ANALYSIS AND INTERPRETATION OF BIO-ELECTRIC SIGNALS–EEG

A DISSERTATION

Submitted in partial fulfilment of
the requirements for the award of the Degree
of
MASTER OF ENGINEERING
in
ELECTRICAL ENGINEERING
(Measurement and Instrumentation)

By
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C E R T I F I C A T E

This is to certify that the dissertation entitled 'Analysis and Interpretation of Bio-electric Signals – EEG' which is being submitted by Mr. R.K. Gupta in partial fulfilment of the requirements for the award of the degree of Master of Engineering in Electrical Engineering (Measurement and Instrumentation) of University of Roorkee, Roorkee, is a record of student's own work carried out under my supervision and guidance. The matter embodied in this dissertation has not been submitted for the award of any degree or diploma.

This is to further certify that he has worked for a period of 5 months from June 79 to Nov. 79, for preparing this dissertation at this University.

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CHAPTERS Page

V.7.1 Segmentation of EEG Wave 47
V.7.1(a) Parameter for Nonstationarities 48
V.7.1(b) Estimating the Boundary Position 49
V.7.2 Characteristic Analytic Parameters in Time Domain 50
V.7.3 Calculation Procedure 51

V.8 Results and Discussion 54

VI. CONCLUSION 60

REFERENCES 62

APPENDICES 66-72

A. Derivative of Tabulated Values 66

B. Data for the Tables (V.1) to (V.4) 67

C O N T E N T S

			Page
	CERTIFICATE		(ii)
	ACKNOWLEDGEMENT		(iii)
	SYNOPSIS		(vi)
CHAPTERS			
I.	INTRODUCTION		1
II.	ANATOMY AND PHYSIOLOGY		7
III.	GENERATION AND CHARACTERISTICS OF THE BIO-ELECTRIC SIGNAL (BRAIN)		16-22
	III.1	Generation of Bio-electric Signal	16
	III.2	Characteristic features of EEG wave	20
IV.	RECORDING TECHNIQUES AND ABNORMALITIES OF EEG		23-33
	IV.1	Recording Techniques	23
	IV.2	Abnormalities	27
V.	METHODS OF ANALYSIS		34-56
	V.1	Histogram Method	36
	V.2	Fourier Analysis Method	37
	V.3	Auto-correlation, Cross-correlation, and Coherence Function Analysis	38
	V.4	Convolved Spectra	42
	V.5	Bispectrum Analysis	43
	V.6	Period Analysis	44
	V.7	Analysis Based on Time-Domain Properties	46

S Y N O P S I S

It is rightly said that,"the beginning of the wisdom is the calling of things by their right names". This dissertation is a small step in this direction i.e. trying to explore a method which describes the abnormality of brain- a centre of wisdom.

The confounded language of brain is studied with the help of bio-electric potentials recorded from the scalp. This recording is termed as the Electroencephalogram (EEG). In this process, the first step should be in the direction of comprehending the physiological process. So, in all fairness, the anatomy and functions of the brain are discussed succintly, followed by a brief description of : the underlying process in the generation of EEG wave and its characteristics; the recording techniques; and then some of the abnormalities of the brain having clinical and pathological background.

At last, the author has discussed a few methods of analysis of random signal. An approach is made towards the time domain analysis for extracting the elementary features of EEG wave. In this approach, the epochs are decided by the segmentation procedure for a random signal. In this way, more discernible, and informative parameters are obtained, which can be correlated with the clinical and pathological conditions of the brain.

As such, the analysis of electroencephalogram does not specify any etiology, but in all sincerety an idea of certain pathological conditions of the brain, e.g., lateralised injury, brain tumor, etc. can be had with the help of this analysis method.

CHAPTER – I

INTRODUCTION

Michael Crichton, in his book and screen play, 'The Terminal Man' spins an entertaining tale about a team of surgeons and engineers who collectively connect a patient's brain to a computer for the express purpose of regulating his behaviour. They achieve the goal in the story as narrated by monitoring the electrical activity of functional groupings of neural units with the help of implanted electrodes. The pattern of electrical activity is processed and recognised by the computer, which in turn stimulates specific locations within the brain. But this is only the beginning of the Crichton's story; it goes on to describe how things can go awry when a human, particularly an ingenious one, sets his mind to circumvent automated machine operated control systems.

With animal subjects in place of humans of Crichton's story, a large number of experiments are underway in various laboratories throughout the world where the computer is not only monitoring the brain waves, but is also programmed to automatically stimulate the brain or sensory modalities in attempts to educe specific behavioural responses.

It has now been just over a century since Dr. Richard Caton, an English Physician, reported in 1875

his observations concerning brain waves recorded from the exposed surface of the brains of rabits and monkeys. He was the first to observe two forms of brain electrical activity, the first is now known as an evoked potential; and the second is the spontaneous, on going electrical activity, the recording of which is now termed as EEG.

At the time of Caton's discovery it had been known for nearly one half century that nerves conducted electrical pulses. Another one half century was to pass following Caton's investigations before brain waves were to be discovered in humans by the German psychiatrist, Hans Berger, in 1929. By this time the technological advancement, beyond the crude galvanometer and optical amplification from Caton's days to the use of electronic amplifiers, allowed Berger to measure potential fluctuations directly from the scalp with two large pad electrodes soaked in saline which he placed on the forehead and at the back of the head over the occipital region. Later, he confirmed his observations at the scalp by measuring directly from the surface of the brain. He then hoped it might be possible to establish relationship between these potentials and the performance of the brain.

In clinical medical practice, these potential fluctuations recorded from the scalp named by Berger as Electroencephalogram (EEG), has been found useful in two main fields. First, the study of abnormality which has been

known from very ancient times – epilepsy, and secondly in helping the early diagnosis of at least certain types of tumors.

Electroencephalography is to this day one of the major techniques in the study of higher nervous activity. In view of the penetration of mathematics into all fields of science, the attempts by physiologists to supplement visual analysis of the electroencephalogram (EEG) by mathematical model is natural. From mathematical analysis of electroencephalographic data, much information on the functional state of the brain can be obtained, so that a deeper understanding of complex physiological phenomena will undoubtedly result, and the possibilities for diagonis in cases of tumor of the brain will be enhanced.

The study of the brain potentials and their rhythms is one of the most complicated tasks that has ever been proposed to physiologists. Brain potentials and their rhythms are the net result of a conjuction of many hetrogeneous physical conditions, anatomical organizations, statistical effects and differential properties of the neuron segments implemented in different ways.

It is the aim of this study to have a pragmatic approach for the analysis of the clinical EEG, with the final goal of an automatic diagonsis. In this case, we do not have any neurophysiological model, either source and generators of the EEG are still not exactly known, so we

have to content ourselves with the phenomenological model.

The evaluation given by the physician consists of two parts; the first one is purely descriptive whereas the second one contains the diagnostic evaluation of the record. This corresponds to the pattern recognition. It should also be noted in this context that only in few cases it is possible to establish a diagnosis from the EEG alone (About 15% of the population show EEG abnormalities but do not suffer any neurological disorder).

The EEG activity is divided into the following categories: spontaneous on going (non paroxysmal) activity, spontaneous paroxysmal activity, and the activity evoked by external sensory stimulation. The EEG signal has a random form which can be described in statistical terms. There are two waveforms of interest. In the first, the observed signal has statistically regular features (e.g. normal α and β rhythm) and signal can be regarded as stationary or stochastic process. In the second type, specific transients such as isolated pulse or complex bursts are observed (i.e. spikes or waves).

EEG is best characterized as continuous wave activity of variable amplitude and frequency, within constant phase relations quite similar to random noise in overall characteristics. Arising spontaneously, the EEG is difficult to correlate in consistent manner with discrete

behavioural events. Superposition and summation, which bring about dramatic improvements in signal to noise ratio in such time locked events as evoked potentials, completely fail with regard to EEG. Generally, when several EEG segments are superimposed, the algebric sum tends to zero in keeping with noise like characteristic of this activity.

Unlike man-made machine communication systems in which noise is an encumberance, the brain may use noise as a desirable or perhaps even essential factor. From the study of statistical properties of EEG, certain inferences may be made about the basic mechanism of the generation of EEG. While the origin of the brain wave is still enigmatic, it is nonetheless evident from the various study that slow wave phenomena are an important indicator of fundamental cerebral process.

Today, the electroencephalographer is not only concerned with the voltage-time derivation from the scalp, considered as the remote field of a single, compact distant generator; but he is also concerned with interaction of many partially correlated voltage-time changes spatially dispersed and not only over the convexity of the surface but in three dimensions; as he is reaching down with his probes into the regions where the generators of these waves actually lie. No wonder he cries for help. To whom he should turn ? The answer is implied in a paragraph taken from a book "The Organisation of the Cerebral Cortex" by 'Sholl, D.A.'.

"It is suggested, however, that whether the cortex is studied by anatomist, the physiologist, or the psychologist, the model employed should be based on the concept of probability and discussed in a statistical language. This would imply that any theory that attempts to account for the properties of the dynamic spatio-temporal system that forms the basis of our behaviour, must employ statistical hypothesis.'

CHAPTER – II

ANATOMY AND PHYSIOLOGY

The fluctuating potential recorded either from the scalp, cortex or depths of the brain, represents a superposition of volume-conductor fields produced by a variety of active neuronal current generators. Unlike the relatively simple bioelectric source i.e. the nerve trunk with its enclosed bundles of circular cylindrical nerve axons, the sources generating the field potentials are aggregates of neuronal elements (dendrites, cell bodies or somata, and axons of nerve cells) with complex inter - connections. The architecture of the neuronal brain tissue is not uniform from one location to another in the brain. So we must first discuss about gross anatomy and functions of the brain, the ultra-structure of the cerebral cortex and other related things before any detailed study of electroencephalography.

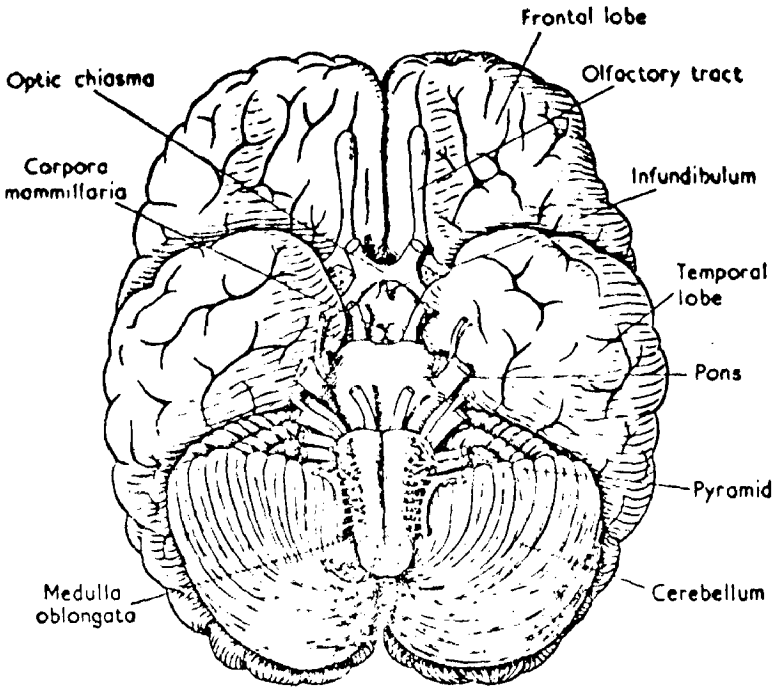
Anatomy and Functions of the Brain [5, 12, 16]

The central nervous system (CNS) consists of the spinal cord lying within the bony vertebral column and its continuation, lying within the skull. The brain is the greatly modified and enlarged portion of the CNS, surrounded by three protective membranes (meninges; e.g. the pia mater, the arachnoid, and the dura mater) and

enclosed within the cranial cavity of the skull. The spinal cord is likewise surrounded by downward continuation of meninges, and is encased within the protective vertebral column. Both brain and spinal cord are bathed in a special extracellular fluid called cerebral spinal fluid (CSF).

Within the CNS, there are ascending (sensory) nerve tracts that run from spinal cord to the various areas of the brain, conveying information regarding changes in the external environment of the body that are reported by various peripheral biological transducers. There are a number of sensory transducers for sensing temperature, pain, fine touch, pressure and various other things on the human body surface.

Similarly, there are descending (motor) nerve tracts that originate in various brain structures such as the cerebrum and cerebellum (Fig. 2.1) and terminate ultimately on motor neurons in the ventral horn of the spinal cord. Thus there exists two-way communication links between the brain and the spinal cord that allow higher centres in the brain to control or modify the behaviour of elemental spinal reflex arc at a given spinal level. In this way, the brain is not only informed of a peripheral event, but also modifies the response of the spinal reflex to that environmental reflex. The transmission of information to brain is by means of a frequency modulated train of nerve impulses which stimulates the neurons of specific area of the brain.



Note: The cranial nerves

The under surface of the human brain.

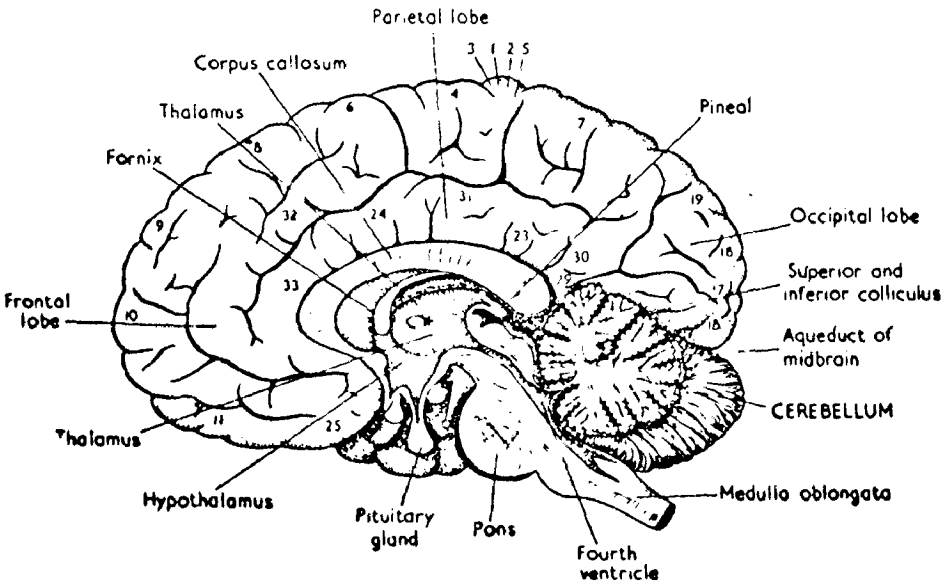


Fig. 2-1 Vertical cross-section of the human brain through its plane of approximate symmetry. In the upper and left-hand parts of the Figure we see part of the surface (cerebral cortex) of the right cerebral hemisphere. The numbers follow the Brodmann numbering system for regions of the cortex

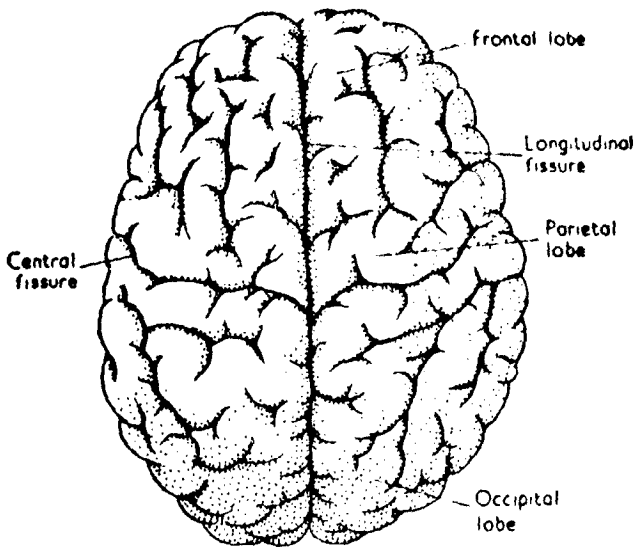


FIG. 2·2a The human brain seen from above.

