DISSERTATION ON

A STUDY ON CLINICAL PROFILE OF NON TRAUMATIC COMA PATIENTS & CORRELATION OF THEIR PROGNOSIS BASED ON ELECTROENCEPHALOGRAM

DISSERTATION SUBMITTED TO

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

IN PARTIAL FULFILMENT OF THE REGULATIONS FOR THE AWARD OF THE DEGREE OF

M.D. - GENERAL MEDICINE- BRANCH – I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004.

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APRIL - 2015

CERTIFICATE

This is to certify that this dissertation entitled

"**A STUDY ON CLINICAL PROFILE OF NON TRAUMATIC COMA PATIENTS & CORRELATION OF THEIR PROGNOSIS BASED ON ELECTROENCEPHALOGRAM.**"

is the bonafide original work of **Dr.SURIYAKANTH.S** in partial fulfilment of the requirements for M.D Branch -I (General Medicine) Examination of the TamilnaduDr. M.G.R. Medical University to be held in APRIL - 2015. The period of study was from October– 2011 - November 2012.

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DECLARATION

I, **Dr.SURIYAKANTH.S** , solemnly declare that the dissertation titled DISSERTATION ON **"A STUDY ON CLINICAL PROFILE OF NON TRAUMATIC COMA PATIENTS & CORRELATION OF THEIR PROGNOSIS BASED ON ELECTROENCEPHALOGRAM"** is a bonafideworkdone by me at Thanjavur Medical College, Thanjavur during Janauary 2014 – september 2014 under the guidance and supervision of **Prof.Dr.C.GANESAN, M.D.,** Unit Chief M-4, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to TamilnaduDr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.D. degree (Branch -I) in General Medicine**.

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ACKNOWLEDGEMENT

I gratefully acknowledge and my sincere thanks to the Dean, **PROF.DR.MAHADEVAN,MS** Thanjavur Medical College, Thanjavur, for allowing me to do this dissertation and utilize the institutional facilities.

I am extremely grateful to **PROF.DR.P.G.SANKARANARAYANAN .,**Head of the Department, Department of Internal Medicine, Thanjavur Medical College, for his full-fledged support throughout my study and valuable suggestions and guidance during my study and my post graduate period.

I am greatly indebted to **PROF.DR.C.GANESAN MD** my Professor and Unit Chief, who is my guide in this study, for his timely suggestions, constant encouragement and scholarly guidance in my study and post graduate period.

I Profoundly thank my professors **PROF.DR.K.NAGARAJAN,M.D PROF.DR.S.MANOHARAN,M.D;PROF.DR.D.NEHRU,DMRD,M..D DR.GUNASEKARAN,M.D D.M.,** (Registrar), for their advice, guidance and valuable criticism which enabled me to do this work effectively.

I would like to express my gratitude to **PROF.DR.M.THANGARAJ MD DM** Department of Neurology for his immense help in the study which enabled me to complete this work and also permitting me to utilize the facilities available in the department.

My sincere thanks to assistant professors **DR.C.PARANTHAKAN MD Dr.GOWTHAMAN.G,M.D.,**; **DR.RAVICHANDRAN.V,MD & DR.A.MAGESH M.D** for their motivation, encouragement and support.

I am also thankful to **DR.BALAMURALI, MD, DM** & **DR.ELANGOVAN MD DM** for spending their valuable time in giving EEG reports for my study.

I also want to express my thanks to all the EEG technician without whom its difficult to complete the task.

A special mention of thanks to all the patients who participated in this study for their kind cooperation without whom the study would not be possible.

I would like to thank my colleagues and friends who have been a constant source of encouragement..

CONTENTS

A STUDY ON CLINICAL PROFILE OF NON TRAUMATIC COMA PATIENTS & CORRELATION OF THEIR PROGNOSIS BASED ON ELECTROENCEPHALOGRAM

ABSTRACT

BACK GROUND:

Coma is an eye closed state of unresponsiveness with severely impaired arousal & cognition. EEG plays a role in establishing the prognosis of the disease states that are capable of causing neuronal death which is frequently underused in acute setting.

AIMS & OBJECTIVES:

To assess the clinical profile of non traumatic coma patients and their EEG patterns & correlate it with outcome of the patient.

MATERIALS AND METHODOLOGY:

Detailed clinical examination & EEG was taken to 50 patients who satisfy our inclusion & exclusion criteria at admission in TMCH casualty . Results analyzed and statistical analysis done by SPSS software .

RESULTS:

Most common etiology of coma in our study is CVA (38 %) followed by metabolic coma. Mortality rate is high in coma secondary to CVA (40.9%) and HIE (23%) where as less in metabolic coma. Alpha coma is the most common EEG pattern (72%) encountered in our study. Other coma like theta and delta are also seen but not specific to particular etiology.

CONCLUSION:

Apart from late hospitalization, initial poor GCS , presence of neuro - ophthalmological signs; poor outcome can be also predicted by **theta and delta coma which has statistically significant high mortality(53.6%).**

Yet it is solely difficult to prognosticate the patient based on EEG alone ,it can be used as a adjunct in many conditions.

KEY WORDS: ELECTROENCEPHALOGRAM , ALPHA COMA,THETA COMA,DELTA COMA.

A STUDY ON CLINICAL PROFILE OF NON TRAUMATIC COMA PATIENTS AND CORRELATION OF THEIR PROGNOSIS BASED ON ELECTROENCEPHALOGRAM

INTRODUCTION

The easier and simplest way of defining consciousness is, the state of the patient's awareness of self and environment and his responsiveness to external stimulation and inner needs [1].

Therefore, unconsciousness exactly has the opposite meaning, that is, state of unawareness about self and environment or a suspension of those mental activities which make them aware of their environment [1], coupled with a diminished responsiveness to environmental stimuli.

Coma as such has plenty of causes including stroke, metabolic encephalopathies, drug intoxication still more, which affects different functions of the brain resulting in varied presentations. Common etiologies accounting for stroke varies in different part of the world, based on their traditional risk factors. Yet it is a prime duty of the physician in the casualty to clinically identify the cause and further evaluate accordingly as soon as possible. Every coma is a Medical emergency.

The famous quotation TIME IS BRAIN [2] applies not only for stroke but for every other acute brain insult irrespective of the etiology. Because longer the

time the brain is in failure the risk of getting a irreversible damage i.e) Permanent neurological invalidism is more.

In spite of this, systematic studies of patients presenting in the ER with unconsciousness are surprisingly few.

Apart from ventilator dependency and infective complications as quoted in many studies [3], coma is one of the major critical conditions which leads to prolonged intensive care and increased mortality.

Even though plenty of neuro imaging techniques are available in the modern medical world for evaluation of coma , electrophysiological studies also play a vital role [4] in assessing thalamic circuits which are inaccessible clinically.

Cerebral electrographic patterns allow distinction of coma from normal sleep[5] and other causes of confusion or unresponsiveness.

Certain EEG patterns tell us deepening or lightening of mental status, though progression of coma with various EEG patterns is inconsistent[6].

Recovery of consciousness is usually very gradual, sometimes marked by emergence of clear behavioral milestones, but more often by subtle improvements. Subtle signs of consciousness have to be recognized early to avoid misdiagnosis.

But plenty of studies are needed to give definitive correlation of EEG findings and prognosis.

Here we are doing one such study to assess the clinical profile of patients and to correlate the outcome with clinical findings and EEG pattern with background activity.

AIMS & OJECTIVES

OVERALL AIM:

Coma , being one of the most common medical emergency we are facing the main Purpose of this study is to improve our Knowledge regarding common Etiologies of Coma & their prognosis, and to relate Clinical Tools To Facilitate Differential Diagnosis Of Patients With Impaired Consciousness.

OBJECTIVES:

1. To study the varied clinical presentation of coma in different etiologies.

2. To compare the mortality rate in each coma.

3. To assess the EEG patterns in coma & correlate it with the recovery of the patient.

HISTORICAL BACKGROUND

It has been documented from pre-Columbian Peru to bronze-age Europe that Archaeological evidence of the occurrence of the trepanation [7] 6000 years back illustrates that a link between brain and consciousness.

The term koma (Greek term for coma) meaning deep sleep had already been used in the Hippocratic corpus [8] ("Epidemica") and then by Galen early in the second century AD. But after that the term literally vanished in literature till middle of the 17th century.

In 4th to 6th century BC Greek philosophers and physicians such as Alcmaeon and Plato proposed a correlation between sensations and the brain [9]. This theory of encephalocentrism considered the brain to be the centre of consciousness, sensation and knowledge.

In 1672 Thomas Willis again quoted this term in his famous literature De anima Brutorum [10] in which he sequentially described deeper form of unconsciousness.

Early in 18th century the term was found in Boerhaave's Lectures in his teaching on European medicine [11]. It was described by him as a perfect image of deep sleep as in a healthy person except for the duration.

In the end of the 19th century John Hughlings – Jackson a Neurologist proposed that consciousness resided in both cerebral hemispheres. He explained the possibility of unconsciousness only if both hemispheres gets damaged

simultaneously [12]. But his opinion was challenged by several clinical observations.

Later it was Baron Constantin von Economo, who demonstrated the relationship of impaired consciousness and brain stem reflex abnormalities in time of the First World War. He correlated the symptoms to lesions found in the para median reticular formation in the midbrain and in the grey matter anterior to the third ventricle in the hypothalamus.

Von Economo [13] proposed that this specific circuitry in the brainstem is responsible for wakefulness and arousal of the forebrain.

Based on the difficulties encountered by Kocher in localizing the lesions in his experimental coma further studies are made to overcome this.

After the discovery of Morruzzi and Magoun [14], in 1949; Marshall and Magoun, 1998,that found that this ‘system of ascending reticular relays’ had an effect ‘generally upon the cortex’ which is now called as ARAS which gradually gained importance in understanding the normal things.

ANATOMY OF CONSCIOUSNESS:

Two important domains of Consciousness are Arousal and Cognition.

Arousal is a primary function maintained by deep brainstem and medial thalamic structures. Cognitive functions need an intact cerebral cortex and major subcortical nuclei.

Our current knowledge on the anatomy and physiology of alertness [14] is contributed from the elegant experiments of Bremer and of Magoun and Moruzzi in the 1930s and 1949 by two separate scientist. They induced a state of coma in a cat & studied further regarding clinical localization of lesion.

After Several years it was Morrison and Dempsey who demonstrated a system of nonspecific projections from the thalamus to cortex without any specific sensory nucleus.

Further refinement of this concept resulted from the observation by Morruzzi and Magoun [14] that electrical stimulation of the medial midbrain tegmentum and above this level caused a anesthetized animal to suddenly become alert and its EEG change correspondingly, i.e., to become desynchronized.

It is the reticular activating system which maintains the alertness of the individual.

RETICULAR FORMATION:

This term was initially used to designate those areas of CNS which do not have a well defined nuclei [16]. It is regarded as phylogenetically ancient representing the primitive nerve network. During evolution a anatomical and functional organization has developed on the primitive network.

Reticular formation is composed of diffuse ill-defined intermingling mass of neurons and nerve fibres. It is a polysynaptic network.

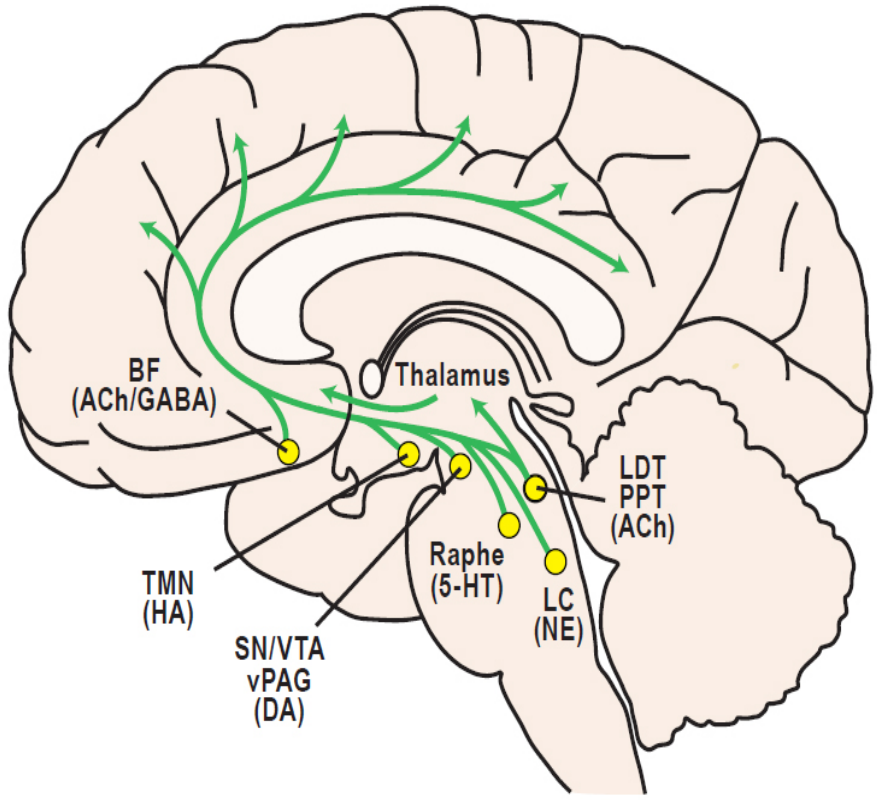
Though the exact upper extent of the reticular system is not known it extends throughout the brain stem and connected above with the subthalamic nucleus and below with the spinal cord reticular formation.

The nuclei of reticular formation is divided in to median; medial; lateral column based on the size of the neurons.

With development of immunoflourescence a number of neurotransmitters have been demonstrated in the reticular formation. Such study is called as **CHEMOARCHITECTONICS**. [17] It creates a logical idea to assume group of neurons with particular neurotransmitter to its functional relevance.

For example most of the neurons in the raphe nuclei are serotonergic and ramifies in to entire CNS.

Functionally it is divided in to ascending and descending system of reticular formation.

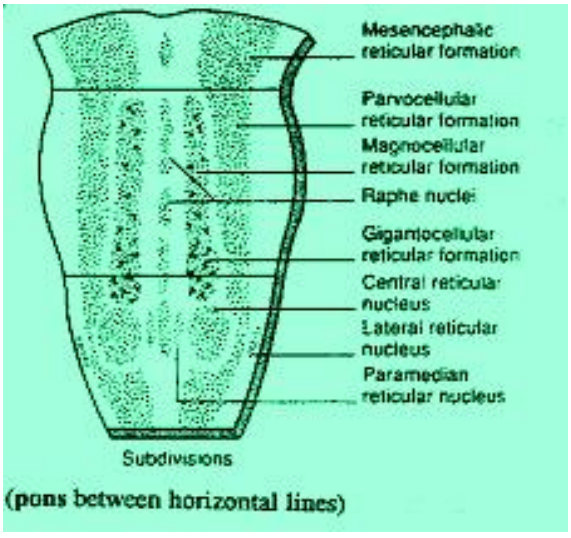


ACH – ACETYL CHOLINE

GABA – GAMMA AMINO BUTYRIC ACID

NE – NOR EPINEPHRINE

5HT – SEROTONIN



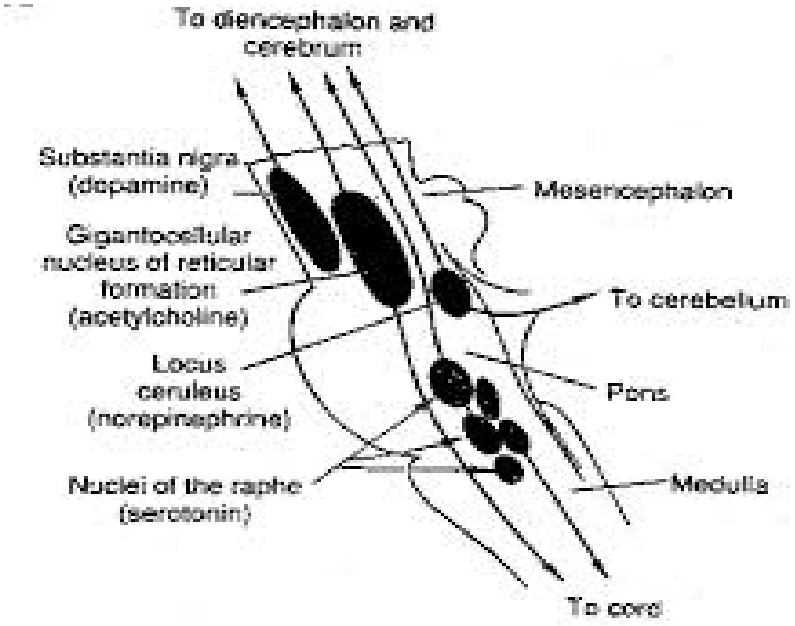
MICROSCOPIC VIEW OF RETICULAR NUCLEI IN CUT SECTION OF BRAIN STEM

DESCENDING RETICULAR SYSTEM:

These are the fibres from the reticular formation to autonomic centers in brainstem and reticulospinal tract.

ASCENDING RETICULAR SYSTEM:

It is called as ARAS (ascending reticular activating system).Fibres arise from spinothalamic, auditory & trigeminal pathway and relay in the lateral part of the reticular system.



CLINICAL DEFINITIONS

COMA is a state of complete unresponsiveness to arousal in which the patient lies with the eyes closed.

STUPOR is defined as a state in which the patient is initially unresponsive but he need repeated vigorous stimuli to achieve arousal.

VEGETATIVE STATE means patient is awake but are unaware of themselves or their environment. If this continues for more than a month it is called as **PERSISTENT VEGETATIVE STATE.**

To diagnose that they should satisfy the criteria given by multi society task force.

MCS: (Giacino et al.,) clearly discernible evidence of self- or environmental awareness should be there. It can be demonstrated by testing the following behaviors:

1. Follows simple commands

2. Gestural or verbal yes/no responses (regardless of accuracy)

3. Intelligible verbalization

4. Purposeful behavior, including movements or affective

Behaviors that occur in contingent relationship to relevant environmental stimuli and are not due to reflexive activity.

CAUSES OF COMA:[12]

(Data from Plum, F., Posner, J.B., 1980. The Diagnosis of Stupor and Coma)

I. SYMMETRICAL-NONSTRUCTURAL:

TOXINS: Lead ; Thallium ; Mushrooms; Cyanide ; Methanol ; Ethylene glycol ; Carbon monoxide

DRUGS: Sedatives ; Barbiturates*; Bromides ; Alcohol ;Opiates ;Salicylate; Anticholinergics ; Amphetamines;Lithium; Monoamine oxidase inhibitors

II. SYMMETRICAL-STRUCTURAL

SUPRATENTORIAL

- Bilateral internal carotid occlusion ; Bilateral anterior cerebral artery occlusion

III. ASYMMETRICAL- STRUCTURAL

SUPRATENTORIAL

- Thrombotic thrombocytopenic purpura
- DIC
- Nonbacterial thrombotic endocarditis ; marantic endocarditis
- Fat emboli
- Unilateral hemispheric mass (tumor, bleed) with herniation

METABOLIC:

- Hypo / Hyperthermia **;**Hypoxia **;** Hypercapnia
- Hypo / Hypercalcemia
- Hyper / Hyponatremia

- Hypo/Hyperglycemic nonketotic coma / Diabetic ketoacidosis

- Wernicke encephalopathy
- Porphyria

- Hepatic encephalopathy
- Uremia
- Dialysis encephalopathy
- Addisonian crisis

INFECTIONS:

- Bacterial meningitis
- Viral encephalitis
- Syphilis
- Sepsis
- Typhoid fever
- Malaria

BLEED:

- Subdural Hemorrhage, *Bilateral*
- Intracerebral bleed
- Pituitary apoplexy
- Massive or bilateral supratentorial infarction

  Multifocal leukoencephalopathy

OTHERS

- Post ictal
- Diffuse ischemia (myocardial infarction, congestive heart failure, arrhythmia)
- Hypotension / Hypertensive encephalopathy
- Fat embolism ; Hypothyroidism

COMA ASSESMENT SCALES:

- To assess the present situation and to determine the of ultimate outcome in a multicenter study of outcome after severe brain damage clinical scales are very important[19,18]. Even though it cannot pick up very minor changes it is very obvious that such scaling systems are required to grade the patient and determine the prognosis. Some of the commonly used scaling systems are the Glasgow Coma Scale (GCS), Rappaport Coma/Near-Coma Scale and JFK Coma Recovery Scale.
- Before Glascow scale early in the time of second world war Britain MRC introduced certain terms to promote exchange of information between medical personnel. But unfortunately it is very difficult to understand the meaning of these terms and to apply it in patients who are unconscious.
- These concerns led to set up studies in Glasgow in 1970 with severe head injury patients along with collaborators. Then it extended to include the monitoring of early severity and of outcome of coma from non-traumatic brain insults. This led to the publication in 1974 of what now called as the Glasgow Coma Scale (GCS).
- The scale comprises three tests: eye, verbal and motor responses. The three values separately as well as their sum are considered. The lowest possible GCS (the sum) is 3 (deep coma or death), while the highest is 15 (fully awake person).

