string galvanometer, as a very sensitive and more accurate measuring instrument, was introduced by Einthoven in 1903. This became a standard instrument for a few decades and enabled photographic recording.

More recent EEG systems consist of a number of delicate electrodes, a set of differential amplifiers (one for each channel) followed by filters [27], and needle (pen)-type registers. The multichannel EEGs could be plotted on plane paper or paper with a grid. Soon after this system came to the market, researchers started looking for a computerized system, which could digitize and store the signals. Therefore, to analyse EEG signals it was soon understood that the signals must be in digital form. This required sampling, quantization, and encoding of the signals. As the number of electrodes grows the data volume, in terms of the number of bits, increases. The computerized systems allow variable settings, stimulations, and sampling frequency, and some are equipped with simple or advanced signal processing tools for processing the signals.

The conversion from analogue to digital EEG is performed by means of multichannel analogue-to-digital converters (ADCs). Fortunately, the effective bandwidth for EEG signals is limited to approximately 100 Hz. For many applications this bandwidth may be considered to be even half of this value. Therefore, a minimum frequency of 200 samples/s (to satisfy the Nyquist criterion) is often enough for sampling the EEG signals. In some applications where a higher resolution is required for representation of brain activities in the frequency domain, sampling frequencies of up to 2000 sample/s may be used.

In order to maintain the diagnostic information the quantization of EEG signals is normally very fine. Representation of each signal sample with up to 16 bits is very popular for the EEG recording systems. This makes the necessary memory volume for archiving the signals massive, especially for sleep EEG and epileptic seizure monitoring records. However, in general, the memory size for archiving the radiological images is often much larger than that used for archiving the EEG signals.

A simple calculation shows that for a one hour recording from 128-electrode EEG signals sampled at 500 samples/s a memory size of $128 \times 60 \times 60 \times 500 \times 16 \approx 3.68$ Gbits ≈ 0.45 Gbyte is required. Therefore, for longer recordings of a large number of patients there should be enough storage facilities such as in today's technology Zip disks, CDs,

large removable hard drives, and optical disks. Although the format of reading the EEG data may be different for different EEG machines, these formats are easily convertible to spreadsheets readable by most signal processing software packages such as MATLAB.

The EEG recording electrodes and their proper function are crucial for acquiring high-quality data. Different types of electrodes are often used in the EEG recording systems, such as:

- disposable (gel-less, and pre-gelled types);
- reusable disc electrodes (gold, silver, stainless steel, or tin);
- headbands and electrode caps;
- saline-based electrodes;
- needle electrodes.

For multichannel recordings with a large number of electrodes, electrode caps are often used. Commonly used scalp electrodes consist of Ag−AgCl disks, less than 3 mm in diameter, with long flexible leads that can be plugged into an amplifier. Needle electrodes are those that have to be implanted under the skull with minimal invasive operations. High impedance between the cortex and the electrodes as well as the electrodes with high impedances can lead to distortion, which can even mask the actual EEG signals. Commercial EEG recording systems are often equipped with impedance monitors. To enable a satisfactory recording the electrode impedances should read less than 5 kΩ and be balanced to within 1 kΩ of each other. For more accurate measurement the impedances are checked after each trial.

Due to the layered and spiral structure of the brain, however, distribution of the potentials over the scalp (or cortex) is not uniform [40]. This may affect some of the results of source localization using the EEG signals.

1.6.1 Conventional Electrode Positioning

The International Federation of Societies for Electroencephalography and Clinical Neurophysiology has recommended the conventional electrode setting (also called 10−20) for 21 electrodes (excluding the earlobe electrodes), as depicted in Figure 1.8 [17]. Often the earlobe electrodes called A1 and A2, connected respectively to the left and right earlobes, are used as the reference electrodes. The 10−20 system avoids both eyeball placement and considers some constant distances by using specific anatomic landmarks from which the measurement would be made and then uses 10 or 20 % of that specified distance as the electrode interval. The odd electrodes are on the left and the even ones on the right.

For setting a larger number of electrodes using the above conventional system, the rest of the electrodes are placed in between the above electrodes with equidistance between them. For example, C_1 is placed between C_3 and C_z. Figure 1.9 represents a larger setting for 75 electrodes including the reference electrodes based on the guidelines by the American EEG Society. Extra electrodes are sometimes used for the measurement of EOG, ECG, and EMG of the eyelid and eye surrounding muscles. In some applications such as ERP analysis and brain computer interfacing a single channel may be used. In such applications, however, the position of the corresponding electrode has to be well determined.

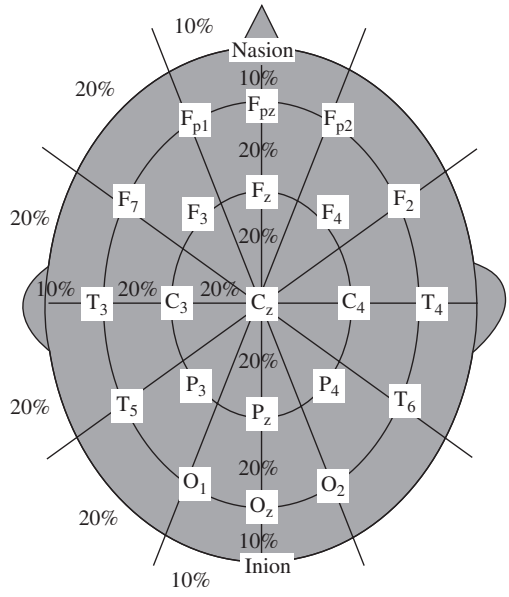

Figure 1.8 Conventional 10–20 EEG electrode positions for the placement of 21 electrodes

For example, C_3 and C_4 can be used to record the right and left finger movement related signals respectively for brain–computer interfacing (BCI) applications. Also F_3, F_4, P_3, and P_4 can be used for recording the ERP P300 signals.

Two different modes of recordings, namely differential and referential, are used. In the differential mode the two inputs to each differential amplifier are from two electrodes. In the referential mode, on the other hand, one or two reference electrodes are used. Several different reference electrode placements can be found in the literature. Physical references can be used as vertex (C_z), linked-ears, linked-mastoids, ipsilateral ear, contralateral ear, C_7, bipolar references, and tip of the nose [28]. There are also reference-free recording techniques, which actually use a common average reference. The choice of reference may produce topographic distortion if the reference is not relatively neutral. In modern instrumentation, however, the choice of a reference does not play an important role in the measurement [41]. In such systems other references such as FP_z, hand, or leg electrodes may be used [42]. The overall setting includes the active electrodes and the references.