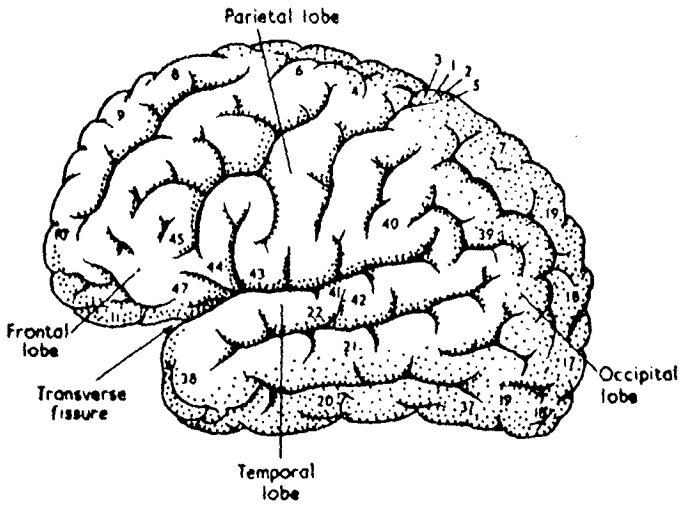


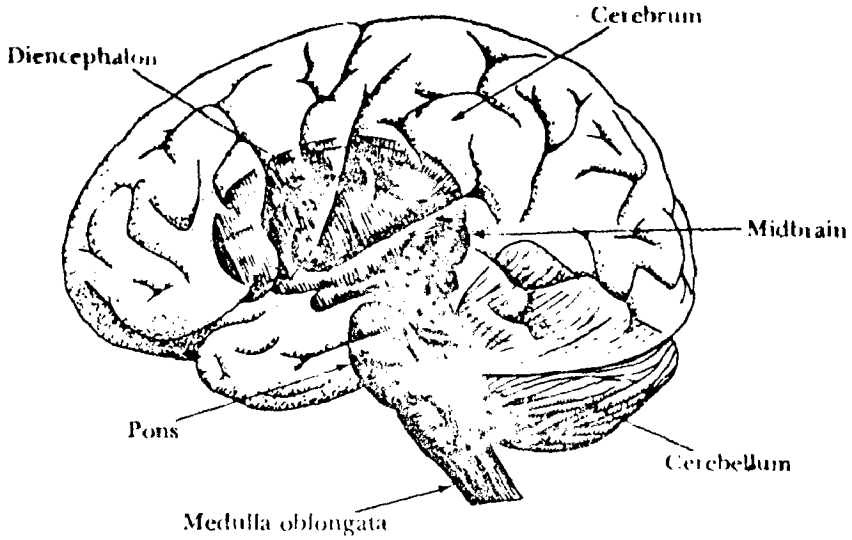
FIG. 2·2b The human brain from a side view.

In turn, the decision to take a particular motor action in response to a stimulus is manifested in the activity of cortical neurons from one of the various areas of brain. Such cortical activity is reflected in changes in the volume-conductor field potentials recorded from the brain as EEG.

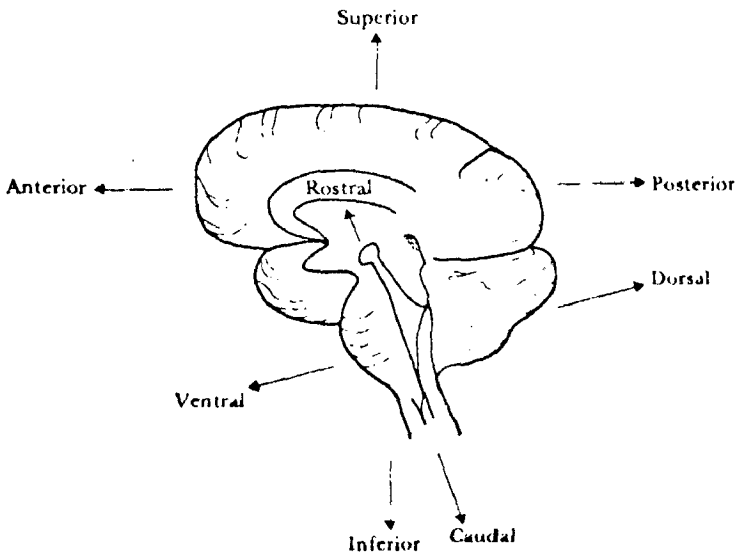
The brain is divided into three main parts - cerebrum, brain stem, and cerebellum (Fig.2.3a) from the point of localisation and functional study of brain. The general anatomic directions of orientation in the CNS are shown in (Fig.2.3b). Here the directions rostral (towards head), caudal (towards tail), dorsal (back), and ventral (front) are associated with the brain stem; remaining terms are associated with the cerebrum. The terms medial and lateral imply nearness and remoteness respectively, to central midline axis of brain.

The Cerebrum

The cerebrum (Fig.2.4) is a paired structure, with right and left cerebral hemispheres, each relating to the opposite side of the body. The surface layer of the hemisphere is called cortex, which receives sensory information from skin, eyes, ears and other receptors located generally on the opposite side of the body, which is compared with the previous experience and produces movements in response to these stimulii.



(a)



(b)

Fig. 2·3 (a) Anatomical relation of brainstem structures (medulla oblongata, pons, midbrain, and diencephalon) to cerebrum and cerebellum. (b) General anatomic directions of orientation in the central nervous system. Here the directions rostral (toward head), caudal (toward tail), dorsal (back) and ventral (front) are associated with the brainstem; remaining terms are associated with the cerebrum. The terms medial and lateral imply nearness and remoteness, respectively, to central midline axis of brain.

Each hemisphere consists of several layers. The outer layer is a dense collection of nerve cells that appear grey in color. So it is called as grey matter. This outer layer, roughly 1 cm. thick is called cerebral cortex. It has a highly convoluted surface consisting of gyri (ridges) and sulci (valleys); the deeper sulci being termed fissures. The deeper layers of the hemisphere (i.e. beneath the cortex) consists of axons (or white matter) and collection of cell bodies, termed nuclei.

The major dividing landmark of the cerebral cortex is the lateral fissure (Fig. 2.4), which runs on the lateral (side) surface of the brain from the open end in front, posteriorly and dorsally (backward and upward). The lateral fissure defines a side lobe of cortex below it, called the temporal lobe. The upper part of this lobe contains the primary auditory cortex, which is the part of the cortex that receives auditory impulses via neural pathways leading from the auditory receptors in the inner ear. For most individuals, the left temporal lobe surrounding the transverse temporal gyrus is involved in more complex interpretation of auditory signals. If cells in this area are damaged and die, the subject is not able to interpret sound as words. This general cortical area surrounding the prime auditory reception area (primary auditory cortex) acts as an area of auditory interpretation.

The visual system is another example of the projection of the senses onto the cerebral cortex. The occipital lobe at the back of the head is the primary visual cortex; on which depends the ability of the visual system to detect spatial organisation of the visual scene. Specific points on the retina are connected with specific points of the visual cortex.

Another dividing line, the central sulcus runs from the medial surface over the convexity of the hemisphere to the lateral fissures. The central sulcus also represents the posterior border of the frontal lobe. The gyrus lying just anterior (forward) to the central sulcus is the precentral gyrus, which functions as the primary motor cortex. From this gyrus, nerve signals run down through the brain stem to the spinal cord for control of skeletal muscles via neural control of motoneurons in the ventral horn of the spinal cord. Lesions of the part of this precentral gyrus cause partial paralysis on the opposite side of the body.

In the area called premotor cortex, more complex movements such as speech are organised. The anterior and inferior portion of the frontal lobe are involved in the control of emotional behaviour. Immediately behind the central sulcus lies the parietal lobe. Its anterior border is the central sulcus; its ventral boundary is the lateral fissure; and its posterior boundary is rather ill defined on the lateral surface (Fig.2-4). Immediately posterior to

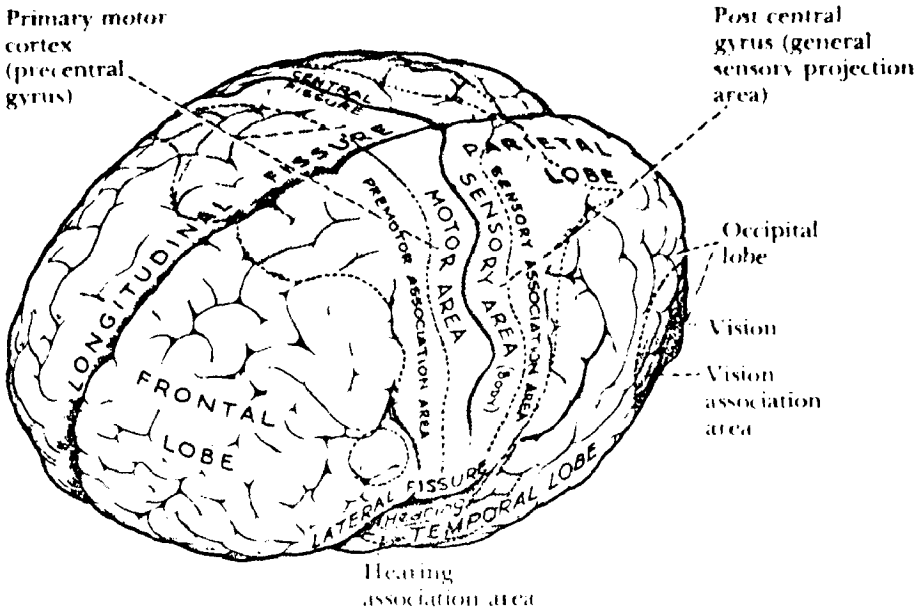


Figure 2·4 The cerebrum, showing the four lobes (frontal, parietal, temporal, and occipital), the lateral and longitudinal fissures, and the central sulcus.

the central sulcus is the primary somatosensory cortex, the postcentral gyrus. Each little area along this gyrus is related to a particular part of body and receives impulses from all general sense receptors of the skin. Higher-order sensory discrimination is organized solely in the parietal lobe. The parietal lobe is also responsible for a person's awareness of the general position of the body and its limbs in space.

The Brain Stem

The brain stem is composed of four regions each having its distinct functions: the medulla oblongata, the pons, the mid brain, and the diencephalon (Fig. 2.3a). Each contains groupings of cell bodies (nuclei) and bundles of nerve axons (tracts) that are intermingled. At the upper border of the medulla is a distinctive bulge; the pons. The medulla contains fiber tracts, as well as motor and sensory nuclei for receiving sensory information from and controlling muscles in the mouth, neck, and throat. It also hosts for the reflex control of the respiratory and cardio-vascular systems.

The nerves that connect directly to the brain are called cranial nerves. There are twelve pairs cranial nerves, eleven of which enter the brain stem and the olfactory nerve from the nose enters the cerebrum.

The pons contains cranial nerve nuclei associated with sensory input and motor output to the face. The midbrain

contains the major nuclei controlling eye-movements. It also contains large tracts carrying signals down from the cerebral hemispheres, as well as sensory tracts arising from various sources (the spinal cord, auditory system etc.) and continuing through the mid brain to higher centres.

The diencephalon is the most superior portion of the brain stem; its chief component and largest structure is the Thalamus. The thalamus serves as a major relay station and integration center for all general and special sensory systems sending information to their respective cortical reception areas. It serves as a gateway to the cerebrum [16].

The brain contains a system of cavaties, known as ventricles, where the cerebrospinal fluid is generated. The fluid passes through a hole in brain stem and surrounds both brain and spinal cord. These, thus float in the fluid which helps to resist the stresses due to acceleration.

Cerebellum

The cerebellum (Fig.2.1) receives information from the spinal cord regarding the position of trunk and limbs in space. It receives information that has originated in the cortex. Fibers descend from the cortex to nuclei in the pons, synapses occur, and postsynaptic fibers carry information to the cerebellum. The spinal cord sends the cerebellum feed back information about the limbs in space.

The cortex sends the cerebellum a command about where it should be. The cerebellum compares information and sends commands to spinal motor neurons. The cerebellum receives a strong input from the vestibular system and is heavily involved in continuous adjustment of muscles to maintain the body's posture under a variety of operating conditions.

The Reticular Formation

Throughout the extent of the brain stem, there is a diffuse collection of neurons and nuclei collectively known as the reticular formation [5] . Many special small nuclei, motor and sensory in function, are interspersed in the reticular formation. Some motor nuclei operate in conjunction with the diffuse reticular neurons to activate the subconscious motor activities of the body. Most of the reticular formation is excitatory in function. Diffuse stimulation in this area increases the muscle tone. A small area in the lower part of reticular formation has inhibitory function.

When the facilitory portion (excitatory) is uninhibited by signals from other sources, it transmits repetitive impulses to skeletal muscles throughout the body. This facilitory area also provides the input to the reticular activating system (RAS). This stimulus to this very important system causes a sleeping animal to awaken instantaneously. Anesthesia and comatose states cause impairment of its function. In sleep, RAS is in dormant state, yet any type

of sensory input signal cause sudden activation of RAS, producing arousal. So there is an accompanying change in typical EEG recordings, from sleeping to a waking pattern of activity.

RAS is a complex polysynaptic pathway. Collateral nerve branches funnel into it not only from the long ascending sensory nerve tracts running from the spinal cord to the thalamus and cortex, but also from sensory nerve input from the face as well as the auditory, visual, and olfactory systems. The system is nonspecific as most reticular neurons are activated with equal facility by different sensory stimulii. In the specific system of ascending sensory neural pathways to the thalamus and cortex, the component nerve fibres are activated by only particular type of sensory stimulation. Activity in RAS, through the thalamus or bypassing it, projects in a diffuse manner to the cerebral cortex.

Some of the quantitative data about a human brain are given as follows. The predominant part of the brain mass is seen to lie in the cerebral hemispheres. This may be the apparent reason for unique efficiency with which man can think abstractly and symbolically [12] .