SCORE	MOTOR	VERBAL	EYE
1	No movement.	None	Does not open eyes
2	Extensor response. Extension of elbow with pronation and adduction	Incomprehensible speech. Moans and groans only – no words	Opens eyes in response to painful stimuli
3	Abnormal flexion. Slow withdrawal with pronation ofwrist, adduction of shoulder	Inappropriate words. Intelligible words but mostly expletives or random	Opens eyes in response to voice
4	Withdraws. Normal flexion of elbow or knee to local painful stimulus	Confused conversation. Attends & responds but answers muddled/wrong	Opens eyes spontaneously
5	Localises. Other limb moves to site of nail bed pressure	Orientated. Knows who, where, when; year, season, month	--
6	Obeys commands. Exclude grasp reflex or postural adjustments	--	--

COMA/NEAR COMA (CNC) SCALE:

It was contributed by was contributed by Santa Clara Valley Medical Center by Rappaport.

It is used to measure small changes in those with severe brain injuries who function at near-vegetative and vegetative states. It provides quick assessment of progress or lack of progress in low-level brain injured patients.

The CNC has five levels, which depends on 11 items, based on numerous stimulation test that can be scored to indicate severity of even a sensory, perceptual, and primitive response deficits.

JFK COMA RECOVERY SCALE-REVISED (CRS-R) :

Previously used CRS not include behavioural criteria which is must to diagnose the minimally conscious state (MCS), so it has a limited diagnostic utility.[20]

CRS-R comprises 6 subscales addressing auditory, visual, motor, oromotor verbal, communication, and arousal processes. Scoring is based on the specific behavioral responses to sensory stimuli that is administered in a standardized manner. The scale can be administered reliably by trained examiners and produces reasonably stable scores over repeated assessments. CRS-R is capable of discriminating patients in an MCS from those in VS

CLINICAL EXAMINATION IN COMA:

Even though there are numerous newer modalities of investigation still complete clinical examination is very essential in every comatose patient. It helps us to guide how to proceed further.

Major things include pupils examination, ocular movements and respiratory pattern in a comatose patient. It helps to localize the lesion and also in predicting the prognosis.

RESPIRATORY PATTERN ABNORMALITIES IN COMA

Although the respiratory pattern of a patient in coma helpful in localizing structural dysfunction in the neuraxis, metabolic abnormalities also affect the respiratory centers of the pons (pneumotaxic and apneustic) and medulla (expiratory and inspiratory) and result in patterns mimic that of a structural disease. Therefore, before interpretation of respiratory changes in a comatose patient thorough evaluation of the metabolic status of the patient is a must .

CHEYNE-STOKES RESPIRATION:[21]

It is characterized by brief periods of hyperpnoea alternating regularly with shorter periods of apnea. After the apneic phase, the amplitude increases gradually then reaches a peak later gradually wanes to apnea.

Apart from periodic fluctuation in respiration, the alertness of the individual also varies. The pupils dilation toward normal from the miosis is characteristic

of diencephalic dysfunction. Sometimes even eyelids open during the rapid breathing phase and close during the slow phase.

Cheyne-Stokes respiration the respiratory drive becomes more closely dependent on the Pco_2. Carbon dioxide accumulation causes hyperpnea, which in turn induces a drop in pCO_2 which stops the respiratory stimulus & period of apnea ensues.

Usually this respiratory pattern seen in bilateral widespread cortical lesions. Metabolic disturbances such as uremia and hypoxic encephalopathy often results in similar breathing pattern. But it is relatively non specific.

Cheyne-Stokes respiration with supratentorial mass lesions indicates that there is transtentorial herniation.

APNEUSTIC BREATHING:[22]

It consist a long inspiratory pause,then air is retained for several seconds then released. Commonly seen in lesions of the lateral tegmentum in the lower half of pons.

CLUSTER BREATHING:

Its also called as short-cycle Cheyne stokes breathing , with less waxing and waning in an irregular sequence. Often it is regarded as an ominous sign of a posterior intracranial lesion or malignant intracranial tension.

ATAXIC BREATHING:

It is also called the atrial fibrillation of respiration which is characterized by a completely irregular type of breathing pattern in which inspiratory gasps of various amplitude and length are intermingled with periods of apnea.

This type of breathing often seen in agonal patients heralds complete respiratory failure & follows damage of the dorsomedial medulla.

The most common etiologies for this pattern include cerebellar or pontine hemorrhages,trauma, and posterior fossa tumors.

In severe meningitis the classic breathing pattern described by Biot called as Biots breathing.

ONDINE'S CURSE[23]:

Pathways from the cerebral cortex controls voluntary respiration where as pathways descending from the medulla subserves automatic respiration; so selective impairment of automatic or voluntary breathing is possible.

Anatomically separate pathways are available for inspiratory and expiratory process, descending pathways that are under voluntary control travel within the dorsal cord in the region of the corticospinal tract, whereas those from primary medullary respiratory centers travel in the ventrolateral cord.

Ondine's curse refers to the loss of automatic breathing during sleep.

This respiratory pattern, obviously absent in comatose patients, because it occurs with lower brainstem dysfunction.

This disorder has also been recorded with high cervical cord lesions after surgical section of the ventrolateral spinal cord for pain relief, probably because of reticulospinal tract interruption.

A selective paresis of voluntary but not automatic respiration has been described with a discrete infarction of the ventral basis pontis.

PUPIL EXAMINATION

Pupillary examination is very important in the evaluation of coma. Because it most of the time helps to rule out whether any major structural lesions with impending herniation there are not. Metabolic coma are resistant to pupillary abnormalities.

To have a clear knowledge on intepretation of pupillary signs, the knowledge on anatomical substrate essential for controlling the pupillary muscles are important[24].

Dilator pupillae by sympathetic; sphincter pupillae by parasympathetic pathway.

Sympathetic first-order neuron arises in the hypothalamus through the posterolateral tegmentum to the ciliospinal center of Budge at the T1 level of the spinal cord, then 2nd neuron from here synapse in the superior cervical sympathetic ganglion. The third-order neuron travels along the internal carotid artery and then through the ciliary ganglion to the pupillodilator muscles.

Parasympathetic starts from Edinger westpal nucleus travels in the oculomotor nerve to the ciliary ganglion, from which it innervates the pupillosphincter muscle.

Abnormalities of this reflex when unilateral , indicate structural lesions of the midbrain or oculomotor nerve. A few exceptions are noteworthy like pharmacological instillation of drugs like anti cholinergics.

Some of the pupillary abnormalities helps in localising the cause of coma & location of lesion.[25]

SIZE & PUPILLARY LIGHT REFLEX	**SITE & CAUSE OF LESION**
Bilateral Small reactive	Sleep or bilateral diencephalic dysfunction (metabolic coma)
Miosis along with anhidrosis on the side of the body ipsilateral to the lesion	Unilateral hypothalamic damage
Midposition unreactive spontaneous hippus	Midbrain tectum or pretectum [26]
Midposition unreactive irregular with corectopia	Midbrain tegmentum [26]
Pinpoint pupils reacting to light	Pontine tegmental lesions
Lateral pontine, lateral medullary, and ventrolateral cervical cord lesions	Ipsilateral Horner syndrome.

VARIOUS EYE MOVEMENTS IN COMA:

The assessment of ocular motility in comatose patients relies heavily on reflex eye movements, including the oculocephalic reflex which is done by the doll's eye maneuver and the oculovestibular reflex [28,27] by instillation of cold or warm water into EAC.

	RESPONSE	INTERPRETATION
Lateral head rotation	Eyes remain conjugate, move in direction opposite to head movement	Normal
	No movement in either eye on rotating head to left or right	Bilateral pontine gaze palsy, bilateral labyrinthine dysfunction, drug intoxication, anesthesia
	Eyes move appropriately when rotated in one direction but do not move when rotated in opposite direction	Unilateral pontine gaze palsy
	One eye abducts, the other eye does not adduct	Third nerve palsy INO
Vertical head flexion and extension	No movement in either eye	Bilateral midbrain lesions

Caloric testing with ice water instilled over 30 seconds then head is raised 30 degrees and an intact tympanic membrane is documented, provides a stronger stimulus than the oculocephalic reflex.

If only the latter reflex is present, either caloric stimulation has been performed inadequately (e.g., hindered by the presence of wax in the external auditory canal) or there is damage to the labyrinth (e.g., by ototoxic antibodies) or the vestibular nuclei in the superolateral medulla.

The following eye movements have been described in coma but it does not indicate any specific etiology but many associations have been described. But various eye movements are described in same patients.

SPONTANEOUS EYE MOVEMENTS:

SHORT-CYCLE PERIODIC ALTERNATING GAZE:

It involves roving of the eyes from one extreme of gaze to the other and back, each cycle taking 2.5 to 8 seconds.

This usually indicates both hemisphere involvement with an intact brainstem, but some cases also been described with posterior fossa hemorrhage, basal ganglia infarcts, hydrocephalus, and overdose of tranylcypromine.

It has also been described in chronic hydrocephalus from infancy with absent vertical eye movements, but have ping-pong gaze since childhood.

Crevits and Decruyenaere [29] described ping-pong gaze in hepatic encephalopathy, carbon monoxide intoxication, and hypoxia. This disorder has no prognostic value. Main differential diagnosis periodic alternating gaze

deviation, which is an alternating horizontal conjugate gaze deviation lasting 1 to 2 minutes in each direction.

REPETITIVE DIVERGENCE:

It is rarely seen in patients with coma from hepatic encephalopathy.At rest the eyes are in midposition or slightly divergent.

There will be repeated cycle of deviating out, then fixed in deviated position slowly returns to original position but the movements will be synchronous in both eyes.

OCULAR BOBBING:[30]

It is a intermittent involuntary movement in the eye in which the downward movement is brisk but the returning movement is slow

Ocular bobbing is described in toxic-metabolic encephalopathies like OPC poisoning and intrinsic pontine lesions like central pontine myelinolysis or pontine infarct. Typical ocular bobbing have preserved horizontal eye movements is some times said to be specific but it is not pathognomonic of pontine insult.

INVERSE OCULAR BOBBING:[31]

It is otherwise called as ocular dipping or fast-upward ocular bobbing. It consists of a slow-downward eye movement with fast return to midposition. It is

mostly seen in which may occur in hypoxic coma secondary to cardiac arrest or after status epilepticus. This eye movement indicates a diffuse brain dysfunction. Horizontal gaze reflexes are usually intact as in ocular bobbing.

REVERSE OCULAR BOBBING (FAST-UPWARD OCULAR BOBBING)[32]:

It is fast-upward eye movement with a slow return to midposition.It occur in patients with metabolic encephalopathy & coma due to combined phenothiazine and benzodiazepine poisoning.

SLOW-UPWARD OCULAR BOBBING (REVERSE OCULAR DIPPING):

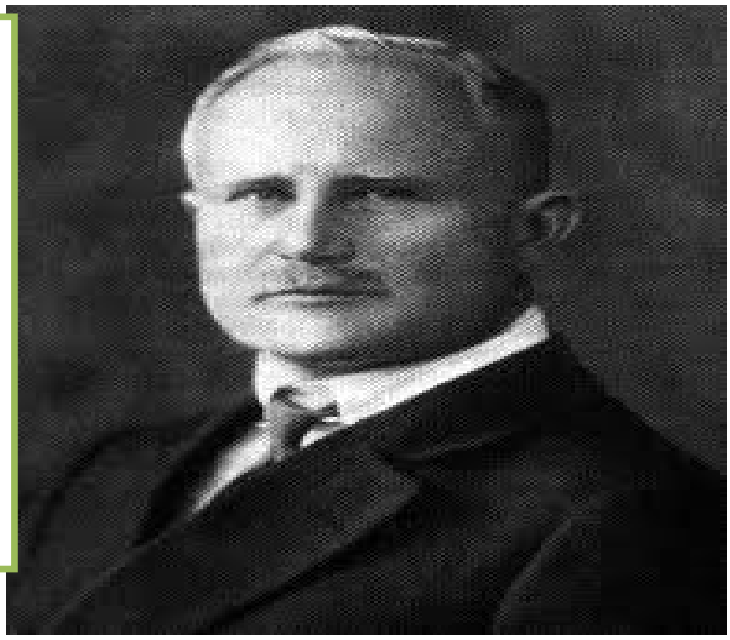
It is characterized by slow-upward eye movements followed by fast return to midposition. It is described in various metabolic encephalopathies.

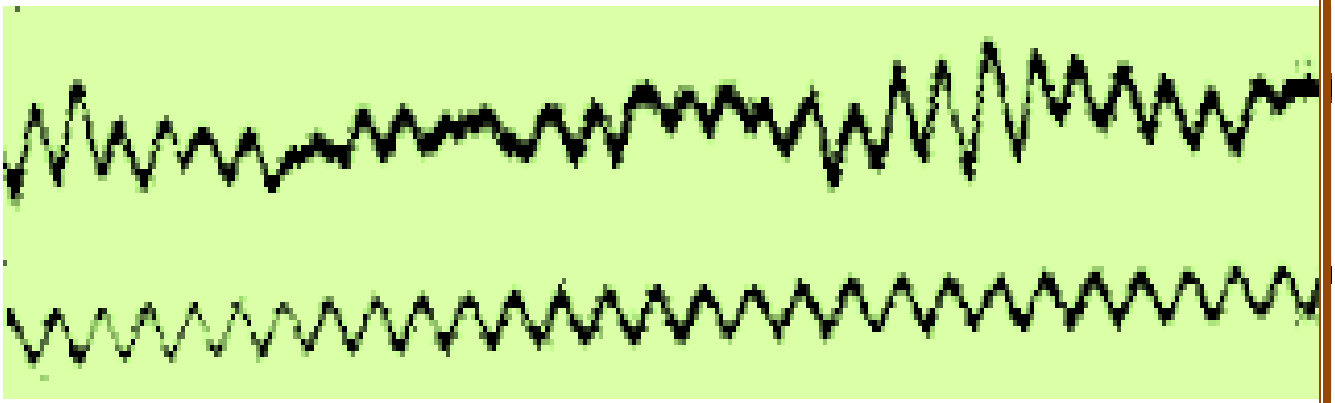
RICHARD CATON

1842-1926

HANS BERGER

(1873-1941) & FIRST EEG RECORED IN HIS SON





HISTORY OF EEG

Limiting the discussion only to history of EEG can only reveal the tip in the iceberg there are many pioneers in this field who contributed much to the modern learning of EEG.

The first attempt at the electrical activity of brain was studied by **RICHARD CATON** (1842-1926) a physician from Liverpool presented his findings in BMJ in 1875[33].

He deserves the credit for the discovery of fluctuating potentials that constitute the EEG. Even he is a research drop out his bold work remains a mile stone.

G.FRITSCH discovered the capability of human cortex can be electrically stimulatable. Initially the spontaneous electrical activity of brain was demonstrated in rabbits & dogs.

PRAVDICHNEMINSKY coined the term "ELECTROCEREBROGRAM" (mixture of Greek and Latin terms :ELECTRO and GRAM from Greek ,CEREBRO from Latin) for tracings done in dog.

It was **HANS BERGER** (1873-1941) ,the discoverer of Human EEG ,a neuropsychiatrist. He proposed the term ELEKTRENKEPHALOGRAM[34,35].

Every electroencephalographer must be familial with his work. The first human record from his son was taken in 1925.The tracings are shown previously.

From then EEG branched out in to world of single neurons & microelectrode technique was introduced in early1950s.

Topical EEG diagnosis has made a comeback of its own in the form of computerized brain mapping. This fascinating thing was done by **FRANK DUFFY** [36] Its a old EEG in a new cloth.

Recently, an interest has been sparked in the use of EEG for establishing a direct channel of communication between the brain and a computerized device. These Brain-Computer Interfaces allow a user to control an external device by altering their mental state while a pattern analysis algorithm simultaneously attempts to identify the corresponding change in the EEG signals. A device such as a mouse can be instructed to take an action that has been previously associated with the detected change in mental state. This procedure effectively bypasses our innate, motor based means of communication.

NEUROPSYCHOLOGICAL BASIS TO EEG[37]

CNS basically consists of nerve cells which are arranged in a specific laminar character with Glial cells located in between. Nerve cells are surrounded by several thousand synapses.

When membrane of nerve cell body is penetrated by microelectrode, a potential of about 60 to 70 milli volt with negative polarity in the intracellular space can be recorded, which is susceptible to fluctuations elicited by the surrounding synapses.

Like nerve cell, Glial cell can also play a role in generating extracellular potentials but since there are no post synaptic potentials Glial cell fail to show any action potentials. Glial cell membrane potential depends on the extracellular pottasium concentration.

GENESIS OF EEG WAVES

If a grouped and synchronous influx takes place in the afferent system towards the superficial generator structures, EEG evolves are of high amplitude. In case of periodic sequence of afferent burst the recording shows sinusoidal potential fluctuations.

DC (direct current /direct coupling)recordings are usually performed in animal reordings, where slower potenials can also be picked up.

Specific findings in conventional EEG are associated with different DC shift[38]. For example Hypercapnia induced disappearance of EEG is associated with monophasic positive DC shift. There are several patterns of characteristic DC fluctuation.

NORMAL EEG[39]

It is a continuous roar or noise of the brain with wide range of frequencies. It is not a hodgepodge of frequencies.

The EEG is typically described in terms of (1) rhythmic activity and (2) transients. Pronounced rhythmicity may be a sign of abnormality. The rhythmic activity is divided into bands by frequency.

The frequency of EEG has a fuzzy lower an upper limit. The range lies between 0.1cps to 100 cps, in a more strict sense between 0.3cps and 70 cps (cycles per second).

In normal adult, slow range and very sparse range are very sparsely represented ,mostly medium and fast ranges predominate in the picture.

The frequencies are broken down in to following and or ranges. The sequence of greek letter is not logical and can be understood only in historical view.

Delta below 3.5/sec

Theta 4 -7.5/sec

Alpha 8 – 13/sec

Beta 14 – 30/sec

Gamma above 30/sec. The exact width of Gamma range is still debated.

Curio proposed following designations: Omega 60 to 120 Hz ; Rho 120 to 500 Hz ; Sigma 500 to 1000 Hz.

ALPHA RHYTHM[41]:

International Federation of Societies for Electroencephlography & Clinical Neurophysiology**(IFSECN)**[42] proposed following definition for alpha rhythm.

‘‘Rhythm at 8 – 13 Hz occuring during wakefulness over the posterior regions of the head, generally in higher voltage in the occipital region.Best seen with eyes closed and under condition of physical relaxation and mental inactivity. Blocked by visual and mental effort’’

The amplitude varies considerably from person to person, moment to moment.

It is usually characterized by rounded or sinusoidal wave forms.

It is clearly a manifestation of posterior half of the head as opposed to Burger classical view of global cerebral rhythm. Alpha drop out is a classical feature of normal adult EEG.

ROLANDIC (CENTRAL) MU RHYTHM:

Frequency & amplitude similar to alpha rhythm but topographical and physiological significance varies. Most common frequency seen is 10/sec.

Mu stands for motor and it is strongly related to function of the motor cortex.

It is historically called as pre central alpha rhythm /alphoid rhythm /high voltage rolandic alpha. C3 & C4 electrode located over the precentral gyrus is the optimal location for picking up the central rhythm.

BREACH RHYTHM:

The presence of cranial bone defect will have a considerable effect on EEG frequency. Mu rhythm will be prominent over that area.

KAPPA RHYTHM[40]:

The anterior temporal rhythm in alpha frequency was reported by LAUGIER.It is possibly an ocular artifact caused by lateral oscillation of eyeball.

BETA RHYTHM[43]:

Any rhythmical activity above 13/sec is called beta rhythm. Encountered mainly in the frontal and central regions. Usually not exceeding 35/sec.

Berger in 1938 reversed his earlier concepts and said that the beta waves not the alpha waves of the EEG are concomitant phenomenon of the mental activity.

According to GIBBS and GIBBS [44], beta waves must be abundant in quantity and of rather high voltage to be termed abnormal.

It has also been found that beta activity has certain relationship with personality traits. In emotionally stable persons beta activity over the central region is absent, in contrast to a central fast rhythm in aggressive dynamic personality.

THETA RHYTHM:

The term theta is selected because of is presumed thalamic origin. It was introduced by WALTER & DAVEY. According to international nomenclature it is the rhythm with frequency of 4 to under 8Hz.

Normal waking record contains only small amount of theta frequencies[43]. It was correlated to emotions of disappointment and frustrations.

A rhythmical 4/sec pattern occurring over the vertex solely in the waking state has been described as vertex spindles by MAGNUS.

LAMDA WAVES[45]:

These are the sharp transients occurring in the occipital region of the head in waking up subjects. Mainly positive waves, bi or tri phasic. Lamda activity is strictly bilateral synchronous.

LOW VOLTAGE RECORD:

IFSECN defines it as a waking record characterized by activity of amplitude not greater than 20 micro volt over all head region.

In a comatose patient its inferred that it is due to true decline of cerebral activity not merely by desynchronization .

RELATIVE ADVANTAGES OF EEG:

- Cost effective hardware
- No need of bulky and immobile equipment as in other modalities it can be used in almost all places.
- Very high temporal resolution, on the order of milliseconds rather than seconds.
- Modern EEG data collection systems are capable of recording at sampling rates above 20,000 Hz if desired.
- EEG is relatively tolerant of subject movement.
- Methods for minimizing, and even eliminating movement artifacts exist
- Better study of the responses to auditory stimuli.
- Does not aggravate claustrophobia like MRI.
- Does not involve exposure to radio ligand like PET.
- Extremely non invasive when compared to electrocorticogram.