In another similar setting, called the Maudsley electrode positioning system, the conventional 10–20 system has been modified to capture better the signals from epileptic foci in epileptic seizure recordings. The only difference between this system and the 10–20 conventional system is that the outer electrodes are slightly lowered to enable better capturing of the required signals. The advantage of this system over the conventional one is that it provides a more extensive coverage of the lower part of the cerebral convexity, increasing the sensitivity for the recording from basal subtemporal structures [43]. Other deviations from the international 10–20 system as used by researchers are found in References [44] and [45].

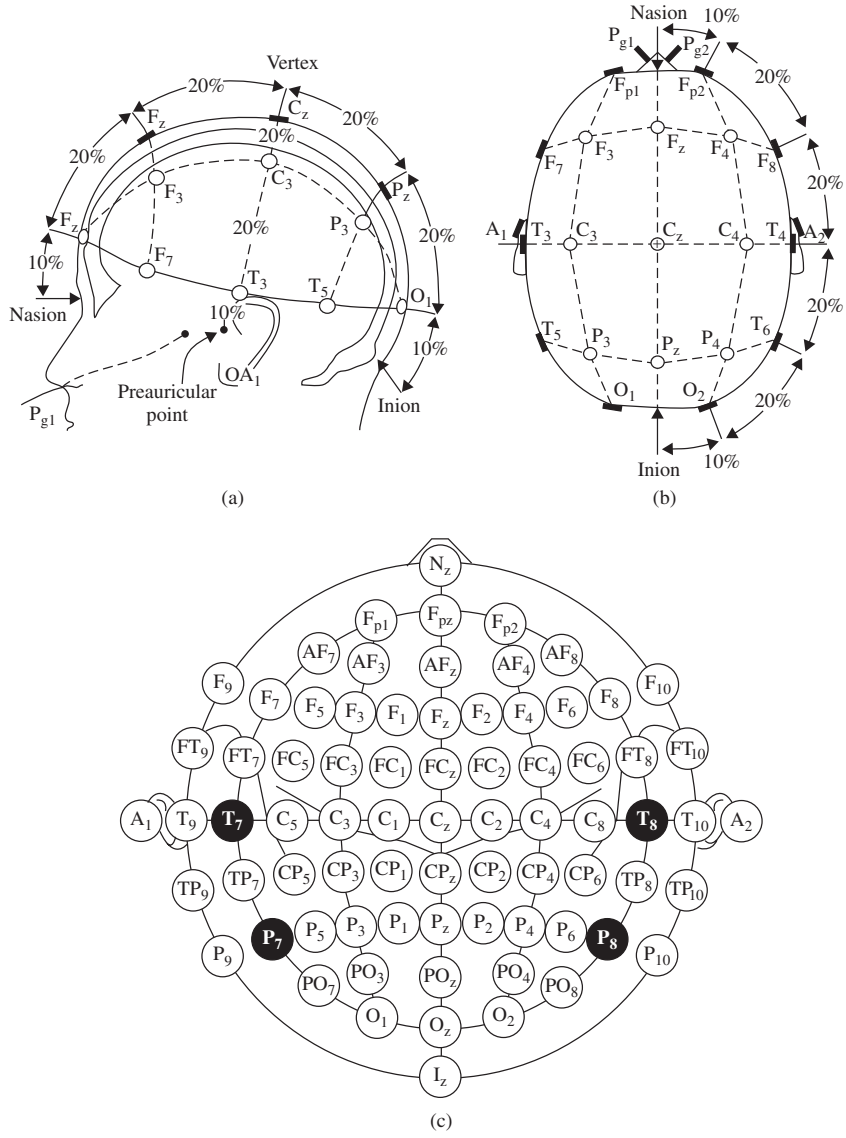

Figure 1.9 A diagrammatic representation of 10–20 electrode settings for 75 electrodes including the reference electrodes: (a) and (b) represent the three-dimensional measures, and (c) indicates a two-dimensional view of the electrode setup configuration

In many applications such as brain–computer interfacing (BCI) and study of mental activity, often a small number of electrodes around the movement-related regions are selected and used from the 10–20 setting system. Figure 1.10 illustrates a typical set of EEG signals during approximately seven seconds of normal adult brain activity.

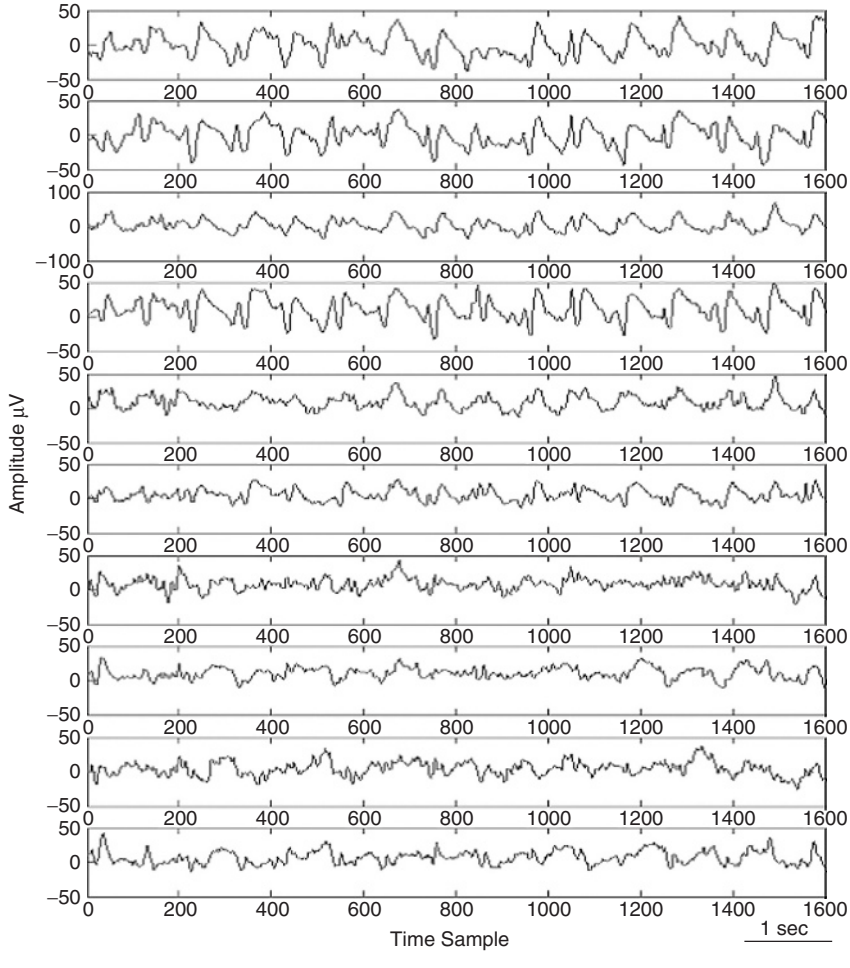

Figure 1.10 A typical set of EEG signals during approximately seven seconds of normal adult brain activity