			% of brain weight
Brain weight, male	1400 gm	Cerebral hemispheres	88
Brain weight, female	1300 gm	Cerebellum	10
Brain volume	1200 ml	Brain stem	2
Spinal cord weight	27–38 gm		
Spinal cord length	42 cm		

CHAPTER – III

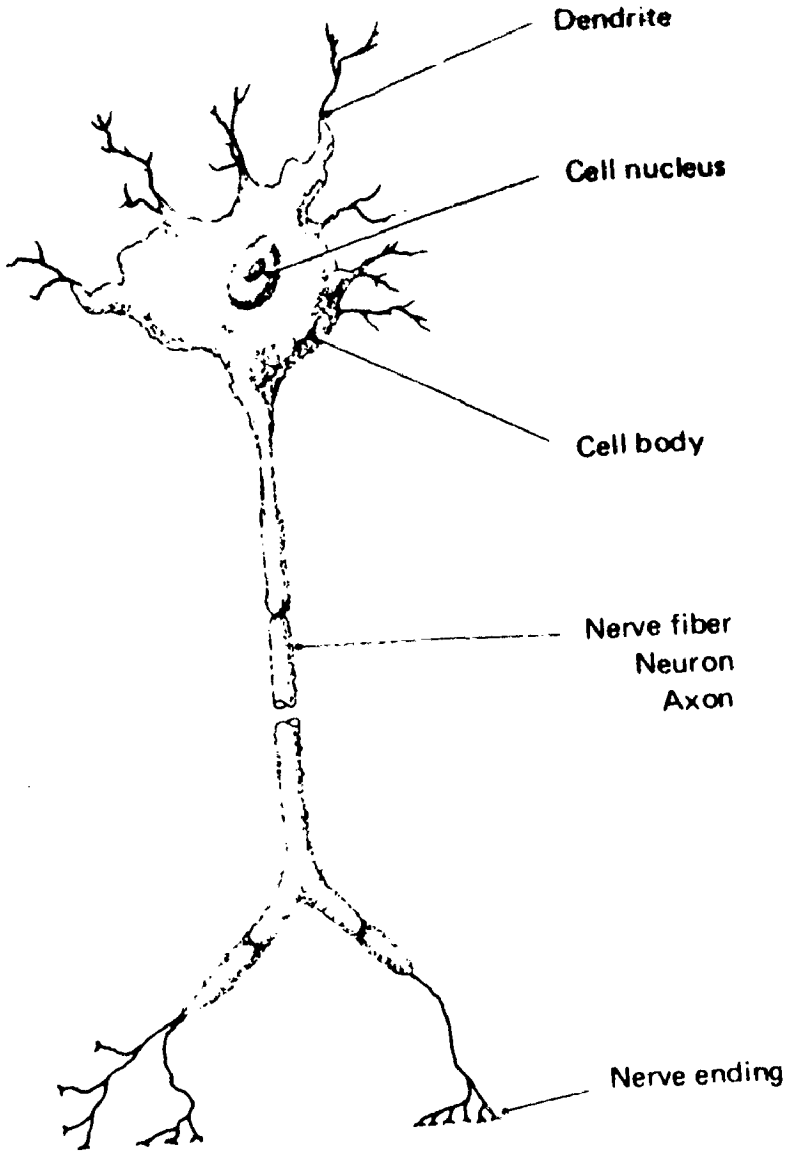
GENERATION AND CHARACTERISTICS OF THE BIOELECTRIC SIGNAL (BRAIN)

III.1 Generation of Bioelectric Signal [5,12]

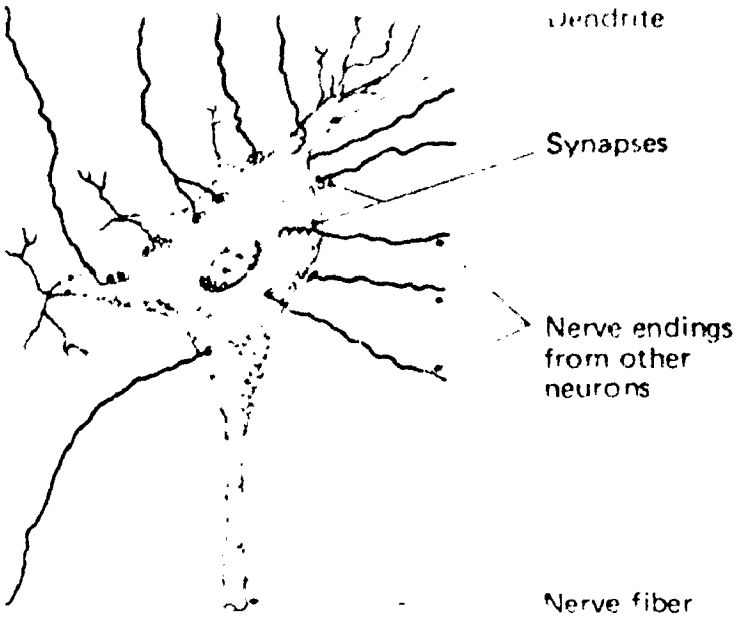
Bioelectric potentials are produced as a result of electrochemical activity of cells known as excitable cells, that are components of nervous, muscular or grandular tissue. The functional unit of the nervous system is the nerve cell or the neuron. The nerve cells are building blocks of signalling system of the brain. They are to it as the logic circuits, wires and elements of the magnetic core store are to a digital computer.

Each nerve cell body has several short processes, or dendrites and a long nerve fiber, an axon, which can have many branches. While the size of the central body of the nerve cell is that of the other cells of the body (10–100 μm), the axon can be a meter in length (Fig.3.1a).

The neurons, like other cells of the body, are electrically polarized at rest. The interior of the neuron is at a potential of about –70 mv relative to the exterior, leading to a very high field strength of about 10^5 volts/cm across the very thin surface membrane (Fig.3.1b). This potential is mainly due to the active transport of the K^+ ion

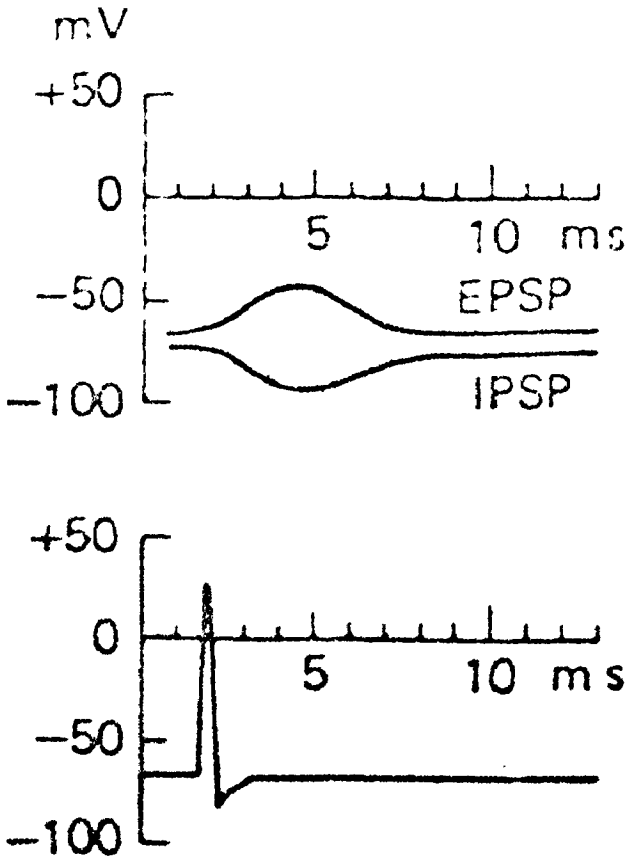


a _ NERVE CELL WITH BRANCHES

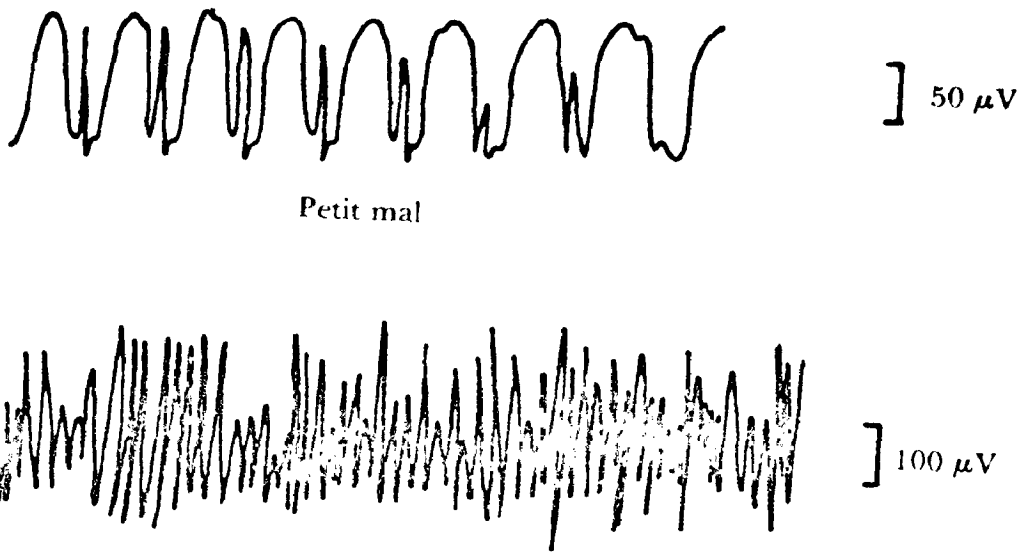


b _ SYNAPSES BETWEEN ONE NEURON AND A NUMBER OF NERVE ENDINGS FROM OTHER NEURONS

FIG. 3.1


mV
+50
0
5
10 ms
−50
EPSP
IPSP
−100
+50
0
5
10 ms
−50
−100

(a)


50 μV
Petit mal
100 μV
Grand mal epilepsy

(b)

FIG. 3·2

to the interior of neuron. At the steady state of resting potential, the cell membrane is said to be polarised. A lessening of the magnitude of this polarisation is called depolarisation and an increase in magnitude is referred to as hyperpolarisation. When a nerve cell is exposed to a stimulus above a threshold – whether electrical, chemical, mechanical, or thermal – a nerve impulse, which seen as a change in membrane potential, is generated and spread in the cell. This is due to a sudden increase in Na^+ ion permeability of the membrane, which results in the depolarisation of the membrane followed by repolarisation.

Information is transmitted through an axon by means of short impulses of constant amplitude, in accordance with the monostable flip-flop. When the stimulation threshold is exceeded, a nerve impulse is generated and conducted along the axon at a speed depending on its diameter. For axons of 20 and 0.5 μm, the velocity is about 100 and 0.5 m/s. The duration of the impulse is about 1m sec and the information is coded through the rate of conducted impulses [16] .

In the nervous system, there are a large number of synapses (i.e. the connection between two excitable cells in the form of a contact surface between a neuron and another neuron, muscle cell, or a sensory cell etc.) between each neuron and cell bodies of the dendrites of other neurons, and branched end fibers of axons (Fig.3-1b). In the synapse a nerve impulse can be transmitted, blocked, or changed from

simple to repetitive pulse or integrated with impulses from other cells leading to a complex pattern, depending on the function of synapse. Two types of chemical substance: acetylcholine and norepinephrine, are diffused across the gap ($\approx$ 200 A^0) in the synapses resulting in two type of nerves known as cholinergic and adrenergic respectively.

In the body of neuron, the two different potentials namely excitatory post synaptic potential EPSP, and inhibitory post synaptic potential IPSP are generated via synapses. The membrane potential is not measurable at the scalp due to the thickness of intervening tissues. So, activity of large number of neurons must be synchronized to have a measurable potential at the scalp. Such synchronization is controlled by subcortical centres probably from the brain stem. But the exact mechanism is still unknown, i.e. why EEG curves are clinically interpreted largely on a purely empirical basis.

The peak to peak amplitude of the waves that can be picked up from the scalp is normally 100 μv or less, while that on the exposed brain is 10-20 times greater - 1 mv. The frequency content ranges from 1 to 50 Hz.

Some quantitative data about nerve cells in various part of the human brain are as follows [12] .

(i) Cerebral hemispheres: The number of cells in the cerebral cortex (both sides) has been thought to be about $5\text{-}8 \times 10^9$.

(ii) Cerebellum : Blinkov and Glezer (1968) gave this figure as about 10^{10} neurons.

(iii) Spinal Cord: 1.3×10^{7} neurons (Blinkov and Glezer,1968).

(iv) Corpus Callosum: (Fig.2.1) This consists the majority of the fibres which connect two sides of cerebral cortex, passing through a plane of approximate bilateral symmetry of head and brain. It contains about 1.4×10^{8} axons.

(v) Total input to brain and spinal cord: There are 1.37×10^{6} fibres into the spinal cord and, apart from optic tract, 2.9×10^{5} into the brain (Bruesch and Arey, 1942).

(vi) Volume of cell bodies: Hyden (1960) finds the following volumes in the rabbit (in μm^3, fixed tissues, except spinal neurons): cerebral cortex, 5×10^{2} to 2×10^{4}; spinal neurons, 2.5×10^{4} to 5×10^{5}; granule cells of cerebellum, 600-700; bipolar cells of retina, 10^{3}-5×10^{3}.

(vii) Number of dendritic branches: Sholl (1956) finds 20-80 in cat cerebral cortex.

(viii) Total number of synapses in human cerebral cortex: This is not known but according to Cragg's and Pakkenberg's work comes out to be something between 1.6×10^{13} to 1.6×10^{14}.

(ix) Mean firing rate per cell: It is measured in the range of 1 - 10 firings/sec as an overall average in cat visual cortex. (Horta et.al, 1969).

(x) The fundamental parameter, the threshold number of impulses needed to fire a cell, is not known for brain cells.

III.2 Characteristic features of EEG Wave [5, 16]

Electrical recordings from the exposed surface of the brain or scalp shows continuously oscillating electrical activity within the brain which varies both in frequency and amplitude. Under certain circumstances e.g. in certain normal mental states and pathological conditions, as epilepsy-definite patterns are seen in EEG signals (Fig.3.2b). Under normal conditions, an inverse relationship exists between amplitude and frequency. It is because an increased cerebral activity leads to a more desynchronized activity of nerve cells.

The intensities of the brain waves on the cortex may be large as 10mv, whereas those recorded from the scalp have a smaller amplitude of 100 μV approx. The frequencies of these brain waves range from 0.5 – 50 Hz and their character is highly dependent on the activity of cerebral cortex. Some of these are characteristics of specific abnormalities of brain; such as epilepsy and others occur in normal persons. The brain waves are classified into four wave groups as alpha (α), beta (β), theta (θ), and delta (δ) (Fig.3.3).

Alpha waves have frequencies between 8-13 Hz. Their voltage is approximately 20-200 μV with approx. mean of 50 μV. They appear over the occipital lobes in the awake, mentally relaxed state with the eyes closed. When the eyes are opened, the alpha activity disappears and waves of a higher frequency and lower amplitude appears, (Fig.3.3). If the patient falls asleep, the alpha activity disappears entirely. It depends upon the individual also – in about 10% of all normal subjects no typical alpha activity can be recorded,[16].

Beta waves normally occur in the frequency range of 13-30 Hz and sometimes – during intense mental activity as high as 50 Hz. They appear over the parietal and frontal regions of scalp. This wave is divided into two major types – β1 and β2. The β1 waves have a frequency about twice that of alpha waves, and show the same behaviour towards mental activity as alpha waves. The β2 waves, appear during intense activation of CNS or during tension.

The frequency range of the theta waves is 4-8 Hz. These occur mainly in the parietal and temporal regions in children but they also occur during emotional stress in adults, during periods of disappointment and frustation. They also appear in light sleep of adults.