ARTIFACTS:

BIOLOGICAL:

Electrical signals that originate from non-cerebral origin are called artifacts. The records almost always are contaminated by such artifacts. The amplitude of artifacts varies based on signals of interest.

It needs lot of experience to correctly interpret EEGs .There is simply no substitute for an adequate training period in joint reading sessions.

Common types of biological artifacts include:

Eye movements induced artifacts[46] :

Mostly caused by the potential difference between the cornea and retina, which is quite large compared to cerebral potentials. When the eyes are completely still, this corneo-retinal dipole does not affect EEG. Saccades also generate transient electro myographic potentials, known as saccadic spike potentials (SPs). It overlaps the gamma-band.

Eyelid fluttering artifacts or Kappa waves usually seen in the prefrontal leads, that is, just over the eyes.

ECG (cardiac) artifacts:

Usually mistaken for spike activity.To avoid this, modern EEG acquisition includes a one-channel ECG from the extremities which is helpful to identify cardiac arrhythmias and in evaluating syncope.

EMG (muscle activation)-induced artifacts:

EEG contamination by muscle has been more prevalent particularly in the gamma range above 20 Hz. However, Surface Laplacian has been shown to be effective in eliminating muscle artifact.

Glosso kinetic artifacts- tongue movements contaminate the EEG, especially in parkinsonian & tremor disorders.

Artifacts can be useful in various applications. The EOG signals are very important in polysomnography & in conventional EEG for assessing possible changes in alertness, drowsiness or sleep.

ENVIRONMENTAL ARTIFACTS:

Besides the ocular artifact movement by the patient or even just settling of the electrodes, may cause *electrode pops* , spikes originating from a momentary change in the impedance of a given electrode.

Poor grounding cause significant 50 or 60 Hz artifact, depending on the power system's frequency.

Other possible interference can be the presence of an IV drip

Such devices can cause rhythmic, fast, low-voltage bursts, confused for spikes.

ARTIFACT CORRECTION:

Recently, independent component analysis techniques, attempt to "un mix" the EEG signals into some number of underlying components have been used to correct or remove EEG contaminants. Separate algorithms are available for specific remixing thereby nullifying (zeroing) the weight of unwanted components resulting in "clean" EEG.

ROLE OF EEG IN DIAGNOSIS OF CEREBROVASULAR LESIONS[44]

HACHINSKI & NORRI have pointed out that EEG is not of much help in the diagnosis of stroke when CT /MRI are available[48].

An imaginative approach to evaluate stroke with EEG has been proposed by VELHO GROENBERG.

The temporal profile of evolution is important, as an acute vascular foci cause a more impressive focal slowing than a neoplasm which is slowly growing.

Sometimes lesions deep from the cortex escape from detection via EEG.

ACUTE CVA:

EEG manifestations depend on the location of the vascular territory and the pathogenetic mechanism involved in causing the lesion.

In the presence of massive neurological deficit pronounced polymorphic delta activity more in the affected side temporo parietal region.

Conclusive phenomenon such as focal motor twitching are common contra laterally in some forms of extra territorial ischemia (water shed infarct).

According to VAN DER DRIFT there will be irregular very slow ipsilateral delta activity with maximum voltage in the fronto temporal region.

EEG changes may not be as good as clinical picture that too in a hemorrhagic stroke due to preservation of cortical garland. Intermixed theta wave is more in hemorrhage rather than in thrombosis.

Preservation of background activity is prognostically a good sign. During the period of recovery decline of slow activity parallel to the neurological improvement.

Recovery phase shows remarkable resolution of initial EEG changes. But it's often difficult to distinguish an ischemic and hemorrhagic lesion from EEG.

THROMBOSIS OF ANTERIOR CEREBRAL ARTERY & ITS PERFORATING BRANCHES [50]:

Ipsilateral frontal lobe delta activity is typical EEG concomitant in this event with sometimes intermittent delta activity.

THROMBOSIS OF POSTERIOR CEREBRAL ARTERY:

Delta activity is pronounced in the ipsilateral parieto occipital region.

WATER SHED INFARCT:

Diffusely disordered severely disorganized mixed delta theta activity with rhythmic periodic spike discharge.

THALAMIC HAEMORRHAGE:

According to JASPER & VAN BUREN there will be a reduction in alpha rhythm in anteroventral thalamic haemorhage.

MIDRAIN HAEMORRHAGE:

Diffuse activity in the upper theta range.

LOWER BRAINSTEM HAEMORRHAGE:

Well preserved alpha rhythm in the posterior aspect that cannot be blocked by different modalities of stimuli.

CEREBELLAR HAEMORRHAGE:

High voltage delta activity, that too in contralateral cerebellar hemisphere.

TIA[49]:

Mostly normal; sometimes minor slowing of activity(mostly theta) and somewhat sharp activity.

SOME RARE PATTERNS OF EEG IN DROWSINESS:

1. ANTERIOR BRADYRHYTMIAS:

2. WICKET SPIKES:

CEREBRAL ANOXIA & EEG IN CEREBRAL ANOXIA [52]

Brain is one of the most metabolically active of all the organs in the body which consumes oxygen at a rate of 3.5ml/100 gm /min.CBF is 57 ml/100gm /min. Human brain which occupies only 2% body weight accounts for 20 % resting body oxygen consumption.[53]

This oxygen is utilized for the oxidation of carbohydrate which is the only substrate of brain. Because of this and lack of tissue stores, interruption of O2 delivery causes immediate cell dysfunction.

The vulnerability of the tissue to O2 lack varies. It is given in the following decreasing order:

Hippocampus > Purkinje Cells In Cerebellum > Stellate & Basket Cells >Striatal Cells (Small >Large) > Neocortical Layers 3, 5, 6 > Layers 2,4.

Hypoxia tends to be accompanied by hypercapnia due to insufficient removal of CO2, which is called as asphyxia.

The ultimate cause of hypoxic encephalopathy is a fall in the partial pressure of tissue oxygen to levels that no longer support mitochondrial respiration.

Pure hypoxic encephalopathy uncomplicated by other effects due to reduced blood flow rarely occur in humans.

Neurological signs of this anoxic injury have been described in two phases i.e. primary & secondary by BINDER.

PRIMARY PHASE	SECONDARY PHASE
NO PUPILLARY LIGHT REACTION	NORMAL PUPIL REACTION
INCONSTANT OCULOCEPHALIC & VESTIBULO CEPHALIC REFLEX	PRESERVED OCULOCEPHALIC & VESTIBULO CEPHALIC REFLEX
FLACCID MOTOR TONE	RIGIDITY
NO SPINAL REFLEX	EXAGGERATED REFLEX
NO SPASTICITY	MYOCLONIC JERKS (OCULAR / PHARYNGOFACIAL)

BASIC MECHANISM OF EEG CHANGES IN ANOXIA[52]

Energy needed to maintain membrane potentials of nerve cells is lost in anoxia. Both hypoxia and hypercapnia occurs simultaneously. This biochemical changes exerts effects on cortical and spinal neurons.

Isolated decrease in Po2 -- membrane potential decrease --- discharge rate rises.

Isolated Increase in Pco2 -- membrane potential increase --- inhibition of spontaneous activity.

Further anoxia it breaks the membrane potential and electrical activity ceases.

Vulnerability of nerve cells can also be explained in electrical means. That is in phylogenetically (newly) developed areas the electrical activity disappears first.

EEG CHANGES DURING ARREST OF CEREBRAL CIRCULATION:

Sequences of electrical events are as follows :

With in first 3-6 sec - no clinical /EEG signs.

Last for 7 -13 sec - slow waves of increasing amplitude & decreasing frequency.

If arrest is still prolonged further attenuation and flattening occur.

Return of normal cerebral activity after restoration is resumed in reverse manner.

The first change to appear after circulatory arrest is increase in alpha power & decrease in beta power which also starts decrease in 15 seconds.

When EEG is used in the evaluation of syncope ECG is also simultaneously important because seizures can lead to syncope but reverse i.e) syncope also can cause seizure.

EEG PATTERNS IN PROLONGED PATTERNS AFTER ANOXIA[54]:

DIFFUSE SLOWING:

Most of them have intermingled spindle activity which resembles physiological sleep. These changes are frequently changed by exogenous stimuli.

FIRDA:

It is Ubiquitous sign of initial defect. Characteristic of hypoxia secondary to pulmonary insufficiency & chronic diffuse ischemia.

CONTINUOUS SPIKING:

Post arrest patients have continuous spike & sharp wave activity with occasional asymmetricity.

PERIODIC SPIKES & BURST SUPPRESSION PATTERN:

These patterns indicate that patients have coma with functions down to midbrain level. During continuous spiking activity many involuntary moments are observed like myoclonus.

MONORHYTMICAL ACTIVITIES:

It is difficult to evaluate a complete alpha or theta coma but seen in anoxic coma.

ELECTROCEREBRAL SILENCE: indicates irreversible depolarization.

ROLE OF EVOKED POTENTIALS IN ANOXIC COMA:

In fact evoked potentials [55] are more advantageous than EEG in the aspect that it can predict the subcortical pathophysiology. Another major thing is EEG which will be affected by the sedative drugs where as it will not affect short latency evoked potential.

PREDICTIVE VALUE OF EEG:

It has been open to controversy since long time. Since the use of drugs has become a major restrictive factor & no systematic testing for modifying this influence has been reported its difficult to conclude prognosis.

Malignant EEG signs as described above has hard core poor prognosis. Long term EEG recordings will further helpful in elucidating the prognosis.

PROGNOSIS[56,57]:

Usually traumatic coma patients will recover soon when compared to that of the non traumatic coma, which also depends on the etiology of the coma. Patients with hypoxic ischemic coma have a intermediate prognosis between a metabolic coma & coma due to cerebrovascular accident.

EEG PATTERNS IN COMA[65]:

Its an essential part of neurological evaluation of coma. These patterns that have been observed are not significant as such. It is helpful for diagnostic and prognostic statement in the context of neurological examination.

DIFFUSE SLOWING [65]:

Regardless of whatever the cause of coma may be the most of them will have a diffuse continuous slowing in the theta or delta range.

Slowing of a reactive basic rhythm suggest a demented record rather than a coma.

Gradual dissolution of alpha rhythm and intermingled theta will be seen in the earlier stages of coma. it sometime mimic normal drowsiness.

GENERALIZED ASYNCHRONOUS SLOW WAVES

Reflect widespread structural damage or dysfunction,that includes subcortical white matter. Examples include Widespread degenerative or cerebrovascular disease, Acute anoxia, post ictal state.

The most common and least specific abnormal EEG pattern.

This is more reactive than focal slow waves attenuated by eye opening and alerting increased by relaxation and hyperventilation.

This pattern is normal in childhood & in 10-15% of normal adults.

INTERMITTENT DELTA RHYTHM:

Bilateral synchronous intermittent rhythmic delta activity is frequently seen in the initial stages. In adults mostly seen over the frontal regions, but in children in the occipital regions the same pattern is observed.

This is termed as FIRDA & OIRDA respectively.

Although it a non specific sign with regard to etiology it is the initial sign of arousal system dysfunction which is seen in earlier stages of coma.

In frontal lesions the delta rhythm (sometimes polymorphous and low voltage) are seen in leads over the contra lateral hemisphere.

Combined focal abnormalities with intermittent delta rhythm is suggestive of a supra tentorial lesion with incipient herniation.

In a otherwise normal EEG occasional delta rhythm indicates a deep seated lesion. Above said rhythm with diffuse slowing suggest metabolic encephalopathy. In contrast to FIRDA / OIRDA , a TIRDA suggest a significant epileptogenic pattern.

In certain types of coma the patterns obtained exactly matches with that of a normal sleep. i.e) sigma waves and K complex. Intermittent appearance of these pattern suggest that the initial phases of the coma the patient is susceptible to sleep induced changes. Later as the coma deepens the patterns get disorganized, which might be due to cortical dysfunction.

ALTERNATING PATTERN:

In patients with cheyne stokes breathing, cycles of high and low voltages are recorded. High voltage activity correlates with hyperventilation. This is due to pacemaker dysfunction due to depression of cortical inhibition.

PROLONGED BURST OF DELTA WAVES AND REACTIVITY OF EEG:

Mainly in patients with head injuries prolonged burst of bilateral high voltage delta activity is seen. It occurs either spontaneously or secondary to some exogenous stimuli lasting for several seconds to minutes.

One of the essential part of EEG examination in coma is to test with simultaneous stimuli. There are two types of responses.

1. Alerting type: Slow wave response to arousal.

2. Blocking type: Reduction of voltage and filtering of remnants.

EPILEPTIFORM ACTIVITIES:

Frequently sharp and spike wave pattern are seen in seizures. When present in the whole record indicates myoclonic status epilepticus.

Coma or confusional states along with PLED indicates that it represent a specific type of non convulsive status epilepticus.

SUPPRESSION BURST ACTIVITY[58]:

In a completely flattened background there is a high voltage burst of slow waves with intermingled sharp transients or spikes. Remnants of cerebral activity in between burst consist of non reactive rhythmical activity.

PERIODIC SPIKING[66,67]:

It is also related to burst related activity but the repetition rate is higher. Slow waves are less prominent and lacking. It is accompanied by myoclonic jerks.

MONORHYTHMICAL ACTIVITIES:

Resembles normal alpha rhythm as in a normal patient but it differs in terms of spatial distribution and reactivity. Almost no fluctuations or no reactivity to stimuli is seen. This alpha coma have to be separated from spindle forms. Always carries a poor prognosis but cases with poor prognosis also have been reported.

LOW VOLTAGE OUTPUT EEG:

Remnants of cerebral activity less than 20 micro volt. In fact this is the precussor of electrocerebral silence. This finding should not be interrupted as in same basis in case of a conscious individual.

FOCAL ABNORMALITIES IN COMA:

These are not rare in case of diffuse encephalopathies .for example non ketotic hyperosmolar coma produces focal deficit and produce corresponding EEG signs.

CORRELATION OF EEG WITH ETIOLOGY:

It can be never specific for a particular etiology. it can show just several correlation with the disease process.

EEG IN SUPRATENTORIAL LESIONS:

Always markedly abnormal. Focal lesion indicate site of lesion ,diffuse slowing parallels the extent of herniation. Other findings of midbrain lesions are described above.

EEG IN INFRATENTORIAL LESIONS:

Usually out of proportion to the extent of the pathology. For example in a patient with apparent coma the EEG appears unexpectedly normal.

For example in patients with locked in syndrome, the reactive alpha rhythm is the only clue that the patient is not in coma rather a locked in syndrome.

EEG IN RELATION TO THE DEPTH OF COMA:

As a functional test it should be actually correlated with the detoriation of the brain function but this most of the time not happen because of the local brain stem coma which leaves the cortex undisturbed so the EEG remains undisturbed.

Irrespective of the etiology the following information guides about the detoriation in cephalocadual direction can be supported by EEG in following manner.

1. Degree of slowing is related to the level of unresponsiveness. (exception to this rule is prolonged burst of delta waves)

2. Response to stimulation varies according to the depth of coma. The initial block type of response is changed to alerting type of response.

3. Sleep potentials progressively worse and finally disappear with deepening coma.

4. Late midbrain syndrome.

PROGNOSTIC CRITERIA FROM EEG: [59,68]

According to Celesia , EEG is of certainly having prognostic value but there are many limiting factors, since the outcome is not only determined by disturbance in the brain itself mainly related to the underlying metabolic and cardiac problems.

But in case of comatose patients where sedative drugs are used neurological examination and EEG examination may be misleading. In such cases evoked potential is helpful.

Some of the Good indicators are as follows:

1. Reactions to exogenous stimuli.

2. Normal looking sleep potentials.

3. Spindle coma(but depends on the etiology causing spindle; best with drugs, seizures)

Poor prognosis indicators:

1. Absence of reactivity.

2. Monotonous high voltage activity.

3. Paroxysmal activities in coma.

4. Triphasic waves in liver failure.

5. Rostral cadual detoriation.

EEG IN METABOLIC COMA[63]

Nutritive and metabolic systems are considered as the fuel for neuronal and glial cells. Any change in these composition may lead to clinical manifestation which is clearly depicted in EEG. These changes are usually reversible.

The history of EEG in metabolic coma begins with Berger observation in schizophrenic patients treated with insulin. Further results are demonstrated by FOLEY et al.,

Let us discuss some of the major metabolic abnormalities:

HYPOGYCEMIA:

Clinical condition i.e. level of awareness, blood sugar level , EEG changes not parallel each other.CNS dysfunction not depends on the level of blood sugar rather it depends on rate of fall in blood glucose[60].

It shows an extremely severe slowing of wave with combined epileptic activity which is more or less severe intensity depending on the individual propensities.

In Spontaneous hypoglycemia the degree of EEG change varies .Sometimes CPS are associated with insulinoma. In these patients surgery will render the patient symptom free.

HYPERGLYCEMIA:

In hyperglycemia there is mixed fast & slow frequencies [60] ; in diabetic coma there is a very pronounced slowing. Hyperosmolar coma seems to be rich in neurological complications, most common manifestation of which is epileptic seizure of focal character. In EEG there is focal seizure activity.

Some times occipital seizure may be the initial manifestation which can be triggered by lateral gaze or triggered by movement.

LIVER DISEASE (HEPATIC ENCEPHALOPATHY)[61]:

Rumpl et al., described 5 stages of conscious level which starts from confusion, lethargy, semicoma, stupor and finally coma. Degree of slowing is reflected correlates with the blood level of ammonia. Sudden shift between a normal alpha & slow substitute are common in liver disease.

TRI PHASIC WAVES[62] :

These are bilateral frontally positive sharp transients, usually of greater than 70 microvolt in amplitude. Usually it is preceded and followed by a smaller negative waveform.

The first negative wave will be mostly of higher amplitude than the second. It occur in bursts of repetitive waves at 1-3 Hz.

No reactivity is the rule, and often an anterior-posterior temporal lag can be observed. The largest deflection is usually frontal. A triphasic morphology is

not sufficient to classify a record as "triphasic waves."These waves will be seen along with deepening of consciousness or in patients who are fully awake.

The presence of triphasic waves cannot be used, for etiological diagnosis of coma rather it can tell it as a metabolic encephalopathy. It is also seen in psychiatric patients treated with Lithium. Sudden shift between a normal alpha & slow waves are common. The reason why EEG hepatic encephalopathy is sensitive still remains a debate.

RENAL DISEASE (URAEMIC ENCEPHALOPATHY):

Though various patterns of EEG are seen there are certain common EEG seen in uremic patients. Irregular low voltage spike with occasional theta rhythm ,slowing of posteriorly placed leads are noted.

It was BRASS who specified the importance of EEG in terminally ill renal failure patients.

Convulsion which may be secondary to cerebral edema or some other etiology in uremic patients occurs suddenly without preceding EEG changes.

EEG in patients with chronic uremic who have undergone dialysis remains normal. Spike wave like burst are seen in 10% patients. Long burst with high voltage with absence spindles have been reported.

HYPOCALCEMIA:

It is one of the highly epileptogenic electrolyte abnormality causing generalized spike and burst. Often a normocalcemic & neurogenic tetany will have normal EEG record.

In most of the Fahr disease patients the EEG is found to be normal.

HYPONATREMIA:

In hyponatremia and water intoxication the EEG abnormalities are usually severe with diffuse slowing. Inspite of correction the time taken for normalization of EEG is slow.

HYPERCALCEMIA:

By Spatz et al., the EEG manifestations will be seen when level exceeds 13mg/dl. It is accompanied by focal deficit & mental impairment. In addition to hepatic encephalopathy these patients also will have triphasic wave patterns.

VITAMIN DEFICIENCIES:

Pyridoxine deficiency mostly seen in children reported as a case of massive generalized spike activity, which shows a typical response to Pyridoxine.

In Thiamine deficiency changes may be mild to severe, initially will have alpha slowing followed by theta and delta frequencies. Same changes will be seen in Pellagra also.

Very pronounced epileptiform EEG abnormalities have been reported in cobalamin deficiency also.

ENDOCRINE DISORDERS:

ADRENAL CORTEX DISORDERS:

In Addisons disease the abnormalities are due to altered metabolism ,usually have slowing but reaches high voltage in case of addisonian crisis. In Cushing syndrome EEG abnormalities are less prominent. But no specific disturbance contributes to these changes.

EEG changes recover well in case of addisonian crisis in response to cortisone where as response is poor in other adrenal disorders.

THYROID DISORDERS: Hyperthyroidism will have acceleration of alpha rhythm frequency , rolandic mu rhythm also will be augmented in hyperthyroidism

PITUITARY DISORDERS: In case of Sheehan syndrome the EEG shows massive diffuse theta and delta activity in association with impaired consciousness. These abnormalities also reflect the secondary depression of adrenocortical function.

MIXED TYPE ENCEPHALOPATHY: The poly etiological condition is separate from all other metabolic condition .It is quite common to have this clinical scenario many times. most of the time it will carry a good prognosis. A

special form of mixed type of encephalopathy occurs in 30% of severe burns, but the prognosis is guarded in this case.