1.6.2 Conditioning the Signals

The raw EEG signals have amplitudes of the order of μvolts and contain frequency components of up to 300 Hz. To retain the effective information the signals have to be amplified before the ADC and filtered, either before or after the ADC, to reduce the noise and make the signals suitable for processing and visualization. The filters are designed in such a way not to introduce any change or distortion to the signals. Highpass filters with a cut-off frequency of usually less than 0.5 Hz are used to remove the disturbing very low frequency components such as those of breathing. On the other hand, high-frequency noise is mitigated by using lowpass filters with a cut-off frequency of approximately 50–70 Hz. Notch filters with a null frequency of 50 Hz are often necessary to ensure perfect rejection of the strong 50 Hz power supply. In this case the sampling frequency can be as low as twice the bandwidth commonly used by most EEG systems. The commonly used

sampling frequencies for EEG recordings are 100, 250, 500, 1000, and 2000 samples/s. The main artefacts can be divided into patient-related (physiological) and system artefacts. The patient-related or internal artefacts are body movement-related, EMG, ECG (and pulsation), EOG, ballistocardiogram, and sweating. The system artefacts are 50/60 Hz power supply interference, impedance fluctuation, cable defects, electrical noise from the electronic components, and unbalanced impedances of the electrodes. Often in the preprocessing stage these artefacts are highly mitigated and the informative information is restored. Some methods for removing the EEG artefacts will be discussed in the related chapters of this book. Figure 1.11 shows a set of normal EEG signals affected by the eye-blinking artefact. Similarly, Figure 1.12 represents a multichannel EEG set with the clear appearance of ECG signals over the electrodes in the occipital region.