Delta waves includs all the EEG activity below 4 Hz. They occur in deep sleep, in infancy, and in serious organic brain disorders. Delta waves can occur solely

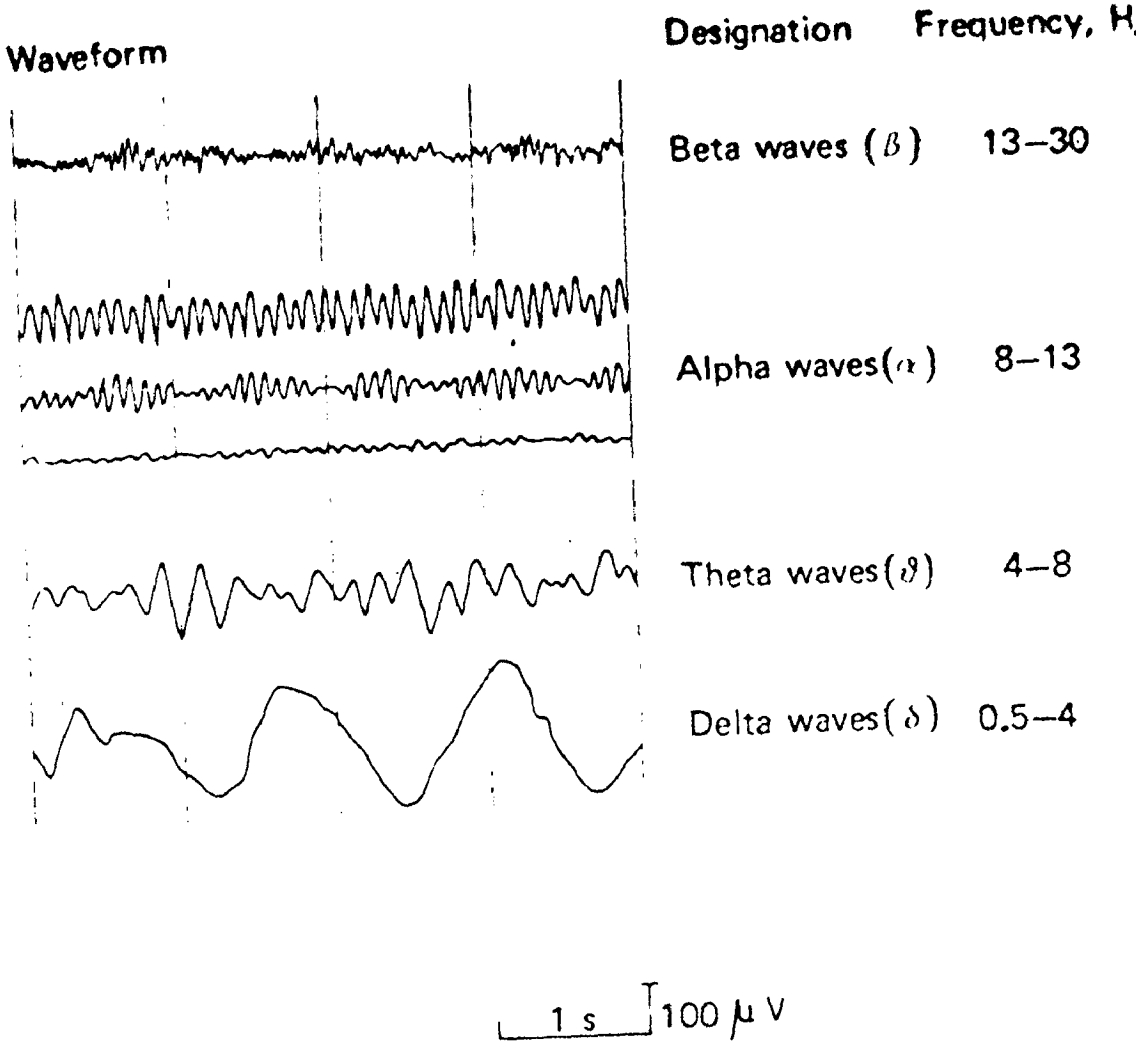


FIG.3.3 _ DIFFERENT TYPES OF NORMAL EEG WAVES

within the cortex, independent of activities in lower regions of the brain.

Sometimes, transients of duration 80–200 m sec also appears as spikes and waves – a pathological waveform is shown in Fig.3.2b. The EEG waves may be altered by pathological processes in the cerebral cortex or brain stem.

CHAPTER – IV

RECORDING TECHNIQUES AND ABNORMALITIES OF EEG

IV.1 Recording Techniques

The system most often used to place electrodes for monitoring the clinical EEG is the International standard 10-20 electrode system; [15] so named because the positions of the electrodes are based on intervals of 10 and 20 percent of the distance between the specific points on the scalp. Reference points are the root of nose (nasion) and the ossification center (bump) on the occipital bone (inion).

The anterior-posterior measurements are based upon the distance between the nasion and the inion over the vertex in the midline. Five points designated as frontal pole (F_p), frontal (F), central (C), Parietal (P), and Occipital (O) are marked in the Fig.4.1 . Lateral measurements are based upon the central coronal plane. The distance is measured from left to right preauricular points.

The electrode locations in different portions of the scalp are designated by even numbers as subscripts for right hemisphere, and odd numbers for left hemisphere. Electrodes at the mid-line in the Frontal, Central and Parietal regions are designated by F_z, C_z and P_z respectively (Fig.4.2).

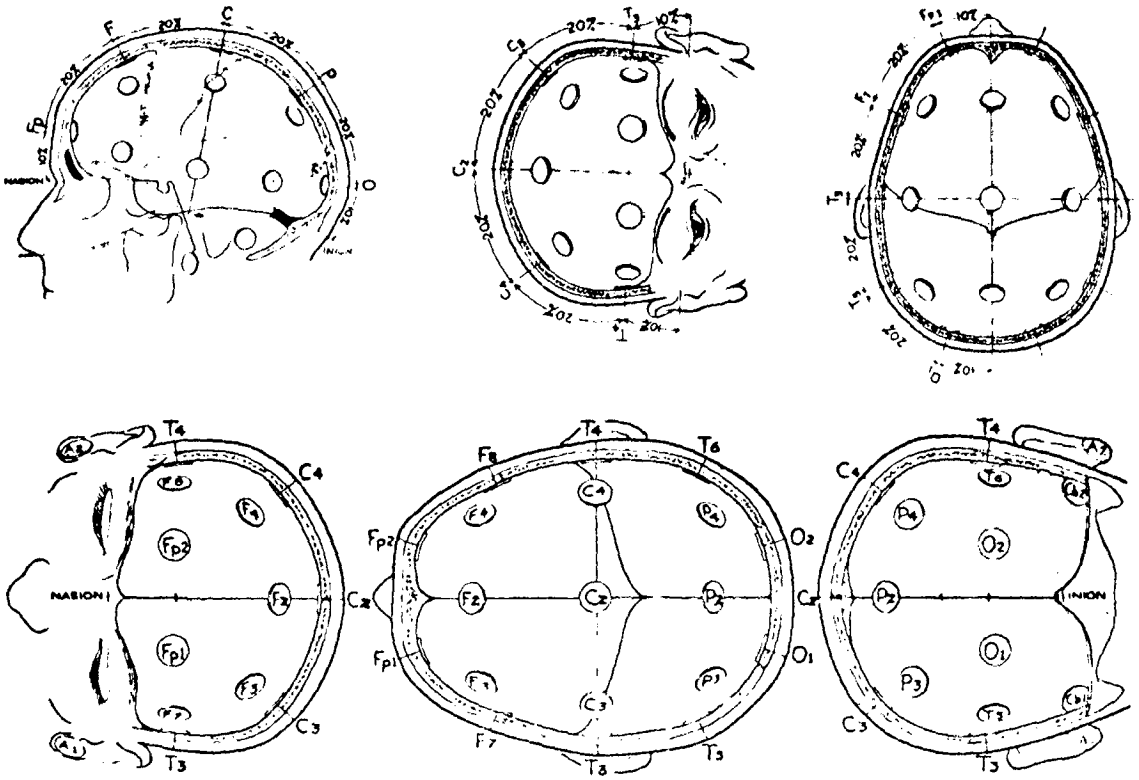


Figure 4-1 The 10-20 electrode system recommended by the International Federation of EEG Societies.

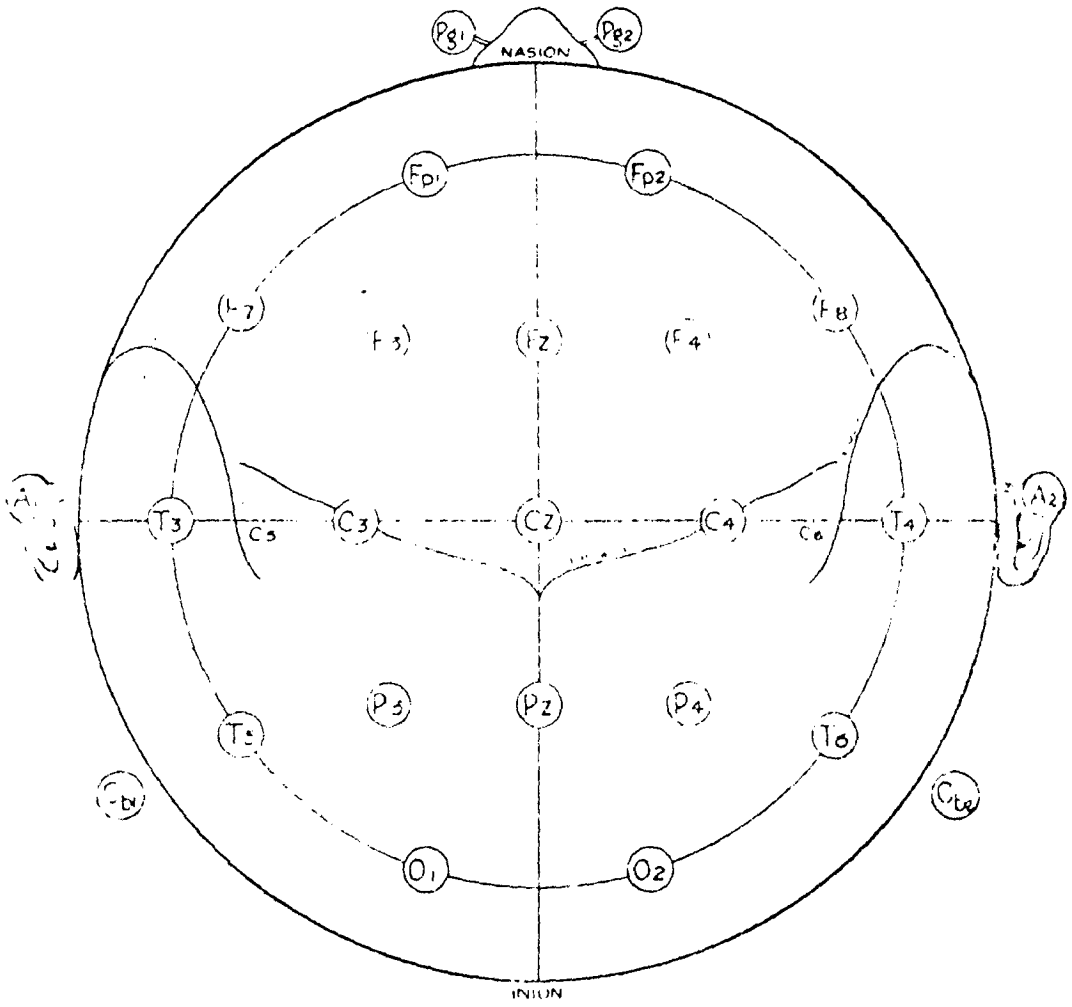


FIG. 42 _ SINGLE PLANE PROJECTION OF HEAD SHOWING ALL STANDARD POSITIONS

Three types of electrode connections are used: (i) Between each of pair (bipolar), (ii) between one monopolar le and a distance reference electrode (usually ear, can be chin, or back of the neck), and (iii) between one monopolar lead and the average of all [5,16]. In average reference mode, the system reference is formed by connecting all scalp recording locations through equal high resistance to a common point. In bipolar system, the differential measurements are advantageous because far field activity common to both electrode is cancelled. These potential undulations are amplified by high gain differential amplifiers and displayed by ink writing of strip chart recorders. The scalp is prepared specially, degreased by cleaning it with alcohal, applying a conducting paste and then non polarisable Ag–AgCl electrodes are glued to the scalp with collodion, or held them in place with rubber straps. The contact impedence is less than 10 KΩ. Sometimes brain potentials are frequency modulated and recorded on a magnetic tape, which can be stored and played back at later times. The speed of the paper in strip chart recorder is set at 3 cm/sec., keeping in view the sampling theorem for the analysis of the waveform containing components of 30 c/s and gives distributions at intervals of 16.7 m sec. or 0.5 mm apart. Any further closer spacing is not warranted as inaccuracies are ushered; like are distortions of pens, practical limits to pen registration, and thickness of ink record.

Electrodes: Two types of electrodes are used in recording the scalp potential as of needle and disc type. Needle electrodes are generally used in open brain surgery. Needle electrodes show much larger changes of impedance with frequency in the ranges of EEG waves, than do surface disc electrodes. So needle electrodes are used with equipments having an input resistance more than 1 MΩ.

The potential picked up on the scalp from a certain cortical center of activity decreases roughly with the square of distance. So the electrode activity picked up by each electrode in a unipolar system, and by each pair of electrode in a bipolar system largely represents the cortical activity below or between the electrodes. The electrode spacing is kept as small as possible.

Artefacts: Since every living organ generates electric currents during recording, there are many sources of electric charge in the head besides the brain [25].

(i) Physiological Sources:

(a) Skin – The generation of slowly changing e.m.fs and the change in resistance during emotional stress etc. of skin are certain factors responsible for psychogalvanic reflex. The effect is associated with activity of sympathetic nervous system.

(b) Muscle – The muscle of scalp, jaws, and neck are in constant tonic activity and are therefore continuously generating a large number of short electrical discharges. Although

the duration of these pulses is about 10 m sec, they often merge into a regular rhythm, which can easily be mistaken as brain rhythm. Clenching of jaws and stiffening of neck greatly enhance this activity. In order to eliminate these artefacts, the subject is asked to relax to the maximum, preferably in a supine position.

(c) Eyes - The eye ball contains two separate media; the aequeous and vitreous humors and since ionic concentrations of these are different, the eye ball forms a concentration cell having a potential difference of about 0.1V between cornea (+ve) and orbit. When the eye ball is stationary, steady potential difference over the surface of head is produced. And with the eye-ball movement, potential difference changes and quite characteristic artefact is produced.

(d) Heart - The voltage changes due to the rhythmic electrical activity of the heart are detectable over the whole body. When the EKG is being recorded with a hand or feet of patient connected to earth, the input of amplifier receives the EKG as an inphase signal. Narrower the R wave of EKG, the less prominent the artefact is. Apart from EKG, the pulse in arteries of head sometimes produces an artefact. More transverse the electrical axis of heart, the large the potential in the transverse EKG run. With ventricular-fibrillation, records produce spikes and sharp waves which cannot be recognised as having a cardiac origin without EKG moniter.

(ii) External Sources:

There may be lot many factors which can effect the scalp recordings; e.g. power frequency poisoning of EEG signal, communications line nearby, faulty electrodes etc. So the competent electroencephalographer must start with the ability to distinguish the activity in tracing which is of cerebral origin from that which is due to any of a number of other factors; movement of the head or eyes, movement of extremities, facial muscle contractions, perspiration, faulty electrodes, defects within the apparatus, and broadcast activity from electrical equipments in the vicinity. The age and level of consciousness must also be taken into account.

IV.2 Abnormalities

Weiner (1961) stated that, 'The mathematician need not have the skill to conduct a physiological experiment, but he must have the skill to understand one, to criticise one, and suggest one. The physiologist need not be able to prove a certain mathematical theorem, but he must be able to grasp its physiological significance and to tell mathematician for what he should look'.

In all the earlier chapters, we have discussed the physiological basis of the brain waves, types of the wave etc. which is necessary for a mathematician to understand. To have a pragmatic approach towards brain wave analysis,

a mathematician should also have an idea of what information he will search for in pathological EEG in the process of quantifying the signal. In this section, the author describes some of the abnormal patterns of EEG [9, 14]. The abnormality of these patterns is validated after a careful statistical studies of the number of patients.