MATERIALS AND METHODOLOGY

PLACE OF STUDY:

This study was conducted at the Department of Internal Medicine at Thanjavur Medical college hospital during JANUARY 2014- AUGUST 2014.

TOTAL NUMBER OF PATIENTS INCLUDED IN THE STUDY:

50 patients (including both males & females).

GEOGRAPHIC DISTRIBUTION:

Patients included in this study were from Urban & Rural areas of Ariyalur, Perambalur , Thanjavur, Thiruvarur and Pudhukottai districts.

INCLUSION CRITERIA:

1. Patients >18 yrs admitted with History of Non Traumatic coma.

2. Post CPR status.

EXCLUSION CRITERIA:

1. Those not given consent

2. History of trauma.

3. Age < 18 yrs

4. Patients treated outside for coma.

5. H/O evidence of SOL.

6. H/O inherited Metabolic disorder.

7. H/O neurosurgeries like VP shunt.

8. Recurrent episodes of coma.

PROCEDURE:

Patients admitted in medicine casualty in a state of coma satisfying the above inclusion and exclusion criteria are selected.

After obtaining informed consent from the patients relative, the patient was evaluated in detail based on history of presenting illness. Associated risk factors are also elicited from history for identifying the cause of coma.

The following investigation are done in all patients admitted with coma and some special investigation in a few patients based on their clinical relevance.

1. RBS
2. Sr. UREA /CREATININE

3. Sr.BILIRUBIN /LIVER ENZYMES
4. Sr.SODIUM & POTTASIUM
5. ECG
6. CT /MRI BRAIN

Ethical committee clearance has been obtained for all the above said procedures.

EEG was taken to all these Patients by the international 10-20 systems.

Procedure to take EEG:

This system need the knowledge about where the electrodes are to be placed. It depends on the distance from definitive landmarks in the skull.

Each site has a letter to identify the site. For example F in frontal lobe. The letter Z indicate zero line in the centre.

Even number denotes the electrode placed on the right hemisphere

Odd number denotes the electrode placed on the left hemisphere.

Where to place electrode?

1. In the centre line of scalp measure the distance between nasion and inion in centimeters.

2. Measure and mark the 50% total as Cz.

3. Measure and mark 10% from the nasion and other from inion as Fpz & Oz.

4. Measure and mark 20% from the Fpz & Oz as Fz & Pz.

5. Measure the total distance between the two pre auricular points just above the indentation in the zygomatic arch. Mark 50% of this distance. Point where it cut in the previous centre line is true Cz.

6. Mark 10% from the preauricular points as T3 & T4

7. Mark 50% between T3 & Cz as C3 ; mark 50% between T4 &Cz as C4

8. Measure the circumference of Fpz & Oz by joining those two points

9. Measure and mark the 5% in the right and left of both Fpz & Oz ;that is marked as Fp1 & Fp2 ;O1 & O2

10. Measure down 20% from Fp1 & Fp2 then mark F7 & F8

11. Calculate the distance between F7 & F8 ;line where it marks 50 % in the Fz is true Fz.

12. The half distance between the F7 & Fz will be F3 & F8 & Fz will be F4.

13. 20% Nasion Inion distance between Fp1 to F3 is true F3 ; 20% Nasion Inoion distance between Fp2 to F4 is true F4.

13.Connect Fp1 and O1 then Fp2 and O2. Make preliminary C3 and C4.

14. Mark 50% between Fp1 and O1 where it intersect the line is true C3; Mark 50% between Fp2 and O2 where it intersect the line is true C4.

This completes all the placement of all 21 leads.

After placing all the leads in correct position the EEG is recorded for 20 minutes. Analysis done by experienced neurophysiologist to assess the pattern in each patient.

The patients were followed up still the patient is hospital, Outcome is determined whether the patient is alive i.e) recovered from coma or death at the time of discharge.

This is correlated with the EEG patterns and analysis done by SPSS software.

RESULTS

TABLE 1: AGE DISTRIBUTION

AGE	No.of Patients (n=50)	Percentage (100%)
Below 30yrs	9	18.0
31 to 40yrs	10	20.0
41 to 50yrs	14	28.0
51 to 60yrs	13	26.0
61yrs & above	4	8.0

MAJORITY OF THE PATIENTS IN THIS STUDY BELONGS TO 41-50 YEARS OF AGE WHO OCCUPIES 28% OF THE POPULATION, FOLLOWED BY 51-60 YEARS CONSTITUTING 26% POPULATION.

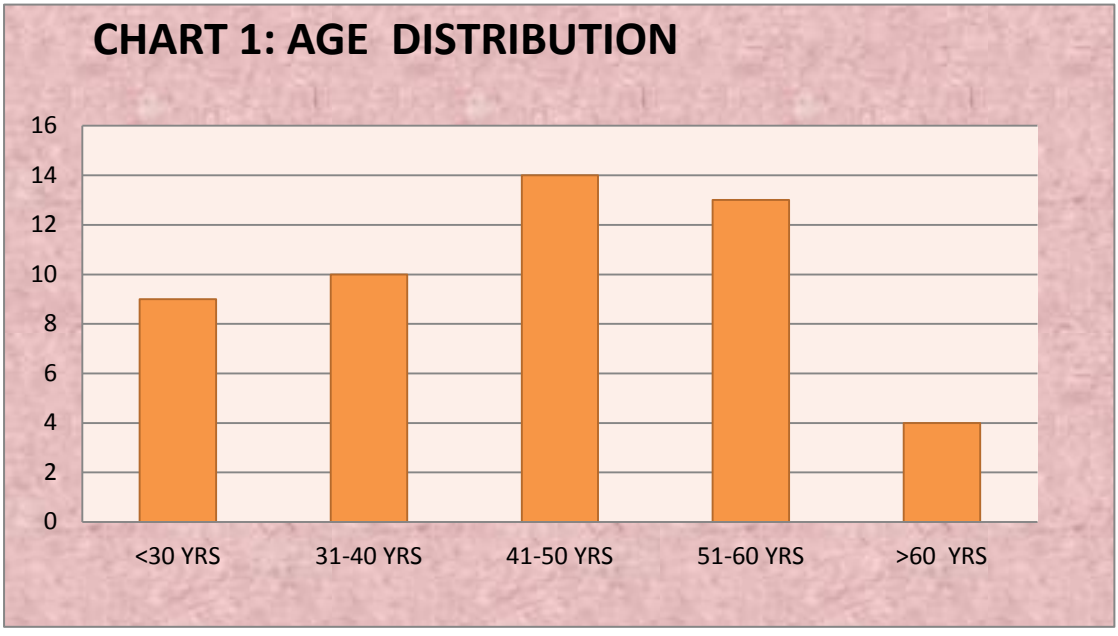


TABLE 2: SEX DISTRIBUTION

SEX	No of patients (n=50)	Percentage (100%)
Male	42	84.0
Female	8	16.0

84 % OF THE STUDY POPULATION ARE MALES.

REMAINING 16% ARE FEMALES.

CHART 2:

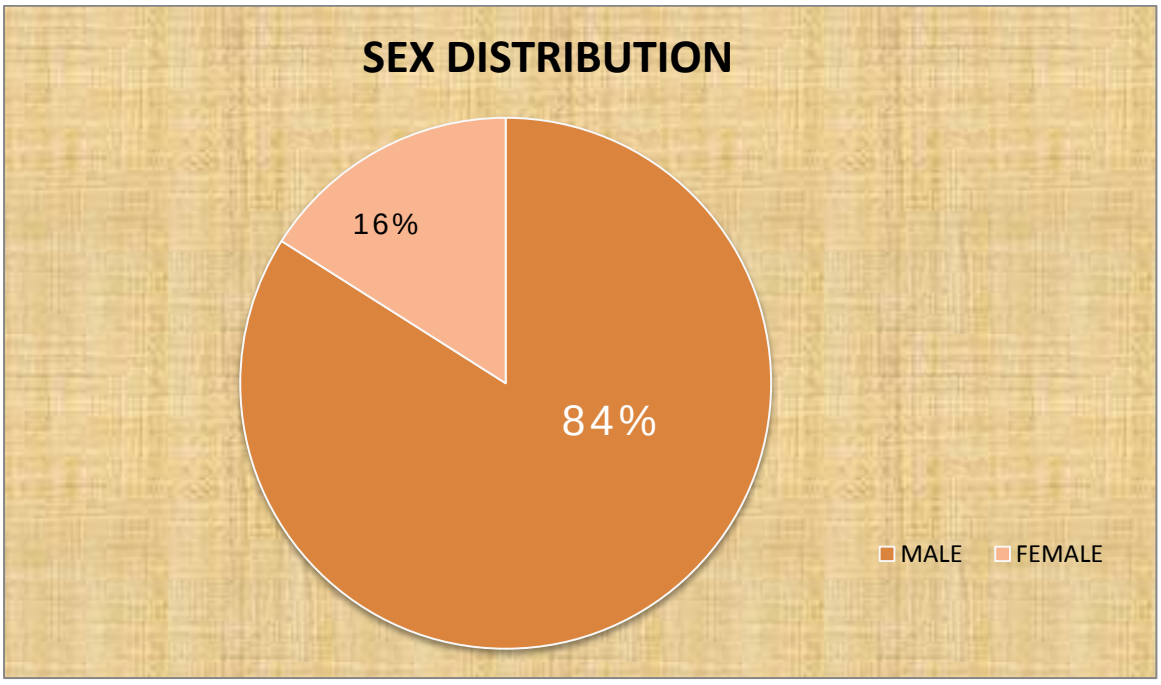


TABLE 3: DISTRIBUTION IN RESPECT TO DURATION OF COMA

DURATION OF COMA IN DAYS	No.of patients (n=50)	Percentage (100%)
<1 day	32	64.0
>1 day	18	36.0

64% OF THE PATIENTS PRESENT WITH IN THE FIRST DAY OF COMA.

REMAINING 36 % ADMITTED AFTER 1 DAY.

CHART 3:

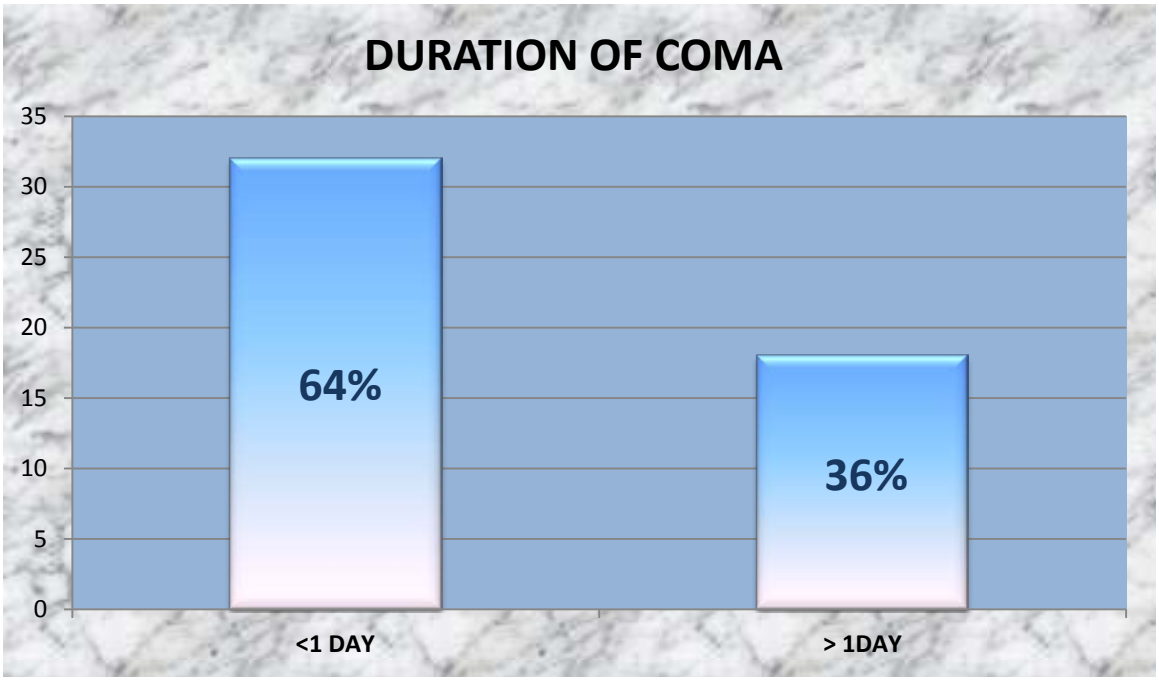


TABLE 4: SYMPTOM DISTRIBUTION

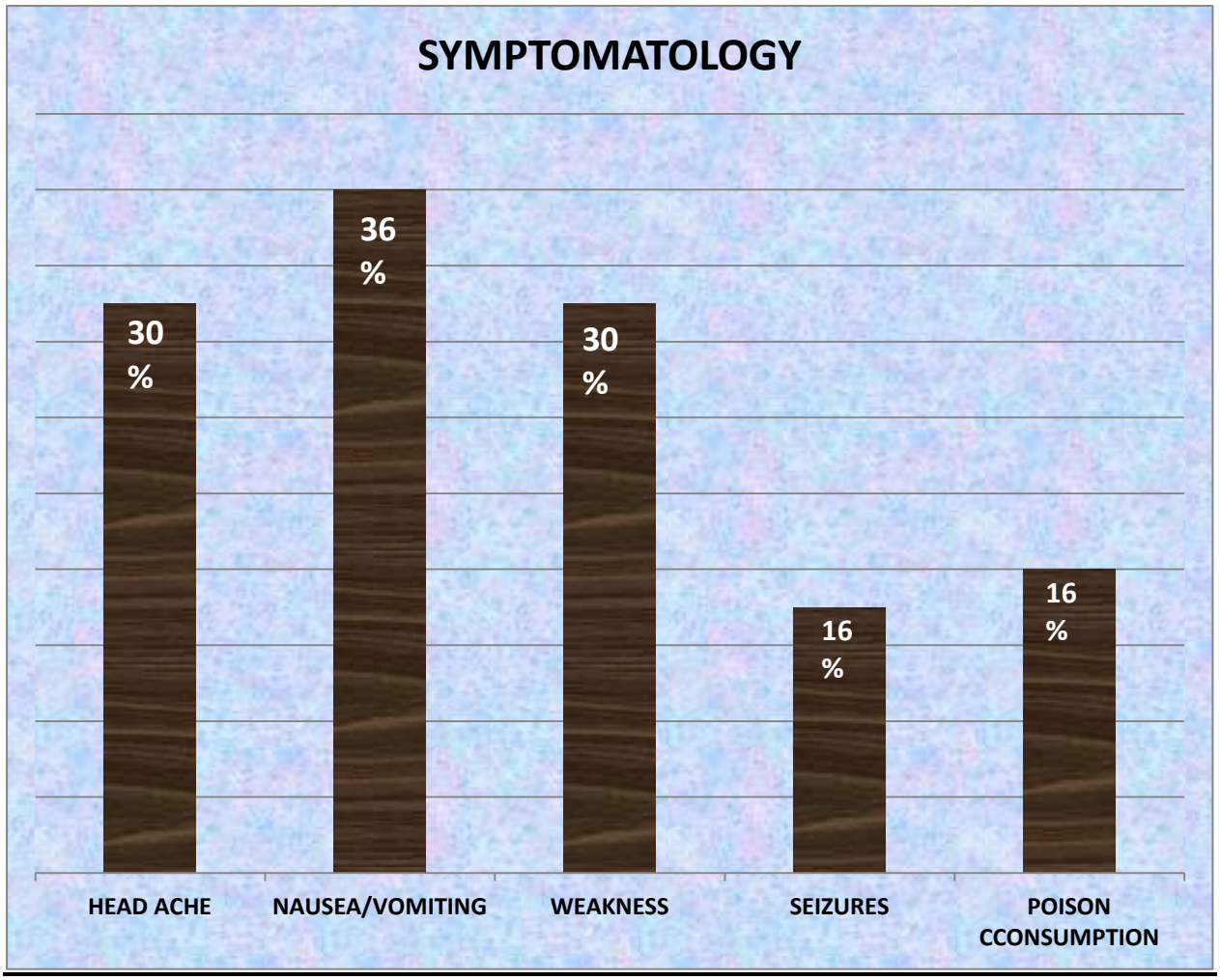
SYMPTOMS	NO OF PATIENTS	PERCENTAGE
HEAD ACHE	15	30 %
NAUSEA/VOMITING	18	36 %
WEAKNESS	15	30 %
SEIZURES	07	14 %
POISON CONSUMPTION	08	16%

THE MOST COMMON PRESENTING SYMPTOM IN THIS STUDY IS NAUSEA,VOMITING FOLLOWED BY HEADACHE, WEAKNESS AND SEIZURES.

HISTORY OF POISON CONSUMPTION IS PRESENT IN 8 PATIENTS.

THE VALUES ARE DEPICTED IN THE ABOVE TABLE AND A BAR DIAGRAM IS GIVEN BELOW.

CHART 4: SYMPTOM DISTRIBUTION



THIS BARS REPRESENT THE PICTORICAL

REPRESENTATION OF THE SYMPTOMS DISTRIBUTION

WITH PERCENTAGE MARKED INSIDE.

TABLE 5: PREVALENCE OF SYSTEMIC HYPERTENSION

SHT	No. of PATIENTS (n=50)	Percentage (100%)
YES	25	50.0
NO	25	50.0

50 % OF PATIENTS INCLUDED IN STUDY ARE HYPERTENSIVE.

PARTICULARLY MALES.

TABLE 6: PREVALENCE OF DIABETES MELLITUS

DIABETES MELLITUS	No.of Patients (n=50)	Percentage (100%)
No	29	58.0
Yes	21	42.0

COMPARED TO HYPERTENSION, THE PREVALENCE OF DIABETES IS LESS AROUND 42%.

TABLE 7: PREVALENCE OF CHRONIC LIVER DISEASE

CHRONIC LIVER DISEASE	No.of respondents (n=50)	Percentage (100%)
ABSENT	34	68.0
PRESENT	16	32.0

AMONG THOSE PRESENTED WITH COMA 32% HAVE HISTORY AND CLINICAL FEATURES OF CHRONIC LIVER DISEASE.

TABLE 8: PREVALENCE OF CHRONIC KIDNEY DISEASE

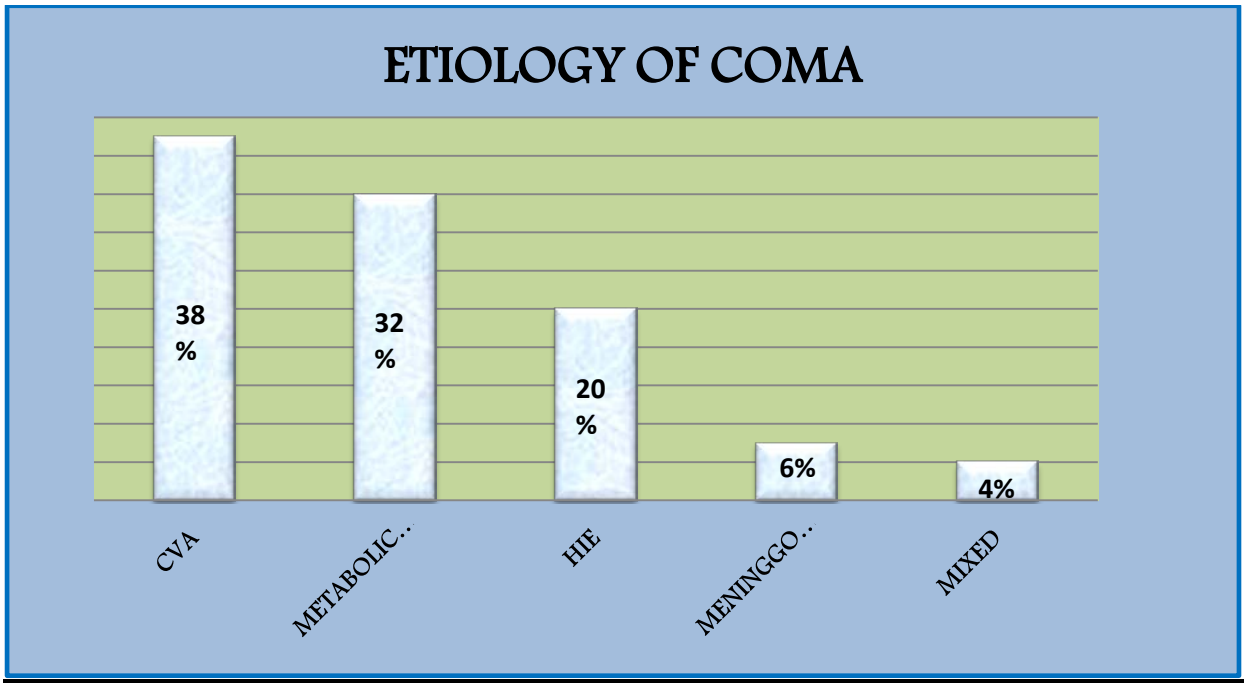
CHRONIC KIDNEY DISEASE	No. of Patients (n=50)	Percentage (100%)
No	41	82.0
Yes	9	18.0

18 % PATIENTS HAVE HISTORY AND LABORATORY EVIDENCE OF CHRONIC KIDNEY DISEASE .