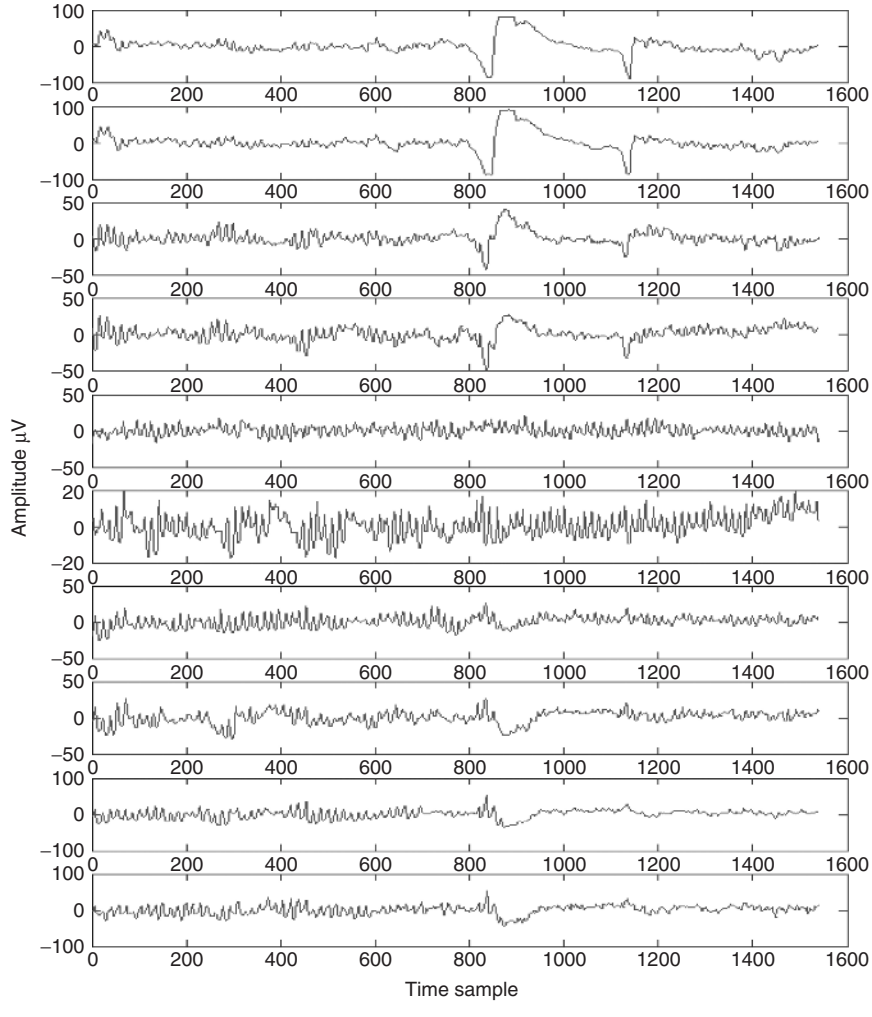

Figure 1.11 A set of normal EEG signals affected by the eye-blinking artefact

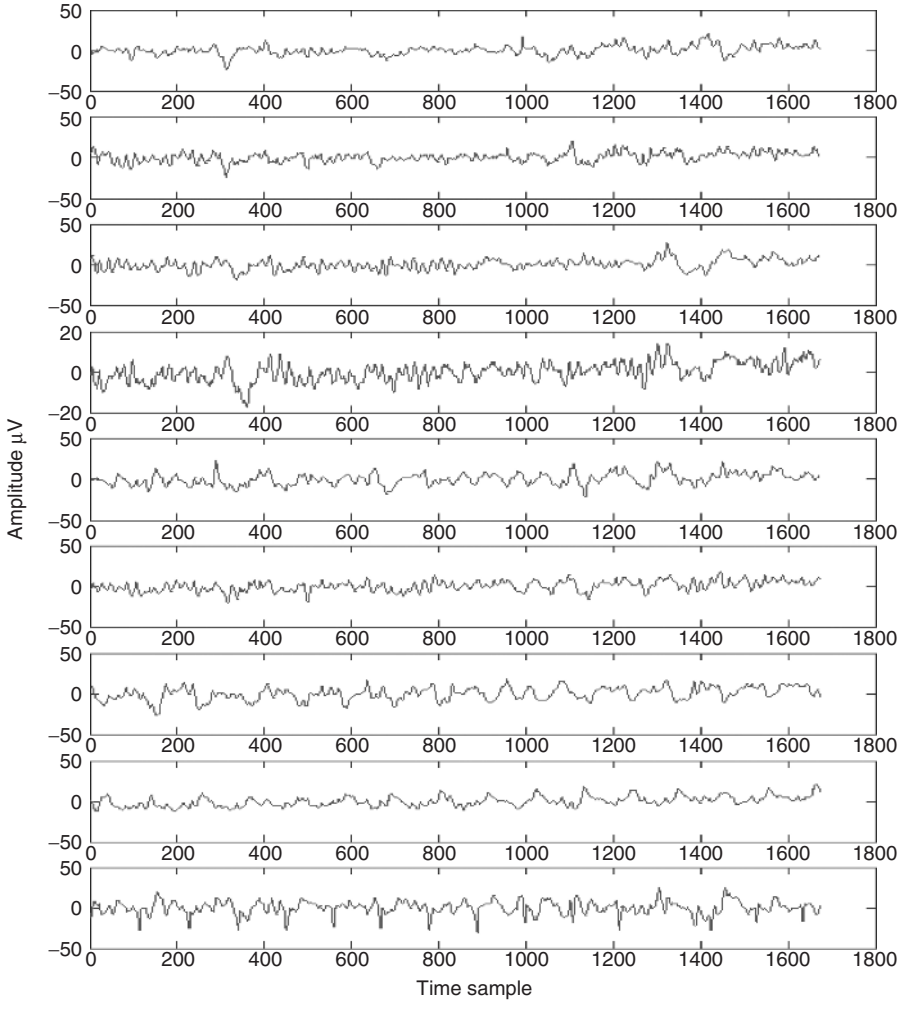

Figure 1.12 A multichannel EEG set with the clear appearance of ECG signals over the electrodes in the occipital region

The next section highlights the changes in EEG measurements that correlate with physiological and mental abnormalities in the brain.

1.7 Abnormal EEG Patterns

Variations in the EEG patterns for certain states of the subject indicate abnormality. This may be due to distortion and the disappearance of abnormal patterns, appearance and increase of abnormal patterns, or disappearance of all patterns. Sharbrough [46] divided the nonspecific abnormalities in the EEGs into three categories: (a) widespread intermittent slow wave abnormalities, often in the delta wave range and associated with brain dysfunction; (b) bilateral persistent EEG, usually associated with impaired conscious

cerebral reactions; and (c) focal persistent EEG usually associated with focal cerebral disturbance.

The first category is a burst-type signal, which is attenuated by alerting the individual and eye opening, and accentuated with eye closure, hyperventilation, or drowsiness. The peak amplitude in adults is usually localized in the frontal region and influenced by age. In children, however, it appears over the occipital or posterior head region. Early findings showed that this abnormal pattern frequently appears with an increased intracranial pressure with tumour or aqueductal stenosis. Also, it correlates with grey matter disease, both in cortical and subcortical locations. However, it can be seen in association with a wide variety of pathological processes varying from systemic toxic or metabolic disturbances to focal intracranial lesions.

Regarding the second category, i.e. bilateral persistent EEG, the phenomenon in different stages of impaired, conscious, purposeful responsiveness are etiologically nonspecific and the mechanisms responsible for their generation are only partially understood. However, the findings in connection with other information concerning etiology and chronicity may be helpful in arriving more quickly at an accurate prognosis concerning the patient's chance of recovering previous conscious life.

As for the third category, i.e. focal persistent EEG, these abnormalities may be in the form of distortion and disappearance of normal patterns, appearance and increase of abnormal patterns, or disappearance of all patterns, but such changes are seldom seen at the cerebral cortex. The focal distortion of normal rhythms may produce an asymmetry of amplitude, frequency, or reactivity of the rhythm. The unilateral loss of reactivity of a physiological rhythm, such as the loss of reactivity of the alpha rhythm to eye opening [47] or to mental alerting [48], may reliably identify the focal side of abnormality. A focal lesion may also distort or eliminate the normal activity of sleep-inducing spindles and vertex waves.

Focal persistent nonrhythmic delta activity (PNRD) may be produced by focal abnormalities. This is one of the most reliable findings of a focal cerebral disturbance. The more persistent, the less reactive, and the more nonrhythmic and polymorphic is such focal slowing, the more reliable an indicator it becomes for the appearance of a focal cerebral disturbance [49–51]. There are other cases such as focal inflammation, trauma, vascular disease, brain tumour, or almost any other cause of focal cortical disturbance, including an asymmetrical onset of CNS degenerative diseases that may result in similar abnormalities in the brain signal patterns.

The scalp EEG amplitude from cerebral cortical generators underlying a skull defect is also likely to increase unless acute or chronic injury has resulted in significant depression of underlying generator activity. The distortions in cerebral activities are because focal abnormalities may alter the interconnections, number, frequency, synchronicity, voltage output, and access orientation of individual neuron generators, as well as the location and amplitude of the source signal itself.

With regards to the three categories of abnormal EEGs, their identification and classification requires a dynamic tool for various neurological conditions and any other available information. A precise characterization of the abnormal patterns leads to a clearer insight into some specific pathophysiologic reactions, such as epilepsy, or specific disease processes, such as subacute sclerosing panencephalitis (SSPE) or Creutzfeldt–Jakob disease (CJD) [46].

Over and above the reasons mentioned above there are many other causes for abnormal EEG patterns. The most common abnormalities are briefly described in the following sections.

1.8 Ageing

The ageing process affects the normal cerebral activity in waking and sleep, and changes the response of the brain to stimuli. The changes stem from reducing the number of neurons and due to a general change in the brain pathology. This pathology indicates that the frontal and temporal lobes of the brain are more affected than the parietal lobes, resulting in shrinkage of large neurons and increasing the number of small neurons and glia [52]. A diminished cortical volume indicates that there is age-related neuronal loss. A general cause for ageing of the brain may be the decrease in cerebral blood flow [52].

A reduction of the alpha frequency is probably the most frequent abnormality in EEG. This often introduces a greater anterior spread to frontal regions in the elderly and reduces the alpha wave blocking response and reactivity. The diminished mental function is somehow related to the degree of bilateral slowing in the theta and delta waves [52].

Although the changes in high-frequency brain rhythms have not been well established, some researchers have reported an increase in beta wave activity. This change in beta wave activity may be considered as an early indication of intellectual loss [52].

As for the sleep EEG pattern, older adults enter into drowsiness with a more gradual decrease in EEG amplitude. Over the age of sixty, the frontocentral waves become slower, the frequency of the temporal rhythms also decreases, frequency lowering with slow eye movements become more prominent, and spindles appear in the wave pattern after the dropout of the alpha rhythm. The amplitudes of both phasic and tonic nonrapid eye movement (NREM) sleep EEG [52] reduce with age. There is also a significant change in rapid eye movement (REM) sleep organization with age; the REM duration decreases during the night and there is a significant increase in sleep disruption [52].