When the brain disorder is suspected, the EEG sometimes gives positive evidence of the presence of organic brain disorder. Firstly, sleep patterns are considered of a normal man. In the drowsy state, alpha activity is recorded with decreasing amplitude and intermittent ceasing, and also theta waves are recorded in as sleep advances. In deep sleep, it consists of irregular theta and delta waves over the scalp. During sleep, not only is consciousness lost, certain bodily changes also occur. The pulse rate, blood pressure, and the respiratory rate fall; the eyes usually deviate upwards, the pupils are contracted, but usually react to light slowly. This level of sleep is fairly regularly interrupted by periods of REM (rapid eye movement) sleep lasting from 5 to 30 minutes.

Excessive fast activity is a mild irritative reaction to some type of injury. Diffuse slow waves in waking EEG suggests a diffuse depressive reaction to injury. A localised focus of slow waves suggests a localised depressive reaction to injury such as usually occurs around tumor, infarct or abcess. Extreme slowing, whether diffuse or focal, is commonly associated with clinical evidence of brain damage.

The nature of the injury producing the electroencephalographic abnormality is almost never indicated by the EEG. The EEG does not specify etiology [9]. Abnormalities are much the same whether produce by trauma, vascular disease, infection or neoplastic disease. Slowly progressing atrophy with inflammation, and gradual demyelinating process; e.g. multiple sclerosis are often associated with little or no electroencephlographic abnormality.

Serial electroencephalograms made at weekly or monthly intervals are sometimes of great value, because the nature of the underlying pathological process can to some extent be surmised from the rate at which the abnormality is changing. As a rule, single EEG has little value in determining etiology, but occasionally certain special patterns suggest a vascular lesion,hepatic insufficiency, or subacute encephalitis.

When focal or slow activity is found in a motor or sensory area, it is clinically evident weakness or a sensory defect. A large part of the brain is 'silent', however, it produces no clinical signs or symptoms, if its function is depressed. One of the chief advantages of the EEG is that it shows disorder in these silent areas [9].

A degree of asymmetry between the left and right hal of the brain, eventhough both show normal patterns, is fairly reliable electroencephalographic sign of lateralised injury.

If a structural lesion is present, it is usually on the side of the reduced voltage.

The EEG may be normal, even though the brain is seriously injured. It reveals disorder activity in malfunctioning neurons that, though injured, are still alive. Inactive or dead neurons are not easy to identify in EEG. Purely destructive injuries to the brain often produce surprisingly little electroencephalographic abnormality.

Extreme disorders of neuronal function may occur in the depth of brain e.g. in cerebellum or hind brain, the report will be normal EEG. In these cases waking EEG is ordinarily normal, but certain sleep patterns, particularly 14 per second spindles are usually reduced on the injured side presumably because they originate in thalamus and are poorly conducted to the cortex through the injured internal capsules.

Epilepsy : From an electroencephalographic point of view, the epilepsy is an irritative reaction to injury or to a developmental defect. The clinical evident seizure is the external manifestation of an excessive or deficient release of nervous energy within the brain. If recordings are made on epileptics in the waking state only, not more than 35% show seizure discharges [9]. If patients of epileptic seizures are electroencephalographically observed while asleep, 80 % have seizure discharges. There are two basic types of

epilepsy; grand mal and petitmal (Fig.3.2b). In grand mal epilepsy, there are large discharges lasting from a few seconds to several minutes and usually spreading the whole CNS, including the spinal cord. In petitmal epilepsy, the seizure usually lasts for 1-20 sec. A typical spike and dome pattern recorded during absence type petitmal epilepsy is shown in (Fig.3.2b). Psychomotor epilepsy is characterised electroencephalographically by focal spike discharges in the anterior temporal area. Generalised tonic-clonic convulsions,in grandmal seizures occur in some cases [5].

Patients with following symptoms: attacks of dizziness, pain, emotional stability, and vegetative disturbances usually have 14 or 6 per second positive spikes during light sleep.

Brain injury : Suppression of the normal frequencies, wide spread abnormally slow waves,and outburst of high voltage 2 to 3 per second waves are seen in the acute stage. In chronic post-traumatic state generalised low voltage 2-7 per second waves,sometimes seen in one or both temporal regions, are the rule and disturbance is on the whole proportional to the severity of the injury and persistence of symptoms [9,14].

Hepatic failure: In severe cases delta waves which may be triphasic will be present in EEG. In milder case, there is a slowing of the dominant frequency. The EEG can be used as a sensitive indicator of the response to treatment and diagnosis.

Lipidosis: It is the collection of fats in tissues. Irregular, generalised spike and wave discharges are the characteristics of this disease [14].

Subacute inclusion body encephalitis : It is reduction in intelligence level. The characteristic EEG changes have been described taking the form of complex generalised slow-wave complexes recurring repetitively and often in time with myoclonic jerks and separated by intervals of electrical silence in the record.

Syncope: A brief and transitory loss of consciousness; due to impairment of the cerebral circulation because of which slow waves of high voltage develop in EEG. Concurrently with the loss of consciousness, and this is followed by complete flattening of EEG record, while clonic or tonic convulsions occur.

Brain Tumors: At last, the most important area of EEG is the localisation of tumors in brain. Brain tumors can affect the EEG in two ways. If the tumor displaces the cortex and if it is large enough, the electrical activity will be absent in that part of the hemisphere. An extinguished or damped EEG, or a focus of large delta waves over a certain part of the cortex can thus be a sign of a tumor. It can also provoke an epileptic attack. A tumor involving the basal ganglia may yield 6 percent theta rhythm [9, 16].

On a macroscopic scale, after a careful studies of the EEG pattern and dysfunction of brain chemistry as well, the factors much sought after by a mathematician to help the physiologist in the analysis of brain's coded language are as follows: the amplitude of the signal, frequency, rhythmicity and repetitiveness of certain characteristic pattern, transient activity as spike, asymmetry in two hemispheres etc.

CHAPTER – V

METHODS OF ANALYSIS

The electroencephalogram was considered as the 'window to the brain' but in reality it is more analogous to fleeting shadows cast upon a translucent screen where the speed and size of the shadow may not be commensurate with the actual motion and dimensions of the object. When we speak of analysis of EEG. We do not mean analysis in the same sense as a chemical analysis where the possible components are known; we require a qualitative or quantitative assessment of them; but we mean something which is perhaps better thought of as simply a quantitative description.

The purpose of this section is to describe mathematical basis of several methods to present the rationale for using the parameters derived from these methods of analysis as a basis for comparing their effectiveness in signalling a change in the state based on the subject's EEG. The critical assumption in the methods of analysis is that instantaneous value of exact form of EEG signal is not of direct interest but rather that information about the process lies in certain average properties of the signal.

The pioneer electroencephalographers, in attempting to quantify the EEG, approached it first as a single voltage-time curve - the most striking feature being its frequency. Paul Hoefer et. al (1949) suggested a method for automatic analysis of EEG treating the signal through a battery of tuned filters. So a number of automatic frequency analyser came into being. Reihl, J.L. (1963) gave one method, considering the unit activity as the inverse relationship of frequency and voltage of EEG [20].

In 1951, Krahau, C.E.T. suggested a very fascinating method of analysing the EEG signal using the principles of optics [17]. He recorded the data on a photographic plate and used that as the transmission diffraction grating. Shaw, J.C. (1955) treated the scalp potentials as a result of electric field generated by a number of dipoles inside the head [23]. He suggested that it was from that gradient distribution the quantitative determination of source depth might be made. As a result of all these early explorations it became clear that any analysis of EEG needs to be of its statistical properties.

The assumption underlying the mathematical model and the procedure applied in different methods of analysis are as follows [22].

Assumptions:

(1) The EEG is considered as a short term stationary process i.e. a very small time (10 sec) epoch of analysis, the basic

causing factors for the process are invariant. It does not mean that contiguous 10 sec. epochs will not fluctuate, if the process is stationary for a much longer period, but rather that the nature of these fluctuations from 10 sec epoch to next 10 sec epoch will fall within statistically predictable limits.

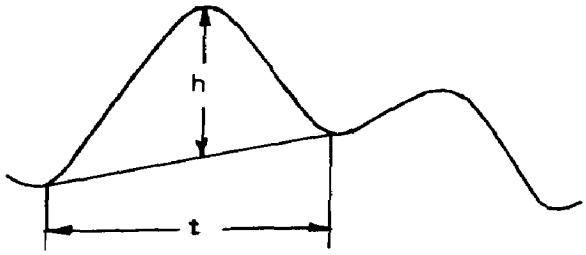
(ii) The changes in state and in the factors underlying the process will persist for periods of time much greater than the epoch of analysis, so that many samples of the derived parameters will be available.

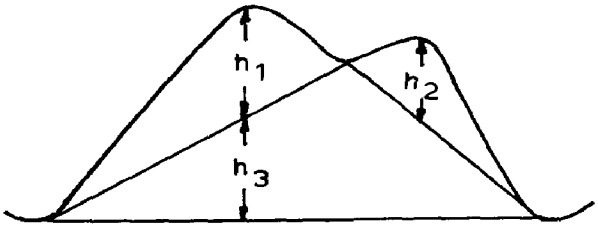
V.1 Histogram Method

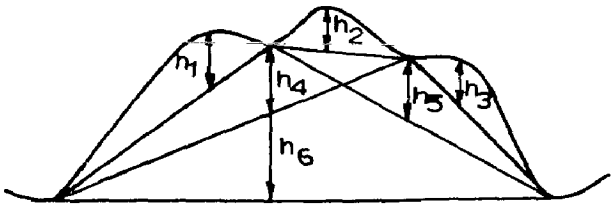
The disadvantage of automatic analysis is the incapability to distinguish between artefacts and original EEG and also the transient characteristics of the analyser.

In making up of histograms, amplitudes and period of waves are measured according to assumed specifications (Fig. 5-1). And the number of waves or the sum of their amplitudes of every 0.5 c/s from 0.5 to 8.0 c/s (δ and θ waves), every 1.0 c/s from 8.0 to 20.0 c/s (α wave), and every 5.0 c/s from 20.0 to 30.0 c/s (β wave) is plotted [8].

In a random signal like EEG, the criteria for making the histograms affects the information content of the signal.







(I) h IS AMPLITUDE AND t IS PERIOD

(II) AS $h_1 > 10\ \mu V$ AND $h_2 < 10\ \mu V$ THIS IS A SINGLE WAVE WITH AN AMPLITUDE OF h_3 (h_3 IS A SLOW WAVE)

(III) $h_1, h_2, h_3 > 10\ \mu V$ $h_4, h_5 < 30\ \mu V$
h_1, h_2, h_3 AND h_6 ARE CHOSEN
(h_4, h_5 AND h_6 ARE SLOW WAVES)

FIG. 5·1

V.2 Fourier Analysis Method

In the frequency analysis by Fourier's series expansion, the electroencephalographic wave is considered as a periodic wave and written as follows[1, 11]:

$$F(t) = \frac{A_0}{2} + \sum_{n=1}^{\infty} (A_n \operatorname{Cos} n\omega t + B_n \operatorname{Sin} n\omega t) \qquad \ldots(V.2.1)$$

where

$$A_n = \frac{2}{T} \int_0^T f(t) \operatorname{Cos} n\omega t \, dt; \text{for } n = 0,1,2, \ldots$$

$$B_n = \frac{2}{T} \int_0^T f(t) \operatorname{Sin} n\omega t \, dt \quad \text{for } n = 0,1,2, \ldots$$

The repetitive waveform has now been broken into a series of component sinusoids whose frequencies are multiples of ω together with constant A_0.

If F(t) is not a periodic signal, and the sufficient condition $\int_{-\infty}^{\infty} |F(t)| \, dt < \infty$ is fulfilled, the Fourier series passes into the Fourier integral $T_0 \to \infty$ as [1]

$$F(t) = \frac{1}{2\pi} \int_{-\infty}^{\infty} F(\omega) e^{i\omega t} \, d\omega \qquad \ldots(V.2.2)$$

where $F(\omega)$ is the Fourier Transform of $F(t)$;

$$F(\omega) = \int_{-\infty}^{\infty} F(t) e^{-i\omega t} \, dt$$

In the case of a stationary signal F(t), the condition $\int_{-\infty}^{\infty} |F(t)| \, dt < \infty$ is fulfilled for the function

$$F_B(t) = F(t) \cdot B(t) \qquad \ldots(V.2.3)$$

with $B(t) = s(t+T) - s(t-T)$

a truncation function (box car function), with $S(x)$ as the step function. And the Fourier Transform of $F_B(t)$ is given as

$$F_B(\omega) = \int_{-T}^{+T} F_B(t)\, e^{-i\omega t}\, dt \qquad \ldots (V.2.4)$$

The Fourier transform is the power spectra of the signal which gives the frequency distribution of the power of the signal. The main feature of the Fourier power spectra is that it is insensitive to phase shifts of input i.e. assuming the signal is periodic, outside the sampled interval, the time origin may be chosen arbitrarily without affecting the Fourier power spectrum. So the Fourier analysis of KEG signal is also not of much interest these days as it treats the wave as periodic and does not give the real time analysis.

V.3 Auto-Correlation, Cross-Correlation, and Coherence Function Analysis

The auto-correlation function for random data describes the dependence of the values of the data at one time on the values at other time. The auto-correlation function is defined mathematically as follows [2]:

$$R_{xx}(\tau) = \lim_{T \to \infty} \frac{1}{T} \int_0^T x(t).\, x(t+\tau)\, dt \quad ; \; 0 < \tau \ll T \qquad \ldots (V.3.1)$$

As a rule of thumb, the maximum τ should not be greater than 10% of T. The one striking property of auto-correlation function is that if the time function contains no periodic component and only random component, then auto-correlation function tends to zero, and the rate at which it tends to zero is a measure of the randomness of the data [2].

The estimated auto-correlation function for discrete data at the displacement rh is defined as [1].

$$R_r = R_{xx}(rh) = \frac{1}{N-r} \sum_{n=1}^{N-r} x_n \cdot x_{n+r} \quad (r = 0,1,2, \ldots m) \qquad \ldots (V.3.2)$$

where

h = sampling interval (N = T/h)

r = lag number

m = maximum lag number

The auto-correlation function can be viewed as a measure of the frequency content of the sample function. The frequency content is given by Fourier Transform of auto-correlation function or power spectral density (PSD) as follows:

$$S_{xx}(f) = 2 \int_{-\infty}^{\infty} R_{xx}(\tau) \exp(-j\pi f\tau)\, d\tau$$

$$= 4 \int_{0}^{\infty} R_{xx}(\tau) \cos(2\pi f\tau)\, d\tau \qquad \ldots (V.3.3)$$

The estimated PSD function for discrete data is given by

$$S_k = 2h\left[R_0 + 2 \sum_{r=1}^{m-1} R_r . \cos\left(\frac{\pi r k}{m}\right) . (-1)^k . R_m \right]$$

$$k = 0, 1, 2, \ldots m. \qquad \ldots (V.3.4)$$

The values of PSD are then smoothed according to the Hanning method as follows: [2]

$$S_0 = 0.5\, S_0 + 0.5\, S_1$$

$$S_k = 0.25\, S_{k-1} + 0.5\, S_k + 0.25\, S_{k+1} ;$$

$$(k = 1, 2, 3, \ldots, m-1)$$

$$S_m = 0.5\, S_{m-1} + 0.5\, S_m$$

The cross-correlation gives the relation between two signal i.e. two channels in case of EEG, as : [2]

$$R_{xy}(\tau) = \lim_{T \to \infty} \frac{1}{T} \int_0^T x(t) . y(t+\tau)\, dt \qquad \ldots (V.3.5)$$

It is a measure of the degree of interdependence between the signals from two or more signals. The Fourier transform of the cross-correlation gives the cross-correlogram, the PSD in frequency domain. This is used to analyse the EEG tracings which reveal waves of apparently the same frequency in different locations of head often having an apparent shift of phase. If the two EEG's are identical in frequency and phase but not truly periodic in character

they give gaussian type of cross-correlation curve around zero lag time. The decay to zero correlation is due to the EEG activity being, although briefly periodic, not continuously so and hence the zero approaching of the curve [3, 4].