TABLE 9: ETIOLOGY OF COMA

ETIOLOGY	NO.OF PATIENTS (N=50)	PERCENTAGE (100%)
CVA	19	38.0
HIE	10	20.0
HEPATIC COMA	6	12.0
URAEMIC COMA	7	14.0
ELECTROLYTEABNORMALITIES	2	4.0
MYXEDEMA COMA	1	2.0
MENINGOENCEPHALITIS	3	6.0
MIXED	2	4.0

CHART 5:



DURING MY STUDY PERIOD MOST OF THE COMA CASES ARE DUE TO CEREBRO VASCULAR ACCCIDENT (38 %) FOLLOWED BY METABOLIC ABNORMALITIES (36 %) THEN HYPOXIC ENCEPHALOPATHY (20%) .

- 2 CASES HAS MULTIPLE CONTRIBUTING FACTORS.
- AMONG THE METABOLIC ENCEPHALOPATHY HEPATIC ENCEPHALOPATHY IS COMMON FOLLOWED BY URAEMIC THEN OTHERS DUE TO ELECTROLYTE ABNORMALITIES.
- ONE CASE IS DUE TO MYXEDEMA COMA

TABLE 10: BACKGROUND SLOWING IN EEG

BACKGROUND SLOWING	No. of respondents (n=50)	Percentage (100%)
PRESENT	33	66.0
ABSENT	17	44.0

66% OF THE PATIENTS WITH COMA IN OUR STUDY HAD BACKGROUND SLOWING IN EEG.

CHART 6: BACK GROUND SLOWING IN EEG

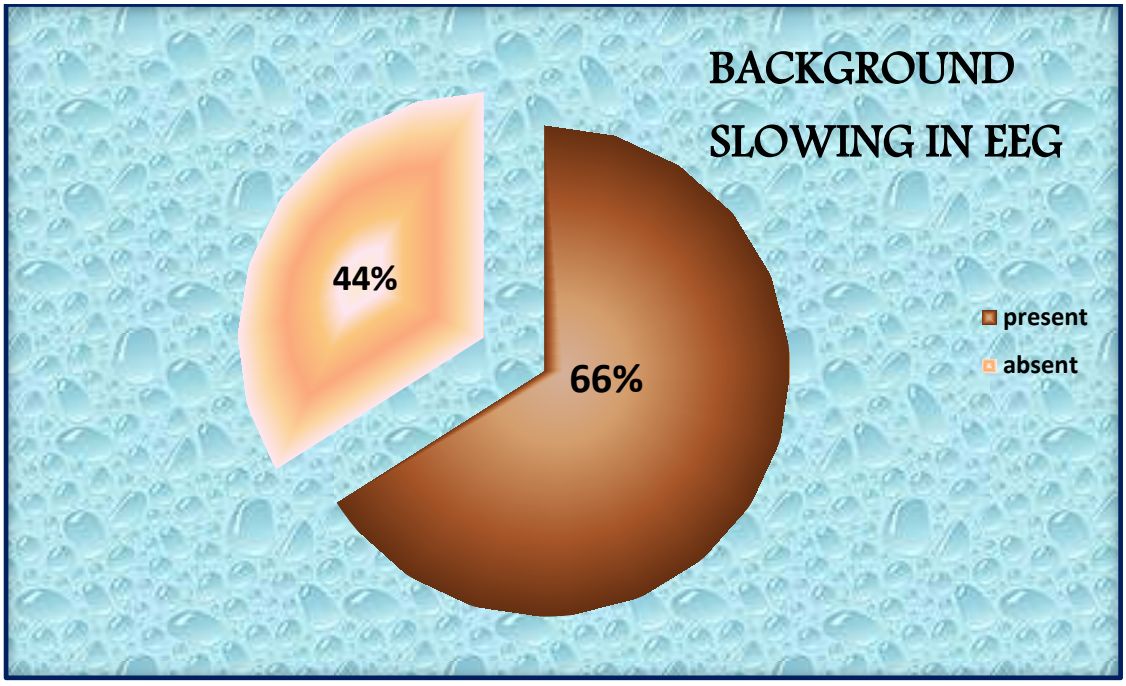


TABLE 11: BACKGROUND WAVES IN EEG

BACKGROUND WAVES IN EEG	No.of Patients (n=50)	Percentage (100%)
A	36	72.0
A/T	1	2.0
D	3	6.0
T	10	20.0

MOST COMMON TYPE OF COMA ENCOUNTERED IS ALPHA COMA IN 72% FOLLOWED BY THETA COMA THEN DELTA COMA.ONLY 1 INDIVIDUAL HAD ALPHA WITH THETA WAVE FORM.

CHART 7: DIFFERENT TYPES OF COMA IN EEG

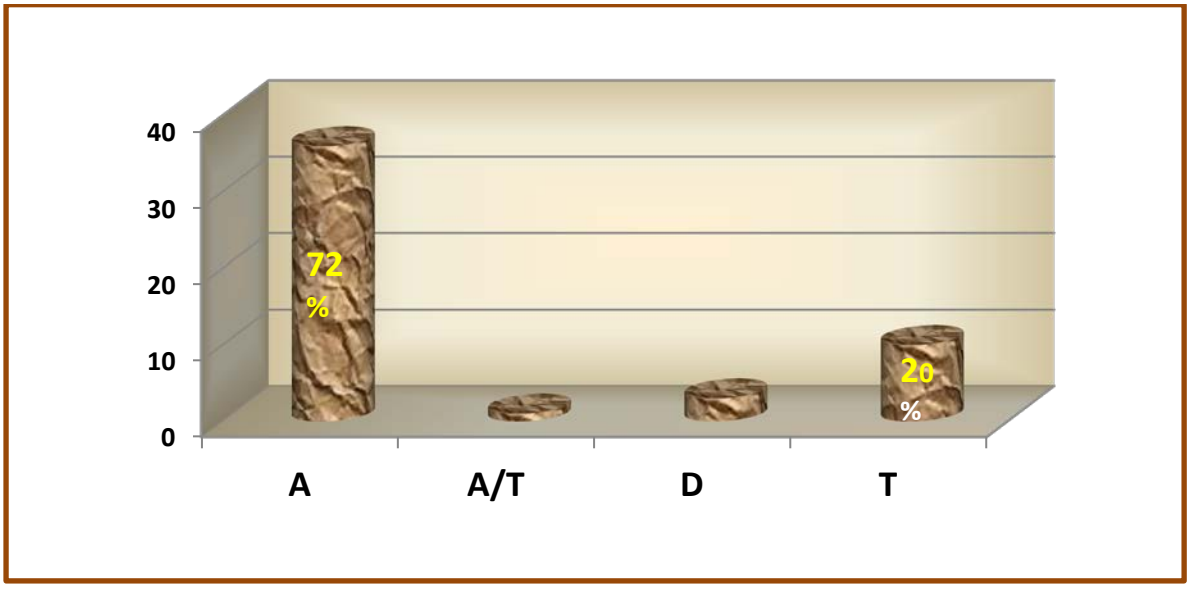


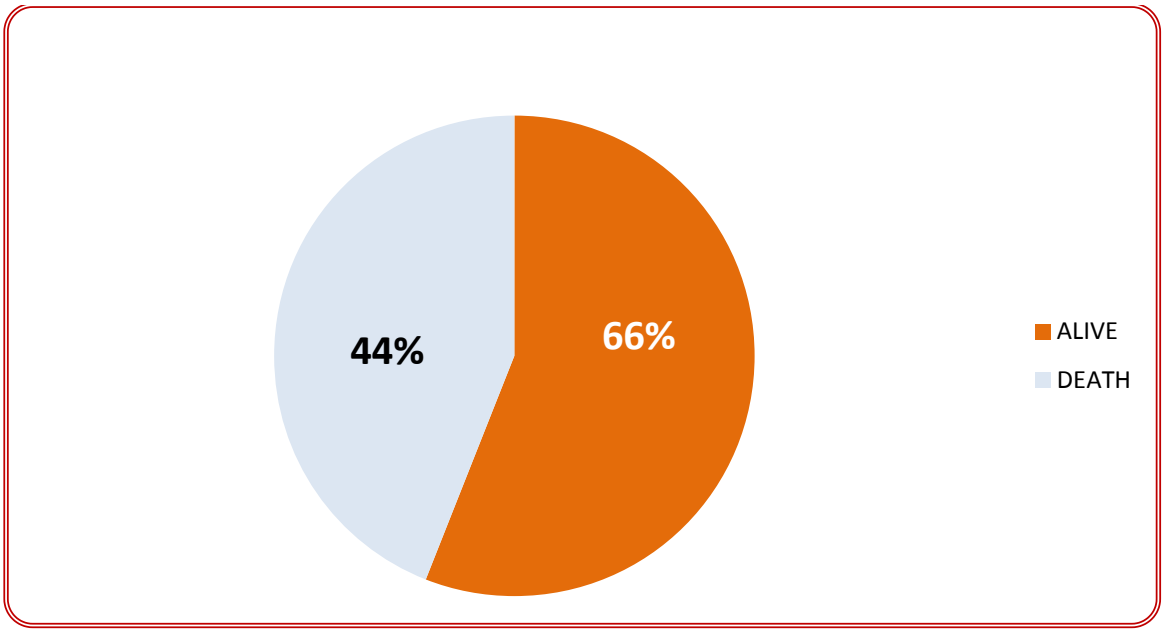
TABLE 12: MORTALITY IN THE STUDY GROUP

OUTCOME	No.of PATIENTS (n=50)	Percentage (100%)
Alive	28	56.0
Death	22	44.0

44% OF THE COMA PATIENTS DIED.

REMAINING 56% SURVIVVED WHICH ALSO INCLUDES THOSE WITH RESIDUAL NEUROLOGICAL DEFICIT.

MORTALITY RATE VARIES WITH RESPECT TO THE CAUSE OF COMA.



ANALYSIS OF RESULTS

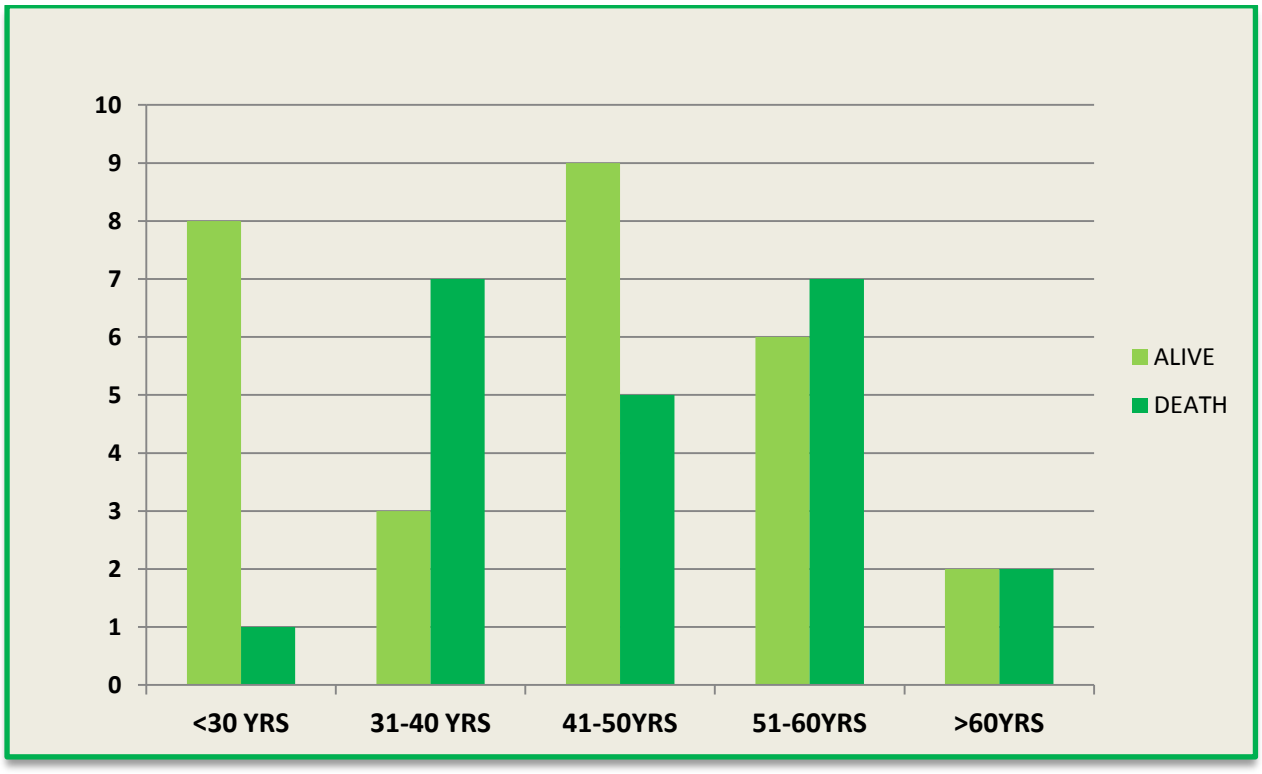
TABLE 13 & 14 : COMPARISON OF AGE WITH OUTCOME

THIS TABLE COMPARES THE MEAN AGE GROUP OF ALIVE PATIENTS WITH THAT OF MEAN AGE OF DEATH PATIENTS.

Age	Mean	S.D	Statistical inference
Alive (n=28)	43.54	14.878	T=-.888 Df=48 .379>0.05 Not Significant
Death (n=22)	46.86	10.526	

AGE	ALIVE		DEATH		TOTAL		STATISTICAL INFERENCE
	n	*%*	*n*	*%*	*n*	*%*	
Below 30yrs	8	28.6%	1	4.5%	9	18.0%	X^2=7.654 Df=4 .105>0.05 Not Significant
31 to 40yrs	3	10.7%	7	31.8%	10	20.0%	
41 to 50yrs	9	32.1%	5	22.7%	14	28.0%	
51 to 60yrs	6	21.4%	7	31.8%	13	26.0%	
61yrs & above	2	7.1%	2	9.1%	4	8.0%	

CHART 9: OUTCOME IN EACH AGE GROUP



ALTHOUGH THE MORTALITY APPEARS TO BE HIGH IN GROUPS 51-60 ;WHILE COMPARING IT TO THE PROPORTIONATE OF CASES IT DOESNOT APPEAR SIGNIFICANT STATISTICALLY.

THAT IS PROVED IN THE ABOVE TABLE BY CHI SQUARE TEST.

TABLE 15:COMPARISON OF OUTCOME WITH RESPECT TO SEX

SEX	ALIVE		DEATH		TOTAL		STATISTICAL INFERENCE
	N	*%*	*N*	*%*	*N*	*%*	
MALE	22	78.6%	20	90.9%	42	84.0%	X^2=1.395 DF=1 .238>0.05 NOT SIGNIFICANT
FEMALE	6	21.4%	2	9.1%	8	16.0%	
TOTAL	**28**	**100.0%**	**22**	**100.0%**	**50**	**100.0%**	

EVEN THOUGH FEMALES OCCUPY 16% IN THE STUDY POPULATION

THE MORTALITY IN FEMALES IS 9% AMONG THE TOTAL DEATH

WHICH IS STATISTICALLY NOT SIGNIFICANT.

THIS IS DONE BY CHI SQUARE TEST.

TABLE 16:COMPARISON OF DURATION OF COMA TO OUTCOME

DURATION	ALIVE		DEATH		TOTAL		STATISTICAL INFERENCE
	N	*%*	*N*	*%*	*N*	*%*	
<1 DAY	**22**	**78.6%**	**10**	**45.5%**	**32**	**64.0%**	**X^2=5.864 DF=1** **.015<0.05** **SIGNIFICANT**
>1 DAY	**6**	**21.4%**	**12**	**54.5%**	**18**	**36.0%**	

THIS TABLE DESCRIBES THE DISTRIBUTION OF PATIENTS WHO PRESENTED WITH COMA OF LESS THAN ONE DAY DURATION WITH THOSE WHO TOOK MORE THAN ONE DAY.

AMONG THE 64% WHO PRESENTED WITH IN ONE DAY ONLY CONTRIBUTED TO 45% OF THE MORTALITY.REMAINING 36% WHO PRESENTED LATE CONTRIBUTES TO REST OF THE 54% MORTALITY.

THUS DURATION DIFFERENCE IS STATISTICALLY SIGNIFICANT BY CHI SQUARE TEST.

TABLE 17 & 18:

COMPARISON OF SYMPTOMATOLOGY TO OUTCOME

WEAKNESS HAVE NO CORRELATION WITH RESPECT TO MORTALITY, WHERE AS SYMPTOMS OF INCREASED ICT LIKE NAUSEA VOMITING CORRELATED WITH MORTALITY.

NAUSEA/VOMITING	**Alive**		**Death**		**Total**		**Statistical inference**
	n	***%***	***n***	***%***	***n***	***%***	
No	22	78.6%	10	45.5%	32	64.0%	X^2=5.864 Df=1 .015<0.05 Significant
Yes	6	21.4%	12	54.5%	18	36.0%	

WEAKNESS	**Alive**		**Death**		**Total**		**Statistical inference**
	n	***%***	***N***	***%***	***n***	***%***	
No	20	71.4%	15	68.2%	35	70.0%	X^2=.062 Df=1 .804>0.05 Not Significant
Yes	8	28.6%	7	31.8%	15	30.0%	

TABLE 19,20,21,22:

COMPARISON OF RISK FACTORS WITH OUTCOME:

1. HYPERTENSION:

SHT	Alive		Death		Total		Statistical inference
	n	*%*	*n*	*%*	*n*	*%*	
No	18	64.3%	7	31.8%	25	50.0%	X^2=5.195 Df=1 .023<0.05 Significant
Yes	10	35.7%	15	68.2%	25	50.0%	
Total	**28**	**100.0%**	**22**	**100.0%**	**50**	**100.0%**	

AMONG THOSE WITH HYPERTENSION i.e. 50% OF THE STUDY POPULATION, 15 PATIENTS DIED WHICH CONSTITUTES ABOUT 68% OF THE DEATH. (STATISTICALLY SIGNIFICANT ASSOCIATION)

WHEN COMPARED TO HYPERTENSION, DIABETES MELLITUS ,CHRONIC LIVER DISEASE ,CHRONIC KIDNEY DISEASE DOES NOT SHOW STATISTICALLY SIGNIFICANT DIFFERENCE IN MORTALITY.

THIS IS DEPICTED IN BELOW TABLES.

2. DIABETES MELLITUS

DM	Alive		Death		Total		Statistical inference
	n	*%*	*n*	*%*	*N*	*%*	
No	19	67.9%	10	45.5%	29	58.0%	X^2=2.538 Df=1 .111>0.05 **NOT SIGNIFICANT**
Yes	9	32.1%	12	54.5%	21	42.0%	

3.CHRONIC LIVER DISEASE

CLD	Alive		Death		Total		Statistical inference
	n	*%*	*N*	*%*	*n*	*%*	
No	20	71.4%	14	63.6%	34	68.0%	X^2=.344 Df=1 .558>0.05 **NOT SIGNIFICANT**
Yes	8	28.6%	8	36.4%	16	32.0%	

4. CHRONIC KIDNEY DISEASE

CKD	Alive		Death		Total		Statistical inference
	n	*%*	*n*	*%*	*N*	*%*	
No	24	85.7%	17	77.3%	41	82.0%	X^2=.595 Df=1 .441>0.05 **NOT SIGNIFICANT**
Yes	4	14.3%	5	22.7%	9	18.0%	

TABLE 23,24:

COMPARISON OF BRAIN STEM REFLEX FINDINGS WITH OUTCOME

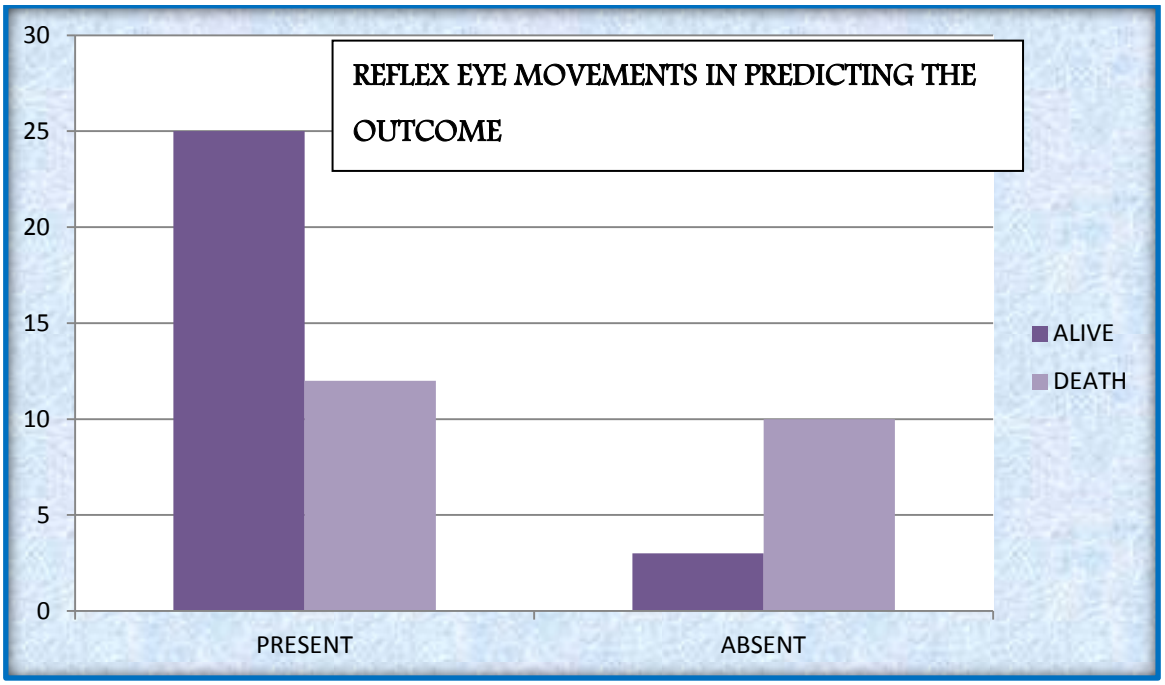
REFLEX EYE MOVEMENTS	Alive		Death		Total		Statistical inference
	n	*%*	*N*	*%*	*N*	*%*	
ABSENT	3	10.7%	10	45.5%	13	26.0%	X^2=7.728 Df=1 .005<0.05 Significant
PRESENT	25	89.3%	12	54.5%	37	74.0%	

THESE TABLES INDICATE THAT THOSE NOT HAVING REFLEX EYE MOVEMENTS AND SLUGGISH PUPILLARY REACTION HAVE MORE MORTALITY THAN THOSE WITH NORMAL SIGNS.