Dementia is the most frequent mental disorder that occurs predominantly in the elderly. Therefore, the prevalence of dementia increases dramatically with ageing of the society. Generally, EEGs are a valuable diagnostic tool in differentiation between organic brain syndromes (OBSs) and functional psychiatric disorders [52], and together with evoked potentials (EPs) play an important role in the assessment of normal and pathological ageing. Ageing is expected to change most neurophysiological parameters. However, the variability of these parameters must exceed the normal degree of spontaneous variability to become a diagnostic factor in acute and chronic disease conditions. Automatic analysis of the EEG during sleep and wakefulness may provide a better contrast in the data and enable a robust diagnostic tool. Next particular and very common mental disorders are described, whose early onset may be diagnosed with EEG measurements.

1.9 Mental Disorders

1.9.1 Dementia

Dementia is a syndrome that consists of a decline in intellectual and cognitive abilities. This consequently affects the normal social activities, mode, and the relationship and interaction with other people [53]. EEG is often used to study the effect of dementia. In

most cases, such as in primary degenerative dementia, e.g. Alzheimer's, and psychiatric disorder, e.g. depression with cognitive impairment, the EEG can be used to detect the abnormality [54].

In Reference [54] dementia is classified into cortical and subcortical forms. The most important cortical dementia is Alzheimer's disease (AD), which accounts for approximately 50 % of the cases. Other known cortical abnormalities are Pick's disease and Creutzfeldt–Jakob diseases (CJD). They are characterized clinically by findings such as aphasia, apraxia, and agnosia. CJD can often be diagnosed using the EEG signals. Figure 1.13 shows a set of EEG signals from a CJD patient. On the other hand, the most common subcortical diseases are Parkinson's disease, Huntington's disease, lacunar state, normal pressure hydrocephalus, and progressive supranuclear palsy. These diseases are characterized by forgetfulness, slowing of thought processes, apathy, and depression. Generally, subcortical dementias introduce less abnormality to the EEG patterns than the cortical ones.

In AD the EEG posterior rhythm (alpha rhythm) slows down and the delta and theta wave activities increase. On the other hand, beta wave activity may decrease. In severe cases epileptiform discharges and triphasic waves can appear. In such cases, cognitive impairment often results. The spectral power also changes; the power increases in delta and theta bands and decreases in beta and alpha bands and also in mean frequency.

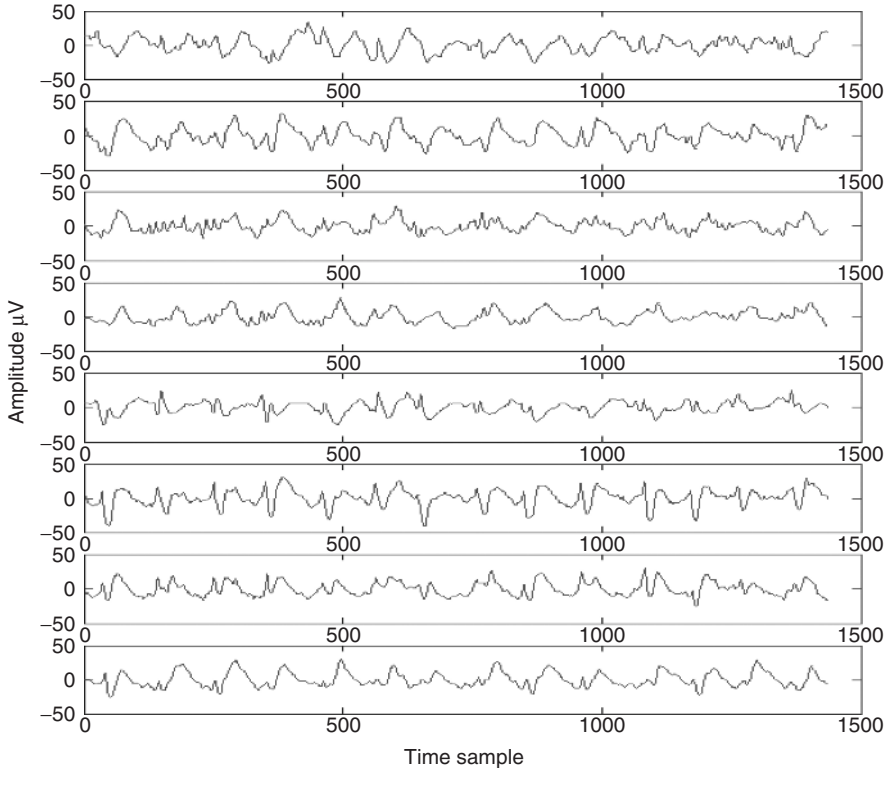

Figure 1.13 A set of multichannel EEG signals from a patient suffering from CJD

The EEG wave morphology is almost the same for AD and Pick's disease. Pick's disease involves the frontal and temporal lobes. An accurate analysis followed by an efficient classification of the cases may discriminate these two diseases. CJD is a mixed cortical and subcortical dementia. This causes slowing of the delta and theta wave activities and, after approximately three months of the onset of the disease, periodic sharp wave complexes are generated that occur almost every second, together with a decrease in the background activity [54]. Parkinson's disease is a subcortical dementia, which causes slowing down of the background activity and an increase of the theta and delta wave activities. Some works have been undertaken using spectral analysis to confirm the above changes [55]. Some other disorders such as depression have a lesser effect on the EEGs and more accurate analysis of the EEGs has to be performed to detect the signal abnormalities for these brain disorders.

Generally, EEG is usually used in the diagnosis and evaluation of many cortical and subcortical dementias. Often it can help to differentiate between a degenerative disorder such as AD and pseudodementia due to psychiatric illness [54]. The EEG may also show whether the process is focal or diffuse (i.e. involves the background delta and theta wave activities). The EEG may also reveal the early CJD-related abnormalities. However, more advanced signal processing and quantitative techniques may be implemented to achieve robust diagnostic and monitoring performance.

1.9.2 Epileptic Seizure and Nonepileptic Attacks

Often the onset of a clinical seizure is characterized by a sudden change of frequency in the EEG measurement. It is normally within the alpha wave frequency band with a slow decrease in frequency (but increase in amplitude) during the seizure period. It may or may not be spiky in shape. Sudden desynchronization of electrical activity is found in electrodecremental seizures. The transition from the preictal to the ictal state, for a focal epileptic seizure, consists of a gradual change from chaotic to ordered waveforms. The amplitude of the spikes does not necessarily represent the severity of the seizure. Rolandic spikes in a child of 4–10 years, for example, are very prominent; however, the seizure disorder is usually quite benign or there may not be clinical seizure [56].

In terms of spatial distribution, in childhood the occipital spikes are very common. Rolandic central–midtemporal–parietal spikes are normally benign, whereas frontal spikes or multifocal spikes are more epileptogenic. The morphology of the spikes varies significantly with age. However, the spikes may occur in any level of awareness including wakefulness and deep sleep.

The distinction of seizure from common artefacts is not difficult. Seizure artefacts within an EEG measurement have a prominent spiky but repetitive (rhythmical) nature, whereas the majority of other artefacts are transients or noise-like in shape. For the case of the ECG, the frequency of occurrence of the QRS waveforms (an element of the ECG) is approximately 1 Hz. These waveforms have a certain shape which is very different from that of seizure signals.

The morphology of an epileptic seizure signal slightly changes from one type to another. The seizure may appear in different frequency ranges. For example, a petit mal discharge often has a slow spike at around 3 Hz, lasting for approximately 70 ms, and normally has its maximum amplitude around the frontal midline. On the other hand, higher frequency spike wave complexes occur for patients over 15 years old. Complexes at 4 Hz and 6 Hz

may appear in the frontal region of the brain of epileptic patients. As for the 6 Hz complex (also called benign EEG variants and patterns), patients with anterior 6 Hz spike waves are more likely to have epileptic seizures and those with posterior discharges tend to have neuroautonomic disturbances [57]. The experiments do not always result in the same conclusion [56]. It was also found that the occipital 6 Hz spikes can be seen and are often drug related (due to hypoanalgetics or barbiturates) and due to withdrawal [58].

Among nonepileptics, the discharges may occur in patients with cerebrovascular disorder, syncopal attacks, and psychiatric problems [56]. Fast and needle-like spike discharges may be seen over the occipital region in most congenitally blind children. These spikes are unrelated to epilepsy and normally disappear in older age patients.

Bursts of 13–16 Hz or 5–7 Hz, as shown in Figure 1.14 (also called 14 and 6 Hz waves), with amplitudes less than 75 μV and arch shapes may be seen over the posterior temporal and the nearby regions of the head during sleep. These waves are positive with respect to the background waves. The 6 and 14 Hz waves may appear independently and

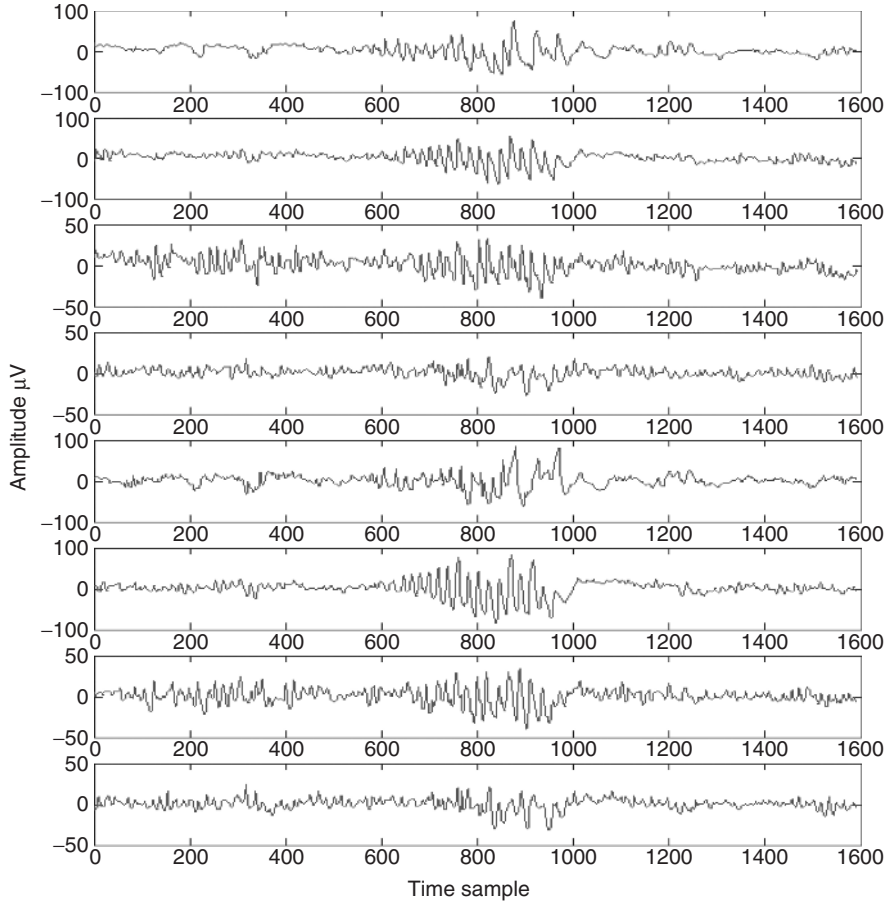

Figure 1.14 Bursts of 3–7 Hz seizure activity in a set of adult EEG signals

be found respectively in younger and older children. These waves may be confined to the regions lying beneath a skull defect. Despite the 6 Hz wave, there are rhythmical theta bursts of wave activities relating to drowsiness around the midtemporal region, with a morphology very similar to ictal patterns. In old age patients other similar patterns may occur, such as *subclinical rhythmic EEG discharges of adults* (SREDA), over the 4–7 Hz frequency band around the centroparietal region, and a wide frequency range (2–120 Hz) *temporal minor sharp transient* and *wicket spikes* over the anterior temporal and midtemporal lobes of the brain. These waves are also nonepileptic but with a seizure-type waveform [56].

The epileptic seizure patterns, called ictal wave patterns, appear during the onset of epilepsy. Although Chapter 4 of this book focuses on an analysis of these waveforms from a signal processing point of view, here a brief explanation of morphology of these waveforms is given. Researchers in signal processing may exploit these concepts in the development of their algorithms. Although these waveform patterns are often highly obscured by muscle movements, they normally maintain certain key characteristics.

Tonic–clonic seizure (also called grand mal) is the most common type of epileptic seizure. It appears in all electrodes but more towards the frontal electrodes (Figure 1.15). It has a rhythmic but spiky pattern in the EEG and occurs within the frequency range of 6–12 Hz. Petit mal is another interictal paroxysmal seizure pattern which occurs at approximately 3 Hz with a generalized synchronous spike wave complex of prolonged bursts. A temporal lobe seizure (also called a psychomotor seizure or complex partial seizure) is presented by bursts of serrated slow waves with a relatively high amplitude of above 60 μV and frequencies of 4–6 Hz. Cortical (focal) seizures have contralateral distribution with rising amplitude and diminishing frequency during the ictal period. The attack is usually initiated by local desynchronization, i.e. very fast and very low voltage spiky activity, which gradually rises in amplitude with diminishing frequency. Myoclonic seizures have concomitant polyspikes, seen clearly in the EEG signals. They can have generalized or bilateral spatial distribution that is more dominant in the frontal region [59]. Tonic seizures occur in patients with the Lennox–Gastaut syndrome [60] and have spikes that repeat with a frequency of approximately 10 Hz. Atonic seizures may appear in the form of a few seconds drop attack or be inhibitory, lasting for a few minutes. They show a few polyspike waves or spike waves with generalized spatial distribution of approximately 10 Hz followed by large slow waves of 1.5–2 Hz [61]. Akinetic seizures are rare and characterized by arrest of all motion, which, however, is not caused by sudden loss of tone as in atonic seizure and the patient is in an absent-like state. They are rhythmic with a frequency of 1–2 Hz. Jackknife seizures, also called salaam attacks, are common in children with hypsarrhythmia (infantile spasms, West syndrome) and are either in the form of sudden generalized flattening desynchronization or have rapid spike discharges [60].

There are generally several varieties of recurring or quasirecurring discharges, which may or may not be related to epileptic seizure. These abnormalities may be due to psychogenic changes, variation in body metabolism, or circulatory insufficiency (which often appears as acute cerebral ischemia). Of these, the most important ones are: periodic or quasiperiodic discharges related to severe CNS diseases; periodic complexes in subacute sclerosing panencephalitis (SSPE); periodic complexes in herpes simplex encephalitis; syncopal attacks; breath holding attacks; hypoglycemia and hyperventilation syndrome due

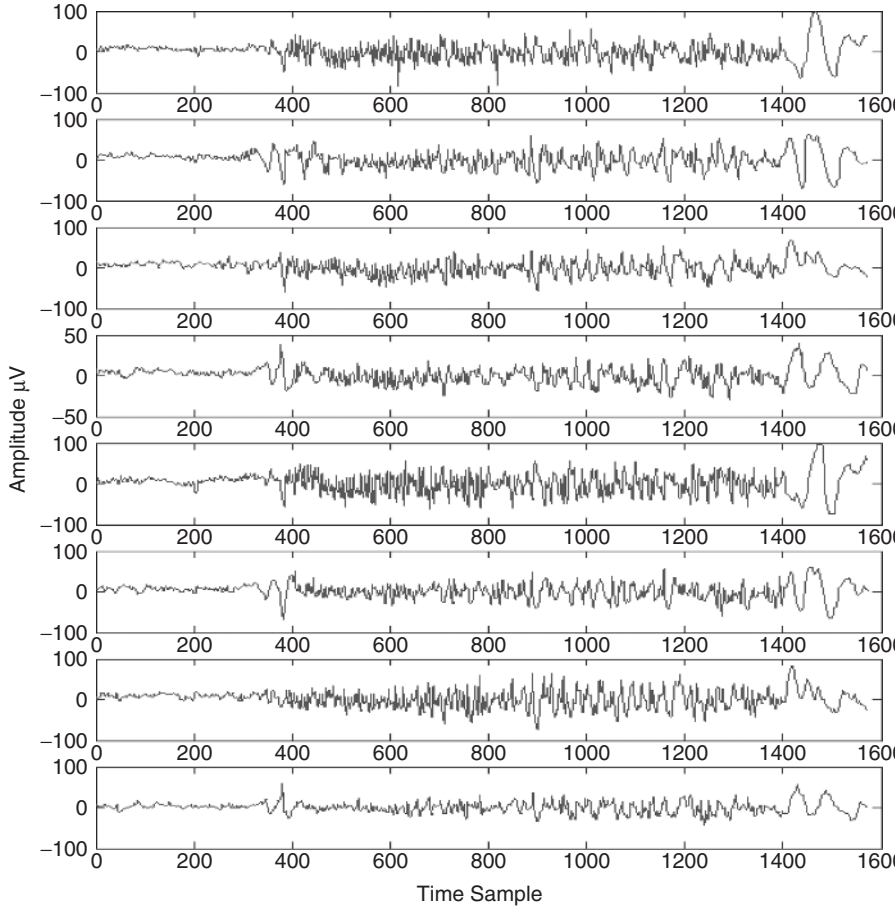

Figure 1.15 Generalized tonic–clonic (grand mal) seizure. The seizure appears in almost all of the electrodes

to sudden changes in blood chemistry [62]; and periodic discharges in Creutzfeldt–Jakob (mad cow) disease [63,64]. The waveforms for this latter abnormality consist of a sharp wave or a sharp triphasic transient signal of 100–300 ms duration, with a frequency of 0.5–2 Hz. The periodic activity usually shows a maximum over the anterior region except for the Heidenhain form, which has a posterior maximum [56]. Other epileptic waveforms include periodic literalized epileptiform discharges (PLED), periodic discharges in acute cerebral anoxia, and periodic discharges of other etiologies.

Despite the above epileptiform signals there are spikes and other paroxysmal discharges in healthy nonepileptic persons. These discharges may be found in healthy individuals without any other symptoms of diseases. However, they are often signs of certain cerebral dysfunctions that may or may not develop into an abnormality. They may appear during periods of particular mental challenge on individuals, such as soldiers in the war front line, pilots, and prisoners.

A comprehensive overview of epileptic seizure disorders and nonepileptic attacks can be found in many books and publications such as References [62] and [65]. In Chapter 5 some recent attempts in application of advanced signal processing techniques to the automatic detection and prediction of epileptic seizures are explained.

1.9.3 Psychiatric Disorders

Not only can functional and certain anatomical brain abnormalities be investigated using EEG signals, pathophysiological brain disorders can also be studied by analysing such signals. According to the *Diagnostic and Statistical Manual (DSM) of Mental Disorders* of the American Psychiatric Association, changes in psychiatric education have evolved considerably since the 1970s. These changes have mainly resulted from physical and neurological laboratory studies based upon EEG signals [66].

There have been evidences from EEG coherence measures suggesting differential patterns of maturation between normal and learning-disabled children [67]. This finding can lead to the establishment of some methodology in monitoring learning disorders. Several psychiatric disorders are diagnosed by analysis of evoked potentials (EPs) achieved by simply averaging a number of consecutive trails having the same stimuli.

A number of pervasive mental disorders cause significant losses in multiple functioning areas [66]. Examples of these are dyslexia, which is a developmental reading disorder; autistic disorder, which is related to abnormal social interaction, communication, and restricted interests and activities, and starts appearing from the age of three; Rett's disorder, characterized by the development of multiple deficits following a period of normal postnatal functioning; and Asperger's disorder, which leads to severe and sustained impairments in social interaction and restricted repetitive patterns of behaviour, interests, and activities.

Attention-deficit hyperactivity disorder (ADHD) and attention-deficit disorder (ADD), conduct disorder, oppositional defiant disorder, and disruptive behaviour disorder have also been under investigation and considered within the DSM. Most of these abnormalities appear during childhood and often prevent children from learning and socializing well. The associated EEG features have been rarely analytically investigated, but the EEG observations are often reported in the literature [68–72]. However, most of such abnormalities tend to disappear with advancing age.

EEG has also been analysed recently for the study of delirium [73,74], dementia [75,76], and many other cognitive disorders [77]. In EEGs, characteristics of delirium include slowing or dropout of the posterior dominant rhythm, generalized theta or delta slow-wave activity, poor organization of the background rhythm, and loss of reactivity of the EEG to eye opening and closing. In parallel with that, the quantitative EEG (QEEG) shows increased absolute and relative slow-wave (theta and delta) power, reduced ratio of fast-to-slow band power, reduced mean frequency, and reduced occipital peak frequency [74].

Dementia includes a group of neurodegenerative diseases that cause acquired cognitive and behavioural impairment of sufficient severity to interfere significantly with social and occupational functioning. Alzheimer disease is the most common of the diseases that cause dementia. At present, the disorder afflicts approximately 5 million people in the United States and more than 30 million people worldwide. A larger number of individuals have lesser levels of cognitive impairment, which frequently evolves into full-blown dementia.

The prevalence of dementia is expected to nearly triple by 2050, since the disorder preferentially affects the elderly, who constitute the fastest-growing age bracket in many countries, especially in industrialized nations [76].

Among other psychiatric and mental disorders, amnestic disorder (or amnesia), mental disorder due to a general medical condition, substance-related disorder, schizophrenia, mood disorder, anxiety disorder, somatoform disorder, dissociative disorder, sexual and gender identity disorder, eating disorders, sleep disorders, impulse-controlled disorder, and personality disorders have often been addressed in the literature [66]. However, the corresponding EEGs have seldom been analysed by means of advanced signal processing tools.

1.9.4 External Effects

EEG signal patterns may significantly change when using drugs for the treatment and suppression of various mental and CNS abnormalities. Variations in EEG patterns may also arise by just looking at the TV screen or listening to music without any attention. However, among the external effects the most significant ones are the pharmacological and drug effects. Therefore, it is important to know the effects of these drugs on the changes of EEG waveforms due to chronic overdosage, and the patterns of overt intoxication [78].

The effect of administration of drugs for anesthesia on EEGs is of interest to clinicians. The related studies attempt to find the correlation between the EEG changes and the stages of anesthesia. It has been shown that in the initial stage of anesthesia a fast frontal activity appears. In deep anesthesia this activity becomes slower with higher amplitudes. In the last stage, a burst-suppression pattern indicates the involvement of brainstem functions, including respiration, and finally the EEG activity ceases [78]. In cases of acute intoxication, the EEG patterns are similar to those of anesthesia [78].

Barbiturate is commonly used as an anticonvulsant and antiepileptic drug. With small dosages of barbiturate the activities within the 25–35 Hz frequency band around the frontal cortex increases. This changes to 15–25 Hz and spreads to the parietal and occipital regions. Dependence and addiction to barbiturates are common. Therefore, after a long-term ingestion of barbiturates, its abrupt withdrawal leads to paroxysmal abnormalities. The major complications are myoclonic jerks, generalized tonic–clonic seizures, and delirium [78].

Many other drugs are used in addition to barbiturates as sleeping pills, such as melatonin and bromides. Very pronounced EEG slowing is found in chronic bromide encephalopathies [78]. Antipsychotic drugs also influence the EEG patterns. For example, neuroleptics increase the alpha wave activity but reduce the duration of beta wave bursts and their average frequency. As another example, clozapine increases the delta, theta, and above 21 Hz beta wave activities. As another antipsychotic drug, tricyclic antidepressants such as imipramine, amitriptyline, doxepin, desipramine, notryptiline, and protriptyline increase the amount of slow and fast activity along with instability of frequency and voltage, and also slow down the alpha wave rhythm. After administration of tricyclic antidepressants the seizure frequency in chronic epileptic patients may increase. With high dosages, this may further lead to single or multiple seizures occurring in nonepileptic patients [78].

During acute intoxication, a widespread, poorly reactive, irregular 8–10 Hz activity and paroxysmal abnormalities including spikes, as well as unspecific coma patterns, are

observed in the EEGs [78]. Lithium is often used in the prophylactic treatment of bipolar mood disorder. The related changes in the EEG pattern consist of slowing of the beta rhythm and of paroxysmal generalized slowing, occasionally accompanied by spikes. Focal slowing also occurs, which is not necessarily a sign of a focal brain lesion. Therefore, the changes in the EEG are markedly abnormal with lithium administration [78]. The beta wave activity is highly activated by using benzodiazepines as an anxiolytic drug. These activities persist in the EEG as long as two weeks after ingestion. Benzodiazepine leads to a decrease in an alpha wave activity and its amplitude, and slightly increases the 4–7 Hz frequency band activity. In acute intoxication the EEG shows prominent fast activity with no response to stimuli [78]. The psychotogentic drugs such as lysergic acid diethylamide and mescaline decrease the amplitude and possibly depress the slow waves [78]. The CNS stimulants increase the alpha and beta wave activities and reduce the amplitude and the amount of slow waves and background EEGs [78].

The effect of many other drugs, especially antiepileptic drugs, is investigated and new achievements are published frequently. One of the significant changes of the EEG of epileptic patients with valproic acid consists of reduction or even disappearance of generalized spikes along with seizure reduction. Lamotrigine is another antiepileptic agent that blocks voltage-gated sodium channels, thereby preventing excitatory transmitter glutamate release. With the intake of lamotrigine a widespread EEG attenuation occurs [78]. Penicillin if administered in high dosage may produce jerks, generalized seizures, or even status epilepticus [78].

1.10 Summary and Conclusions

In this chapter the fundamental concepts in the generation of action potentials and consequently the EEG signals have been briefly explained. The conventional measurement setups for EEG recording and the brain rhythms present in normal or abnormal EEGs have also been described. In addition, the effects of popular brain abnormalities such as mental diseases, ageing, and epileptic and nonepileptic attacks have been pointed out. Despite the known neurological, physiological, pathological, and mental abnormalities of the brain mentioned in this chapter, there are many other brain disorders and dysfunctions that may or may not manifest some kinds of abnormalities in the related EEG signals. Degenerative disorders of the CNS [79], such as a variety of lysosomal disorders, several peroxisomal disorders, a number of mitochondrial disorders, inborn disturbances of the urea cycle, many aminoacidurias, and other metabolic and degenerative diseases, as well as chromosomal aberrations, have to be evaluated and their symptoms correlated with the changes in the EEG patterns. The similarities and differences within the EEGs of these diseases have to be well understood. On the other hand, the developed mathematical algorithms need to take the clinical observations and findings into account in order to enhance the outcome of such processing further. Although a number of technical methods have been well established for the processing of the EEGs with relation to the above abnormalities, there is still a long way to go and many questions to be answered.

The following chapters of this book introduce new digital signal processing techniques employed mainly for analysis of EEG signals followed by a number of examples in the applications of such methods.

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