Weiner introduced a coherence function similar to correlation for pairs of function possessing generalised harmonic decompositions. Its a measure of the correlation between two signals at frequency ω and is defined as:

$$C^2(\omega) = \frac{|P_{xy}(\omega)|^2}{P_{xx}(\omega) \cdot P_{yy}(\omega)} \qquad \ldots (V.3.6)$$

Where P represents the spectral density of the corresponding signals. It is a measure of how well a particular spectral component in one signal (x) can be estimated by a linear function of spectal component in another signal (y).

The advantage of correlation analysis over filter techniques is that it can detect the periodic signal irrespective of its frequency components. The methods of PSD and frequency correlation plots are very difficult to use in the monitoring of long data runs (e.g., EEG), since they make unusual demands on the perceptiveness, skill, and experience of the evaluator in associating the subtle changes in the function shape with change in state. And also the extent to which the basic assumptions of these methods are met is controvertial [22].

V.4 Convolved Spectra

When the two signal processes are multiplied to produce a single resultant, it is useful for interpretive purposes to note the relationship of the resultant spectrum to the spectra of separate factors [2], e.g. the EEG can be written as:

$$S(t) = S_1(t).\ S_2(t)$$

The Fourier transform is given by

$$g(\omega) = \int_{-\infty}^{\infty} S(t) \exp(-i\omega t)\, dt$$

$$= \int_{-\infty}^{\infty} S_1(t).\ S_2(t) \exp(-i\omega t)\, dt$$

The Fourier transform of $S_1(t)$ and $S_2(t)$ are denoted by $g_1(\omega)$ and $g_2(\omega)$ respectively. Substituting the inverse Fourier transform of g_1 in the expression for $g(\omega)$ -

$$g(\omega) = \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} g_1(\beta) \exp(i\beta t)\, d\beta \times S_2(t) \exp(-i\omega t)\, dt$$

Rearranging:

$$g(\omega) = \int_{-\infty}^{\infty} g_1(\beta) \left(\int_{-\infty}^{\infty} S_2(t) \exp(-i(\omega-\beta)\, t)\, dt \right) d\beta$$

or

$$= \int_{-\infty}^{\infty} g_1(\beta).\ g_2(\omega-\beta)\, d\beta$$

i.e. the amplitude spectrum of resultant signal S(t) is obtained by convolving the amplitude spectra of two factors $S_1(t)$ and $S_2(t)$. Convolution implies spectral spreading, then bandwidth constraints characteristic of EEG data constrains the period analytic coding points to conform to EEG wave shape[22]

V.5 Bispectrum Analysis

The power spectrum gives the complete information about the statistical properties of an EEG sample only under the assumption that underlying process is Gaussian. There may be a phase relationship between different frequency bands although the model of a stationary Gaussian process assumes independence between different frequency bands. This suggests the observation of higher order spectra. So the first is the bispectrum [7].

Analogous to power spectrum $S(\omega)$, which is the Fourier transform of autocovariance;

$R_j = E[X_t, X_{t+j}]$, the bispectrum $B(\omega_1, \omega_2)$ represents the spectral counterpart of second order auto covariance

$R_{j,k} = E[X_t, X_{t+j}, X_{t+k}]$, and is estimated by smoothing the triple product $Y(\omega_1).\ Y(\omega_2).\ Y(\omega_1 + \omega_2)^*$. Therefore the bispectrum may be considered as the spectral decomposition of the third central moment (which determines the skewness of the amplitude distribution). The calculation of bicoherence

$$C(\omega_1,\omega_2) = \frac{B(\omega_1, \omega_2)}{(S(\omega_1).\ S(\omega_2).S(\omega_1+\omega_2))^{1/2}}$$

demonstrates the degree of relationship between different frequency bands within the same EEG. The expected values of bicoherence is zero for a truly random signal and in case of a completely phase locked system, this value is unity. The deviations from the expected value depends upon the factor

such as ensemble size, the length of each epoch, and stationarity of the signal [21].

The computation time needed for the estimation of bispectra is about 20 times longer than for power spectra.

V.6 Period Analysis

In applications, in which the information to be abstracted from long term EEG recordings appear in the form of generalized changes in the complex structure of the signal, a parameter is chosen which effectively monitor or track these changes. The tracking parameters are derived from the EEG's autospectrum, its auto correlation function, and from the average zero crossing rates of EEG and its time derivatives. The parameters represent even ordered moments of Power spectral density and for complex EEG signal, the parameters are equivalent statistical measure of EEG.

The data points in period analysis are selected to correspond to the following event times [21].

(i) the time at which the signal passes through its average value (called primary zero crossing rate).

(ii) The time at which the signal reaches an extremal value (called intermediate zero-crossing points).

(iii) the time at which the signal has an inflexion point (called mirror zero crossing points).

PSD, ACF, and their relation to Period Analysis Parameters

The following equation relates the average rate of zero crossings to the power spectral density (PSD) for a normally distributed mean process

$$\left(\frac{N_k}{2}\right)^2 = \frac{\int_0^\infty f^{2k+2} P(f)\, df}{\int_0^\infty f^{2k} P(f)\, df} \qquad \ldots (V.6.1)$$

where N_k = average rate of zero crossings of the k^{th} derivative of the signal process $S(t)$

$P(f)$ = Power Spectral density (PSD) of the signal $S(t)$

The auto-correlation function (ACF) and (PSD) function are related as follows:

$$\phi(\tau) = \int_{-\infty}^{\infty} P(f) \exp(i2\pi f\tau)\, df$$

$$= 2\int_0^\infty P(f) \cos 2\pi f\tau\, df \qquad \ldots (V.6.2)$$

Since only even moments do not vanish, considering the even ordered derivatives of $\phi(\tau)$

$$\frac{d^{2n}\phi(\tau)}{d\tau^{2n}} = 2\int_0^\infty (-1)^n (2\pi f)^{2n} P(f) \cos 2\pi f\tau\, df$$

setting $\tau = 0$ and rearranging the terms

$$\int_0^\infty f^{2n} P(f)\, df = \frac{(-1)^n}{2(2\pi)^{2n}} \frac{d^{2n}\phi(0)}{d\tau^{2n}} \qquad \ldots (V.6.3)$$

In this way, instead of computing the components of $P(f)$ and then integrating $f^{2n} P(f)$ to derive moments, we can compute the values of $\phi(\tau)$ at $(2n+1)$ increments of lag $\phi(0), \phi(\Delta\tau), \ldots, \phi(2n\Delta\tau)$, and perform simple finite difference operations to compute spectral moments.

We have for the average rate at which $S(t)$ passes through its zero mean value

$$\frac{N_0}{2} = \frac{1}{2\pi\Delta\tau} \left(1 - \frac{\phi(\Delta\tau)}{\phi(0)}\right)^{1/2} \qquad \ldots (V.6.4)$$

Period analysis does not scale signal amplitude. It would be completely unsuitable to represent the waveshape of a signal of unlimited bandwidth by period analytic methods, since there would be no correlation between the time spacing of events and relative signal amplitude. The choice for epoch also is a compromise for obtaining stable short term statistics without smoothing the variations of interest associated with the long term non-stationary effects reflected in EEG record [22].

V.7 Analysis Based on Time-domain Properties

The need for quantitative method in the description of EEG wave has prompted many mathematicians to define suitable parameters. The first step in EEG analysis is to divide the EEG into manageable lengths. These stationary segments of variable lengths give the data reduction and are also described

by the same small number of parameters. These parameters should quantify the EEG characteristics more perfectly.

Hjorth[13], derived these parameters in time domain taking the epochs of same length. However, if the epoch lengths are modified by the segmentation procedure [18], to have one particular non-stationarity in one segment, the same parameters in these new epochs will be more distinguished and more informative. The author applied the same approach towards EEG analysis i.e. segmenting the EEG wave for deciding the unequal epochs for the purpose of time domain analysis.

Hence the problem of EEG wave analysis is divided into two sections viz; (i) segmentation of the EEG trace, (ii) and the description of EEG wave characteristics by minimum number of parameters in each segment. Firstly, the method is discussed at length and then certain EEG traces are analysed numerically based on the present analysis.

V. 7.1 Segmentation of EEG Wave

Bodenstein and Praetorius (1977) used an auto-regressive filter method for boundary detection of the segments [19]. The present method, by Michael and Houchin; 1978, for the segmentation of EEG wave is based on auto-correlation function (ACF) [18]. The basic idea of segmentation is as follows:

The EEG wave is observed, through a moving window with respect to a reference window. The criteria for the window is to reveal even the slowest frequency components i.e. the length of the window (WL) is less than the shortest expected segment but long enough also to satisfy the above criteria. The position of the reference window is fixed at the first position of each scan of the EEG wave. And the test window is moved in steps (e.g. 200 m sec in present analysis) to scan the EEG wave. When the difference between the EEG characteristics seen through the two windows is large enough, a new segment line is drawn. Now a new reference window is placed at the beginning of the next segment and the process is repeated.

V. 7.1(a) Parameter for Non Stationarities

A difference measure between the test and reference is assumed as the linear sum of first order terms of amplitude and frequency change on percentage basis, as the visual inspection of EEG records involves information about amplitude and frequency. The difference equation is as follows:

$$DIFF = \frac{ADIFF}{ATHR} + \frac{FDIFF}{FTHR} \qquad \ldots (V.7.1)$$

Where ADIFF and FDIFF are difference in amplitude and frequency respectively. And also ATHR and FTHR, the threshold values for amplitude and frequency

respectively, are assumed independently but made equal for best results.

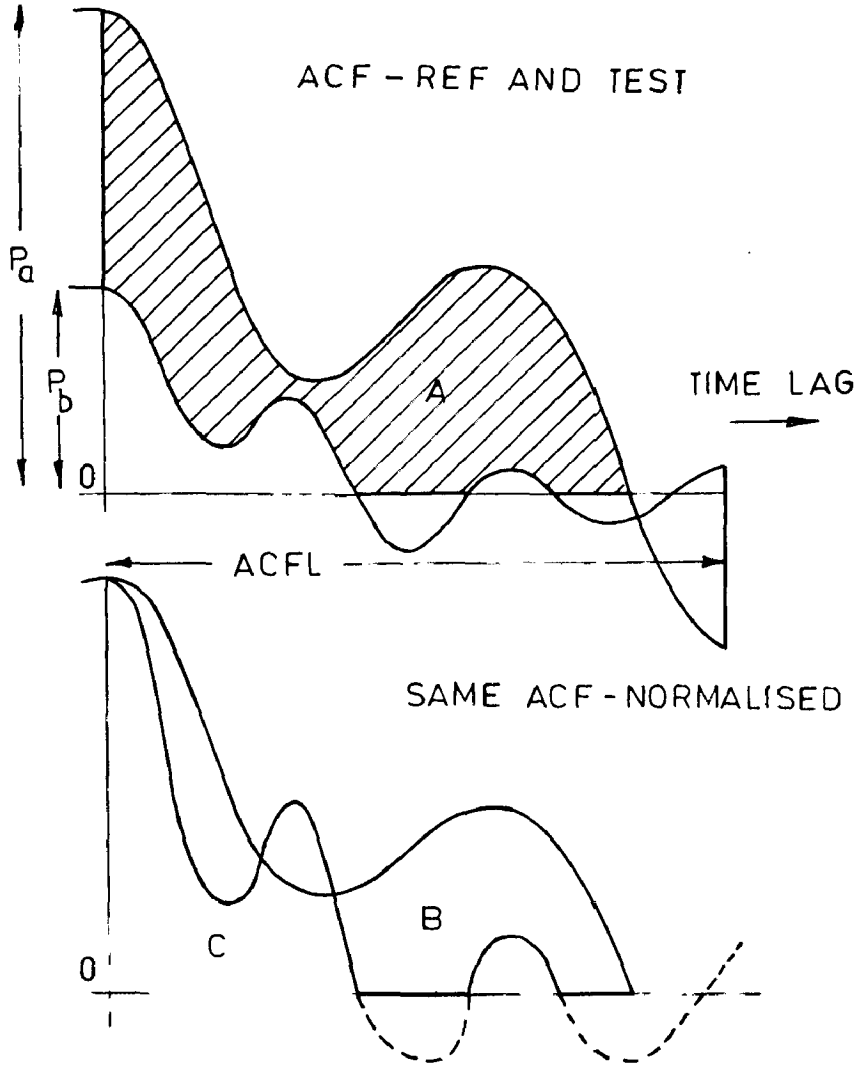
Both amplitude and frequency differences (ADIFF and FDIFF) are calculated from the auto-correlation functions for reference and test windows.

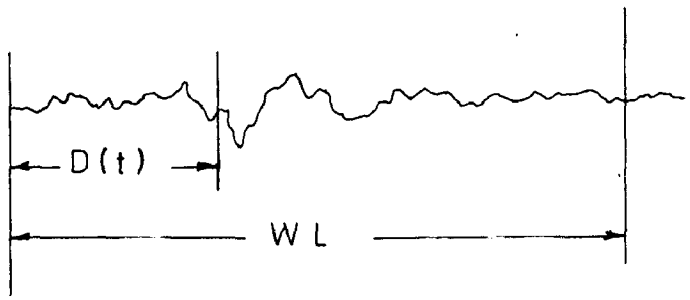
The standard deviation of the signal amplitude equals the square root of the power (P) obtained from the non-normalised auto-correlation function at zero lag. The percentage change in amplitude (ADIFF) is given by the absolute value of the difference between the auto-correlation at zero lag divided by their minimum.

Similarly, FDIFF is the difference between the two frequencies divided by their minimum. For EEG, it measures the overall frequency change. The two auto-correlation functions are normalised and superimposed (Fig.5.2). To obtain this parameter, the area lying between the two normalised auto-correlograms, for a particular length of the curve (ACFL), is divided by the area common to both the curves. Only the positive sections of the auto-correlograms are considered. The length of the auto-correlogram (ACFL) is decided by the slow wave changes consideration and is taken as the length; at least one fourth of the largest cycle length to be considered.

V.7.1(b) Estimating the Boundary Position

The point in time at which any difference measure reaches threshold is taken as the reference point.





$$ADIFF = \frac{\sqrt{P_a} - \sqrt{P_b}}{\sqrt{P_b}}$$

$$FDIFF = B/C$$

FIG. 5·2.

It is displaced from the boundary concerned. Even if there are sharp step like changes in the wave, this displacement D(t) can vary from zero to one window length (Fig.5.2). The author has estimated the boundary as the ratio of two non-normalised auto-correlation function at the zero lag of reference window and the test window giving difference measure as threshold 1.

As the detection of non-stationarity depends upon the reference window position, so a very small delay (e.g. 120 m sec in present analysis) is introduced between the boundary line of the segment and the next reference window position.

V.7.2 Characteristic Analytic Parameters in Time Domain

The statistical methods involving the frequency considerations are used to define descriptive qualities for the general characterization of an amplitude-time pattern of an EEG wave. The parameters, characterising the EEG pattern in terms of amplitude, time scale, and complexity, within each epoch in the time domain are defined as follows [13].

(a) Activity : The activity is quantified by means of the variance of amplitude (or squared standard deviation) $(\sigma_g)^2$, which has the necessary additive property to allow integration of different observations during the epoch into one representative figure.

(b) Mobility : The mobility is defined as the ratio of the standard deviation of the first derivative (i.e. slope) to the standard deviation of the amplitude ($\frac{\sigma_d}{\sigma_a}$). It is expressed as a ratio per time unit and may also be conceived as mean frequency. Since these quantities are equally dependent on the mean amplitude, the ratio will be dependent on the curve shape only in such a way that it measures the relative average slope.

(c) Complexity : This parameter is dimensionless and estimated as the ratio between the mobility of the first derivative of EEG wave and the mobility of the EEG wave itself (i.e. $\frac{\sigma_{dd}/\sigma_d}{\sigma_d/\sigma_a}$). It gives a measure of excessive details with reference to the 'softest' possible curve shape i.e. sine wave, this corresponding to unity. It expresses the number of standard slopes actually generated during the average time required for generation of one standard amplitude as given by the mobility.

V. 7.3 <u>Calculation Procedure</u>

The actual procedure for estimating the parameters for a particular EEG wave is enumerated in the following steps:

1. Segmentation of EEG wave:

   (a) Assume the threshold values for amplitude and frequency (ATHR and FTHR), the window length (WL), step size (ST), the auto-correlation function length (ACFL), and the delay between two segments (D).

(b) Digitise the wave shape at fixed interval (H), and then calculate the auto-correlation functions to have auto-correlograms for assumed ACFL from reference and test window data points. And also calculate the normalised values for the above ACFL.

(c) ADIFF is calculated from the non-normalised auto-correlation functions at zero lag for reference and test windows (Fig.5.2).

(d) FDIFF is calculated from normalised auto-correlograms bounded by ACFL (assumed). To calculate the area (as illustrated in the Fig.5.2) bounded by the two curves, the author has used the trapezoidal rule and for simplicity and brevity the auto-correlograms are assumed to be formed by straight lines between two points and the curves are not a smoothed one as the calculation needs only the ratio of the two areas.

(e) Next, the DIFF values are calculated from the equation (V.7.1),

$$DIFF = \frac{ADIFF}{ATHR} + \frac{FDIFF}{FTHR}$$

If the values from the above equation reaches the threshold value 1, then we move to the next step (f) for estimating the boundary, otherwise we repeat the above steps for the test window data points after moving the window decided by the stepsize (ST) assumed

(f) The boundary is estimated by calculating the parameter D(t) as the ratio of the auto-correlation function at zero lag for reference and the test window decided by the step (e).

(g) The next reference window is placed after the assumed delay (D) and the whole procedure is repeated to decide the other segments.

2. Calculation of Parameters

(a) The data points for one segment are taken and the first derivative at each point is calculated from the differentiation formula applied for tabulated values given in the Appendix A.

(b) Similarly the second derivative is calculated at each point of the segment from the earlier calculated values of the first derivative of the function.

(c) Next the standard deviation of all the values for one segment is calculated for amplitude (σ_x), the first derivative ($\sigma_{\dot{x}}$), and the second derivative ($\sigma_{\ddot{x}}$) of the data points. Then the three parameters e.g. activity (σ_x^2), mobility ($\sigma_{\dot{x}}/\sigma_x$), and complexity $\left(\frac{\sigma_{\ddot{x}}/\sigma_{\dot{x}}}{\sigma_{\dot{x}}/\sigma_x}\right)$ are calculated.

(d) The same procedure is repeated for other segments also.

V.8 Results and Discussion

The method described in the section V.7 offers a way of quantifying the general characteristics of an EEG trace. The author has derived the three parameters (e.g. activity, mobility, and complexity) for EEG waves in different conditions of abnormality as slow waves focus, abnormal very slow, and abnormal moderately slow wave.

To characterise the grand mal and petit mal type of epilepsy, the specific pattern in each case for one channel is analysed by the author. A case of asymmetry in occipital area of two hemispheres is also analysed.

The EEG wave patterns for all cases, shown in Fig.5.3(a,b), are taken from the book [9, 10]. The analysis can be extended to other channels of the EEG recordings as well. The segmentation of each wave is depicted by the arrows in the figure. In all these individual segments, the three parameters (σ_a^2, σ_d/σ_a, $\sigma_{dd}/\sigma_d/\sigma_d/\sigma_a$) defined earlier are enumerated and tabulated in the Tables (V.1) to (V.4).

Data for these tables are given in Appendix B.

The values of the parameters listed in the above tables are calculated manually. The data points are also limited in number as the EEG wave taken in each case is very small. The segmentation procedure gives the error

below 20% WL [18], and detection of non-stationarity also depends on the position of the reference window of the seam.

The values from the first row of the Tables (V.1), and (V.2) show that in a particular segment (III), the activity is increased and the mobility is reduced appreciably as compared to the mobility in other segments. It means a slow wave is recorded in that particular segment, which can said to be focussed. This fact is clinically corroborated as the pathological wave is for slow wave focus, which suggests a localised depressive reaction to injury such as usually occurs around tumor, infarct, or abcess [9].

The second and third rowsof the Tables (V.1) to (V.3) compares the activity, mobility,and complexity in case of diffused slow activity of the electroencephalogram. It is perspicuous that activity and mobility in abnormal very slow is less than the abnormal moderately slow EEG wave. And the complexity is larger in the first case.

In petit mal epilepsy the seizure usually lasts for a very short duration. This is corroborated by the fact that activity is increased in (III and IV) segments - Table (V.1), alongwith very high mobility in (III) segment - Table (V.2), in comparison to the activity and mobility in other contiguous segments. In this case the wave becomes less complex conforming to the softest curve.

Whereas in Grandmal, the activity is on the higher side and the mobility is also varying from low to higher values appreciably. In this way all the above pathological conditions are corroborated by the mathematical analysis.

In Table (V.4), the parameter for left and right occipital area of the brain are compared. The activity and mobility in right occipital area is less than the left occipital area in all segments except the segment IV - Table (V.4). The complexity of EEG wave of right hemisphere is more excepting the segment IV. It means there is a asymmetry between right and left occipital area of the brain, which corroborates the fact that EEG waves were specified for voltage amplitude asymmetry in the right occipital area. The analysis correctly specify the asymmetry between two hemispheres which may be helpful in indicating the lateralized brain injury, even though both hemispheres show normal pattern.

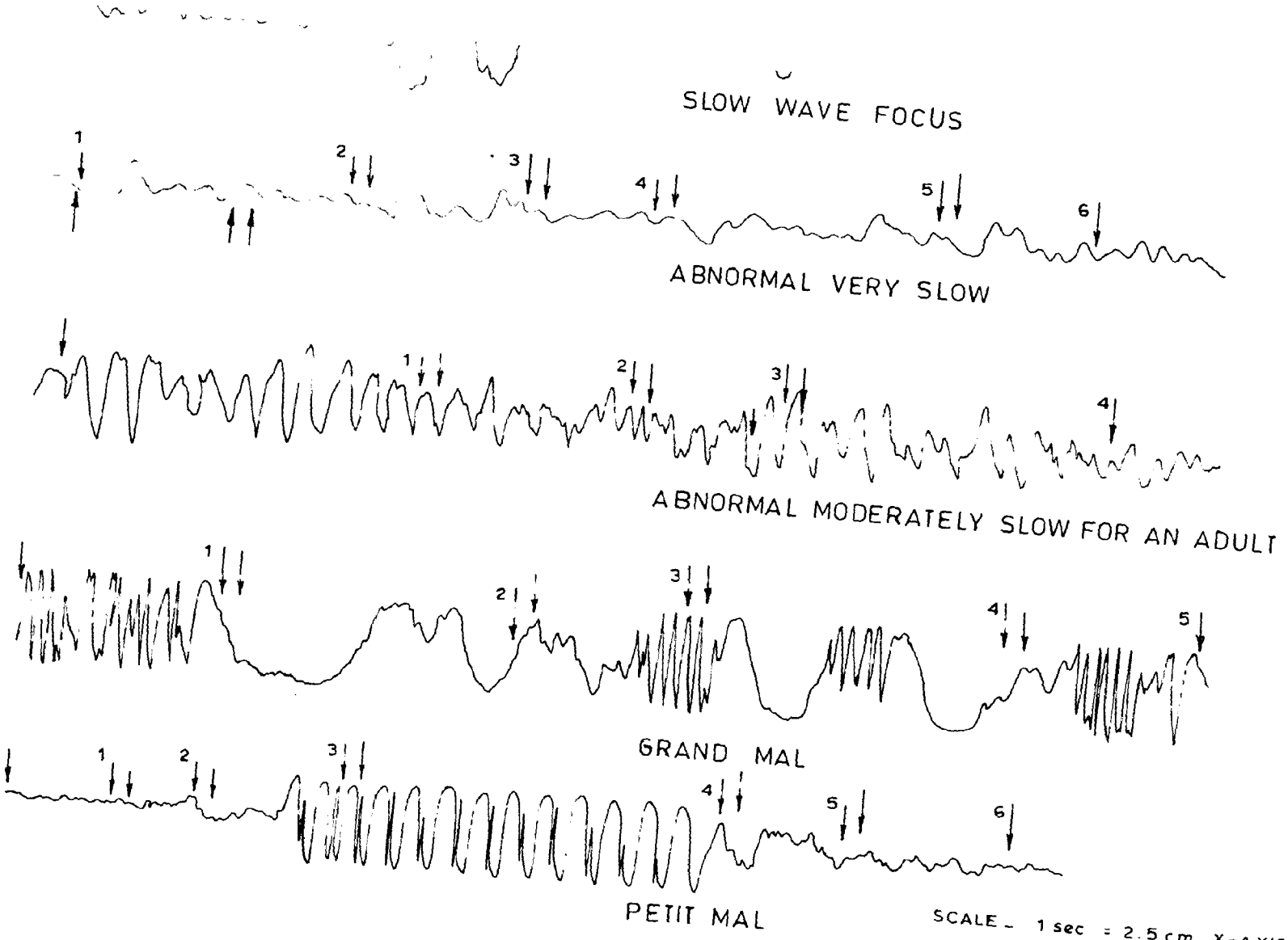
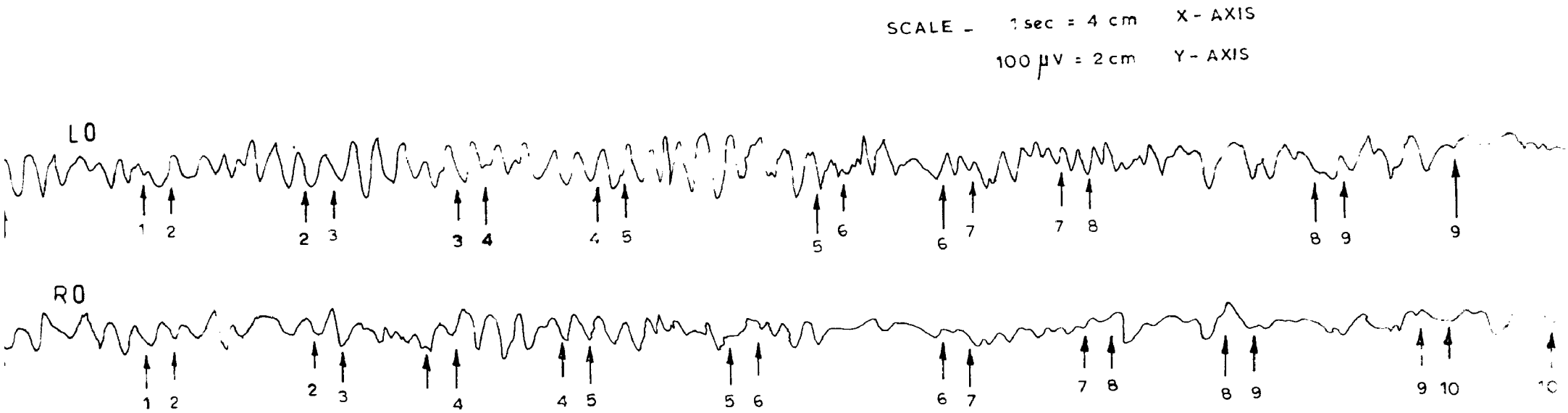

SLOW WAVE FOCUS
1
2
3
4
5
6
ABNORMAL VERY SLOW
1
2
3
4
ABNORMAL MODERATELY SLOW FOR AN ADULT
1
2
3
4
5
GRAND MAL
1
2
3
4
5
6
PETIT MAL
SCALE - 1 sec = 2.5 cm X-AXIS
100 μV = 1 cm Y-AXIS

FIG. 5.3 a



VOLTAGE AMPLITUDE ASYMMETRY

FIG. 5.3b

TABLE – V.1

Activity (σ_a^2) in various EEG waves; Units : $(\mu V)^2$

Types of Wave	SEGMENTS					
	I	II	III	IV	V	VI
Slow wave focus	108.3333	80.5555	3451.0941	299.6017	328.2222	216.6091
Abnormal very slow	185.4019	78.3950	247.0344	68.6874	237.0155	315.9999
Abnormal moderately slow	1273.7772	943.9446	1069.2707	864.5061	–	–
Grandmal	3391.3494	2900.2495	1761.1796	5836.788	2662.4349	–
Petit mal	22.9166	89.4097	1486.6729	3729.7085	570.7756	67.4556

TABLE – V.2

Mobility ($\frac{\sigma_d}{\sigma_a}$) in various EEG waves Units : $\frac{1}{\text{sec}}$ (=mm)

Types of Wave	SEGMENTS					
	I	II	III	IV	V	VI
Slow wave focus	1.1700	.8200	.5215	.5965	.6501	.6800
Abnormal very slow	.6913	.8968	.6606	1.0453	.5541	.6668
Abnormal moderately slow	.9256	.9723	.9708	.8646	–	–
Grandmal	.7228	.2965	.9836	.7258	.5787	–
Petit mal	1.1824	.8101	1.5173	.7983	.5062	.8648

TABLE – V.3

Complexity ($\frac{\sigma_{dd} / \sigma_d}{\sigma_d / \sigma_s}$) in various EEG waves

Types of Waves	SEGMENTS					
	I	II	III	IV	V	VI
Slow wave focus	1.1180	1.0677	1.4114	1.9228	1.5446	1.3278
Abnormal very slow	1.1285	1.0859	1.4136	.7336	1.3757	1.1696
Abnormal moderately slow	1.0324	.9793	1.3427	1.1494	–	–
Grandmal	1.6435	2.5961	1.6679	1.3850	1.4394	1.4870
Petit mal	1.1865	1.4489	.9011	1.0848	1.5994	

TABLE – V.4

Parameters for asymmetry in two hemispheres

Segment No.	Activity σ_s^2 $(\mu V)^2$		Mobility $\frac{\sigma_d}{\sigma_d}$ $\frac{1}{\text{sec}(=\text{HZ})}$		Complexity $\frac{\sigma_{dd}/\sigma_d}{\sigma_d/\sigma_s}$	
	LO	RO	LO	RO	LO	RO
I	122.9659	83.2840	1.0388	0.9942	1.0587	1.1263
II	116.5599	82.8402	1.0115	.8971	1.0826	1.1624
III	178.0718	43.3593	1.0537	.8151	1.0624	1.0666
IV	107.1428	182.1874	.9592	.9843	1.1687	.8860
V	204.7755	61.3905	1.0126	.7637	1.0921	1.2514
VI	112.4999	34.8832	.8229	.7438	1.3536	1.2351
VII	89.6193	22.9024	1.0977	.6739	1.0640	1.2357
VIII	107.7929	48.2993	.7005	.9119	1.1799	.7829
IX	104.5351	34.0270	.7654	.5453	1.2160	1.6916
X	–	44.7368	–	.8766	–	1.7552

Note : LO = Left Occipital; RO = Right Occipital

CHAPTER – VI

CONCLUSION

This dissertation has been concerned with the extraction of elementary features from EEG records, and the desired end result is a medical diagnosis. It should be mentioned at this point of time that electro-encephalogram does not specify etiology. Though the tracings of repeated measurements in the same biological state need not be the same, however they contain something in common which might be characteristic for the state of investigation. So serial electroencephalograms made at weekly or monthly intervals are of great value to specify the etiology, because the nature of the underlying pathological process can to some extent be surmised from the rate at which the abnormality is changing.

Here, only one channel is used for the analysis purpose. However, in routine clinical recordings, signals are derived from at least 8, typically 12, frequently even 16 leads simultaneously, where the electrodes are distributed over the scalp of the patient according to a fixed pattern. We can apply the same procedure of analysis on all channels in parallel. A computer program can be made and results can be obtained by feeding the data directly to the computer. The rapidly increasing availability of low cost

fast acting digital lab-computers equipped with necessary converters for analog input and output will allow the physician to compare the activity and mobility in different parts of the brain, especially in contralateral sites, and in particular the focal distribution of paroxysmal activity is of utmost importance for the diagnosis of tumors and local lesions. The complexity of the EEG wave does not give any concrete information with respect to abnormalities of the brain, at present.

A further scope of this study may be in the field of the geniuses, and the personality of man. Because a healthy brain probably guards its secrets jealously. So the secrets of the brain of genii should be explored assidiously. Nevertheless, in the present circumstances, most facets of the brain are better described by the novelist than the mathematician.

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APPENDIX A

Derivative of Tabulated Values:

Suppose f_0, f_1, f_2 ... f_{i-1}, f_i, f_{i+1}, ... are the tabulated values of the function at interval of h. The derivative at each tabulated points, also the end points, is calculated according to the differentiation formula,

$$f_i' = \frac{-f_{i-1} + f_{i+1}}{2h} \quad \text{(equation 3.2.11 of Ref.)}$$

The error term in this expression is $(-\frac{1}{6} h^2 f''')$. The derivative at the initial point is calculated as follows (equation 3.2.8 of Ref.),

$$f_0' = \frac{-3f_0 + 4f_1 - f_2}{2h}$$

While the derivative at the last point is derived by its companion slanted in opposite direction and is given by the equation,

$$f_n' = \frac{f_{n-2} - 4f_{n-1} + 3f_n}{2h}$$

The error term is $\pm \frac{1}{3} h^2 f'''$ in the above case.
The second order derivative can also be derived from the tabulated values for the first derivative in the manner analogous to the first derivative.

(Ref: McCormick, John M., and Mari G. Salvadori: "Numerical Methods in FORTRAN", Prentice Hall of India Pvt. Ltd.,1968)

APPENDIX B

B:1 Data for Tables (V.1), (V.2), and (V.3)

Time interval between two samples (H) = 40 m sec.

X - axis 1 sec = 2.5 cm

Y - axis 100 μV = 1 cm ... Fig. 5.3a

For Segmentation:

ATHR = 50% ; PTHR = 40%; AOFL = 160 m sec.;

Step Size (ST) = 200 m sec.; WL = 1.0 sec.;

Delay between two segments (D) = 120 m sec.

B:1.1 Slow wave focus (SF)

Segments

I +10, 0, 0, 0, -10, -20, -30, -10,
-20, 0, +10, 0, -20, 0, 0, -5, 0,
+5, 0, -20, 0, 0, -10, -10, -10,
+10, -10, +10, -10, -15, +15, -10.

II -10, -15, -5, +10, -10, +15, 0, -10,
-15, 0, 0, 0.

III 0, +40, +30, +30, +50, +70, +50, +20,
-20, -40, +25, -70, -100, -110, -100, -100,
-30, +30, +70, +80, +95, +20, +10, -20,
-60, -70, -60, -80, -90, -75, -40, -10,
+20, +50, +60, +50, +55, 0.

IV 0, -10, -15, -20, -5, +5, +10, +10, 0, +10,
+20, +20, +10, +30, +10, 0, +30, +30, +30,
+40, +20, +10, -20, 0, 0, 0, 0, -10, -20,
-10, +10, +10, -10, 0, 0, -10, -40, -30.

Segments

V
+20, +30, +20, +40, +40, +30, +30, +10, +10,
-10, - 5, +10, -20, -10.

VI
0, 0, +10, +10, -10, 0, +10, 0, 0, 0,
+10, + 5, 0, -10, +10, 10, +10, +20, +40,
+20, +30, +40, +40, 0, -10, +10, +10, -10,
-20, +10, 0, -30, +10, +10, -10, +10, +10,
+10, +20.

B:1.2 Abnormal very slow (LP)

Segments

I
-5, -15, -20, -15, -15, -10, -10, -10, +10,
+40, +30, +10, 0, -10, -5, +0, +5, +10,
-15, 0, 0, 0, +10, +10, -10, -10, 0.

II
+20, +15, 0, -5, 0, +20, 0, 0, -10,
-10, 0, -5, +5, +5, 0, 0, 0, +15

III
0, -10, 0, -5, -15, 0, +25, +30, +20,
+20, +15, -10, -10, -10, -10, 0, +10, 0,
0, -10, -20, -20, +5, +35, +30, +15, +20,
+20.

IV
+10, 0, -10, -5, 0, 0, +5, 0, 0,
0, +5, +10, +20, +10, 0, +5, +15, +20,
+20, 0.

V
+10, +15, +10, +5, 0, -15, -25, -30, -20,
0, +20, +10, +5, +25, +30, +30, +25, +10,
+5, +10, +10, +10, +20, 0, +5, 0, -10,
-10, 0, 0, 0, +5, -5, 0, +25, +45,
+40, +30, +20.

VI
+15, 0, -10, -15, -10, -10, +25, +50, +35,
+25, +25, +40, +30, 0, 0, +5, 0, -10,
- 0, 0, +10, 0, +25, +15, 0.

B:1.3 Abnormal moderately slow (LP)

Segments

I
+10, -10, -20, +10, +50, +20, -50, -100, -10,
+50, +40, +40, -30, -100, -10, +60, +40, +50,
-10, -20, +10, +30, -10, -40, -30, +20, +40,
0, -20, -30, -30, +20, +40, -10, -70, +10,
+40, +70, 0, -80, -20, +60, +100, -10, -30,
-60, +10, +30, +50, -20, -10, -50, +30, +60,
-30, -40, 0, +50, +40, 0, -40, +10.

Segments

II
-20, -50, 0, +20, +40, +10, -10, -30, +10,
+70, -30, -60, -20, +20, 0, +30, 0, -30,
-10, +20, -10, 0, -10, -50, -10, -20, +20,
+30, 0, +50, +70, 0, -10, +40.

III
-30, -10, +10, -10, +20, -50, -30, -50, 0,
+10, -30, -50, -20, +20, +10, 0, +30, -30,
-80, 0, +60, +40, -50, -30.

IV
0, -10, -60, -30, +30, +20, +10, -30, -10,
+30, +60, 0, -60, 0, +60, +40, -10, -20,
-20, -10, -40, 0, +30, -10, -10, +20, -20,
-40, -10, -10, +20, +70, +30, +20, -40, +20,
+30, -30, -50, +10, +40, +30, 0, +20, -30,
-10, +40, 0, +10, -10, +10, -20, -30, 0

B:1.4 Grandmal (LF)

Segments

I
+70 +50 -80, +80, -70, +80, -40, +30, 0,
-30, +20, +90, -10, -60, +40, +90, +10, -70,
+50, -10, -70, +90, -70, +20, +70, -40, +90,
-20, -60, +50, +90, +90, +60, +40.

II
-20, -50, -40, -50, -60, -65, -65, -60, -60,
-50, -60, -70, -70, -75, -75, -70, -70, -70,
-60, -50, -35, -10, +10, +10, +50, +70, +75,
+80, +75, +80, +60, +45, +30, +10, +30, +60,
+55, +80, +60, +30, -20, -40, -50, -60, -60,
-50, -40, -20.

III
+60, +80, +40, +30, +10, +50, +20, +40, +20,
-5, -30, -55, -55, 0, -10, -10, +10, -20,
+60, -20, +20, -60, +50, -50, +90, -30, +60.

IV
+90, -70, -20, +40, +60, +95, +95, +95, +60,
+20, -10, -30, -60, -55, -65, -70, -70, -70,
-70, -40, -10, +20, +60, +95, -30 +90, +10,
-95, +10, -95, +30, +70, +90, +80, +70, +70,
+50, +30, -20, -50, -70, -70, -70, -70, -70,
-70, -65, -40, -15, -30, -5.

V
+50, +45, +30, +55, +35, +10, +20, +50, +70,
+50, +95, -20, +80, -50, +100, -20, -60, +100,
-60, +100, -60, +50, +20, +60, +40, +70, +90,
-60, +50, +90, +90.

B: 1.5 Pelit mal (LF)

Segments

I
0, +10, 0, 0, -10, 0, 0, 0, -10,
0, 0, 0, 0, 0, -10, +5, 0, 0,

II
0, -10, 0, 0, +10, +10, 0, 0, 0,
+10, +20, +25.

III
-20, -10, -10, -15, 0, -10, 0, 0, 0,
0, -10, -10, +40, +30, +70, +10, +40, -80,
+40, +60, -50, +60, -80.

IV
-70, -80, -50, +60, +65, -30, +65, -80, +50,
+70, +60, -50, -70, -30, +60, +70, +50, -70,
-85, 0, +60, +65, -20, +60, -80, +50, +70,
+60, -50, -80, -80, +65, +70, -30, +70, -80,
+40, +60, +65, +20, -80, -80, +50, +65, +60,
-60, -80, -80, +50, +70, +60, -50, -80, -30,
+50, +65, +30, -50, -80, -40, 0, +40.

V
-10, -20, -30, -30, +10, +40, +30, +40, +30,
+40, +20, +20, +20, +40, 0, +15, -5, -10,
-20.

VI
0, 0, +15, -5, 0, -10, -20, -10, 0,
0, +10, 0, -20, 0, -10, 0, +10, 0,
0, -10, -10, 0, 0, 0, +10, 0.

B: 2 Data for Table (V.4)

X - axis 1 sec = 4 cm
Y - axis 100 μV = 2 cm

... Fig. 5.3b

Time interval between two samples (H) = 25 m sec.

For Segmentation:

ATHR = 40%; PTHR = 40%; ACFL = 100 m sec.;

WL = 0.5 sec; Step size (ST) = 125 m sec.;

Delay between two segments (D) = 125 m sec.

B: 2.1 Left Occipital (LO)

Segments

I
+5, +10, -5, -25, -10, +15, +10, -20, -10,
+10, +15, -15, -5, +5, +10, +5, 0, +5,
0, -20, -10, +5, -5, -15, +5, -5.

Segments

II	+5,	+10,	-5,	-10,	-10,	-5,	+10,	+5,	-5,
	0,	+10,	-10,	+5,	0,	+15,	+25,	-15,	0,
	+10,	+20,	-10,	-20,	0,	+10,	-5.		
III	0,	-10,	-15,	+5,	+20,	-15,	-30,	-5,	+20,
	-10,	-15,	-20,	+10,	+15,	-10,	-15,	-5,	0,
	-20,	-15,	-5,	+15,	0.				
IV	-5,	-5,	0,	+10,	-10,	-5,	0,	+15,	+10,
	-15,	-15,	-10,	0,	+5,	-20,	-25,	-5,	+5,
	-5,	-20,	-10.						
V	-10,	+10,	-10,	-20,	-10,	+5,	-25,	-15,	-25,
	-5,	+15,	-10,	-30,	-5,	+15,	+5,	-30,	-35,
	-10,	+15,	0,	-20,	-10,	+10,	+15,	0,	-15,
	-15,	-5,	+5,	-5,	-35,	-20,	0,	0.	
VI	-15,	-20,	-10,	-5,	+15,	-10,	-10,	+5,	+10,
	-25,	-15,	-20,	-25,	-15,	0,	-20,	-10,	-5,
	-15,	-10.							
VI	-15,	-5,	-25,	-30,	0,	-5,	-5,	+5,	-10,
	0,	-10,	0,	-5,	-20,	-5,	-15,	-20.	
VIII	-20,	-15,	-20,	-15,	-10,	-15,	0,	-10,	-25,
	-10,	-10,	0,	-5,	-10,	-5,	-25,	-40,	-30,
	-20,	-10,	0,	-5,	0,	-10,	-30,	-20,	-15,
	-30,	-25,	-10,	0,	-10,	-15,	-10,	-15,	-25,
	-30,	-25,	-30,	-25,	-15.				
IX	0,	-10,	-10,	-15,	-35,	-25,	-15,	0,	+5,
	-10,	-25,	-20,	-15,	-5,	-5,	-10,	0,	0,
	+5,	-5,	-5.						

B: 2.2 Right Occipital (RO)

Segments

I	+5,	-5,	+10,	+15,	+10,	+5,	-5,	+25,	+20,
	+15,	+10,	+5,	+5,	+10,	+25,	+20,	+10,	-5,
	+10,	+20,	+10,	-10,	0,	+15,	+10,	0,	
II	0,	+10,	+15,	+10,	0,	+5,	+15,	0,	-10,
	-5,	-10,	+15,	0,	+5,	+15,	+20,	+20,	+20,
	+20,	+15,	+5,	0,	+10,	+10,	+20,	+5.	
III	-5,	0,	+5,	+15,	+10,	+5,	+5,	-5,	+10,
	0,	+5,	0,	-5,	+5,	-10,	-5.		

Segments									
IV	0,	+15,	+25,	+15,	-10,	-5,	+30,	+25,	+5,
	-20,	-5,	+20,	+15,	-15,	-5,	+5,	+5,	+15,
	+10,	0.							
V	-5,	0,	+10,	+15,	0,	-10,	0,	+10,	0,
	-15,	0,	+10,	0,	+15,	0,	+5,	+5,	+5,
	-5,	0,	0,	-5,	+5,	-15,	-10,	-5.	
VI	+5,	0,	+5,	0,	-5,	+5,	-5,	+5,	-5,
	+5,	0,	-15,	-10,	0,	0,	0,	0,	0,
	0,	+5,	+5,	+10,	+5,	-5,	0,	-5,	-5,
	-10,	-5,	+5,	-5,	0,	-5,	-10.		
VII	-5,	-10,	-15,	-10,	0,	-5,	0,	-10,	-5,
	0,	-5,	- 5,	-10,	-5,	0,	-5,	0,	+5,
	0,	0,	0.						
VIII	-10,	0,	0,	-5,	0,	+5,	+5,	+5,	0,
	-5,	0,	0,	-15,	-10,	0,	+5,	+15,	+10,
	+5,	0,	-10.						
IX	0,	0,	+5,	0,	0,	+5,	-5,	-5,	-10,
	-15,	-5,	-10,	-15,	-5,	0,	0,	-5,	-10,
	-10,	-10,	-15,	-10,	-5,	+5,	0,	0,	+5,
	0,	-5,	-5,	-5.					
X	0,	0,	0,	-10,	-20,	-15,	-10,	0,	0,
	+5,	0,	0,	0,	-5,	-10,	0,	-10,	-15,
	-5.								