THESE VALUES ARE STATISTICALLY SIGNIFICANT IN PREDICTING THE MORATLITY AS SHOWN IN CHI SQUARE TEST.

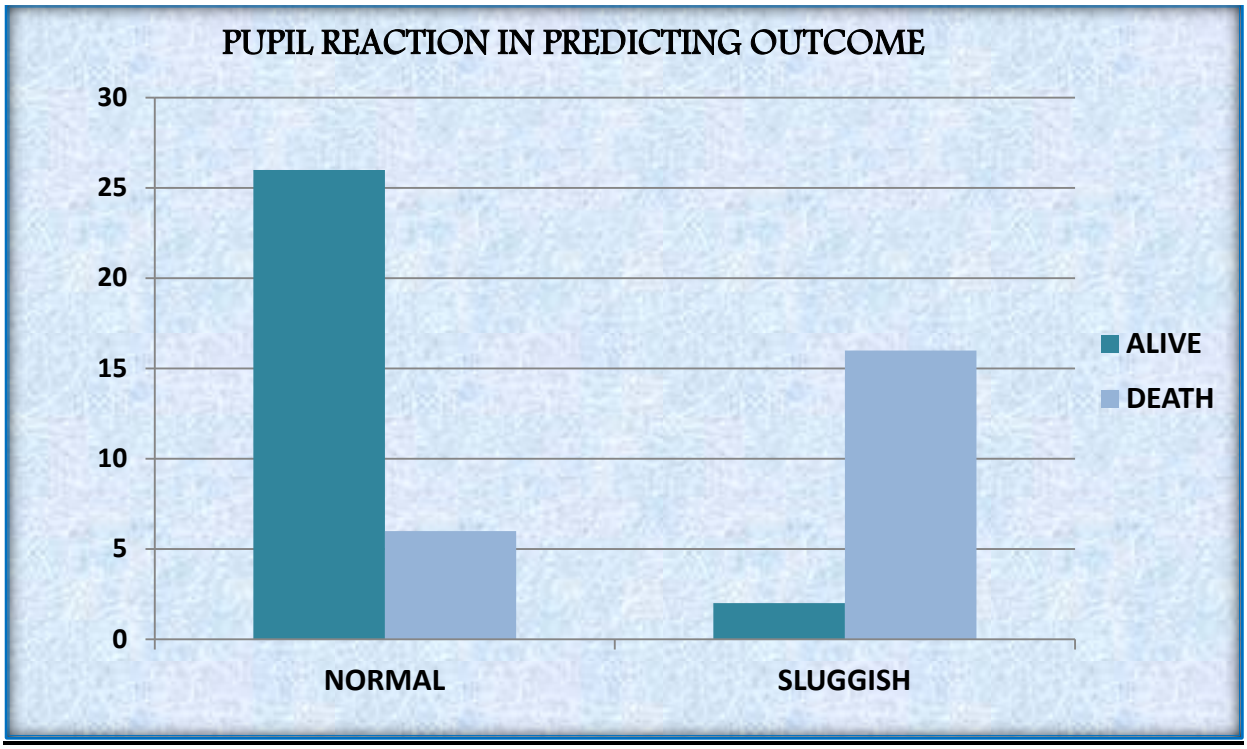
PUPIL REACTION	Alive		Death		Total		Statistical inference
	n	*%*	*n*	*%*	*n*	*%*	
NORMAL	26	92.9%	6	27.3%	32	64.0%	X^2=23.000 Df=1 .000<0.05 Significant
SLUGGISH	2	7.1%	16	72.7%	18	36.0%	

CHART 10: REFLEX EYE MOVEMENTS IN PREDICTING THE OUTCOME



THESE CHARTS INDICATE THE SIGNIFICANCE OF BRAIN STEM REFLEX IN OUTCOME OF THE PATIENT.FOR EXAMPLE THE DEATH IS HIGHER IN THOSE WITH REDUCED REFLEX EYE MOVEMENTS OR SLUGGISH PUPPILARY REACTION.

CHART 11: PUPILLARY REACTIONS IN PREDICTING THE OUTCOME



THOSE WHO ARE HAVING SLUGGISH PUPILLARY REACTION HAVE COMPARATIVELY MORE MORTALITY THAN THOSE HAVING NORMAL PUPILS.

IT IS ALSO PROVEN STATISTICALLY SIGNIFICANT.

TABLE 25: COMPARISON OF OUTCOME IN RELATION TO PAPILLEDEMA

PAPILLEDEMA	Alive		Death		Total		Statistical inference
	n	*%*	*n*	*%*	*n*	*%*	
NO	28	100.0%	11	50.0%	39	78.0%	X^2=17.949 Df=1 .000<0.05 Significant
YES	0	0%	11	50.0%	11	22.0%	

22 % PATIENTS IN THE STUDY HAD PAPILLEDEMA. 50% AMONG THOSE WITH PAPILLEDEMA DIED. THIS FINDING HAS SIGNIFICANT ASSOCIATION WITH MORTALITY AS PROVEN STATISITICALLY.

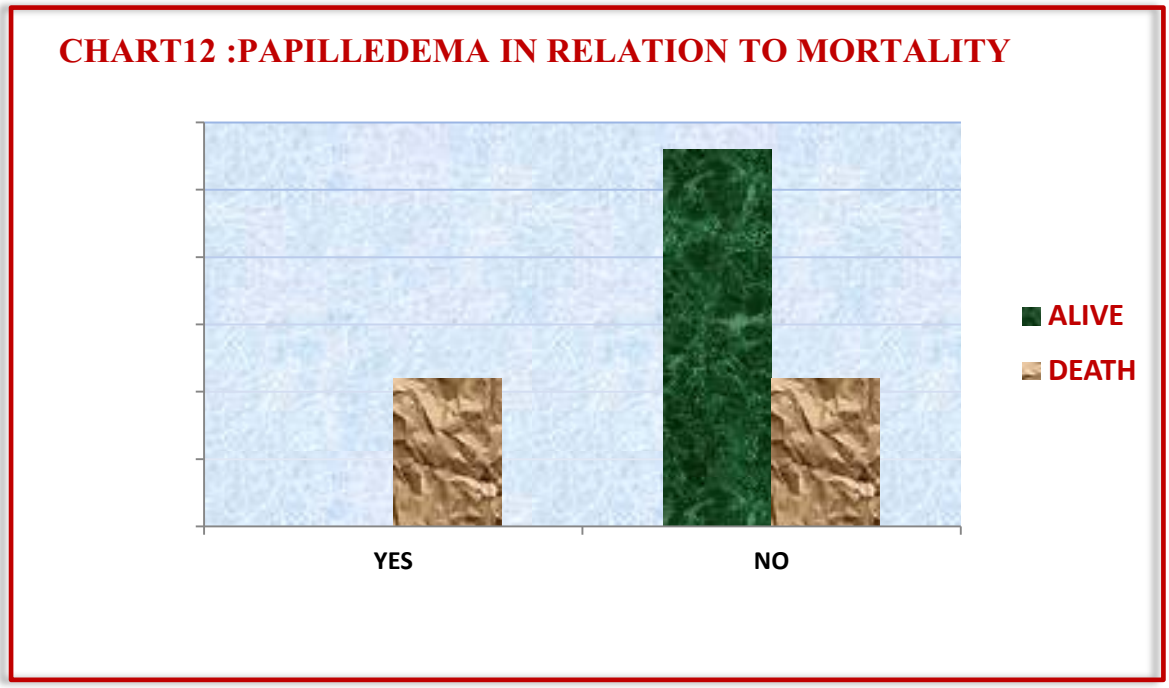


TABLE 26: COMPARISON OF REPIRATORY MOVEMENTS IN PREDICTING THE OUTCOME

RESPIRATORY MOVEMENTS	Alive		Death		Total		Statistical inference
	n	*%*	*n*	*%*	*n*	*%*	
NORMAL	22	78.6%	6	27.3%	28	56.0%	X^2=13.158 Df=1 .000<0.05 Significant
ABNORMAL	6	21.4%	16	72.7%	22	44.0%	

RESPIRATORY MOVEMENTS IN MOST OF THE COMATOSE PATIENTS ARE NORMAL.

44 % HAD ABNORMAL MOVEMENTS. THEY CONSTITUTE 72 % OF THE TOTAL DEATH.

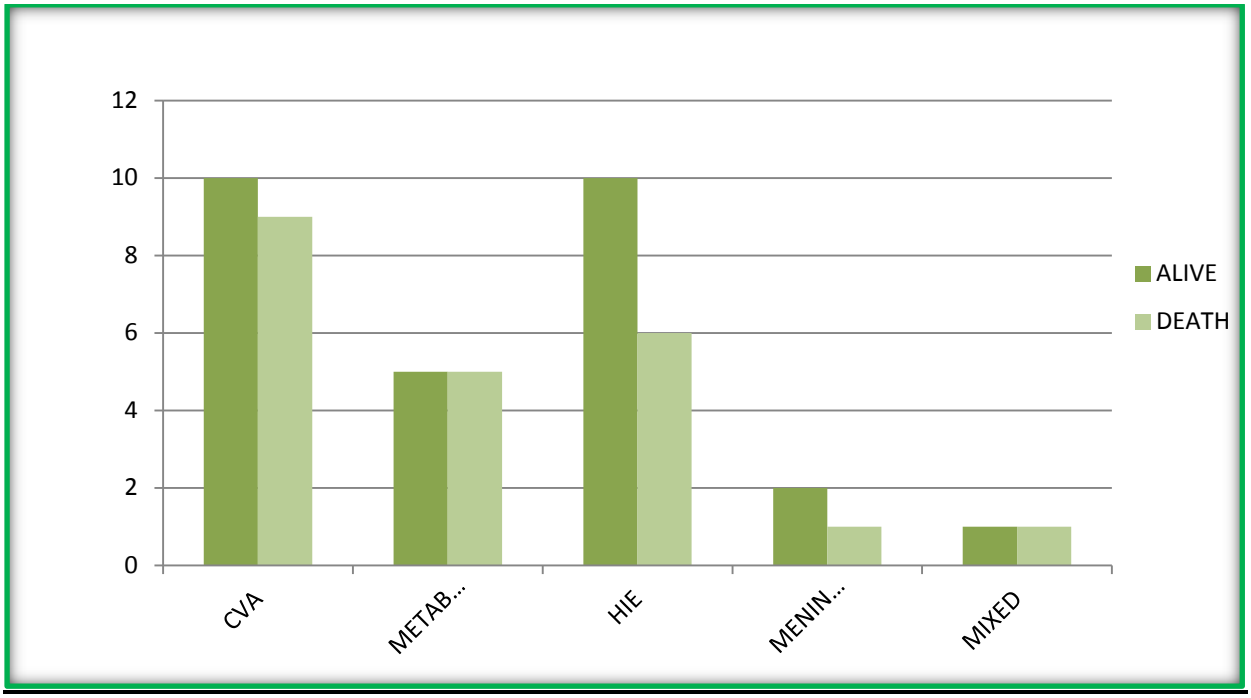
THIS ASSOCIATION IS ALSO PROVEN STATISTICALLY SIGNIFICANT.

TABLE 27: COMPARISON OF OUTCOME WITH RESPECT TO ETIOLOGY

ETIOLOGY	ALIVE		DEATH		TOTAL		STATISTICAL INFERENCE
	n	*%*	*N*	*%*	*n*	*%*	
CVA	10	35.7%	9	40.9%	19	38.0%	X^2=1.497 Df=7 .982>0.05 Not Significant
HIE	5	17.9%	5	22.7%	10	20.0%	
HEPATIC COMA	4	14.3%	2	9.1%	6	12.0%	
URAEMIC COMA	4	14.3%	3	13.6%	7	14.0%	
ELECTROLYTE DISTURBANCE	1	3.6%	1	4.5%	2	4.0%	
MYXEDEMA COMA	1	3.6%	0	.0%	1	2.0%	
MENINGOENCEPHALITIS	2	7.1%	1	4.5%	3	6.0%	
MIXED	1	3.6%	1	4.5%	2	4.0%	

THERE IS A DIVERSE DISTRIBUTION FOR CAUSE OF COMA IN OUR STUDY.

CHART 13: COMPARISON OF MORTALITY IN RESPECT TO PARTICULAR ETIOLOGY



IT IS CLASSIFIED UNDER 4 DIFFERENT HEADINGS.

1. CEREBROVASCULAR ACCCIDENT.

2. METABOLIC (HEPATIC COMA, URAEMIC COMA, MYXEDEMA COMA, HYPONATREMIA)

3. HYPOXIC ISCHEMIC ENCEPHALOPATHY.

4. MENINGOENCEPHALITIS.

IN OUR STUDY COMA SECONDARY TO CEREBROVASCULAR ACCIDENT (40.9%) REMAINS THE MOST COMMON CAUSE.

FOLLOWED BY METABOLIC ENCEPHALOPATHY (27.2%) THEN HIE (22.7 %.). 2 CASES IN OUR STUDY HAVE MIXED ETIOLOGIES.

TABLE 28

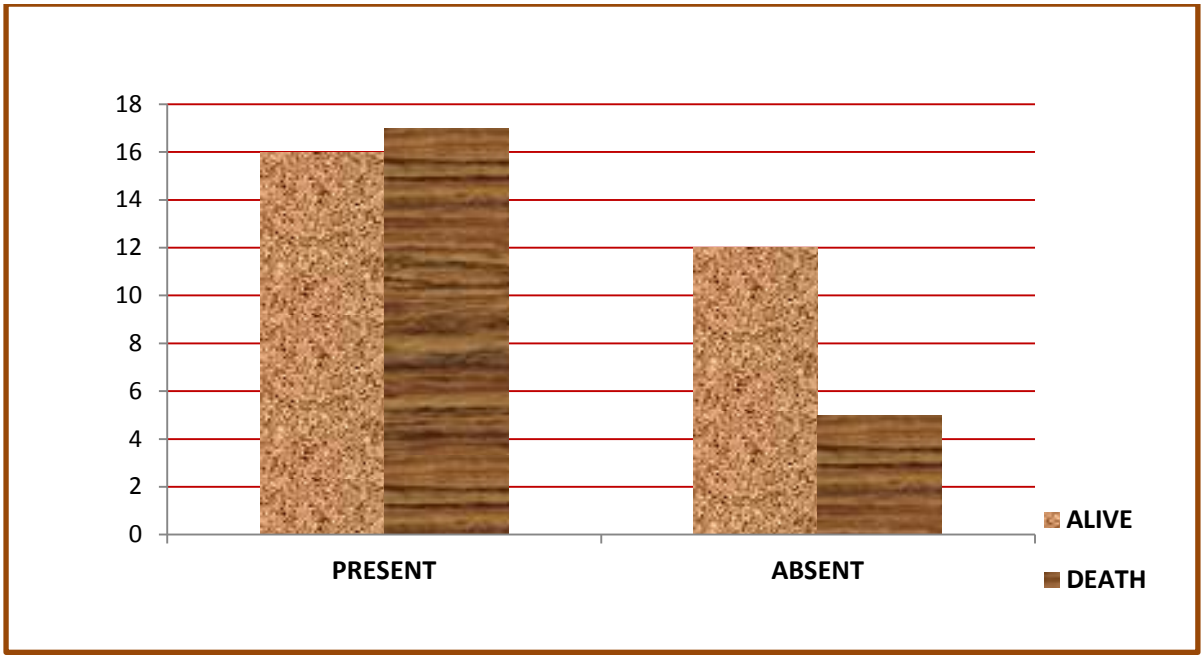
BACKGROUND SLOWING OF EEG IN RELATION TO MORTALITY

SLOWING	ALIVE		DEATH		TOTAL		STATISTICAL INFERENCE
	N	*%*	*N*	*%*	*N*	*%*	
ABSENT	12	42.9%	5	22.7%	17	34.0%	X^2=2.225 DF=1 .136>0.05 NOT SIGNIFICANT
PRESENT	16	57.1%	17	77.3%	33	66.0%	

EVEN THOUGH 66% OF THE PATIENTS HAVE SLOWING IT DOES NOT SHOW ANY SIGNIFICANT ASSOCIATION WITH MORTALITY.

AMONG THE 28 ALIVE PATIENTS 16 PATIENTS HAD SLOWING OF BACKGROUND RHYTHM.BUT IN 22 DEATH PATIENTS 7 HAD SLOWING.EVEN IT APPEARS TO BE HIGHER, IT IS NOT STATISTIALLY SIGNIFICANT.

CHART 14: BACK GROUND SLOWING IN RELATION TO OUTCOME



THERE IS NO STATISTICALLY MUCH SIGNIFICANT DIFFERENCE IN MORTALITY WITH RESPECT TO BACKGROUND SLOWING WHICH IS ALSO SHOWN IN THE ABOVE BAR CHART.

TABLE 29: COMPARING THE OUTCOME IN RESPECT TO INITIAL GCS

GCS	Alive		Death		Total		Statistical inference
	N	*%*	*n*	*%*	*n*	*%*	
3 to 5	8	28.6%	21	95.5%	29	58.0%	X^2=22.624 Df=1 .000<0.05 Significant
6 to 8	20	71.4%	1	4.5%	21	42.0%	

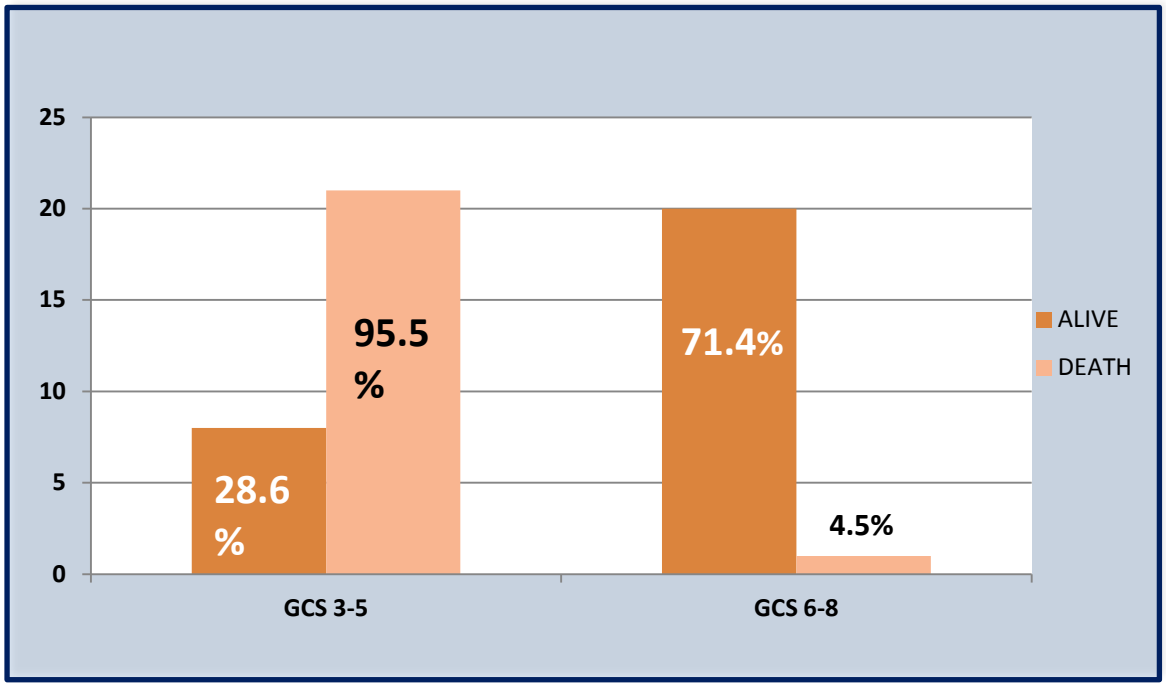
BASED ON GCS THE STUDY GROUP IS CLASSIFIED IN TO 2 GROUPS ONE FROM 3-5 & ANOTHER FROM 6-8.

.

THERE IS A SIGNIFICANT ASSOCIATION BETWEEN THE INITIAL GCS AND MORTALITY.

IT IS PROVEN STATISTICALLY BY CHI SQUARE TEST.

CHART 15: COMPARISON OF MORTALITY IN RESPECT TO INITIAL GCS



95.5% DEATH ARE IN THE FIRST GROUP WHICH INDICATES THAT CHANCE OF REOVERY IS VERY LESS IN 3-5 GROUP.

BUT AROUND 28.6% IN THE GCS OF 3- 5 SURVIVED WHICH INDICATES THERE ARE CHANCES OF RECOVERY EVEN WITH POOR GCS.

IT IS DETERMINED BY FACTORS OTHER THAN GCS LIKE ETIOLOGY.

STILL GCS IS SIGNIFICANTLY STRONG FACTOR IN DETERMINING THE MORTALITY.

TABLE 30: COMPARISON OF MORTALITY WITH RESPECT TO BACKGROUND

EEG

BACKGROUND	ALIVE		DEATH		TOTAL		STATISTICAL INFERENCE
	n	*%*	*n*	*%*	*n*	*%*	
A	27	96.4%	9	40.9%	36	72.0%	X^2=18.953 Df=3 .000<0.05 Significant
A/T	0	0%	1	4.5%	1	2.0%	
D	0	0%	3	13.6%	3	6.0%	
T	1	3.6%	9	40.9%	10	20.0%	

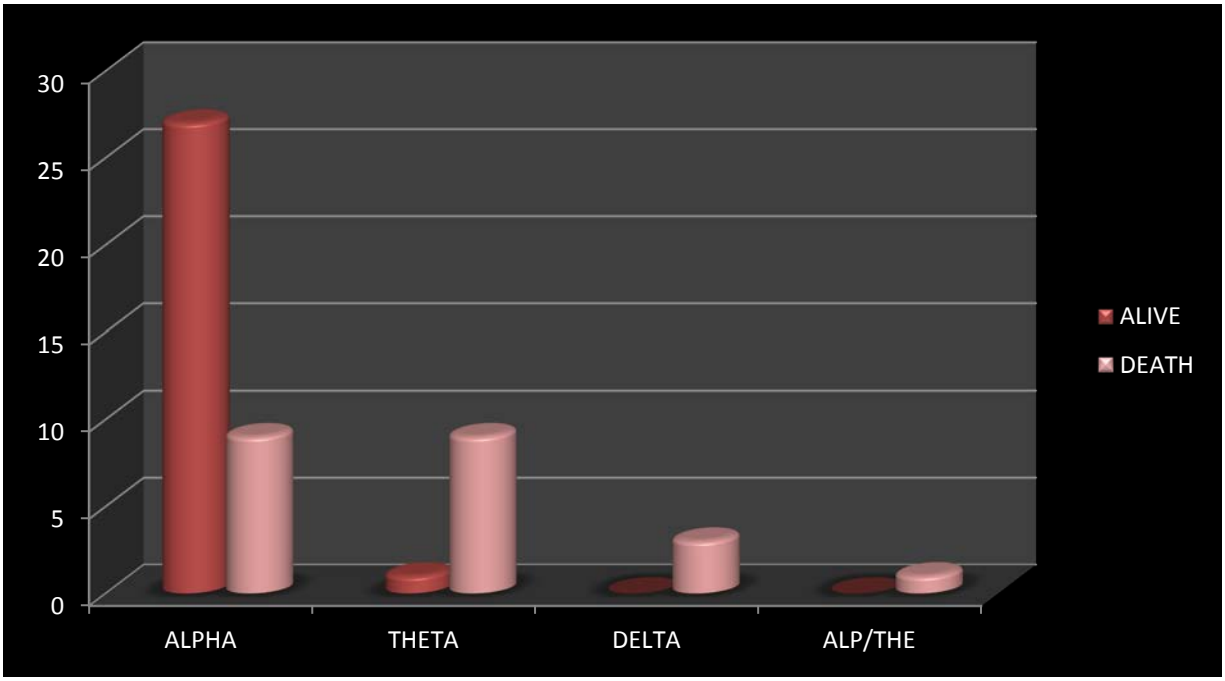
72 % OF THE STUDY POPULATION HAVE ALPHA COMA. 20% HAD THETA COMA. REMAINING 6% HAS DELTA COMA.

AMONG THE 10 PATIENTS WHO HAD THETA COMA ALMOST 9 DIED WHICH CONTRIBUTES TO 40% MORTALITY.

ONLY 3 PATIENTS HAD DELTA COMA BUT ALL THE THREE PATIENTS EXPIRED.THIS DELTA COMA PATIENTS CONTRIBUTES TO 13.6% MORTALITY.

AMONG THE 36 PATIENTS WITH ALPHA COMA ,9 DIED WHICH CONTRIBUTES TO 40.9% MORTALITY.

CHART 14: COMPARISON OF MORTALITY IN RESPECT TO TYPE OF COMA IN EEG



THIS GRAPH INDICATES ALMOST NO PATIENTS WITH DELTA AND THETA WAVE SURVIVED.

SO THERE IS A SIGNIFICANT ASSOCIATION OF THE BACKGROUND TYPE OF COMA WITH THE MORTALITY.

TABLE 31: COMPARISON OF BACKGROUND EEG WAVE IN RESPECT TO ETIOLOGY OF COMA.

ETIOLOGY	Background										Statistical inference
	A		A/T		D		T		Total		
CVA	14	38.9%	0	.0%	1	33.3%	4	40.0%	19	38.0%	X^2=36.276 Df=21 .020<0.05 Significant
HIE	8	22.2%	0	.0%	0	.0%	2	20.0%	10	20.0%	
M1	5	13.9%	0	.0%	0	.0%	1	10.0%	6	12.0%	
M2	5	13.9%	0	.0%	1	33.3%	1	10.0%	7	14.0%	
M3	1	2.8%	0	.0%	0	.0%	1	10.0%	2	4.0%	
M4	0	.0%	0	.0%	0	.0%	1	10.0%	1	2.0%	
ME	2	5.6%	0	.0%	1	33.3%	0	.0%	3	6.0%	
MIXED	1	2.8%	1	100.0%	0	.0%	0	.0%	2	4.0%	

M1 – HEPATIC COMA; M2 – URAEMIC COMA; M3 – ELECTROLYTE DISTURBANCES M4 – MYXEDEMA COMA.

.

THE MOST COMMON PATTERN ENCOUNTERED IS ALPHA COMA.IT IS SEEN IN COMA DUE TO ALMOST ALL ETIOLOGIES.

DELTA COMA IN OUR STUDY IS SEEN IN THREE DIFFERENT TYPES OF COMA. COMA DUE TO INCREASED ICT, URAEMIC COMA, MENIGOENCEPHALITIS.

THETA COMA IS SEEN IN HYPOXIC ISCHEMIC ENCEPHALOPATHY.

CHART 15: DISTRIBUTION OF COMA IN EEG WITH RESPECT TO ETIOLOGY

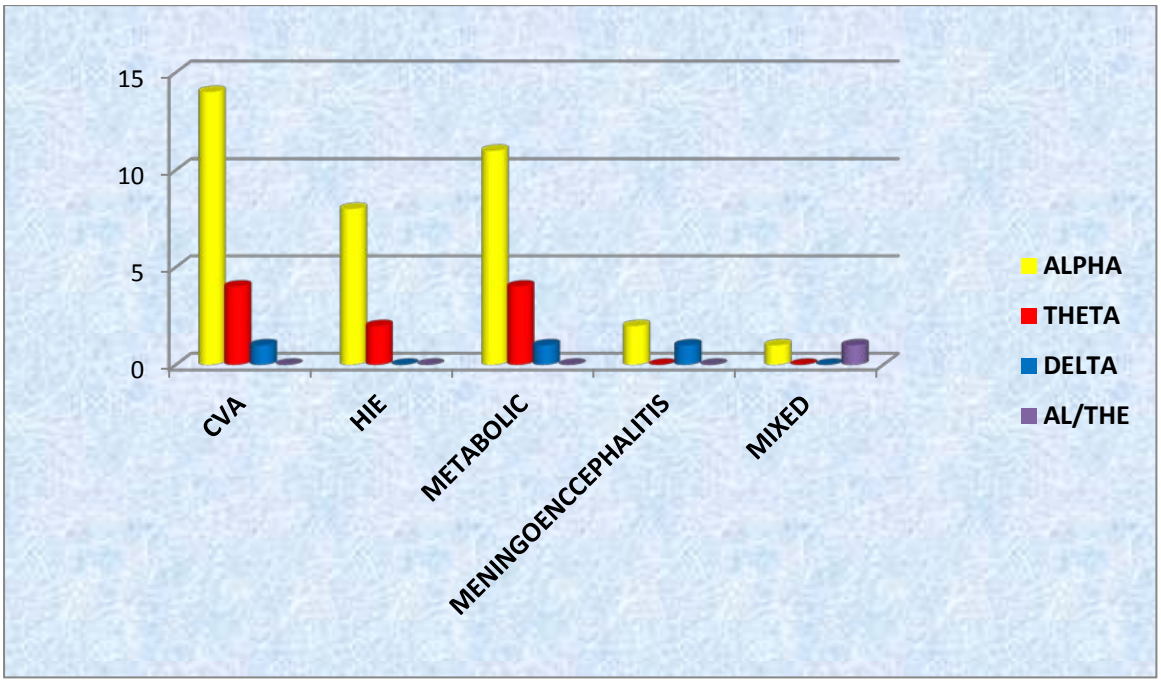


TABLE 32: DISTRIBUTION OF ALIVE PATIENTS WITH RESPECT TO ETIOLOGY AND BACKGROUND WAVES.

ETIOLOGY	A		T		Total		Statistical inference
CVA	10	37.0%	0	.0%	10	35.7%	X^2=28.000 Df=7 .000<0.05 SIGNIFICANT
HIE	5	18.5%	0	.0%	5	17.9%	
M1	4	14.8%	0	.0%	4	14.3%	
M2	4	14.8%	0	.0%	4	14.3%	
M3	1	3.7%	0	.0%	1	3.6%	
M4	0	.0%	**1**	**100.0%**	1	3.6%	
ME	2	7.4%	0	.0%	2	7.1%	
MIXED	1	3.7%	0	.0%	1	3.6%	
Total	***27***	***100.0%***	***1***	***100.0%***	***28***	***100.0%***	

ONLY ONE PATIENT HAVING THETA WAVE SURVIVED.THAT PATIENT IS A CASE OF MYXEDEMA COMA WHO RECOVEREDTO STEROIDS.

TABLE 33: DISTRIBUTION OF DEATH PATIENTS WITH RESPECT TO ETIOLOGY AND BACKGROUND WAVES

ETIOLOGY	BACKGROUND										STATISTICAL INFERENCE
	A		A/T		D		T		TOTAL		
CVA	4	44.4%	0	.0%	1	33.3%	4	44.4%	9	40.9%	X^2=32.158 DF=18 .021<0.05 SIGNIFICANT
HIE	3	33.3%	0	.0%	0	.0%	2	22.2%	5	22.7%	
M1	1	11.1%	0	.0%	0	.0%	1	11.1%	2	9.1%	
M2	1	11.1%	0	.0%	1	33.3%	1	11.1%	3	13.6%	
M3	0	.0%	0	.0%	0	.0%	1	11.1%	1	4.5%	
ME	0	.0%	0	.0%	1	33.3%	0	.0%	1	4.5%	
MIXED	0	.0%	1	100.0%	0	.0%	0	.0%	1	4.5%	

THERE IS A STATISTICALLY SIGNIFICANT ASSOCIATION BETWEEN THE WAVE PATTERN AND ETIOLOGY PROVEN BY CHI SQUARE TEST.

DISCUSSION

Coma still being one of the important un adventured medical emergency. Many studies are needed to clarify the concepts in diagnosis and management of coma.

Our study is one such study about the clinical profile of non traumatic coma and use of electroencephalogram along with clinical findings to correlate the outcome.

AGE & SEX DISTRIBUTION:

This is a cross sectional observation study consist of 50 cases of non traumatic coma.

Among them 42 were males and 8 females. Most of them fall within 40 – 60 age group.

Even more number of death occur in age group more than 50. There was not statistically significant association with respect to age and sex.

Most of studies quoted this similar male sex predominance in non traumatic coma[68].

THE OUTCOME IN OUR STUDY IS NOT AFFECTED BY SEX AS WELL AS AGE.

Lukman Femi Owolabi et al.,[64] conducted a similar study in northern Nigeria regarding the factors predicting the mortality at the end of one month.

In their study also there is a male predominance. The mean age group in their study is 53.7 where as in our study the mean age group is 45.

ETIOLOGY

Coming next to the etiology of coma in our study CVA remains the most common cause of coma followed by metabolic encephalopathy which includes hepatic , uraemic, electrolyte disturbances and miscellaneous later hypoxic ischemic encephalopathy then CNS infection finally coma of mixed etiology.

This descending order for the cause of coma is seen in many Indian studies (Sharma et al., & Thacker et al.,) [71,70] & also in famous Plum and Posner literature, but the there is a gross difference in percentage.

In our study even cerebrovascular accident remains the most common cause around 38% , metabolic encephalopathy contributes to 32% i.e. almost equal.

Though the type and causes of CVA varies among the study population the traditional risk as well as causative factor hypertension was assessed. Among the 19 patients of CVA 12 had hypertension almost 64%.

In the total study population 50% had hypertension, 42% had diabetes mellitus.

IN OUR STUDY HYPERTENSION HAS A SIGNIFICANT ASSOCIATION IN THE OUTCOME WHICH IS PROVEN STATISTICALLY.

Other risk factors like diabetes mellitus , chronic liver disease ,chronic kidney disease has no statistical association with the outcome.

DURATION OF COMA

In our study we have taken two groups of patients, those who presents with in a day and those after 1 day. The time duration has significantly affected the outcome as proven statistically.

Obiako et al.,[73] from Nigeria studied the delayed diagnosis of cause of coma after 24 hours has significant mortality of 85.4% compared to 70% in the group presented with in a day. Apart from the duration of coma in his study he compared the role of family both in terms of support and on the knowledge on the cause of coma had a significant on the outcome of coma.

SYMPTOMATOLOGY

In our study except for symptoms of increased intracranial tension other symptoms has no significant correlation with the outcome.

There are no exact studies to correlate the symptoms with outcome of the patient.

GCS

In our study the morality rate was very high in those with GCS from 3-5 almost 96% of the death. Whereas only one patient died even with GCS of more than 5.

Most of the poor GCS in our study are due to intra cranial hemorrhage.

Various studies has shown similar results (Thacker et al.,)[70].

The chance of waking up normally is 7 times higher than those who presents with poor GCS initially.

As with all other studies in our study also GCS remains a reliable predictor of mortality.

SIGNS

Any abnormal neuro-ophthalmological sign at the time of presentation is always a precursor to worst outcome. In this study we have taken 4 things in to account. They are spontaneous eye movements, pupillary reaction , papilledema and abnormal respiratory movements.

All the 4 components has been significantly associated with mortality which has been proved statistically.

Before considering these signs two things should be ruled out. One is inter observer variability in pupil observation. Other thing to be ruled out is oculomotor nerve involvement.

Most of the studies regarding brain stem reflexes as a prognostic indicator are in traumatic patients. But their significance is clearly depicted in those studies.

Brakman et al., [75] in his study identified 5 promising prognostic indicators in the outcome of coma which include age, duration of coma, GCS, pupillary reaction to light, spontaneous reflex eye movements. He also found a set of days (0,1, 3, 7, 14, 28) in which the above said items to be assessed periodically for better assessment of prognosis.

In his study there was a significant correlation between all the parameters described above with the coma outcome.

IN OUR STUDY EXCEPT FOR AGE ALL THE OTHER HAVE A STATISTICALLY SIGNIFICANT CORRELATION IN PREDICTING THE OUTCOME.

David E et al., [76]did a follow up study for one year in five hundred non traumatic comatose patients for identifying prognostic factors. He concluded that functional recovery depends not on the age and to some extent on the cause of the coma. According to him the recovery is best in miscellaneous cause and in hepatic coma and poor in SAH and other CVDs. This study also identifies the significance of early oculo vestibular response and pupillary response as a predictor of outcome.

IN OUR STUDY THE MOST COMMON CAUSE IS CVA FOLLOWED BY METABOLIC WITH RECOVERY BEST IN METABOLIC COMA LIKE HEPATIC AND MYXEDEMA COMA.

Levy et al., [74] selected a group of patients in vegetative state from a collaborative study containing a group of five hundred non traumatic coma patients. These patients have preserved brain stem function to some extent. But they will have cortex dysfunction. The most common cause being HIE. In these patients' roving eye movements remains the predictor of coma outcome in the one year of follow up.

IN OUR STUDY WE HAVE NOT INCLUDED THE PATIENTS IN VEGETATIVE STATE GROUP.

Arun bansal et al., in a study of non traumatic coma patients in pediatric age group identified that abnormal respiratory pattern and poor pupillary response as a significant sign to predict the mortality. He concludes that simple bed side test are good predictors of coma outcome.

IN OUR STUDY 44 % HAD ABNORMAL MOVEMENTS. THEY CONSTITUTE 72 % OF THE TOTAL DEATH. THERE IS A STATISTICALLY SIGNIFICANT ASSOCIATION BETWEEN ABNORMAL RESPIRATORY MOVEMENTS AND OUTCOME

MORTALITY

THE MORTALITY RATE IN OUR STUDY IS 44%.

There is a gross difference in mortality rate in various studies which is 24% in a study from Sweden to 85% in south Nigeria[64] .

This difference is mainly attributed to varied cause of coma in these studies. Those studies in which the contribution from metabolic coma is higher the mortality rate will be less.

The very high rate in Nigeria is attributed to the non availability of sufficient intensive care facilities even in a tertiary care set up.

SIGNIFICANCE OF ELECTROENCEPHALOGRAM

Bagnato S et al ., [78] investigated the significance of electroencephalogram in disorders of consciousness then followed by coma. They classified the patients under two groups those with or without trauma.

Clinical assessment was done by LCF scaling. EEG done and their severity was classified by Synek. Both these scaling system was done at the time of admission and after three months. Both these scores correlate with each other. So this scoring system can be taken for consideration in assessing the severity.

Filippo Donati et al .,[79] selected a set of 14 coma patients following cardiac arrest of diverse etiologies. Clinical assessment was done by a fixed protocol. Serial Clinical examinations, Electroencephalogram, evoked potentials (SEP) were done.

The EEG patterns are classified in to complete alpha theta activity and incomplete activity. The patients are followed up for 1 year.

They concluded that most of the patients with complete Alpha Theta Coma have very poor prognosis, where as those with incomplete alpha theta coma have the chance of getting full recovery at one year. Combination of above said clinical findings, EEG findings & evoked potentials improve the accuracy of prognosis in all post anoxic states.

Retrospective analysis was done on triphasic waves. Three components were assessed one is slowness, dominancy of location, time lag. Most common cause encountered is hepatic coma but it is also seen in uraemic coma, anoxic, hyper osmolar state. It is not specific to any etiology of coma.

Young B et al., [80] analyzed the importance of electroencephalogram in coma patients because it allows to assess the thalamo cortical circuits which cannot be tested clinically. He concluded that in post cardiac arrest coma electroencephalograms are hundred percent specific to assess the probability of recovering. If there is a generalized suppression 24 hours after the arrest no chance of recovery is there.

Kaplan PW quoted the works of **Fischgold and Mathis** who identified the specific changes in EEG patterns as the level of coma deepens. They also suggested that correlation is best with anoxic injuries to the brain following arrest. It act as a rough guide to differentiate cortical dysfunction from sub cortical dysfunction.

Ricardo JA et al., assessed the frequency of EEG requisition from casualty and they also studied the influence of EEG in identifying the cause of coma and in its management. In acute situations EEGs are grossly unutilized. EEG helps in confirming or ruling out certain clinical diagnosis.

Abhijit Das et al., analyzed the transition of changes from alpha to spindle coma in hepatic coma. As the coma worsens there was a change from low frequency alpha then to theta followed by slowing of theta delta waves. Patient have better outcome with spindle coma. But the prognosis with alpha wave is debated.

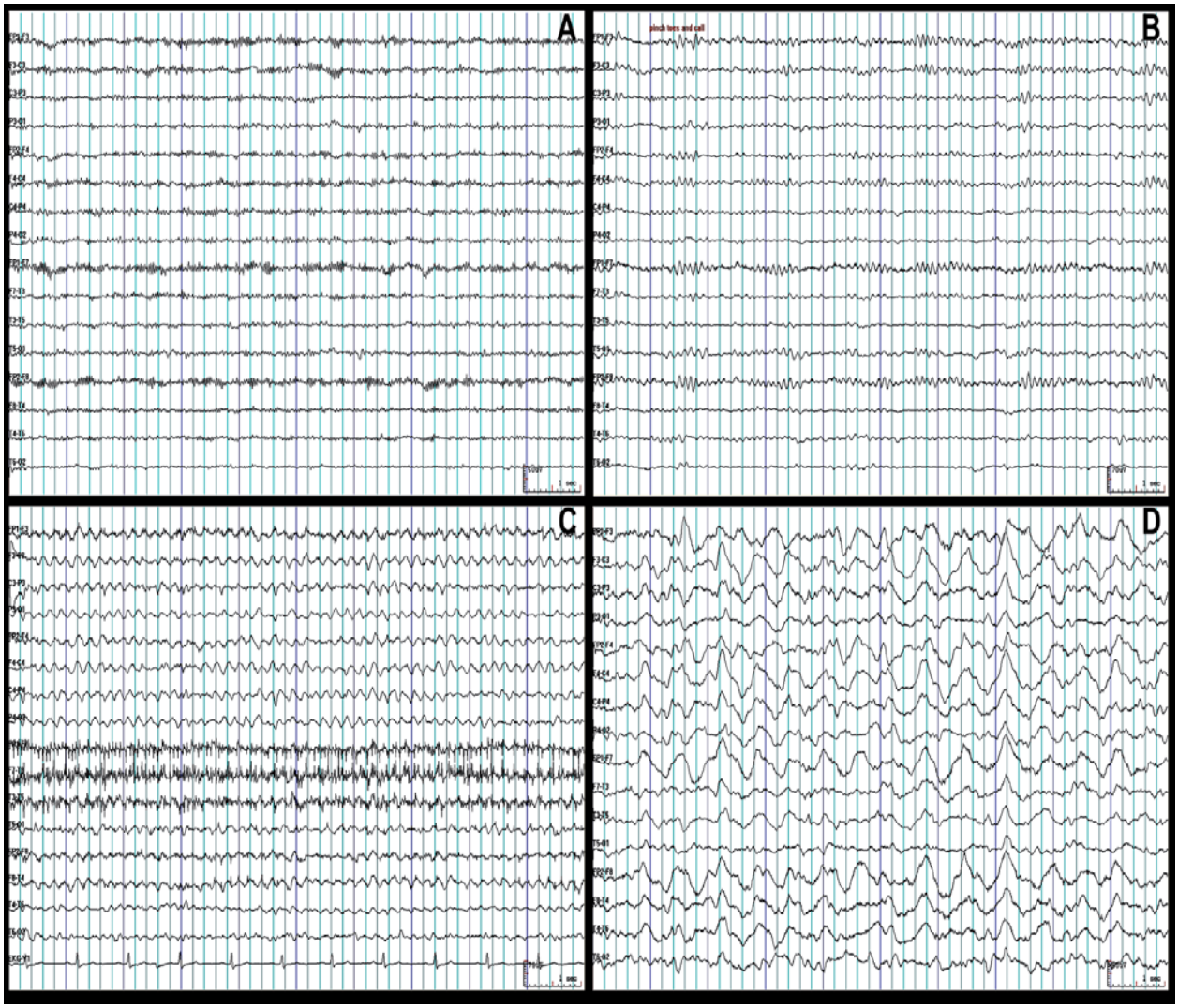
Theta coma refers to a diffuse background activity of 4-7 Hz in coma. Aside from "benign" theta dominant patterns in patients with dementia or encephalopathy [81], it can be seen HIE.

It occurs prominently over the anterior regions ultimately poor prognosis.

High-voltage delta activity in coma is defined as a background activity of 1-3 Hz with amplitudes maximum up to 100 μV. Predominately subcortical white matter pathologies will cause this coma. Seen in metabolic encephalopathies associated with poor outcome[**82**].

Alpha coma prognosis depends on the etiology and the prediction depends on the background reactivity. No background reactivity indicates a very poor prognosis.

EEG COMA PATTERNS



A. BETA COMA PATTERN

B. ALPHA COMA PATTERNS

C. THETA COMA PATTERN

D. **HIGH-VOLTAGE DELTA COMA PATTERN**

COMA PATTERN	ETIOLOGIES	EEG BACKGROUND REACTIVITY*	MOST FREQUENT OUTCOME
BETA COMA INTERMINGLED WITH ALPHA ACTIVITY	INTOXICATIONS OR WITHDRAWAL (BARBITURATES BZD); SEVERE HYPERTHYROIDISM	+/-	FAVORABLE
INTERMINGLED WITH DELTA ACTIVITY	BRAINSTEM LESIONS	-	UNFAVORABLE
ALPHA COMA MORE DIFFUSELY	INTOXICATION (drugs)	+	FAVORABLE
MONOMORPHIC POSTERIOR	BRAINSTEM LESIONS, LOCKED-IN SYNDROME	+/-	UNFAVORABLE
MORE DIFFUSELY	HIE	+/(-)	UNFAVORABLE
THETA COMA	HIE,METABOLIC SYSTEMIC INFECTIONS		UNFAVORABLE
HIGH-VOLTAGE DELTA COMA ANTERIOR PREDOMINANCE OR FOCAL,U/L	METABOLIC ENCEPHALOPATHIES, FOCAL OR UNILATERAL WHITE MATTER LESIONS	+	FAVORABLE
MORE DIFFUSELY	SEVERE METABOLIC ENCEPHALOPATHY,ENCEPHALITIS,VASCULITIS, LARGE WHITE MATTER LESIONS, MARKEDLY ELEVATED ICT	(+)/-	UNFAVORABLE
SPINDLE COMA THETA AND DELTA ACTIVITY WITH PAROXYSMAL BURSTS	POST-ICTAL STATES, INTOXICATION	+	FAVORABLE
SYMMETRIC SPINDLES	HIE, SEVERE TBI, ICH	(+)/-	UNFAVORABLE

BURST-SUPPRESSION WITH INTERRUPTION	INTOXICATION HYPOTHERMIA DRUG USE, HYPOTHERMIA	+/ (-)	FAVORABLE
NO INTERRUPTION	HIE, SEVERE INTOXICATION	(+) / -	UNFAVORABLE
LOW-VOLTAGE DELTA COMA THETA AND DELTA ACTIVITY WITH INTRUSIONS OF ALPHA AND BETA ACTIVITY	TRAUMATIC BRAIN INJURY, HEALTHY INDIVIDUALS	+	FAVORABLE
WITHOUT INTRUSIONS OF HIGHER FREQUENCY ACTIVITY	HIE, SEVERE TBI	(+) /-	UNFAVOURABLE
ELECTRO-CEREBRAL INACTIVITY NO SPONTANEOUS NEURONAL ACTIVITY DETECTABLE	MARKEDHYPOTHERMIA, SEVERE INTOXICATIONS (NERVOUS SYSTEM DEPRESSANT DRUGS)	--	UNFAVOURABLE
	HIE	--	FAVOURABLE

In our study theta and delta coma contributes to 26 % of the total cases. But their contribution to mortality remains 55 % which says that mortality is definitely more if the patient had delta or theta coma.

CONCLUSION

1. Out of the 50 non traumatic coma cases 42 are males 8 are females. There is no statistical significance in mortality in respect to sex.

2. Most of them belong to age group of 40-60 years. Age has also no statistically significant role in predicting the outcome.

3. Time to start treatment i.e) **DELAY IN HOSPITALIZATION** plays a significant role in mortality

4. The preceeding symptomatology helps in localizing the site of lesion and for further examination rather than providing a clue for predicting the prognosis.

5. **INITIAL GCS** is one of the best predictor of final outcome. More than 90% have poor outcome if the GCS is from 3-5. If GCS is from 6-8 the chance of recovery is very good.

6. **ABNORMAL RESPIRATORY MOVEMENTS** of whatever type it is considered as predictor of poor outcome.

7. **NEURO-OPHTHALMOLOGICAL SIGNS** like sluggish pupillary response, presence of papilledema , absence of spontaneous eye movements has a very significant role in predicting the mortality.

8. The most common etiology of coma in our study is Cerebro Vascular Accident followed by metabolic coma then hypoxic ischemic encephalopathy , infection and finally mixed etiology.

9. **HYPERTENSION** is associated in 50% individuals and it is one of the predictor of mortality.

10. Even background slowing is feature of coma it is not statistically significant in predicting the mortality.

11. Alpha coma is the common type encountered in this study.

12. **THETA AND DELTA COMA** has definitely poor outcome. Only one patient survived. In alpha coma the mortality depends on the etiology.

ALL THE ABOVE SAID POOR PREDICTORS HAVE BEEN ASSOCCIATED WITH SIGNIFICANT MORTALITY WHICH IS PROVEN STATISTICALLY.

LIMITATIONS OF THE STUDY

1. Small sample size

2. Only single EEG is taken (serial EEGs are needed)

3. Varied etiology of coma (separate detailed study on coma of each etiology should be carried out)

4. No specific EEG abnormalities are taken in to account

5. No specific reactivity test done (reactivity is more specific in predicting the prognosis)

ANNEXURE – I

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ANNEXURE – II

A STUDY ON CLINICAL PROFILE OF NON TRAUMATIC COMA PATIENTS & CORRELATION OF THEIR PROGNOSIS BASED ON ELECTROENCEPHALOGRAM

NAME: DATE OF ADMISSION:

AGE/SEX: DATE OF DIS /DEATH:

OCCUPATION:

IP NO:

COMPLAINTS:

1.

2.

1.LOC DURATION

2. ASSOCIATED SYMPTOMS:

	PRESENT	ABSENT
HEAD ACHE		
NAUSEA/VOMITING		
VISUAL DISTURBANCE		
WEAKNESS		
SEIZURES		
FEVER		
CHEST PAIN/PALPITATIONS		

3. H/O CARDIORESPIRATORY ARREST (CPR GIVEN /NOT)

4. PAST H/O:

H/O TIA

H/O SIMILAR ILLNESS

H/O TYPE 2 DM / SHT / IHD / TB / COPD / LIVER DISEASE /RENAL DISEASE

H/O PSYCHIATRIC ILLNESS

4. H/O SMOKING /ALCOHOLISM

FREQUENCY/QUANTITY /LAST INTAKE

EXAMINATION:

G/E:

BUILT / NOURISHMENT:

PALLOR /ICTERUS /PEDAL EDEMA:

EXTERNAL MARKERS OF LIVER DISEASE:

EXTERNAL MARKERS OF HIV:

EVIDENCE OF TRAUMA:

EVIDENCE OF POISON CONSUMPTION:

VITALS:

PR: BP:

RR: TYPE OF RESPIRATION: N/ABNORMAL (ANY SPECIFIC CHARACTER)

TEMP:

NEUROLOGICAL EXAMINATION:

1. GCS: E M V
2. PUPILS

	RIGHT	LEFT
SIZE		
SHAPE		
REACTIVITY TO LIGHT		
EQUALITY		

3. FUNDUS EXAMINATION:

4. DOLLS EYE MOVEMENT:

5. CRANIAL NERVE ABNORMALITIES:

6. SIGNS OF MENINGEAL IRRITATION:

7. MOTOR SYSTEM:

ATTITUDE: DECORTICATE / DECEREBRATE / HEMIPLEGIC / NOT SPECIFIC

TONE:

CORNEAL REFLEX:

DEEP TENDON REFLEX:

PLANTAR:

OTHER SYSTEM EXAMINATION:

CVS:

RS:

PER ABDOMEN:

INVESTIGATIONS:

1. RBS :

2.Sr.UREA:

3. Sr.CREATININE :

4.SrSODIUM:

5.SrPOTTASIUM:

6 .Sr.BILIRUBIN:

7. SGOT/SGPT:

8. ECG:

9.CT BRAIN/ MRI BRAIN:

FINAL DIAGNOSIS:

CAUSE OF COMA:

EEG FINDINGS:

BACK GROUND RHYTHM: ALPHA / BETA / THETA

PAROXYSMAL ACTIVITY: SHARP / SPIKE

ANY SPECIFIC PATTERN:

FINAL OUTCOME:

ANNEXURE – III

ANNEXURE III MASTER CHART

ANNEXURE III MASTER CHART

S.NO	IP No	AGE	SEX	PRESENTING SYMPTOMS							PAST H/O				SIGNS						ETIOLOGY	EEG		OUTCOME
				DURATION	HEADACHE	NAUSEA	WEAKNESS	SEIZURES	POISON	CPR	SHT	DM	CKD	CLD	MENINGEAL SIGNS	EYE.MOV	PUPIL REACTION	P.EDEMA	RS PATTERN	GCS		SLOWING	BACKGROUND	
1	24054	50	M	<1	N	N	N	N	N	N	N	Y	N	N	N	p	NL	N	nl	6	CVA	p	A	Alive
2	35153	56	M	>1	Y	Y	Y	N	N	N	Y	N	N	Y	N	p	S	Y	ab	3	CVA	p	T	Death
3	24709	53	M	>1	Y	Y	N	N	N	N	Y	Y	N	N	N	p	S	Y	ab	3	CVA	p	T	Death
4	33261	61	M	>1	N	N	N	N	N	N	Y	N	N	Y	N	A	NL	N	ab	4	CVA	a	A	Death
5	42288	45	F	>1	Y	Y	Y	N	N	N	Y	Y	N	N	N	P	S	Y	ab	5	CVA	p	A	Death
6	42335	45	M	<1	N	N	Y	N	N	N	Y	N	Y	N	N	P	NL	N	ab	4	CVA	p	T	Death
7	40224	60	M	<1	Y	Y	Y	N	N	N	Y	N	N	N	N	A	NL	Y	ab	5	CVA	a	A	Death

8	1491284	60	F	<1	N	N	y	N	N	N	Y	N	N	N	N	P	NL	N	ab	8	CVA	a	A	Alive
9	1493738	80	M	<1	N	N	N	N	N	N	N	Y	N	N	N	P	NL	N	ab	5	CVA	a	A	Alive
10	36159	48	M	<1	N	N	Y	N	N	N	Y	N	N	N	N	P	NL	N	nl	6	CVA	a	A	Alive
11	1491334	53	F	<1	N	N	y	N	N	N	N	N	N	Y	N	P	NL	N	nl	5	CVA	a	A	Alive
12	1492965	60	M	>1	N	N	y	N	N	N	Y	Y	N	N	N	P	NL	N	nl	6	CVA	p	A	Alive
13	42362	55	M	<1	N	N	Y	N	N	N	N	N	N	N	N	P	NL	N	nl	8	CVA	p	A	Alive
14	42335	45	M	<1	N	N	Y	Y	N	N	Y	Y	N	N	N	P	NL	N	nl	6	CVA	a	A	Alive
15	42205	60	M	>1	Y	Y	Y	N	N	N	Y	Y	N	N	N	P	S	Y	nl	4	CVA	p	A	Death
16	42223	40	M	>1	Y	Y	Y	N	N	N	Y	Y	N	N	Y	A	NL	Y	ab	5	CVA	p	T	Death
17	1492317	45	M	>1	Y	Y	Y	N	N	N	N	Y	N	Y	N	P	S	Y	nl	3	CVA	a	D	Death
18	36261	58	M	>1	N	N	N	N	N	N	N	N	N	N	N	P	NL	N	nl	4	CVA	p	A	Alive
19	1491996	56	M	>1	N	N	y	N	N	N	N	Y	N	Y	N	P	NL	N	nl	5	CVA	p	A	Alive
20	33045	22	F	<1	N	N	N	N	Y	N	N	N	N	N	N	P	NL	N	nl	7	M1	a	A	Alive
21	41021	40	M	>1	N	N	N	N	N	N	Y	Y	N	Y	N	P	S	N	nl	5	M1	p	A	Death
22	42262	65	F	>1	Y	Y	N	N	N	N	Y	Y	N	N	N	P	S	Y	ab	5	M3	p	T	Death
23	38528	70	M	<1	Y	Y	Y	Y	N	N	N	Y	N	N	N	P	NL	N	nl	6	M3	p	A	Alive
24	40974	42	M	<1	Y	N	N	N	N	N	Y	N	N	Y	N	P	NL	N	nl	6	M1	p	A	Alive
25	46342	26	M	<1	N	N	N	N	N	N	N	N	N	Y	N	P	NL	N	nl	4	M1	a	A	Alive
26	32346	48	F	<1	N	N	N	N	N	N	Y	Y	N	N	N	P	S	N	nl	4	M4	p	T	Alive
27	33456	42	M	<1	N	Y	N	N	N	N	Y	Y	N	Y	N	A	S	N	ab	3	M1	a	T	Death

28	40964	26	M	<1	Y	Y	N	N	N	N	N	N	N	Y	N	P	NL	N	nl	6	M1	p	A	Alive
29	39462	32	M	<1	N	Y	N	N	N	N	Y	N	Y	N	N	P	NL	N	ab	6	M2	p	A	Alive
30	36542	40	M	>1	N	N	N	N	N	N	Y	Y	Y	N	Y	A	NL	N	ab	5	M2	a	D	Death
31	40501	38	F	<1	N	Y	N	N	N	N	Y	Y	Y	Y	N	P	NL	N	nl	5	M2	p	A	Alive
32	32681	36	M	<1	N	Y	N	N	N	N	Y	N	Y	N	N	A	S	N	ab	5	M2	p	T	Death
33	36312	29	M	>1	Y	N	N	N	N	N	N	N	Y	N	N	P	NL	N	nl	6	M2	a	A	Alive
34	40541	51	M	<1	N	N	N	N	N	N	N	Y	Y	N	Y	P	NL	Y	nl	5	M2	p	A	Death
35	36241	36	M	<1	N	N	N	N	N	N	Y	N	Y	Y	N	P	NL	N	nl	6	M2	p	A	Alive
36	36342	28	M	<1	N	N	N	N	Y	Y	N	N	N	N	N	A	S	N	ab	3	HIE	p	T	Death
37	39838	32	M	<1	N	N	N	N	Y	Y	N	N	N	Y	N	A	S	N	ab	3	HIE	p	A	Death
38	36382	25	M	<1	N	N	N	N	Y	Y	N	N	N	N	N	P	NL	N	nl	6	HIE	p	A	Alive
39	40546	40	M	<1	N	N	N	Y	Y	Y	N	N	N	N	N	P	S	N	ab	4	HIE	p	A	Death
40	40502	27	M	<1	N	N	N	N	Y	Y	N	N	N	N	N	A	NL	N	nl	6	HIE	p	A	Alive
41	36322	41	M	<1	N	N	N	N	Y	Y	N	N	N	N	N	P	NL	N	ab	6	HIE	a	A	Alive
42	38704	34	M	<1	N	N	N	Y	Y	Y	N	N	N	N	N	P	S	N	nl	6	HIE	p	T	Death
43	40764	50	M	<1	N	N	N	N	N	Y	N	N	N	N	N	P	NL	N	nl	6	HIE	p	A	Alive
44	32865	28	M	<1	N	N	N	Y	N	N	N	N	N	N	N	A	S	N	nl	5	HIE	a	A	Alive
45	35345	60	M	<1	N	N	N	Y	N	Y	N	N	N	Y	N	A	S	N	ab	5	HIE	p	A	Death
46	32321	42	F	>1	Y	Y	N	N	N	N	Y	N	N	N	Y	p	NL	N	nl	6	ME	a	A	Alive
47	43211	44	M	>1	Y	Y	N	N	N	N	Y	Y	N	N	Y	A	S	Y	nl	5	ME	p	D	Death

48	**48564**	**26**	**M**	**>1**	**Y**	**Y**	**N**	**N**	**N**	**N**	**N**	**Y**	**N**	**N**	**Y**	**P**	**NL**	**N**	**ab**	**6**	**ME**	**a**	**A**	**Alive**
49	**45642**	**46**	**M**	**<1**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**N**	**Y**	**Y**	**A**	**NL**	**N**	**ab**	**6**	**MIXED**	**p**	**A**	**Alive**
50	**45214**	**54**	**M**	**>1**	**N**	**Y**	**N**	**Y**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**N**	**P**	**S**	**Y**	**ab**	**5**	**MIXED**	**p**	**A/T**	**Death**

MASTER CHART & KEY TO MASTER CHART

A	Absent
A	Alpha coma (in background column)
ab	Abnormal
B	Beta coma
CLD	Chronic Liver Disease
CKD	Chronic Kidney Disease
CPR	Cardio Pulmonary Resuscitation
D	Delta coma
DM	Diabetes Mellitus
Ey.mov	Eye movements
GCS	Glascow Coma Scale
M1	HEPATIC COMA
M2	URAEMIC COMA
M3	ELECTROLYTE DISTURBANCES
M4	MYXEDEMA COMA
N	No
nl	Normal
P	Present

P.edema	Papilledema
RS pattern	Respiratory pattern
S	Sluggish
SHT	Systemic hypertension
T	Theta coma
Y	Yes

ANNEXURE : V

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR S.SURIYAKANTH,** Post graduate in the Department of General Medicine ,Thanjavur Medical College & Hospital, Thanjavur –613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations

Place :

Date : Signature of participant

INFORMATION SHEET

We are conducting a prospective study on

“ A STUDY ON CLINICAL PROFILE OF NON TRAUMATIC COMA PATIENTS AND CORRELATION OF THEIR PROGNOSIS BASED ON ELECTROENCEPHALOGRAM ”

in the Department of General Medicine ,Thanjavur Medical College & Hospital, Thanjavur – 613004.

At the time of announcing the results and suggestions, name and identity of the patients will be confidential.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Date: