DISSERTATION ON

COMPARISON OF GLASGOW COMA SCALE AND THE FULL OUTLINE OF UNRESPONSIVENESS SCORE IN PREDICTING MORTALITY AND NEUROLOGICAL OUTCOME IN PATIENTS WITH ALTERED SENSORIUM ADMITTED IN A MEDICAL INTENSIVE CARE UNIT

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CERTIFICATE

This is to certify that the dissertation titled **"COMPARISON OF GLASGOW COMA SCALE AND THE FULL OUTLINE OF UNRESPONSIVENESS SCORE IN PREDICTING MORTALITY AND NEUROLOGICAL OUTCOME IN PATIENTS WITH ALTERED SENSORIUM ADMITTED IN A MEDICAL INTENSIVE CARE UNIT"** is a bonafide work done by **Dr.M. SATHISH KUMAR, Registration Number - 201911018** at Madras Medical College, Chennai in partial fulfilment of the university rules and regulations for the award of M.D., Degree in General Medicine (Branch-I) under our guidance and supervision during the academic year 2019-2022.

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DECLARATION

I, **Dr. M. SATHISH KUMAR, Registration Number – 201911018,** solemnly declare that this dissertation titled **"COMPARISON OF GLASGOW COMA SCALE AND THE FULL OUTLINE OF UNRESPONSIVENESS SCORE IN PREDICTING MORTALITY AND NEUROLOGICAL OUTCOME IN PATIENTS WITH ALTERED SENSORIUM ADMITTED IN A MEDICAL INTENSIVE CARE UNIT"** was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai between 2019-2022 under the guidance and supervision of my chief **Prof. Dr. NALINI KUMARAVELU M.D.**

This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree in General Medicine (Branch-I).

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ABBREVIATIONS

GCS – Glasgow Coma Scale

FOUR – Full Outline of Un Responsiveness Score

ATME – Acute Toxic Metabolic Encephalopathy

ICU – Intensive Care Unit

TBI – Traumatic Brain Injury

ICH – Intracranial Hemorrhage

PPRF – Para Pontine Reticular Formation

ARAS – Ascending Reticular Activating System.

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INTRODUCTION

The International Classification of Diseases -10[1] defined altered consciousness as "any level of arousal other than normal". Altered sensorium and coma constitute one of the most important diagnostic challenges for any physician. The causes are varied and diverse. One can confidently claim that any busy hospital will always have atleast one patient with altered sensorium at any point of time in their emergency ward. The incidence of altered sensorium in India is said to be around 70 to 80/100,000 persons per year. The most important challenge for the physician in the emergency room confronting a patient with altered sensorium is to think and act "on their feet" with requirements of prioritising Airway, Breathing and Circulation for emergency care while simultaneously evaluating for the varied and diverse causes of coma. The most common cause of altered sensorium worldwide is surprisingly Metabolic Encephalopathy and not trauma! However, identification of the etiology cannot be made with the presenting complaints, adding to the challenge.

The second most important challenge for the physician after early stabilisation is to prognosticate the patient and decide regarding need for specialist care. Upto the early 1970's, there was no universal tool for this purpose which contributed to poor outcomes. The Glasgow Coma Scale (GCS)[2] was invented in 1974 for this purpose and has since revolutionised patient care. However, the pitfalls of the Glasgow coma scale have been studied and documented over time – such as inability to assess intubated patients adequately, lack of incorporation of important neurological examination such as brainstem reflexes etc. Newer scales and scores were introduced

to overcome these fallacies but they were too complex and did not gain popularity beyond the regional centres which invented them. It was at this juncture, the FOUR (Full Outline of Un Responsiveness) score[3] was invented in 2005 to overcome the shortcomings of the Glasgow Coma Scale. The FOUR score is easy to perform, incorporates important diagnostic data and is as useful as GCS in triage centres. Studies conducted so far have acknowledged the advantages of the FOUR score while specifying the need for conducting multiple studies across different patient populations to prove its validity[4].

The aim of this thesis is to compare the FOUR score with the Glasgow Coma Scale to predict mortality and neurological outcome in patients with altered sensorium. This will contribute to patient care in the form of better prognostication and better communication to the attenders of patients admitted with altered sensorium.

REVIEW OF LITERATURE

Altered mental status is one of the common conditions for which medical care is sought as an emergency. An organised and structured approach is needed from the Physician in order to diagnose and treat Altered sensorium as there are multiple causes for the same. The different states of Altered sensorium are described in the following section.

Altered mental status exists as a "continuum of states"[5] with the most severe form being **Coma**, which is defined as a deep sleep like state of the patient with eyes closed from which the patient could not be aroused. **Stupor**, in turn, refers to higher degrees of arousability wherein the patient can be transiently awakened by vigorous stimuli like shaking. It is accompanied by motor behaviour that leads to avoidance or withdrawal from aggravating and uncomfortable stimuli. **Drowsiness** represents a form of a "milder Stupor". It simulates light sleep and is characterized by the presence of easy arousal and persistence of alert state, albeit for only brief periods. **Vegetative state** refers to an awake but unresponsive patient. It is often found in patients recovering from coma. In the vegetative state, the eyelids of the patient occasionally open periodically, which simulates wakefulness. Respiratory and Autonomic functions are retained. Movements such as yawning, coughing, limb movements, swallowing persist, but these are not meaningful responses to the external or internal environment. Signs indicating extensive damage to the cerebral hemispheres are always present,

such as decorticate or decerebrate posturing and loss of response to visual stimuli. **Minimally conscious state** is closely related but less severe than that of the vegetative state; here the patient displays rudimentary motor or vocal behaviours, most of which are spontaneous, but some purposeful, in the form of response to touch, command or visual stimuli. Cardiac arrest leading to cerebral hypoperfusion and traumatic brain injury are the most common causes of Vegetative state and minimally conscious states. Prognosis is usually poor in both Vegetative and minimally conscious states, with recovery after 12 months extremely unusual.

There are certain syndromes which are misinterpreted as stupor or coma; Important syndromes include Akinetic mutism, Catatonia and Locked in syndrome**. Locked in syndrome** refers to an awake patient who is unable to move his limbs or speak but retains voluntary control over his eye lids and vertical eye movements, allowing the patient to signal his needs with a clear mind (as long as it is diagnosed and interpreted). The pupils are reactive. The most common cause for a Locked in state is a basilar artery thrombosis causing an infarction of the ventral pons that transects all descending motor pathways. Ventral pontine hemorrhage can also lead to Locked in syndrome, though hemorrhagic stroke is usually extensive and involves other areas also. Osmotic demyelination syndrome (central pontine myelinolysis) can also lead to the Locked in state. Horizontal eye movements are affected because of the involvement of the Para pontine reticular formation (PPRF). The EEG of the patient is normal, which indicates that the patient is awake.

Catatonia is a hypomobile and mute syndrome; it usually occurs as part of major psychiatric disorders such as schizophrenia or major depression. These patients do not

exhibit or exhibit very few voluntary movements. The characteristic feature is the presence of "waxy flexibility or catalepsy", characterised by limbs retaining postures in which they have been placed by the examiner. Catatonia is partly similar to akinetic mutism, but there is no organic brain disease. Careful examination often reveals that these patients are still responsive – for example, eyelid elevation is actively resisted, eyes move concomitantly with head rotation and such patients blink in response to visual threat. These responses are inconsistent with organic brain lesions.

Akinetic mutism denotes a partially or fully awake state wherein the patient is able to form impressions and think (identified by recounting of events) but remains immobile and mute. This is due to damage involving medial thalamic nuclei or involvement of frontal lobes from extreme hydrocephalus. Abulia denotes a milder form of akinetic mutism where there is mental and physical slowness and decrease in the ability to initiate activity. This state is due to lesions involving the medial frontal lobes and their connections.

Acute Toxic- Metabolic encephalopathy (ATME)[6] is characterised by acute global cerebral dysfunction presenting in the form of impaired consciousness, behavioral changes and seizures. The main caveat is the absence of primary structural brain disease or direct central nervous system infections. Acute Toxic-Metabolic encephalopathy commonly presents as either confusion or delirium. **Confusion** is defined in Neurology textbooks as an inability to maintain a coherent stream of thought or action. **Delirium** is a state of confusion with superimposed hyperactivity of the sympathetic nervous system. It is characterised by the presence of tremor, tachycardia, mydriasis and diaphoresis. Acute Toxic- Metabolic encephalopathy is

common among patients who are critically ill. It is said that Acute Metabolic encephalopathy is often under recognized and under treated as it often occurs in patients who are in need of mechanical ventilation. Acute Toxic- Metabolic Encephalopathy is usually a consequence of systemic illness, but it in itself can cause death by predisposing to aspiration, bed ridden state and infections. Certain metabolic encephalopathies like Hypoglycemia and Thiamine deficiency must be promptly recognised and should always be part of the basic diagnostic algorithm as these patients show complete recovery when treated on time, but if untreated and unrecognised, can be fatal.

ANATOMY AND PHYSIOLOGICAL BASIS OF ALTERED SENSORIUM AND COMA:

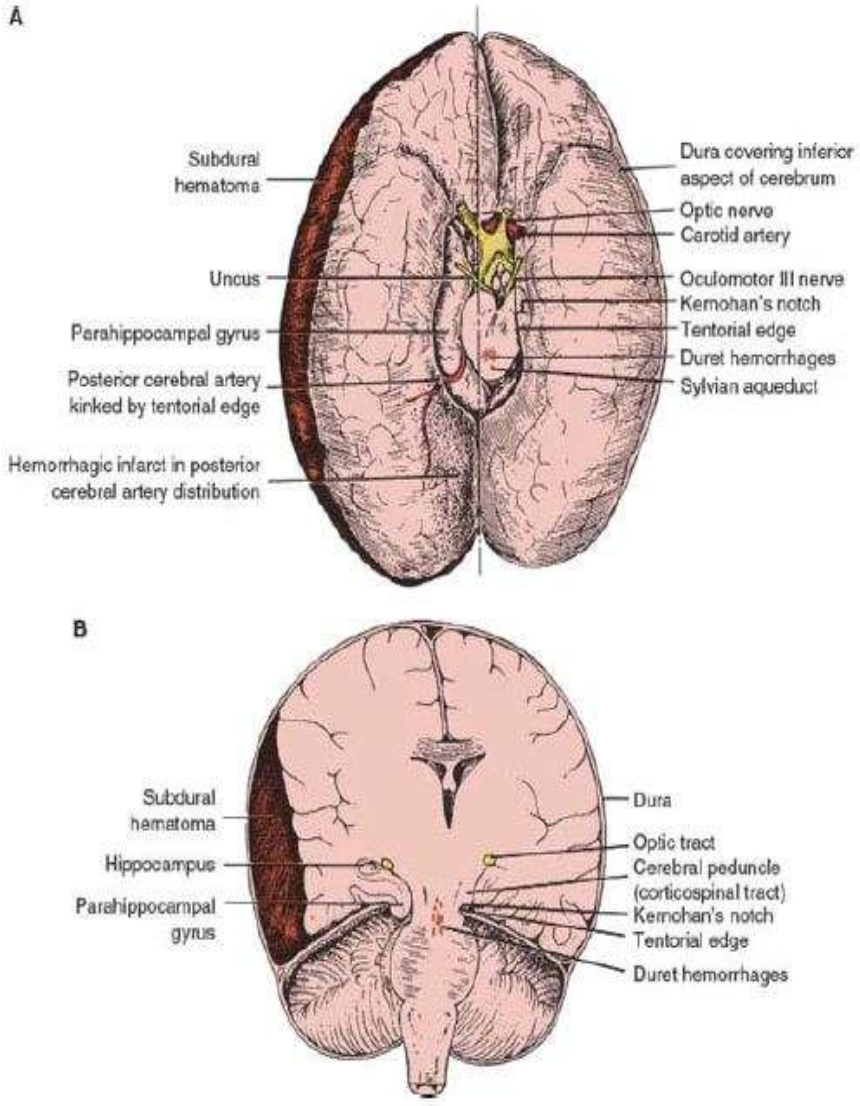
In simple terms, **Arousal** is determined by Ascending Reticular Activating System and **Awareness** is determined by Cerebral Cortex. The main pathophysiology of coma can be explained by 1) Widespread abnormalities of the Cerebral hemispheres, 2) Reduced activity of the Reticular activating system (also known as thalamocortical alerting system). Hence, for patient's mental status, there needs to be a proper functioning of the Reticular activating system, the ascending projections of the Reticular Activating system to the cortex and the function of the Cerebral cortex. Coma can be caused by either structural damage or suppression of the Reticular activating system by drugs, toxins or metabolic derangements. Metabolic causes of coma and altered sensorium are more common than structural injuries.

ALTERED SENSORIUM AND COMA DUE TO CEREBRAL MASS LESIONS AND HERNIATION SYNDROMES:

The skull is a closed cavity. The cranial cavity is separated into multiple compartments by modifications of the dura mater in the form of infoldings. Falx cerebri separates the two cerebral hemispheres; Tentorium cerebelli separates the anterior and posterior cranial fossae. The displacement of brain tissue due to an overlying or adjacent mass into a contiguous compartment is known as Herniation. Herniated brain tissue occupies compartments of the cranial cavity which it does not occupy usually. Altered sensorium, coma and many of their associated signs can be attributed to these shifts of brain tissue.

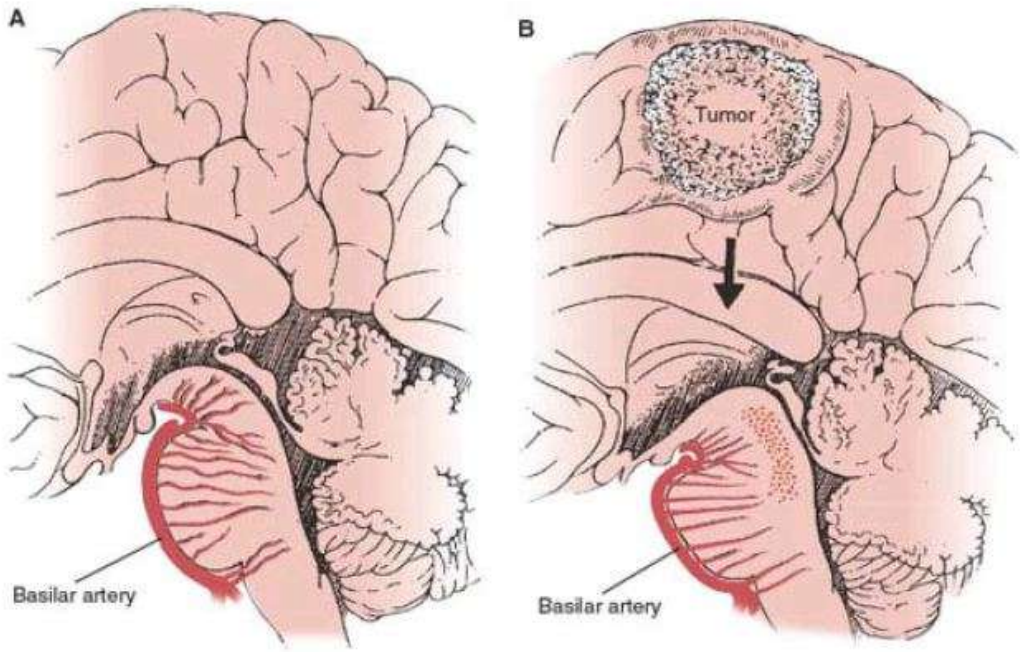
The **four types of cerebral herniation** are Uncal (Lateral transtentorial), Central, Transfalcial and Foraminal. **Lateral Transtentorial herniation** is the most common form of herniation. Here, the brain tissue is displaced from the supratentorial to the infratentorial compartment via the tentorial opening. The most common brain structure involved in such herniation is known as uncus. Uncal herniation denotes the impaction of the “uncus” (anterior medial temporal gyrus) into the tentorial opening just anterior and adjacent to the midbrain. The oculomotor nerve (Cranial Nerve III) is compressed when the nerve passes via the subarachnoid space, leading to enlargement of the pupil on the same side of the lesion (ipsilateral pupil). This is the first sign. This happens because the fibres which cause constriction of the pupil – the parasympathetic fibres of the oculomotor nerve are situated peripherally and are hence more prone to compression and being involved initially. Drowsiness and Coma follows pupillary dilation and this is due to the compression of the Reticular activating system of the

midbrain against the opposite tentorial edge (this is done by the displaced parahippocampal gyrus). Sometimes, the midbrain gets displaced laterally leading to compression of the opposite cerebral peduncle against the tentorial edge- leading to the eponymous **Kernohan Woltman sign** (Extensor plantar response and hemiparesis ipsilateral to the side of the actual lesion). Compression of the posterior and anterior cerebral arteries can occur as they pass over the tentorium cerebelli leading to brain infarction. Sometimes, hydrocephalus can also occur when the ventricular system gets involved and obstruction to CSF flow occurs.



The image shows a Lateral transtentorial herniation: (A) basal view, (B) coronal view. In this example, a subdural hematoma is causing a marked shift of the midline structures and herniation of the parahippocampal gyrus through the tentorial notch. Occlusion of the posterior cerebral artery, which is pinched between the herniated hippocampal tissue and the rigid end of the tentorium, has resulted in medial temporo-occipital infarction. The midbrain is compressed against the contralateral free tentorial edge, causing a laceration of the crus cerebri (Kernohan notch). Stretching of the slender perforating branches of the basilar artery has produced petechial hemorrhage in the tegmentum of the midbrain (Duret hemorrhage).

In **Central transtentorial herniation** there is a symmetric downward movement of the thalamus via the tentorial opening along with compression of the structures of the upper midbrain. Drowsiness and Miotic pupils are the main features (this is in contrast to the unilateral dilated pupil seen in uncal herniation). There is progressive compression of the brainstem and the Reticular activating system in uncal and central herniations, characterised clinically by sequential involvement of the midbrain, pons and the medulla. Thus, the respiratory centres of the brain, which are present in the medulla are involved only late in the course of herniation. In **Transfalcial herniation** there is displacement of the cingulate gyrus across the midline under the falx cerebri. In **foraminal herniation**, there is a downward displacement of the cerebellar tonsils into the Foramen Magnum, leading to early involvement of the respiratory centres of the medulla- leading to death.



The image shows a Central transtentorial herniation. A: Normal sagittal section of the brainstem. The vascular perforators, which are branches of the basilar artery are intact here. B: Mass effect from a high parietal tumor, resulting in downward displacement and superoinferior flattening of the midbrain and upper pons. The increased cross-sectional diameter of these structures is associated with stretching and rupture of the perforators, with subsequent hemorrhage in the tegmentum of the midbrain and upper pons.

COMA DUE TO METABOLIC DISORDERS AND TOXINS (ACUTE TOXIC-METABOLIC ENCEPHALOPATHY):

Research has now found that the phylogenetically newer structures of the brain are more sensitive to metabolic injury. This has been found true via experience, even though the drugs and toxins causing the injury may vary. Brain functions dependent

on complex polysynaptic pathways are affected earlier by such metabolic disturbances than those which are mediated only by a few neurons. Hence, Higher cortical functions and attention are affected early to metabolic insults, while the pupillary light reflex continues to remain in patients who are almost of the brink of brainstem destruction ("brain death").

Metabolic abnormalities mainly cause drowsiness by interrupting and impairing the delivery of sources of energy such as oxygen and glucose. They also cause altered mentation by causing alterations in the neuronal excitability – commonly done by drugs, anesthesia, alcohol and epilepsy.

The neurons of the cerebral cortex are dependent on the Cerebral Blood flow for delivery of the energy substrates such as oxygen and glucose. Brain stores of glucose can provide energy lasting for around 2 minutes after blood flow is interrupted. However, oxygen stores last only for 8 to 10 seconds. Ischemia thus causes both hypoxia and reduction in cerebral blood flow which exhausts the glucose reserves in the brain rapidly.

Metabolic disturbances such as hypoglycemia, hyponatremia, hypercapnia, hepatic failure and renal failure do not cause neuronal destruction like ischemia. They also cause altered mentation which is commonly reversible on identification and correction of the underlying metabolic abnormality. How such reversible changes are made by these metabolic disturbances are not clear, but it is postulated that it could be due to neurotransmitter abnormalities leading to changes in neuronal excitability or changes in ion fluxes across the nerve cell membranes. For example, in **Hepatic encephalopathy** there is a high ammonia concentration in the blood stream. This

leads to increased synthesis of glutamine in astrocytes causing osmotic swelling of the neuronal cell, production of reactive oxygen radicals and synthesis of "false" neurotransmitters. Over a period of time, structural changes such as diffuse astrocytosis occurs in patients with chronic hepatic encephalopathy. In **Renal Failure**, there is an increase in the accumulation of neurotoxic substances such as creatinine, guanidine and related compounds, leading to depletion of catecholamines and altered glutamate and GABA (Gamma amino butyric acid) balance. There is also an associated disruption of the blood brain barrier. Disturbed blood brain barrier leads to accumulation of systemic toxins as well as normal plasma constituents in the brain and CSF. This interferes in neuronal function. Recent research has indicated that large neutral amino acids such as tryptophan and tyrosine are involved in the pathogenesis of delirium in critically ill patients (especially those needing mechanical ventilation). The pathophysiology of **Septic encephalopathy** is multifactorial, characterised by altered blood brain barrier, inflammatory cytokines, reductions in monoamine neurotransmitters and increase in the concentration of false neurotransmitters such as octopamine.

Seizures commonly occur in metabolic encephalopathies associated with large and rapid shifts in sodium and water balance in the brain. Examples include DKA (Diabetic Keto Acidosis), non ketotic hyperosmolar coma and acute hyponatremias.

The pathophysiology of Metabolic and Toxic Encephalopathies can be summarised by the following mechanisms:

- Cerebral edema – in acute fulminant hepatic encephalopathy and hypo osmolar encephalopathies.

- Disruption of the integrity and the balance of neurotransmitters – drug induced altered sensorium/delirium due to Dopamine, acetylcholine, glutamate, GABA etc.
- Alteration in membrane excitability – electrolyte disturbances.
- Alteration in cellular energy and metabolism – in Nutritional disorders like Vitamin B12, folate deficiency.
- Impaired oxygen delivery and mitochondrial dysfunction – in Exogenous toxins like carbon monoxide and cyanide poisoning.

CAUSES OF ALTERED SENSORIUM AND COMA:

The differential diagnosis of coma is vast and extensive:

1. Diseases that cause no focal brainstem or lateralizing neurologic signs (normal CT brain usually)

- Intoxications – alcohol, sedative drugs, opiates etc.
- Metabolic disturbances- anoxia, hyponatremia, hypernatremia, hypercalcemia, diabetic acidosis, non ketotic hyperosmolar hyperglycemia, hypoglycemia, uremic encephalopathy, hepatic encephalopathy, hypercapnia, Addisonian crisis, hypothyroidism, hyperthyroidism, profound nutritional deficiency.
- Severe systemic infections: pneumonia, septicemia, malaria, typhoid fever, Waterhouse Friedrichsen syndrome.
- Shock
- Status epilepticus, post ictal states

- Hypertensive encephalopathy, eclampsia, Posterior reversible encephalopathy syndrome.
- Hyperthermia and hypothermia
- Concussion
- Hydrocephalus.

2. Diseases that cause focal brainstem or lateralizing cerebral signs (CT scan is typically abnormal)

- Hemispheral hemorrhage (basal ganglionic, thalamic) or infarction (large middle cerebral artery territory) with secondary brainstem compression.
- Brainstem infarction due to basilar artery thrombosis or embolism.
- Brain abscess, subdural empyema.
- Epidural and subdural hemorrhage, brain contusion.
- Brain tumor with surrounding edema
- Cerebellar and pontine hemorrhage and infarction
- Widespread traumatic brain injury
- Metabolic coma with pre-existing structural damage.

3. Diseases that cause meningeal irritation with or without fever, and with an excess of RBC or WBCs in the CSF

- Subarachnoid hemorrhage from ruptured aneurysm, arteriovenous malformation and trauma.
- Infectious meningitis and meningoencephalitis
- Paraneoplastic and autoimmune meningitis

- Carcinomatous and lymphomatous meningitis.

APPROACH TO A PATIENT WITH ALTERED SENSORIUM/ COMA:

Airway, breathing and circulation precedes importance in the acute management and assessment of altered sensorium and coma. Following stabilisation of the patient, history, general examination and focussed neurological examination can be proceeded with to evaluate the cause for altered sensorium.

HISTORY TAKING:

Certain causes of altered sensorium should always be queried for, like trauma and drug toxicity. The following points should be kept in mind by the physician while taking history:

- The circumstances and rapidity with which the neurological symptoms of the patient developed is to be questioned.
- Prior symptoms like confusion, weakness, headache, fever, dizziness, double vision and vomiting should be queried.
- The use of drugs, medications and alcohol is to be ruled out.
- Comorbid diseases like chronic liver disease, kidney disease, lung, heart and other diseases are to be questioned.

  Textbooks of Core Neurology state that various terminologies of altered sensorium like coma, stupor, drowsiness, vegetative state etc., although fancy, fail to convey the requisite information needed for neurological localisation of the illness and management. It is suggested that although time consuming, a description of the patient's level of responsiveness aids

in localisation and management of the illness as well as aiding in communication among members of the health care team. It also enhances the consistency in successive evaluations of the unresponsive patient. For e.g.: Rather than using terms such as “the patient is comatose”, it can be conveyed as “Mr. B is lying motionless in the bed and remains so unless vigorously shaken following which he opened his eyes and looked to his left briefly. He did not answer questions or followed any instructions”.

GENERAL EXAMINATION:

Assessment of Vital Signs:

- Presence of fever suggests a systemic process like bacterial meningitis, encephalitis, heat stroke, neuroleptic malignant syndrome etc.
- Hypothermia can be seen in patients with alcohol intoxication, barbiturate and sedative usage.
- Hypotension is present in patients with alcohol and barbiturate intoxication, internal hemorrhage leading to hypovolemia, Myxedema coma and Addisonian crisis.
- Hypertension is present in patients with hypertensive encephalopathy, cerebral hemorrhage, large cerebral infarction or head injury.
- Cutaneous petechiae suggests TTP (Thrombotic Thrombocytopenic Purpura), meningococcemia or the presence of a bleeding disorder which could have caused intracranial hemorrhage.

The following table[7] summarises the general examination findings that can be present in patients with altered sensorium, which will help to identify an underlying cause.

CLINICAL FINDINGS	POSSIBLE UNDERLYING CAUSE
SKIN CHANGES	
1. Bruising over the mastoid process (Battle sign)	Head injury with skull base fracture.
2. Cherry red discoloration of skin.	Carbon monoxide poisoning.
3. Marked pallor	Internal hemorrhage.
4. Excessive sweating/diaphoresis.	Thyrotoxicosis, Hypoglycemia, Organophosphorus poisoning, Shock.
5. Excessively dry skin.	Hypothyroidism, Diabetic Ketoacidosis, Uremia.
6. Eschar	Scrub typhus.
ODOUR OF BREATH	
1. Spoiled fruit odour	Diabetic ketoacidosis
2. Uriniferous odour	Uremic encephalopathy
3. Musty odour	Hepatic encephalopathy

4. Burnt almond odour 5. Onion odour 6. Garlic odour	Cyanide poisoning Paraldehyde poisoning Organophosphorus poisoning
FUNDUS EXAMINATION 1. Papilledema	Hypertensive encephalopathy, Cerebral venous sinus thrombosis, Raised Intracranial Pressure.
2. Roth spots	Infective endocarditis.
3. Subhyaloid hemorrhage	Aneurysmal intracranial bleed.
4. Retinal whitening and retinal vessel changes (orange or whitish discoloration)	Cerebral malaria.
5. Toxic optic neuropathy (swollen or hyperemic disc with associated hemorrhage in early stages and temporal disc pallor in late stages)	Methanol poisoning Mercury poisoning Carbon monoxide poisoning

SIGNS WITH LOCALIZING VALUE IN PATIENTS WITH ALTERED SENSORIUM:

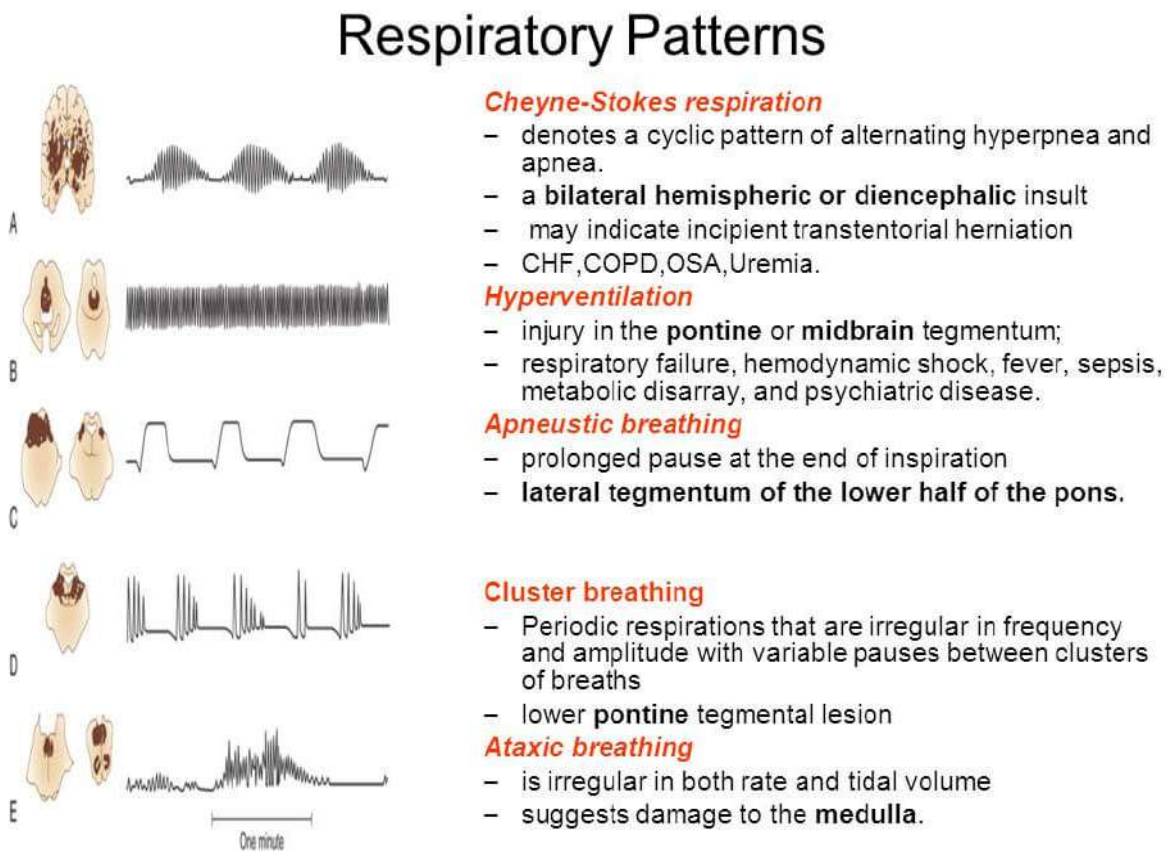
The following examination should be carried out in all patients with altered sensorium as they provide important anatomical clues regarding the nature and extent of the injury:

- Respiratory pattern
- Temperature changes
- Pupils
- Eye movements
- Motor activity of the body and limbs

RESPIRATORY PATTERNS:

Respiratory patterns can help in identifying the location and level of the lesion in the brain, but metabolic abnormalities like hyponatremia can also affect the respiratory centres of the brainstem and medulla like Pons and Medulla and resemble structural neurologic disease. The Pons contains the Pneumotaxic and the Apneustic centre for respiration while the Medulla contains the inspiratory and the expiratory centres – hence involvement of these structures by pathological processes lead to characteristic respiratory patterns. The most important named patterns of respiration that are classically described in medical literature include Cheyne Stokes respiration, Apneustic ventilation, Cluster breathing and Biot's breathing. The following image

shows the patterns of breathing which may help in identification of the location of the lesion:



TEMPERATURE CHANGES:

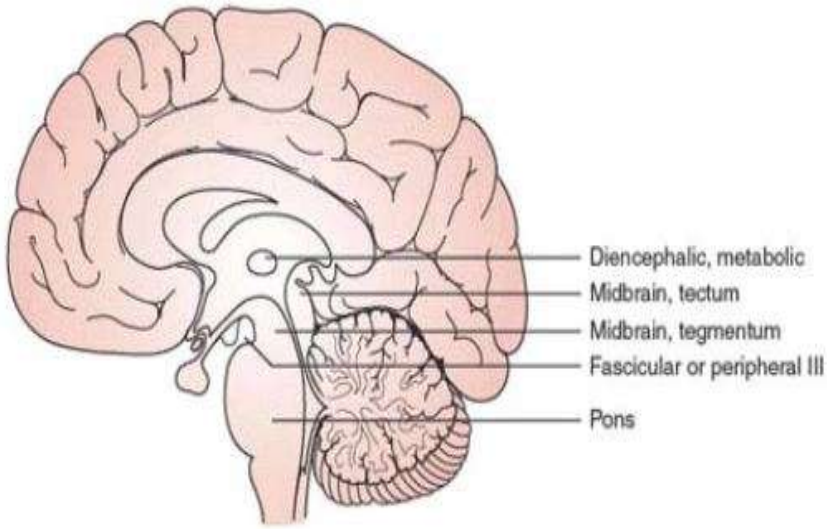
As mentioned earlier, fever is an important sign and indicates systemic processes like bacterial meningitis, encephalitis, heat stroke etc. However, hyperthermia can also be neurogenic, as in hypothalamic dysfunction and in lesions of the pontine tegmentum.

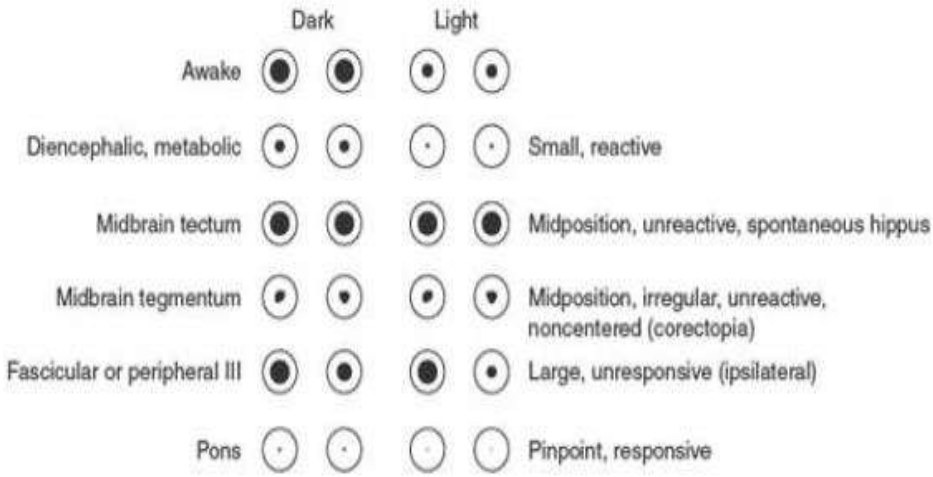
PUPILS:

Important structures like the IIIrd, IVth and VIth cranial nerve nuclei, the Medial Longitudinal Fasciculus and the Para Pontine Reticular Formation (PPRF) are situated in the brainstem amid the Reticular activating system. Hence, Lesions of the Reticular

activating system can also involve the above structures and lead to eye signs which can help in localisation and identification of the disease.

The following pupillary responses are characteristic of lesions at different levels of the brain:



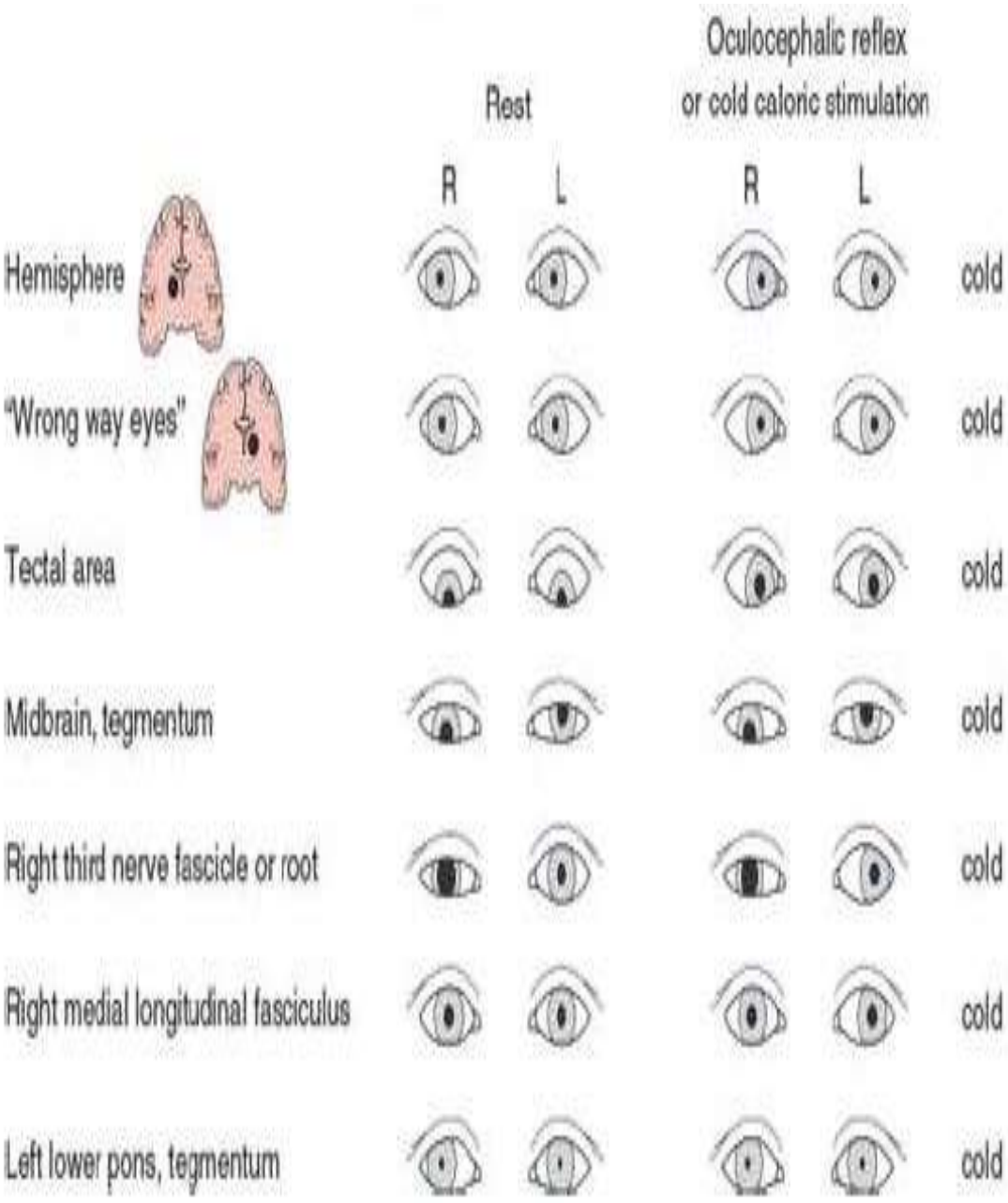


EYE MOVEMENTS:

The eyes are observed by elevating the lid if the patient is in coma and the resting position and spontaneous position of the globes should be observed. Horizontal

divergence of the eye at rest is normal in patients who are drowsy. As long as the brainstem is intact, the eyelids are closed, the eyes are slightly divergent and drift slowly from side-side, known as Roving eye movements. As coma deepens, the roving eye movements disappear first, followed by oculocephalic and oculovestibular reflex.

Conjugate horizontal roving eye movements are the most common spontaneous eye movements. If these are present, it indicates that there is no extensive damage to midbrain and pons. Conjugate horizontal ocular deviation to one side indicates damage to the frontal lobe on the same side or less commonly the pons on the opposite side. The cardinal rule is “Eyes look towards the side of the Hemispheral lesion and away from a brainstem lesion”. Exceptions include seizures involving the frontal lobe, where the eye may deviate to the opposite side and a condition known as “wrong way eyes”, in which the eyes deviate paradoxically away from the side of a deep Hemispheral lesion (reason unknown). The following image helps in identifying the patterns of ocular movements which can help in localising the disease.


Rest
Oculocephalic reflex
or cold caloric stimulation
R
L
R
L
Hemisphere
cold
"Wrong way eyes"
cold
Tectal area
cold
Midbrain, tegmentum
cold
Right third nerve fascicle or root
cold
Right medial longitudinal fasciculus
cold
Left lower pons, tegmentum
cold

Term	Description	Causes
Classical ocular bobbing	Rapid, conjugate downward movement and slow return to primary position Absence of reflex horizontal eye movements	Highly specific but not pathognomonic for an acute pontine lesion
Atypical ocular bobbing	Rapid, conjugate downward movement and slow return to primary position Reflex horizontal eye movements are preserved	Usually occurs with anoxia, and is considered a nonlocalizing finding
Ocular dipping (inverse ocular bobbing)	Slow downward eye movement is followed by a rapid return to primary position	Nonlocalizing, usually follows hypoxic insult or metabolic disorders
Reverse ocular bobbing	Initial rapid upward movement is followed by slow return to primary position	Nonlocalizing, usually follows hypoxic insult or metabolic disorders
Reverse ocular dipping (converse ocular bobbing)	Slow upward movement is followed by rapid return to primary position	Nonlocalizing, usually follows hypoxic insult or metabolic disorders
Vertical nystagmus	Vertical pendular oscillations with a frequency of 2-3 cycles/sec Differentiated from bobbing by the absence of a latency between the corrective saccade and the next slow deviation	Suggestive of pontine strokes

Corneal reflex:

Patients who are in altered sensorium/coma have a higher threshold for the corneal reflex. In patients who have impaired eye closure (as in VIIth cranial nerve lesions and lower pontine lesions), it may also lead to deviation of the jaw to the opposite side (Corneopterygoid reflex). If the upper pons and midbrain are intact, the eyes may roll upward (Bell's phenomenon).

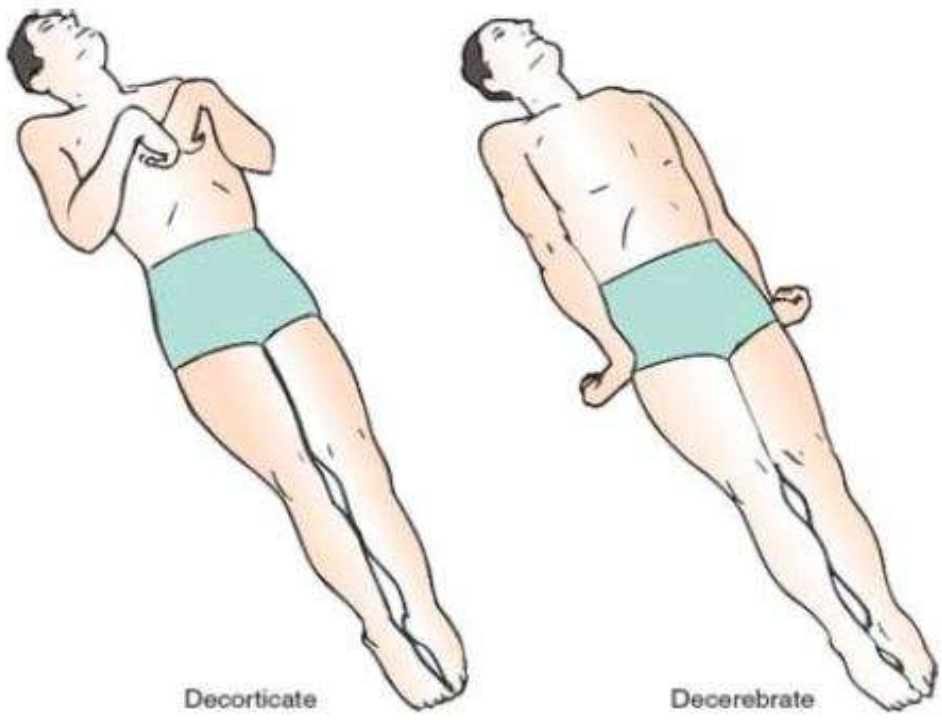
MOTOR ACTIVITY OF THE BODY AND LIMBS:

Certain motor responses act as clues to the level of the lesion; unlike the other localising signs, certain motor responses can also help in identification of the etiology of the altered sensorium/coma. Important motor activity signs that need to be looked for include:

1. Movements suggestive of Decorticate and Decerebrate rigidity.
2. Features suggestive of "Man in the barrel syndrome".
3. Tremors
4. Asterixis
5. Multifocal and generalised Myoclonus.

DECORTICATE AND DECEREBRATE RIGIDITY:

DECORTICATE RIGIDITY	DECEREBRATE RIGIDITY
The upper limbs are flexed and the lower limbs are extended.	Both the upper and lower limbs are extended.
The lesion can be localised to above the level of Midbrain.	The lesion can be localised below the level of Midbrain.
Temperature regulation is intact.	Temperature regulation is lost.
Prognosis is comparatively better.	Prognosis is poor.



MAN IN THE BARREL SYNDROME:

Anoxic lesions of the “cerebral border zones” can result in significant damage to the area of the motor cortex which has the maximum representation for the arms. Thus, these patients will have bilateral weakness of the arms with relative sparing of the lower limbs.

TREMORS:

Tremors are common in Metabolic Encephalopathy and are coarse and irregular, lasting for 8 to 10 cycles per second. The amplitude of the tremor is supposed to be the highest when the patient holds his hand outstretched.

ASTERIXIS:

Asterixis can be defined as a “sudden, brief loss of postural tone which gets translated in the form of a flapping movement; the movement becomes prominent when the hand is held in dorsiflexion at the wrist; fingers are to be extended and abducted. Thus, asterixis needs some degree of co-operation from the part of the patient. However, asterixis can also be elicited in the lower limb passively by flexion and abduction of the hip at about 60 to 90 degrees to the thighs. The resultant “flap” is a result of involuntary contraction of the adductor muscles of the hip, such as Adductor longus, Adductor brevis against gravity. Asterixis is present in patients with slight stupor and wanes as coma worsens. Unilateral asterixis can appear when a toxic encephalopathy coexists with a structural lesion of the motor pathways. “Midbrain asterixis” is characterised by lapses in postural control by the involvement of reticular activating system. This presents as frequent “drop attacks”.

MYOCLONUS:

Myoclonus can be either multifocal or generalised. Multifocal myoclonus refers to sudden, non-rhythmic twitching that affects one muscle first, then another, but without any particular pattern. It commonly involves the facial and proximal limb muscles. Causes include Uremia, Carbon dioxide Narcosis and large doses and rapid injection of I.V Penicillin. Generalised myoclonus, in contrast, involves mainly the axial musculature and is characterised by sudden contraction of the axial muscles which may cause the person to jump periodically. It is commonly seen in Hypoxic encephalopathy following Cardiac arrest and is prominent on the first day post resuscitation. Generalised Myoclonus often has a "burst suppression pattern" on EEG.

OTHER MOTOR CLUES TO AID IN DIAGNOSIS:

Lazarus sign:

When the entire brain, including the brainstem has undergone total or subtotal irreversible damage, spontaneous reflex movement of spinal origin can be witnessed in about 40% of the patients post cardiac arrest. Lazarus sign denotes complex movements of spinal origin, sometimes suggesting purposeful activity. For example, passive flexion of the neck may elicit a jerk that raises all 4 limbs off the bed. However, ancillary procedures done confirm total destruction of the brain.

Seizures:

Generalised seizures often cause transient coma and is an important differential diagnosis in the evaluation of a patient with altered sensorium. A febrile illness can also be followed by refractory status epilepticus, often in children.

CLINICAL FINDINGS IN LATERAL TRANSTENTORIAL HERNIATION (UNCAL HERNIATION):

Here, due to raised intracranial pressure, the mesial temporal lobe, consisting of the uncus anteriorly and the parahippocampal gyrus posteriorly are pushed between the ipsilateral midbrain and the sharp free edge of the tentorium cerebelli. The following series of event are sequential:

- The third cranial nerve is compressed and the ipsilateral pupil becomes progressively dilated and responds sluggishly to light. Prompt recognition and decompression surgery is mandatory at this stage, otherwise the usual progression is deadly.
- The posterior cerebral artery is compressed between the parahippocampal gyrus and the free edge of the tentorium, leading to mesial occipital infarct.
- The herniated hippocampus pushes the midbrain against the sharp edge of the dura on the opposite side. This carves out a literal notch known as Kernohan's notch on the lateral midbrain. Thus, this notch interrupts the cerebral peduncle (the corticospinal tract is involved) on the side opposite to the original lesion. This leads to hemiparesis ipsilateral to the same side of the lesion – but this hemiparesis is a "false localising sign". Thus, a dilated pupil and a hemiparesis on the same side should always raise the suspicion of a Kernohan's notch phenomenon.
- Downward displacement of the midbrain occurs by this stage causing tearing of the paramedian perforating vessels that feed the midbrain tegmentum (Duret hemorrhage). By this stage, chances of recovery are remote. The pupil that was

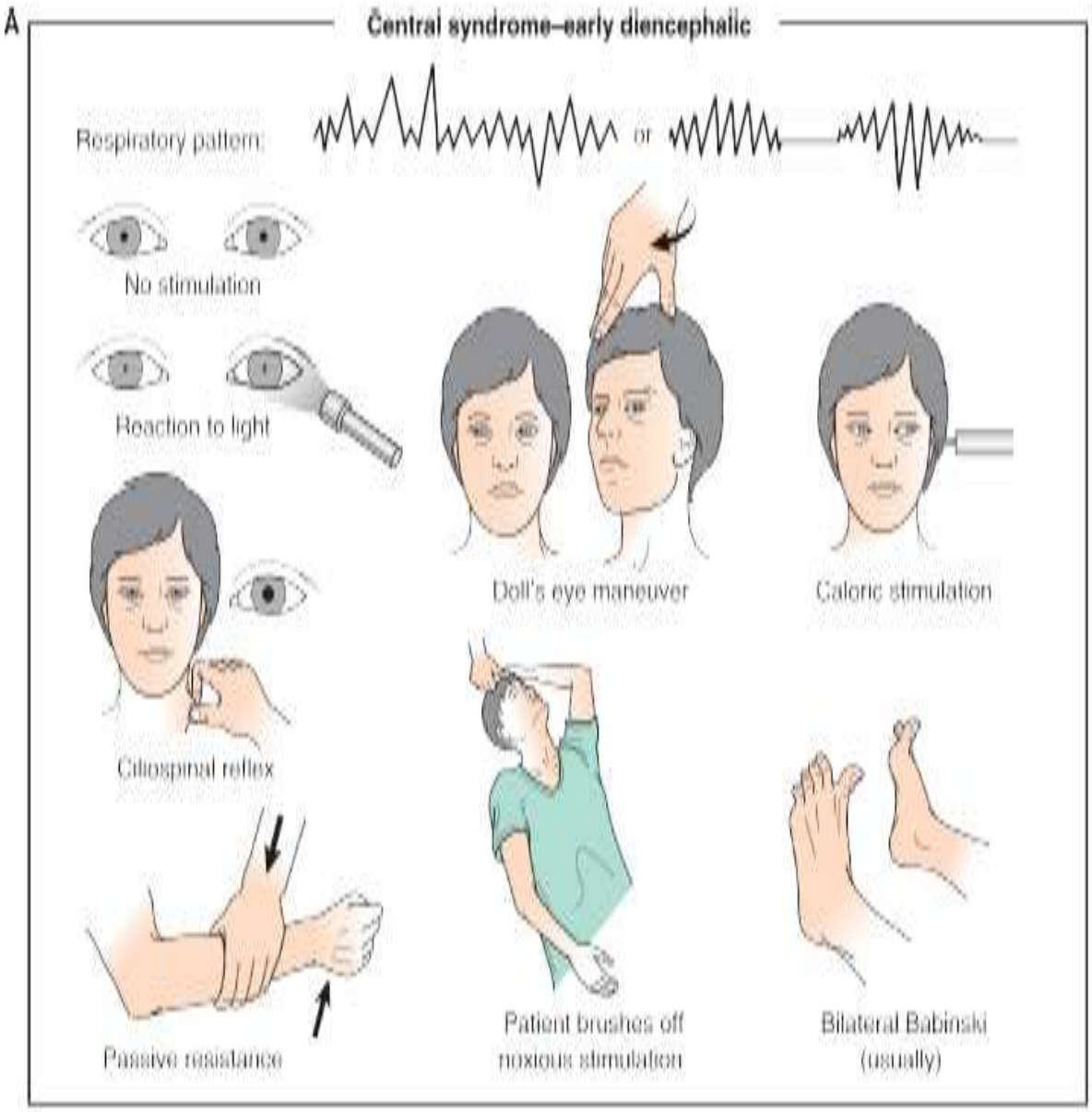
initially large becomes a little smaller while the other pupil becomes midsize and unresponsive.

CLINICAL FINDINGS IN CENTRAL TRANSTENTORIAL HERNIATION:

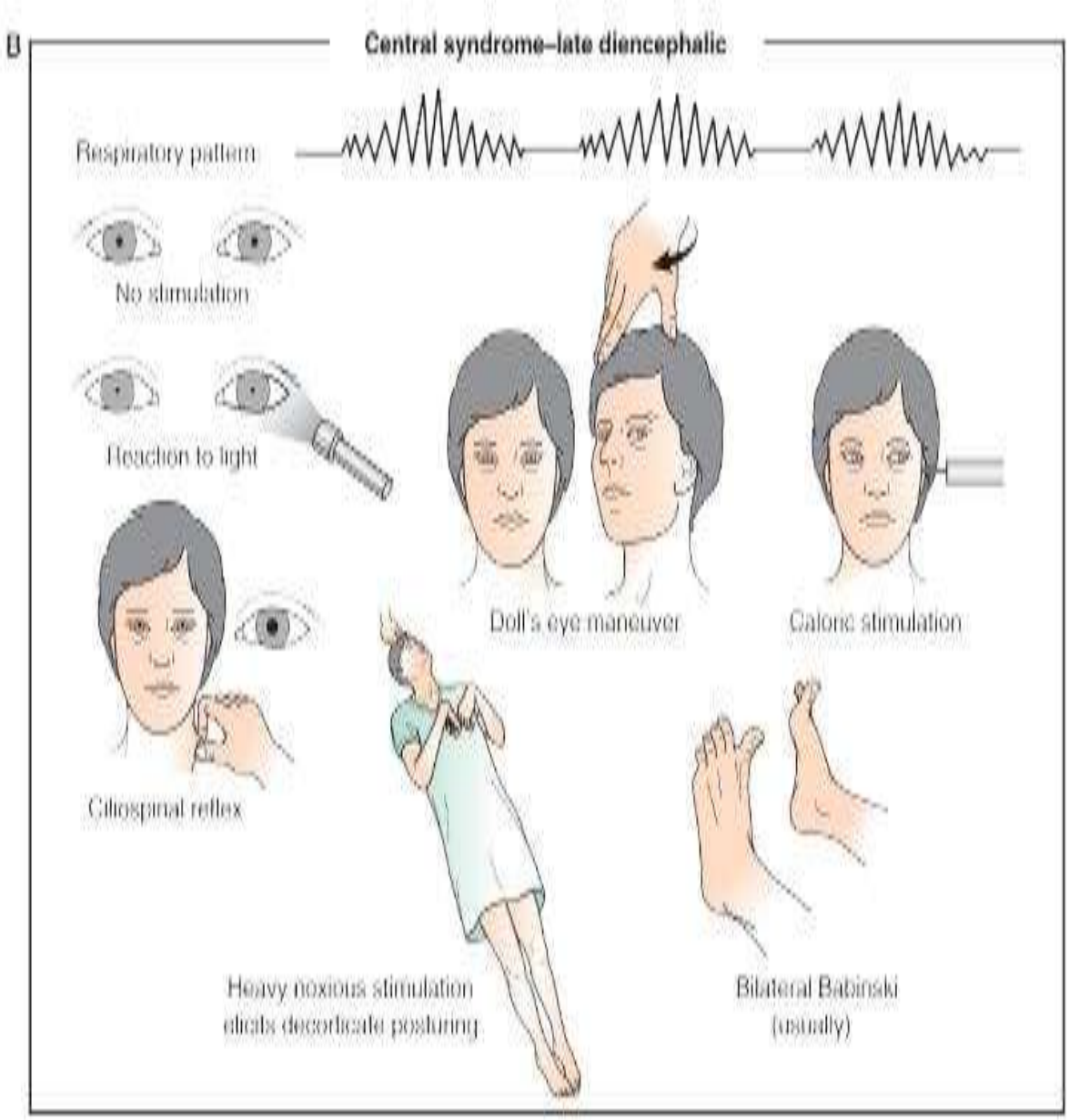
Frontal, parietal or occipital masses first compress the diencephalon which shift downwards and buckles over the midbrain. This leads to flattening of the midbrain and pons in rostro caudal direction. The characteristic evolution of this clinical picture in central transtentorial herniation is described as the “central syndrome of rostro caudal deterioration”, as the signs and symptoms evolve sequentially in the order of involvement of the brainstem structures, midbrain, pons and the medulla. It can be explained in four stages:

1. Early diencephalic stage
2. Late diencephalic stage
3. Midbrain – upper pons involvement stage.
4. Lower pons- upper medulla involvement stage.

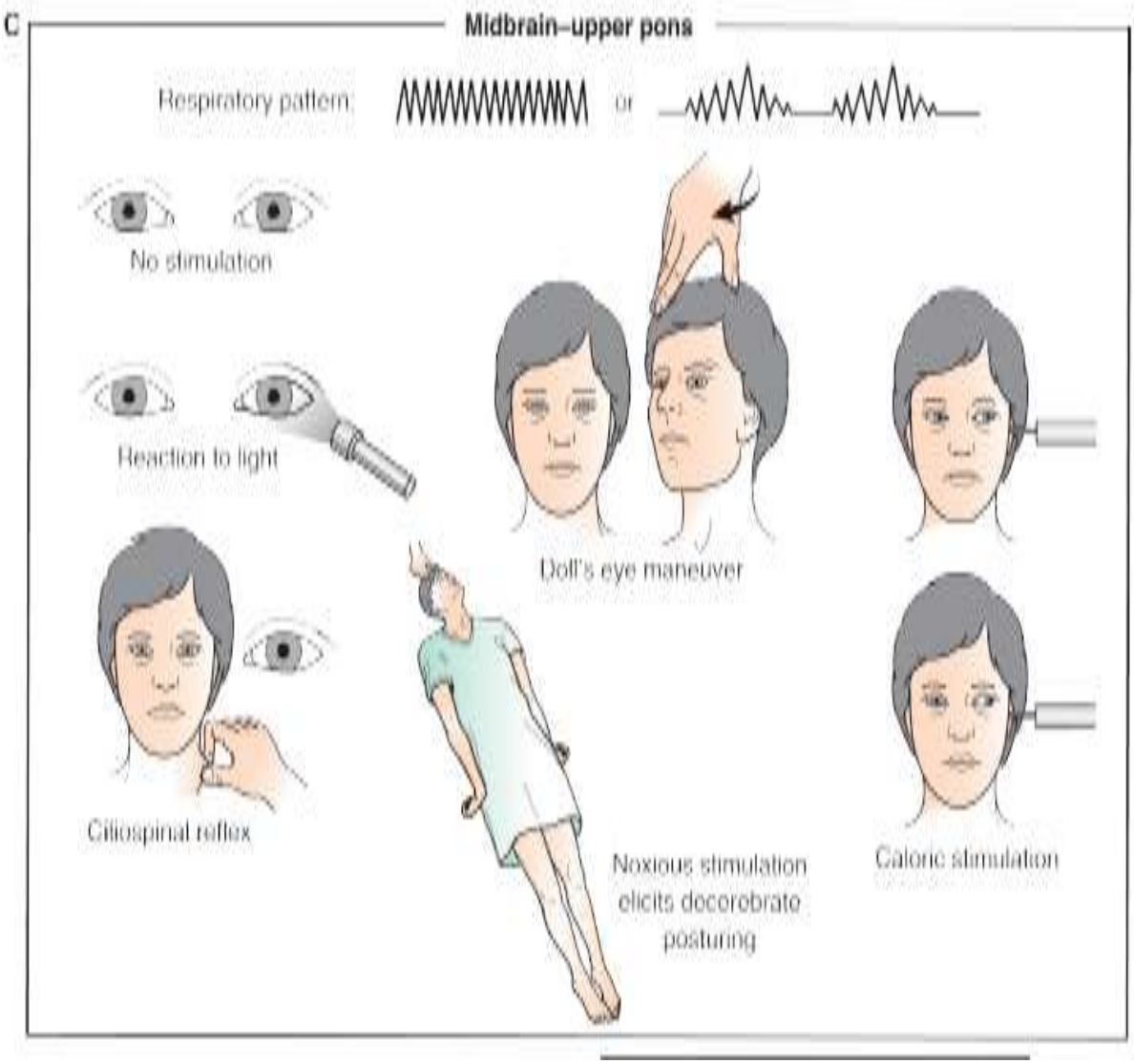
The following images are explanatory:



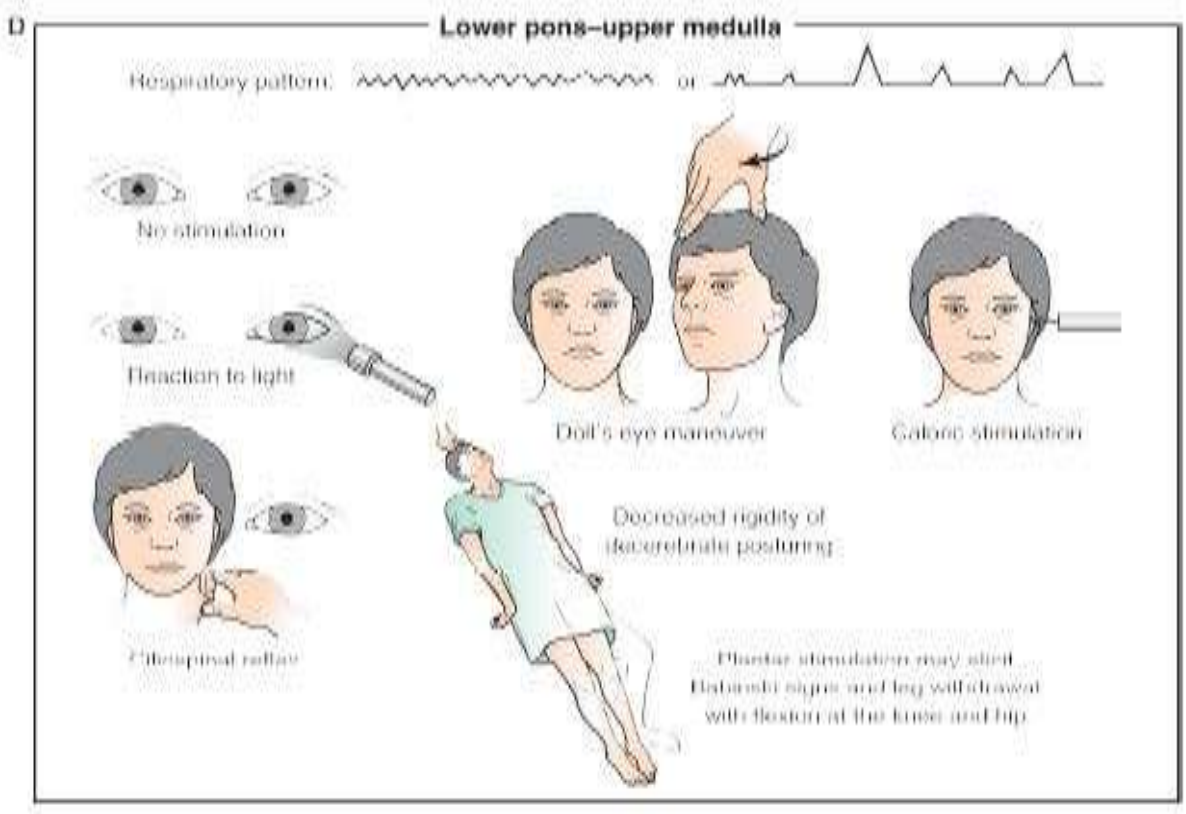
In the early diencephalic stage, there is impaired attention and somnolence. Respiratory pattern is usually normal. Pupils are tiny but react to light. Roving eye movements are present. Motor signs include Paratonia and bilateral extensor plantar.



In the late diencephalic stage, the patient cannot be aroused. Cheyne stokes respiration occurs. The pupil continues to be small yet reactive but roving eye movements disappear. Doll's eye movements are present. Patient shows decorticate posturing and bilateral extensor plantar responses. Neurological function can still be good if the patient is intervened at this stage.



When the midbrain and upper pons are compressed, temperature oscillations and diabetes insipidus can be noted because of involvement of the hypothalamus. Pupils become midsized, irregular or eccentric and do not react to light. Doll's eye response is restricted and the patient shows decerebrate posturing.



When the lower pons and upper medulla are compressed, the patient may have Apneustic breathing rarely. This is accompanied absent pupillary responses to light, absent Doll's eye movements, extensor plantar response associated with flexion of the knee and flexion of the hip (Triple response) and Decerebrate posturing (with decreased rigidity compared to the previous stage.

INVESTIGATIONS FOR DIAGNOSING ALTERED SENSORIUM/COMA:

The most important investigations used in evaluating the cause for altered sensorium include imaging such as CT/MRI, chemical and toxicological analysis of blood and urine, CSF examination and EEG. Metabolic encephalopathies are

readily identified by investigations done routinely during admission, such as Renal function tests, Liver function tests, Serum electrolytes, Serum calcium etc help in early identification. Easy availability of imaging in hospitals has shifted the focus to identification of causes of coma that can be detected by imaging (mass, cerebrovascular disease etc). But it is important to know that most of the causes of altered sensorium are metabolic or toxic in origin. Also, a normal CT scan does not rule out anatomic lesions such as acute brainstem infarction, meningitis or encephalitis. Lumbar puncture is to be performed when no cause could be readily identified. An imaging study should be carried out prior to performing lumbar puncture to exclude large intracranial mass lesion which could lead to herniation.

EEG often provides clues to metabolic or drug induced states but is rarely diagnostic. Predominant high voltage slowing in the frontal regions is typical of metabolic coma.

APPROACH TO TREATMENT:

The immediate goal in a comatose patient is stabilisation of airway, breathing and circulation and prevention of further nervous system damage. Hypotension, hypoglycemia, hypercalcemia, hypoxia, hypercapnia, and hyperthermia should be corrected rapidly. An oropharyngeal airway is adequate to keep the pharynx open in a drowsy patient who is breathing normally. Tracheal intubation is indicated if there is apnea, upper airway obstruction, hypoventilation, or emesis, or if the patient is at risk for aspiration. Mechanical ventilation is required if there is hypoventilation or a

need to induce hypocapnia in order to lower ICP. IV access should be established, and naloxone and dextrose are administered if narcotic overdose or hypoglycemia is a possibility; thiamine is given along with glucose to avoid provoking Wernicke's encephalopathy in malnourished patients. Certain toxic and drug-induced comas have specific treatments such as fomepizole for methanol and ethylene glycol ingestion.

Administration of hypotonic intravenous solutions should be monitored carefully in any serious acute brain illness because of the potential for exacerbating brain swelling. Cervical spine injuries must not be overlooked, particularly before attempting intubation or evaluation of oculocephalic responses. Fever and meningismus indicate an urgent need for examination of the CSF to diagnose meningitis. Whenever acute bacterial meningitis is suspected, antibiotics including vancomycin and a third-generation cephalosporin should be administered along with dexamethasone.

Stepwise approach in the management of Raised ICP (Intracranial Pressure):

Insert ICP monitor—ventriculostomy versus parenchymal device

General goals: maintain ICP <20 mmHg and CPP ≥60 mmHg. For ICP >20–25 mmHg for >5 min:

1. Elevate head of the bed; midline head position
2. Drain CSF via ventriculostomy (if in place)
3. Osmotherapy—mannitol 25–100 g q4h as needed (maintain serum osmolality <320 mosmol) or hypertonic saline (30 mL, 23.4% NaCl bolus)
4. Glucocorticoids—dexamethasone 4 mg q6h for vasogenic edema from tumor, abscess (avoid glucocorticoids in head trauma, ischemic and hemorrhagic stroke)
5. Sedation (e.g., morphine, propofol, or midazolam); add neuromuscular paralysis if necessary (patient will require endotracheal intubation and mechanical ventilation at this point, if not before)
6. Hyperventilation—to $Paco_2$ 30–35 mmHg (short-term use or skip this step)
7. Pressor therapy—phenylephrine, dopamine, or norepinephrine to maintain adequate MAP to ensure CPP ≥60 mmHg (maintain euvolemia to minimize deleterious systemic effects of pressors). May adjust target CPP in individual patients based on autoregulation status.
8. Consider second-tier therapies for refractory elevated ICP
   a. Decompressive craniectomy
   b. High-dose barbiturate therapy ("pentobarb coma")
   c. Hypothermia to 33°C

PROGNOSIS:

Prognostication of the patient is very important as it helps to convey to the attendants and family members of the patient the actual status of the patient and help them to mentally prepare for the eventualities. Metabolic comas usually have a better prognosis compared to traumatic causes. The Glasgow Coma Scale was devised to collect prognostic data from patients with head injuries. Since then, multiple prognostic scales have been developed; data collected from these models should be taken as approximations and the actual status of the patient in question, his age and underlying systemic illness should be taken into account.

GLASGOW COMA SCALE:

The most popular and well-known scoring system used for assessment of patients with altered sensorium and coma is the Glasgow Coma Scale – created by Dr. Graham Teasdale and Dr. Bryan Jennett in 1974. According to Dr. Teasdale, upto the early 1970s, the evaluation of patients with altered sensorium were subjective and the terminology used to convey information about the status of the patients were confusing. There had been a tendency to try to understand patients with altered sensorium with discrete levels, with such arbitrary levels made on the basis of one or two responses. This creation of arbitrary levels to describe patients with altered sensorium such as stupor, obtundation, sub comatose (with some terminologies still in use occasionally in medical literature) created confusion among physicians and led to chaos in the form of morbidity and mortality among patients which could have been avoided. Scales such as the AVPU scale[8] (Alertness, Verbal Response, Pain response and Unresponsiveness) were in vogue prior to the creation of the Glasgow Coma

Scale but were too simplistic to aid in making decisions beyond triage. Recognising this need, the development of the Glasgow Coma Scale began in earnest in the year 1971 at the Neurosurgical unit of the Institute of Neurological Sciences, Glasgow. An advantage was that this was a multidisciplinary unit catering to specialist services to a population of more than 3 million, hence there was no shortage of patients requiring specialist care. The main aim of the creators in creating the scale were to provide clear and effective communication between the referring centres and the specialist centres regarding the current status of the patient. An added aim was to link the knowledge about the patient's state of admission with its outcome. The scale was built upon a foundation of three responses – Motor, Verbal and Eye responsiveness. At the time of creation of the scale, the creators felt that the level of expertise among the practicing clinicians and nurses were low, so they did not distinguish between findings such as 'flexion and abnormal flexion'. The scale was initially envisaged to be communicated as three separate responses (eye, verbal and motor) and not as a composite single score as it is in use currently. Each component has a minimum score of 1 while the maximum scores vary according to the component (eye response has a maximum of 4, verbal response has a maximum of 5 and motor response has a maximum score of 6).

Over the last 47 years, the scale has been subjected to intense scrutiny and multiple validity and inter observer reliability trials[9] and it has to be said that, generally the scale has withstood the test of time. It has proven its worth in being a simple scale of whose components can be examined quickly in the emergency ward or the triage centre and to give an effective and quick communication not only to the specialist but also to the attenders and family members of the patient. The incorporation of GCS

into scoring systems in the ICU and the Trauma centre like the APACHE score and the Revised Trauma score is a nod to its importance. However, the creator himself does not deny that the score has some deficiencies and some of the criticism is warranted. The score is employed in more than 80 countries as the only method in use for assessment of head injury. However, it should be used with caution and as the creator recommends, it is not a substitute for proper neurological examination.

In order to address the criticisms of the Glasgow Coma Scale, new modifications such as the GCS-P[10] score have been created, incorporating the pupillary response to light to the Glasgow coma scale. In contrast to the Glasgow coma scale, where normal responses are associated with higher scores, the poorer responses in the Pupillary component of the GCS-P score are awarded higher scores so that it can be subtracted from the main score. To improve the inter-rater reliability, a website has been created with multiple video and visual aids for the assessment of the Glasgow coma scale to help physicians and nurses update and educate themselves regarding the correct technique, which was not emphasized earlier. The updated Glasgow coma scale[11] is as follows:

GLASGOW COMA SCALE : Do it this way

GCS EYES VERBAL MOTOR

Institute of Neurological Sciences NHS Greater Glasgow and Clyde

CHECK

For factors Interfering with communication, ability to respond and other injuries

OBSERVE

Eye opening , content of speech and movements of right and left sides

STIMULATE

Sound: spoken or shouted request
Physical: Pressure on finger tip, trapezius or supraorbital notch

RATE

Assign according to highest response observed

Eye opening

Criterion	Observed	Rating	Score
Open before stimulus	✔	Spontaneous	4
After spoken or shouted request	✔	To sound	3
After finger tip stimulus	✔	To pressure	2
No opening at any time, no interfering factor	✔	None	1
Closed by local factor	✔	Non testable	NT

Verbal response

Criterion	Observed	Rating	Score
Correctly gives name, place and date	✔	Orientated	5
Not orientated but communication coherently	✔	Confused	4
Intelligible single words	✔	Words	3
Only moans / groans	✔	Sounds	2
No audible response, no interfering factor	✔	None	1
Factor interferring with communication	✔	Non testable	NT

Best motor response

Criterion	Observed	Rating	Score
Obey 2-part request	✔	Obeys commands	6
Brings hand above clavicle to stimulus on head neck	✔	Localising	5
Bends arm at elbow rapidly but features not predominantly abnormal	✔	Normal flexion	4
Bends arm at elbow, features clearly predominantly abnormal	✔	Abnormal flexion	3
Extends arm at elbow	✔	Extension	2
No movement in arms / legs, no interfering factor	✔	None	1
Paralysed or other limiting factor	✔	Non testable	NT

Sites For Physical Stimulation

Finger tip pressure | Trapezius Pinch | Supraorbital notch

Features of Flexion Responses

Modified with permission from Van Der Naalt 2004
Ned Tijdschr Geneeskd

Abnormal Flexion
Slow Sterotyped
Arm across chest
Forearm rotates
Thumb clenched
Leg extends

Normal flexion
Rapid
Variable
Arm away from body

For further information and video demonstration visit www.glasgowcomascale.org

The major advantages of Glasgow Coma Scale include:

- Ease of assessment as it has only three components.
- Aids in rapid assessment and is not time consuming.
- It is the most studied scale with respect to altered sensorium and has proven validity.

The major disadvantages of Glasgow Coma Scale are:

- There is a lacuna in the assessment of the verbal component in aphasic and intubated patients; this can skew the score on addition of the components scale.
- Patients who are aphasic or have a language barrier cannot be assessed properly.
- The motor component of the score has been accorded more points, leading to a mathematical bias.
- There is an inconsistency in inter rater reliability which has been well documented in literature over a period of time.
- It does not include important neurological examination like assessment of respiratory patterns and brainstem reflexes.

In order to overcome the above shortcomings, newer coma scales such as the Maryland Coma scale, the Bouzarth Coma scale and the Clinical Neurologic Assessment Tool were introduced to supplant the GCS. However, these scales were more complex and hence did not gain popularity or widespread use.

FULL OUTLINE OF UNRESPONSIVENESS (FOUR) SCORE:

In view of the shortcomings of the Glasgow coma scale, the Full Outline of Unresponsiveness score was created by Wijdicks et al. The creators state that the main reason for creating another score was that the GCS did not detect subtle changes in the sensorium of the patients; Important neurological parameters such as brainstem reflexes and breathing patterns were not included under GCS. The creators of the score have stated that as most of the patients who are admitted with coma and altered sensorium are intubated, the verbal response could not be assessed; Physicians hence resort to assigning an arbitrary value of 1 which is not right in anyway. Hence, the new score, the Full Outline of Unresponsiveness score was introduced, which incorporated 4 main components:

- Eye response
- Motor response
- Brainstem reflexes
- Respiratory Patterns

Unlike the GCS, the FOUR score gives a minimum of 0 for each of its individual components. The maximum score is 4 for each of the components, which is uniform across all the four components, again, unlike the GCS where the Motor component has a maximum score of 6, the verbal component has a maximum score of 5 and the eye response has a maximum of 4.

FOUR Score

Eye Response

4= eyelids open or opened, tracking, or blinking to command
3= eyelids open but not tracking
2= eyelids closed but open to loud voice
1= eyelids closed but open to pain
0= eyelids remain closed with pain

Motor Response

4= thumbs-up, fist, or peace sign
3= localizing to pain
2= flexion response to pain
1= extension response to pain
0= no response to pain or generalized myoclonus status

Brainstem Reflexes

4= pupillary and corneal reflexes present
3= one pupil wide and fixed
2= pupillary or corneal reflexes absent
1= pupillary and corneal reflexes absent
0= absent pupillary, corneal, and cough reflex

Respiration

4= not intubated, regular breathing pattern
3= not intubated, Cheyne-Stokes breathing pattern
2= not intubated, irregular breathing pattern
1= intubated, breathes above ventilator rate
0= intubated, breathes at ventilator rate or apnea

Advantages of the FOUR score:

- The FOUR score can be tested in intubated patients unlike the GCS.
- Brainstem reflexes are elicited and looked for in the FOUR score which informs about the status/stage of the brainstem injury. Brainstem reflexes aren't part of GCS.
- Locked in syndrome can be recognised by FOUR score as it incorporates eye tracking as part of it.
- Signs of uncal herniation, such as unilateral dilated and fixed pupil, are incorporated as part of the FOUR score.
- Respiratory patterns are assessed as part of the FOUR score which can decide the need for intubation or other respiratory support.
- The in-hospital mortality was higher in patients with the lowest FOUR score compared to the lowest GCS, as the FOUR score can categorise the severity of the patients with the lowest GCS into different levels.
- Each component has a similar score 0 to 4, with higher scores representing better consciousness states; This is unlike the GCS which can tend to be mathematically skewed as different components have different scores.
- The absence of the verbal component in this score avoids difficulty in assessment of patients with language problems.

A few studies have been conducted comparing the validity of the GCS and FOUR score in predicting mortality; some have favored the FOUR score to be better while some have concluded that both are equivalent. A review of the studies published in the literature is as follows:

REVIEW OF PREVIOUS RELATED STUDIES:

Teasdale and Jennett (1974) published the seminal paper "Assessment of Coma and Impaired Consciousness" in Lancet. They described the Glasgow Coma scale and the need to perform individual components of the scale separately and demonstrated its use in the Neurosurgical ward, especially for Traumatic Brain injury patients. They found that the GCS was superior to the previously used AVPU scale not only in terms of better communication of the patient's condition between physicians for referral services but also for prognostication.

Wijdicks et al (2005) published an interesting study named "Validation of a New Coma Scale: The FOUR score" in the Annals of Neurology. They introduced the Full Outline of Unresponsiveness score (FOUR) as an improvement to the GCS. 120 patients were enrolled in the study and covered a wide range of causes of altered sensorium and coma such as ischemic stroke, hemorrhagic stroke, traumatic brain injury etc. They found that the FOUR score had advantages such as ability to test intubated patients, incorporation of brainstem reflexes which helps in early identification and prognostication of brain death, inclusion of signs suggestive of uncal herniation such as single dilated and fixed (unresponsive) pupil and information about respiratory patterns and documentation of the presence of respiratory drive. They suggested that the study be conducted over a wide range of population so that the validity can be determined and advised that this score replace GCS in Neurology ICUs.

Fischer et al (2010)[12] did a prospective observational study to compare the reliability of Neurologists and ICU staff in performing GCS and FOUR score (inter rater reliability). 267 patients admitted to the ICU were included in the study. They concluded that the FOUR score was better than the GCS in predicting inter rater reliability with adequate training of the participants. They also found that the reliability of rating the scores were better among Neurologists than Intensive care unit staff. The limitations of the study include the fact that Neurologists are better trained and better understand the nuances of the FOUR score as it involves testing extra components such as brainstem reflexes and respiration than GCS compared to the ICU staff who are trained in performing only the GCS.

Büyükcam et al (2012)[13] compared the GCS and FOUR score in the pediatric age group (among 100 children) admitted with trauma. Their main aim was to identify the predictive ability of GCS and FOUR score in predicting morbidity and mortality among the pediatric age group. The study included 100 children admitted to the emergency department with trauma. They found that the cut off scores for predicting mortality were 7 for GCS and 9 for FOUR score. They found no significant differences between GCS and FOUR score for predicting mortality and morbidity. However, their logistical regression analysis indicated FOUR score was better in predicting mortality and discharge of patients than GCS.

Khajeh et al (2014)[14] compared GCS and FOUR score among pediatric patients. 200 patients were selected from the Pediatric Intensive Care Unit. They found that the Four score was better than GCS in predicting mortality in the Intensive care unit.

Saika et al (2015)[15] conducted a prospective study comparing the GCS and FOUR score for predicting the mortality in patients admitted with traumatic brain injury. 138 patients were included in the study. They found that the GCS and FOUR score were comparable in predicting the mortality of patients with traumatic brain injury. Limitations of the study included a small sample size and involving only patients with traumatic brain injury.

Mouri et al (2015)[16] conducted a study to identify the ability of the FOUR score in predicting mortality in patients with Hepatic encephalopathy. They included 94 patients who were known cases of Decompensated Liver disease. They found that the FOUR score was able to identify the different stages of hepatic encephalopathy and was able to predict the onset of overt hepatic encephalopathy in these patients. They concluded that the FOUR score be put into widespread use among patients with Decompensated Liver disease for predicting hepatic encephalopathy.

Said et al (2016)[17] conducted a pilot study among intubated patients to predict the ability of GCS and FOUR score to predict extubation at the end of fourteen days as the outcome. 86 patients were included in the study. They found that FOUR score was better in predicting the outcome. Both GCS and FOUR score were equal in predicting mortality at the end of 28 days and neurological outcome at the end of 3 months.

Surabenjawong et al (2017)[18] conducted a prospective study in Thailand comparing the GCS and FOUR score in patients admitted with acute stroke. 60 patients were included in the study. They found that the FOUR score was better than GCS in predicting neurological outcome; They also found FOUR score to be better than GCS in predicting mortality at the end of three months for acute stroke.

Zeiler et al (2017)[19] did a prospective study in patients with subarachnoid hemorrhage. 64 patients were included in the study. The main aim of the study was to identify the ability of the FOUR score in predicting mortality in patients who developed subarachnoid hemorrhage following aneurysmal rupture. They found that the P value was <0.05 indicating that the ability of FOUR score in predicting mortality was statistically significant. The main limitation of the study like others were the small sample size and involvement of patients with only one diagnosis.

Ramazani et al (2019) compared the GCS and FOUR score in the Medical intensive care unit. They did an observational study with 300 patients who were admitted consecutively in the Medical ICU. The study was conducted over a period of 14 months. They concluded from their data analysis that there were significant statistical differences in GCS and FOUR score between non survivors and survivors. The discrimination power was good for both scores with Area under the curve being 87.3% for FOUR score and 82.6% for GCS. They concluded that both GCS and FOUR scores were good for predicting outcomes in the ICU; they also concluded that the FOUR score had better discrimination and was better than GCS in predicting outcomes in the ICU. The limitations of the study included a mix of cases being used with cases of stroke being more compared to other cases. They also found difficulty in selecting an appropriate model of statistics for improving predicting ability of the models.

STUDY JUSTIFICATION

- The Glasgow coma scale is widely used all over the world for assessment of patients admitted with altered sensorium and coma. However, the shortcomings of the Glasgow coma scale are well known, including variations in inter rater reliability, difficulty in assessing intubated patients, non-assessment of brainstem reflexes etc.
- The FOUR score (Full Outline of Un Responsiveness score) was introduced in 2005 by Wijdicks et al. The score included new components such as brainstem reflexes and respiration while making away with the verbal component assessment. The score thus did not have many of the disadvantages associated with GCS and could be performed in quick time with adequate training.
- Multiple studies have been conducted comparing the validity of GCS and FOUR score in predicting mortality. However, they have been hampered by small sample sizes; Comparison studies such as these should always be conducted across different population groups to identify validity.
- Hence the purpose of this study is to compare the ability of the FOUR score in relation with the Glasgow Coma Scale in predicting mortality and neurological outcome. If the FOUR score consistently proves better outcomes across different populations, then recommendations can be given to incorporate the score in assessing patients with altered sensorium universally.

AIM OF THE STUDY

To compare the Full Outline of Un Responsiveness score (FOUR) with the Glasgow Coma scale (GCS) in predicting mortality and neurological outcome in patients admitted with altered mental status in the Medical ICU.

MATERIALS AND METHODS

STUDY DESIGN:

Comparative diagnostic study

STUDY PLACE:

Medical ICUs of Rajiv Gandhi Government General Hospital, Institute of Internal Medicine, Madras Medical College and COVID ICU, Rajiv Gandhi Government General Hospital, Chennai.

STUDY PERIOD:

May 2021 to October 2021.

STUDY POPULATION:

Patients admitted with altered mental status in the Medical ICUs and COVID ICU of Rajiv Gandhi Government General Hospital, Chennai.

CASE DEFINITION:

Patients admitted with altered sensorium (defined by International Classification of Diseases – ICD 10 as “any measure of arousal other than normal”) and patients/legal attenders consenting to the study.

INCLUSION CRITERIA:

1. Patients above 18 years of age presenting with altered sensorium (defined by ICD 10 as “any measure of arousal other than normal”).
2. Patients/Legal representatives giving consent to the study.

EXCLUSION CRITERIA:

1. Patients/Legal representatives not willing to participate in the study.
2. Patients less than 18 years of age.
3. Patients with Traumatic brain injury/ polytrauma.
4. Patients diagnosed as “brain dead” at the time of admission (determined by the American Academy of Neurology Criteria).

SAMPLE SIZE:

250 patients (above 18 years of age admitted with altered sensorium, who met the eligibility criteria during the study period.

SAMPLING TECHNIQUE:

Nil.

METHODS:

250 patients admitted with altered sensorium were selected from Medical ICUs (with a subset of patients from COVID ICU). These patients were selected after confirming that they met the eligibility criteria (inclusion and exclusion criteria) and after obtaining informed consent from either the patient or the legal attender. After obtaining detailed history, patients admitted with altered level of consciousness were

examined. FOUR score and GCS of the patients were calculated at the time of admission and after 24 hours. Traumatic Brain injury was excluded by Non contrast CT scan of the Brain. Detailed clinical and neurological examination was performed for all the patients enrolled in the study. Basic blood investigations were recorded. The outcome of the patients was divided into Survivors and Non survivors. Among survivors, the Neurological Outcome was calculated at the time of discharge and at the end of three months using the Modified Rankin scale. The two scores were compared to identify which score predicted mortality and neurological outcome accurately.

ASSESSMENT OF THE GLASGOW COMA SCALE:

1)Assess Eye Response:

Spontaneous Eye Opening – 4

Eye opens in response to speech – 3

Eye opens in response to pain – 2

No response – 1.

2) Assess Verbal Response:

Oriented to time, place and person – 5

Confused – 4

Words – 3

Sounds – 2

No response – 1.

3) Assess Motor response:

Obeys oral commands – 6

Localises pain – 5

Flexion response to pain – 4

Abnormal flexion response to pain – 3

Extension response to pain – 2

No response to pain – 1.

Total score of the Glasgow coma scale – 15.

ASSESSMENT OF FOUR SCORE (FULL OUTLINE OF UN RESPONSIVENESS SCORE):

1)Assessment of eye response:

Opens eye spontaneously, tracks, blinks to command – 4

Opens eye, does not track or blink to command – 3

Eyes closed, open to loud voice – 2

Eyes closed, open to painful stimulation – 1

Eyes remain closed after painful stimulation – 0.

2)Assessment of motor response:

Obeys commands, makes thumbs up, fist or peace sign – 4

Localises painful stimulus – 3

Flexion response to pain – 2

Extension response to pain – 1

No response/ generalised myoclonus – 0

3)Assessment of brainstem reflexes:

Pupil and corneal reflexes present – 4

One pupil wide and fixed – 3

Pupil or corneal reflexes absent – 2

Pupil and corneal reflexes absent – 1

Absent pupil, corneal and cough reflex – 0

4) Assessment of Respiratory pattern:

Not intubated, regular breathing pattern – 4

Not intubated, Cheyne Stokes breathing pattern -3

Not intubated, irregular breathing – 2

Intubated, Breathes above ventilator rate – 1

Intubated, does not breathe above ventilator rate/ apnea – 0.

MODIFIED RANKIN SCALE FOR NEUROLOGIC DISABILITY[20]

0 – No symptoms.

1 – No significant disability, despite symptoms; able to perform all usual duties and activities.

2 – Slight disability, unable to perform all previous activities but able to look after own affairs without assistance.

3 – Moderate disability; requires some help, but able to walk without assistance.

4 – Moderately severe disability, unable to walk without assistance and unable to attend to own bodily needs without assistance.

5 – Severe disability, bedridden; incontinent and requires constant nursing care and attention.

6 – Death.

The above scores are determined by the following standardized questionnaire (yes/no questions):

- Do you have any symptoms that are bothering you?
- Are you able to do the same work as before?
- Are you able to keep up with your hobbies?
- Have you maintained your ties to friends and family?
- Do you need help making a simple meal or doing household chores?
- Do you need help with eating, going to the toilet or bathing?
- Do you need help with shopping or travelling close to home?

- Do you need another person to help you walk?
- Do you stay in bed most of the day and need constant nursing care?

STATISTICAL ANALYSIS:

The data obtained were analysed with Statistical analysis software (SPSS 23) and the following statistical methods were used to arrive at a conclusion:

Descriptive statistics (frequency tables, mean and standard deviation), graphical analysis, correlation and comparative analysis, Chi square test, assessment of sensitivity and specificity and Receiver Operator Characteristic curves. The results of the study are described in the following pages.

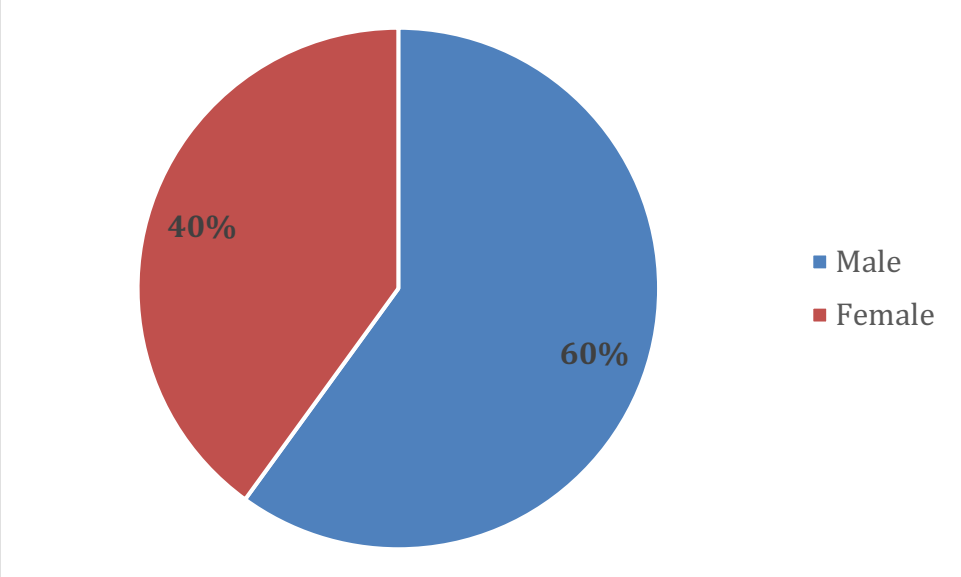
RESULTS

Descriptive analysis – Baseline Characteristics of the Study Population:

Table 1: Descriptive analysis of Gender Distribution in study population (N=250)

Gender Distribution	Frequency	Percentage
Male	150	60%
Female	100	40%
Total	250	100%

Figure 1: Pie chart of Gender Distribution (N=250)

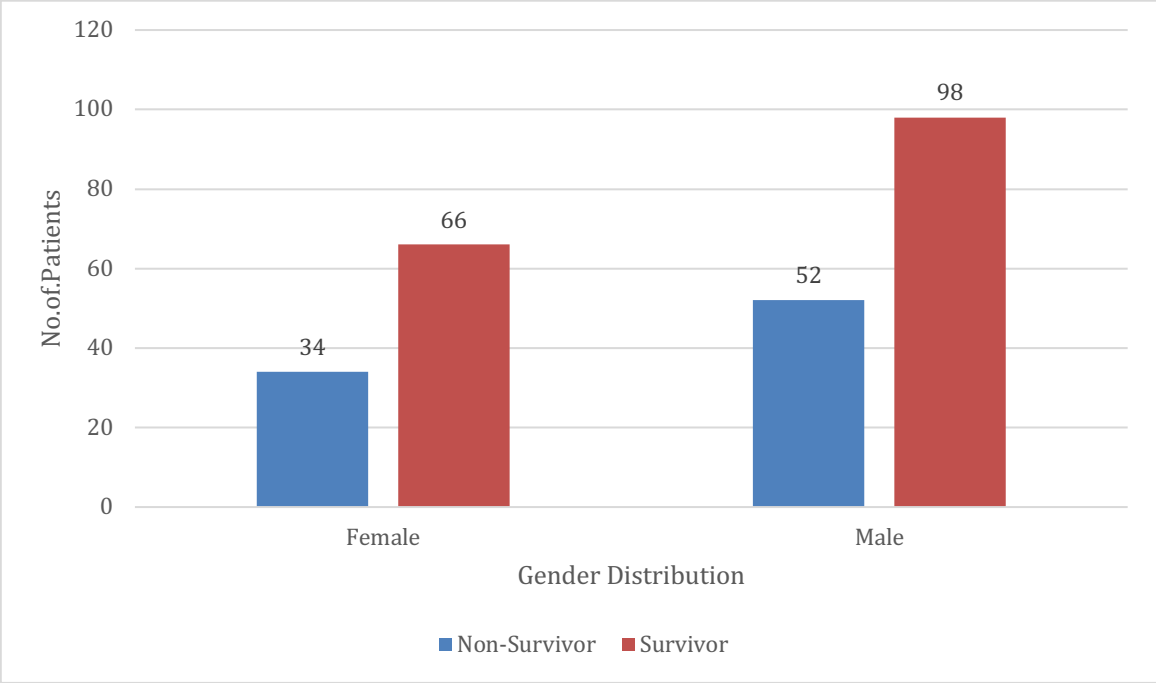


A total of 250 participants were enrolled in the study. In that 60% of the population were males (n= 150) and 40% of the population were females (n=100).

Table 2: Comparative analysis of Gender and Outcome in study population (N=250)

Gender	Outcome		Total	P-value
	Non-Survivor	Survivor		
Female	34(39.53%)	66(40.24%)	100(40%)	0.913
Male	52(60.47%)	98(59.76%)	150(60%)	
Total	86(100%)	164(100%)	250(100%)	

Figure 2: Bar chart for Gender and Outcome (N=250)

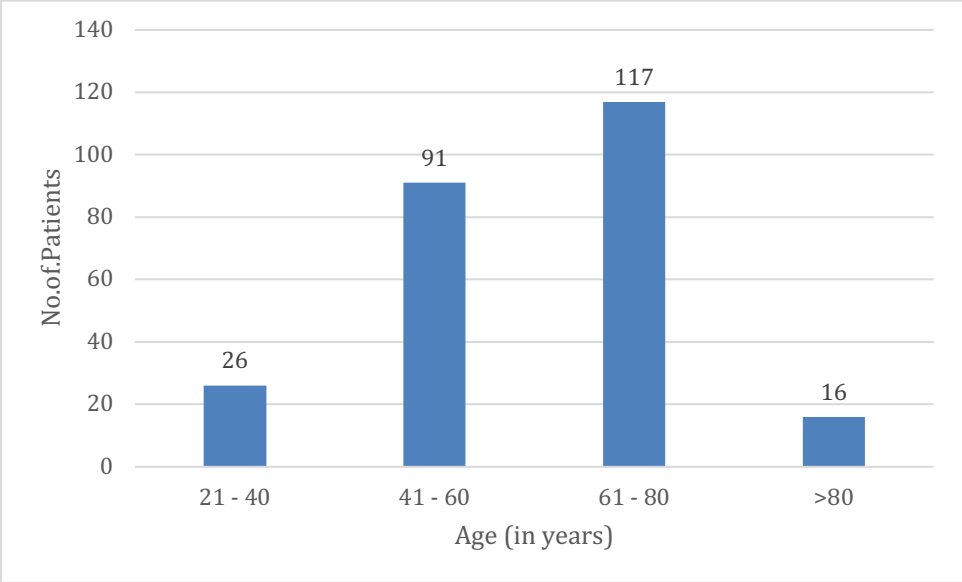


In the study, 164 patients survived (65.60%), while 86 patients expired (34.40%). 66% of the females enrolled in the study survived (n=66), while 34% of the females expired (n=34). Among males, 65.33% of the males enrolled in the study survived (n=98), while 34.67% of the males expired (n=52). There was no statistical significance between the gender of the study population and the outcome, as given by the p value of 0.913.

Table 3: Descriptive analysis of Age Distribution in study population (N=250)

Age Distribution	Frequency	Percentage
21 - 40	26	10.40%
41 - 60	91	36.40%
61 - 80	117	46.80%
>80	16	6.40%
Total	250	100%

Figure 3: Bar chart of Age Distribution (N=250)

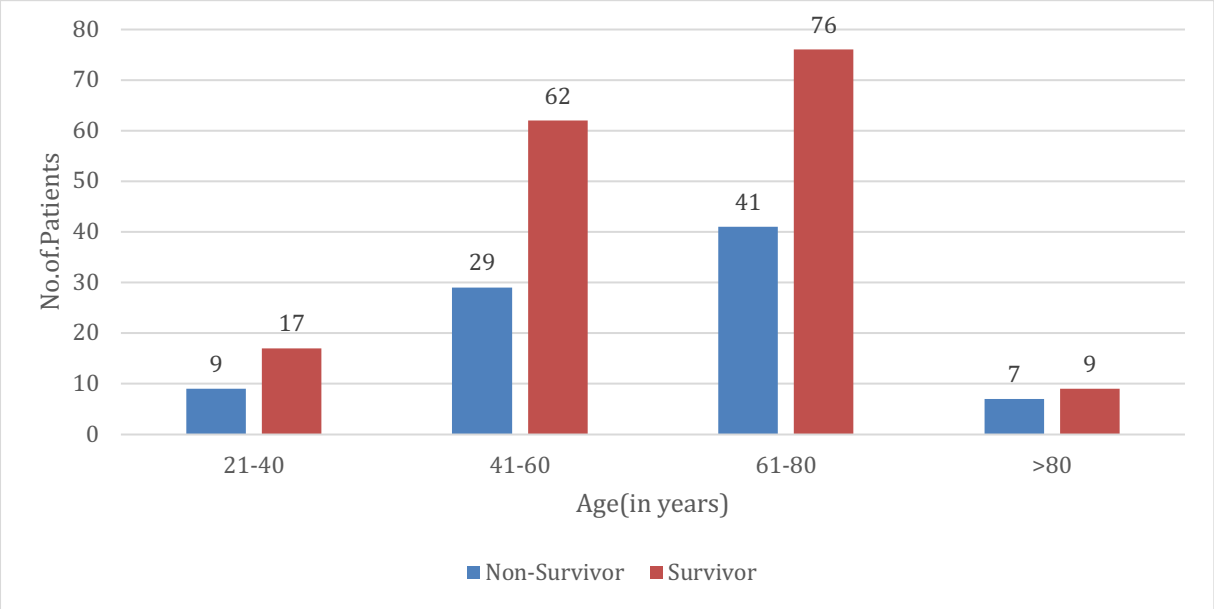


Among the 250 participants in the study, most of the enrolled study population were between the age group 61-80 (46.80%, n=117). This reflects the fact that the common causes of altered sensorium included in the study population – metabolic encephalopathies and ischemic stroke are common in this age group.

Table 4: Comparative analysis of Age and Outcome in study population (N=250)

Age	Outcome		Total	P-value
	Non-Survivor	Survivor		
21-40	9(10.47%)	17(10.37%)	26(10.40%)	0.825
41-60	29(33.72%)	62(37.80%)	91(36.40%)	
61-80	41(47.67%)	76(46.34%)	117(46.80%)	
>80	7(8.14%)	9(5.49%)	16(6.40%)	
Total	86(100%)	164(100%)	250(100%)	

Figure 4: Bar chart for Age Vs Outcome (N=250)

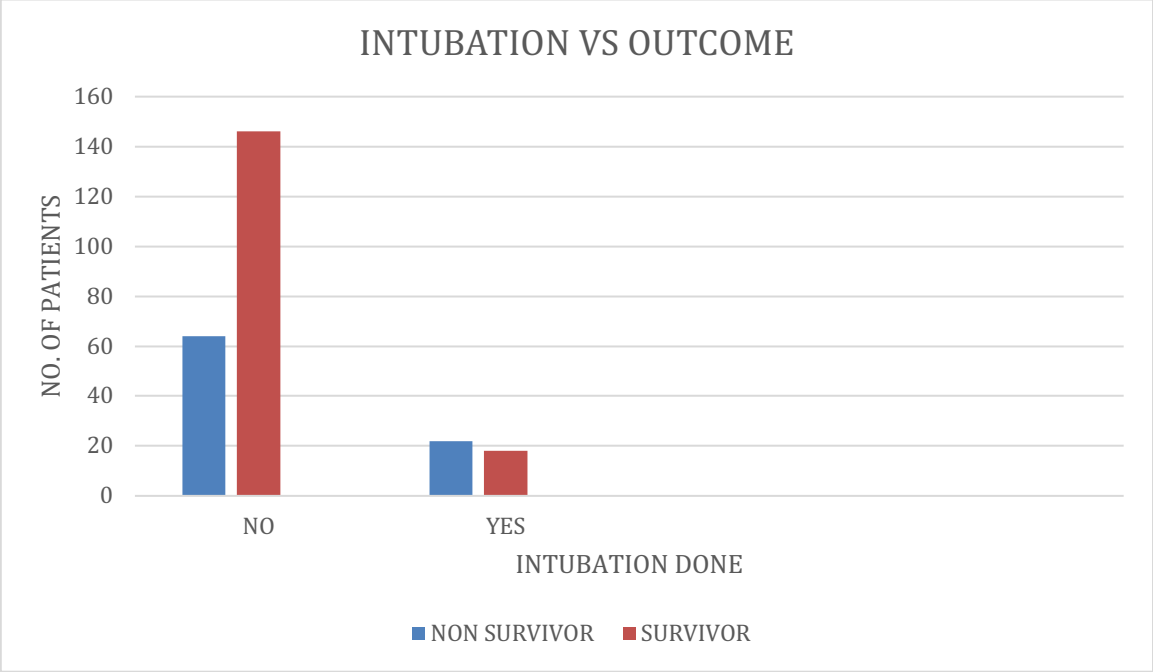


In the study population, maximal mortality was seen in the age group of 61-80 years (n=41, 47.67%). However, there was no statistical significance between the age group of the study population and the outcome, as determined by the p value of 0.825. Interestingly, the mortality in the age group 21-40 years (total participants 26, mortality - 10.47%) was higher than that in the patients more than 80 years (total participants 16, mortality -8.14%). This can be explained by the fact that most of the deaths in the younger age group were due to enrolment of patients admitted with Paraquat poisoning, which generally has a poor prognosis.

Table 5: Comparative analysis of Intubation and Outcome in study population (N=250)

Intubation	Outcome		Total	P-value
	Non-Survivor	Survivor		
No	64(74.42%)	146(89.02%)	210 (84%)	**0.121**
Yes	22(25.58%)	18(10.98%)	40 (16%)	
Total	86(100%)	164(100%)	250(100%)	

Figure 5: Bar chart for intubation Vs Outcome (N=250)



40 intubated patients were enrolled in the study (16% of the total study population). Interestingly, the non survivors were higher than survivors in the intubated population. This could be due to the fact that the intubated patients are usually the sickest. With 84% of the study population not intubated, this led to a skew and resulted in no statistical significance between intubation and outcome (p value = 0.121)

Table and Figure 6: Descriptive analysis of Glasgow Coma Scale at admission

Glasgow Coma Scale	Eye Response	Verbal Response	Motor Response
1-2	80(32%)	85(34%)	34(13.60%)
3-4	170(68%)	127(50.80%)	62(24.80%)
5-6	0(0%)	0(0%)	154(61.60%)
Not Testable	0(0%)	38(15.20%)	0(0%)
Total	250(100%)	250(100%)	250(100%)

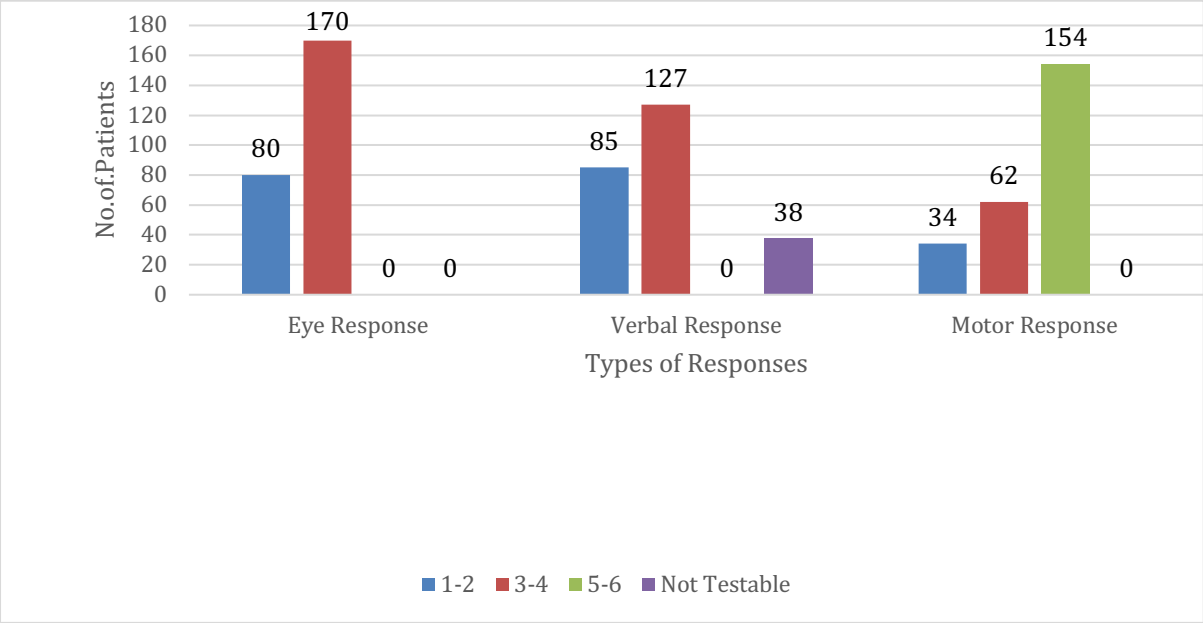
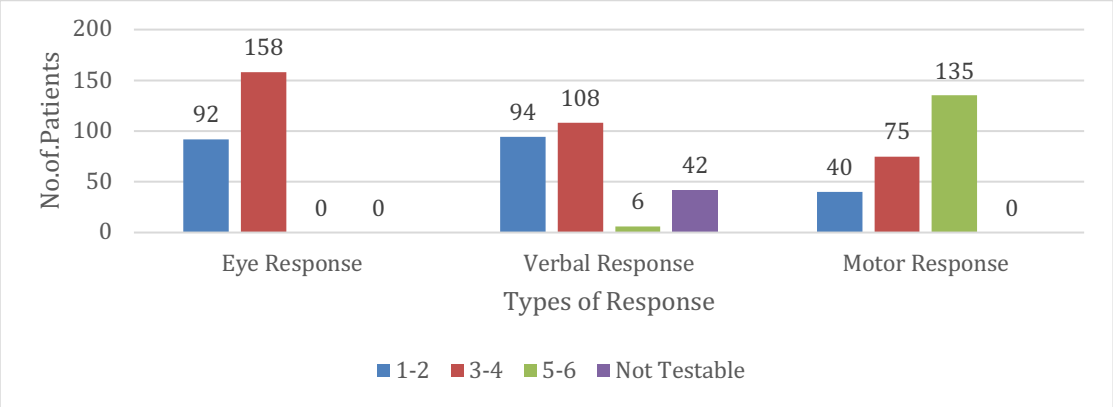


Table and Figure 7: Descriptive analysis of Glasgow Coma Scale 24 hours after admission

GCS	Eye Response	Verbal Response	Motor Response
1-2	92(36.80%)	94(37.60%)	40(16%)
3-4	158(63.20%)	108(43.20%)	75(30%)
5-6	0(0%)	6(2.40%)	135(54%)
Not Testable	0(0%)	42(16.80%)	0(0%)
Total	250(100%)	250(100%)	250(100%)



The tables and figures 6 and 7 show the Eye, motor and verbal responses of the patients at the time of admission and 24 hours after admission (as part of the Glasgow coma scale). The important aspect that can be seen here is that the Verbal responses could not be quantified for around 40 patients because the verbal responses could not be tested in patients with aphasia and in those who are intubated. This is one of the fallacies of the GCS.

Table 8: Descriptive analysis of the FOUR Score at the time of admission (N=250)

FOUR Score	Eye Response	Brainstem Reflexes	Motor Response	Respiration
0	9(3.60%)	1(0.40%)	19(7.60%)	9(3.60%)
1-2	222(88.80%)	34(13.60%)	91(36.40%)	36(14.40%)
3-4	19(7.60%)	215(86%)	140(56%)	205(82%)
Total	250(100%)	250(100%)	250(100%)	250(100%)

Figure 8: Bar chart of the FOUR Score at Admission (N=250)

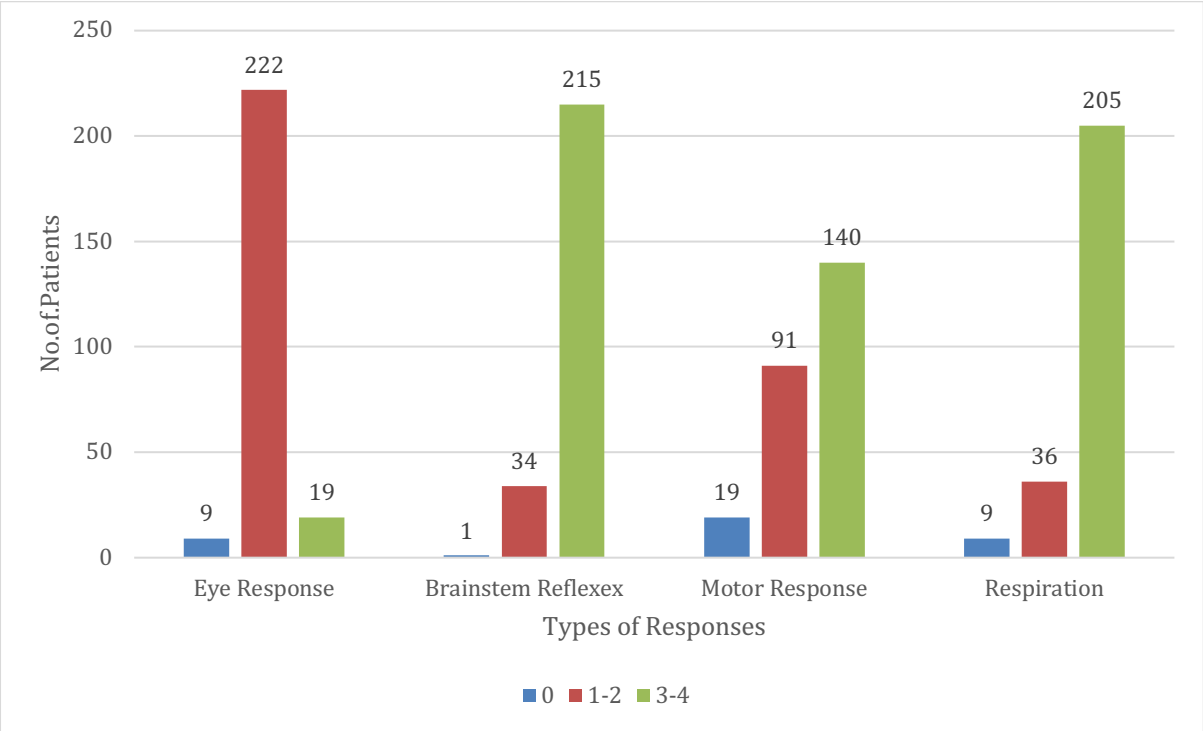
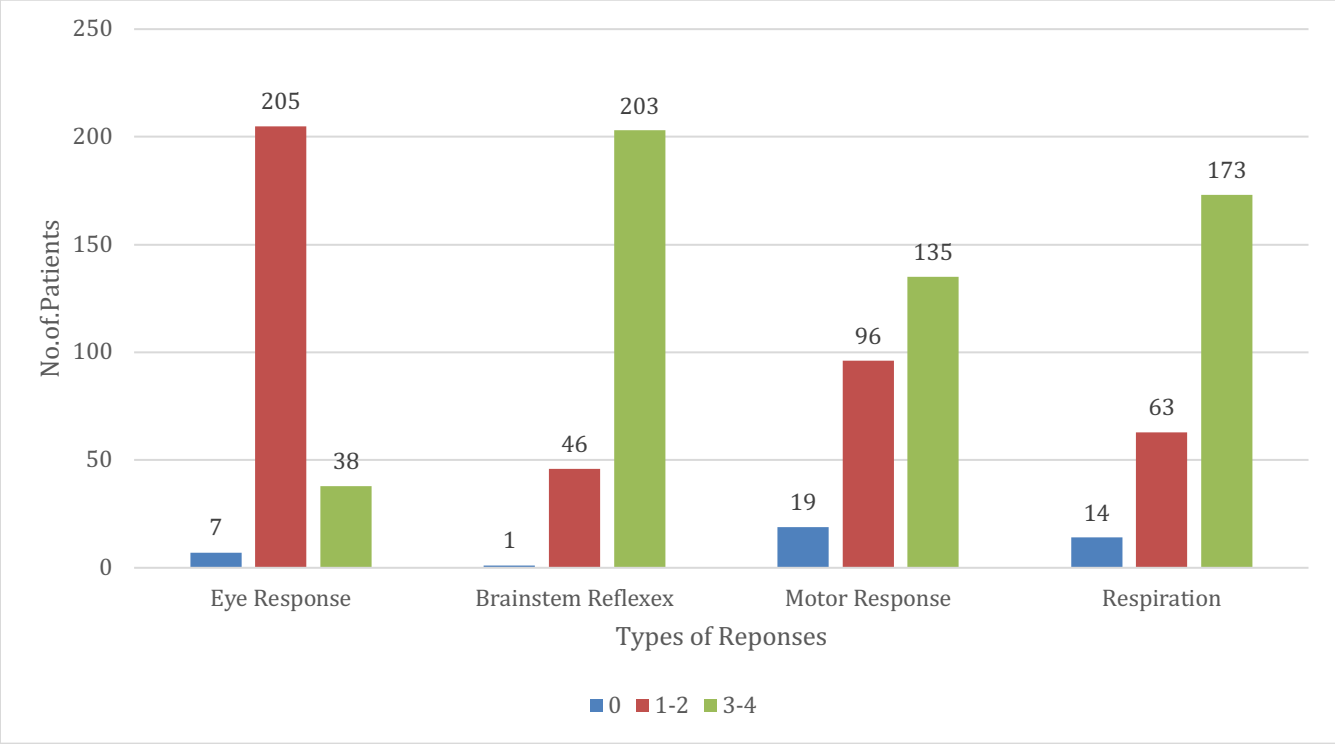


Table 9: Descriptive analysis of the FOUR Score at 24 hours after admission

FOUR Score	Eye Response	Brainstem Reflexes	Motor Response	Respiration
0	7(2.80%)	1(0.40%)	19(7.60%)	14(5.60%)
1-2	205(82%)	46(18.40%)	96(38.40%)	63(25.20%)
3-4	38(15.20%)	203(81.20%)	135(54%)	173(69.20%)
Total	250(100%)	250(100%)	250(100%)	250(100%)

Table 9: Bar chart of the FOUR Score at 24 hours after Admission (N=250)



All the component of the FOUR score are uniform, with a maximum score of 4 and a minimum score of 0, unlike the GCS. This helps in better comparison and prevents skewing of the results towards one of the components.

Table 10: Comparative analysis of the Motor Response of GCS at 24hrs after admission and Outcome in study population (N=250)

Motor Responses	Outcome		Total	Modified Rankin scale for Survivors At 3 months after Discharge	P-value
	Non-Survivor	Survivor			
1-2	36(41.86%)	4(2.44%)	40(16%)	4±0	**<0.001 ***
3-4	42(48.84%)	33(20.12%)	75(30%)	1.60±1.41	
5-6	8(9.30%)	127(77.44%)	135(54%)	1.66±1.59	
Total	86(100%)	164(100%)	250(100%)		

Figure 10: Bar chart for Motor Response of GCS Vs Outcome (N=250)

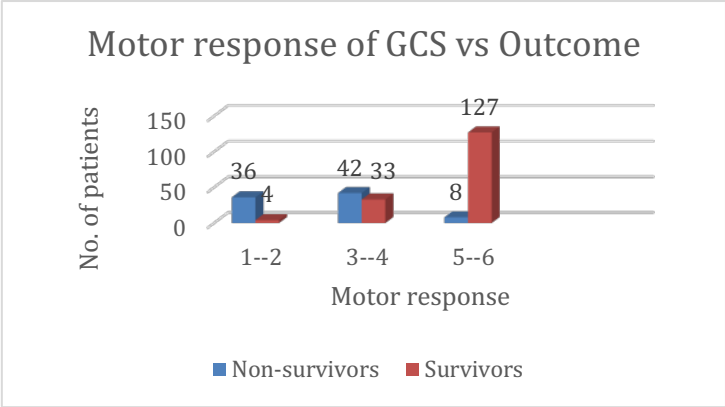


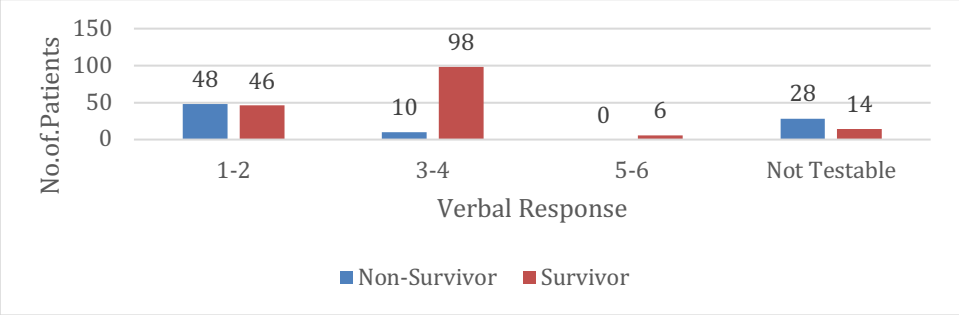
Table 11: Comparative analysis of Eye Response of GCS at 24 hours after admission and Outcome in study population (N=250)

Eye Responses	Outcome		Total	Modified Rankin scale for Survivors At 3 months post Discharge	P-value
	Non-Survivor	Survivor			
1-2	70(81.40%)	22(13.41%)	92(36.80%)	1.54±1.10	**<0.001***
3-4	16(18.60%)	142(86.59%)	158(63.20%)	1.73±1.64	
Total	86(100%)	164(100%)	250(100%)		

Table 12: Comparative analysis of Verbal Response of GCS at 24 hours after admission and Outcome in study population (N=250)

Verbal Responses	Outcome		Total	Modified Rankin scale for Survivors At 3 months post Discharge	P-value
	Non-Survivor	Survivor			
1-2	48(55.81%)	46(28.05%)	94(37.60%)	1.80±1.51	**<0.001 ***
3-4	10(11.63%)	98(59.76%)	108(43.20%)	1.79±1.63	
5-6	0(0%)	6(3.66%)	6(2.40%)	0	
Not Testable	28(32.56%)	14(5.54%)	42(16.80%)	1.57±1.39	
Total	86(100%)	164(100%)	250(100%)		

Figure 12: Bar chart for Verbal Response of GCS Vs Outcome (N=250)



From the table and Figures above, we can conclude that there is a statistically significant relationship between the various individual components of the GCS in predicting the outcome (lower scores are associated with increased mortality in each individual score), as given by the p value of <0.001 for each of the individual components. For predicting the neurological outcome of the survivors, we can see that the Motor component of the GCS is better compared to the other two components. Lower scores in the motor component of the GCS are clearly associated with poor neurological outcome (Modified Rankin scale ≥4). Neurological outcome could not be predicted from the Eye and Verbal components.

Table 13: Comparative analysis of the Eye Response of the FOUR score at 24 hours after admission and Outcome in study population (N=250)

Eye Responses	Outcome		Total	Modified Rankin scale for Survivors At 3 months post Discharge	P-value
	Non-Survivor	Survivor			
0	7(8.14%)	0(0%)	7(2.80%)	NIL	**<0.001***
1	61(70.93%)	20(12.20%)	81(32.40%)	1.65±1.08	
2	16(18.60%)	108(65.85%)	124(49.60%)	1.74±1.70	
3	2(2.33%)	5(3.05%)	7(2.80%)	0.8±0.83	
4	0(0%)	31(18.90%)	31(12.40%)	1.80±1.49	
Total	86(100%)	164(100%)	250(100%)		

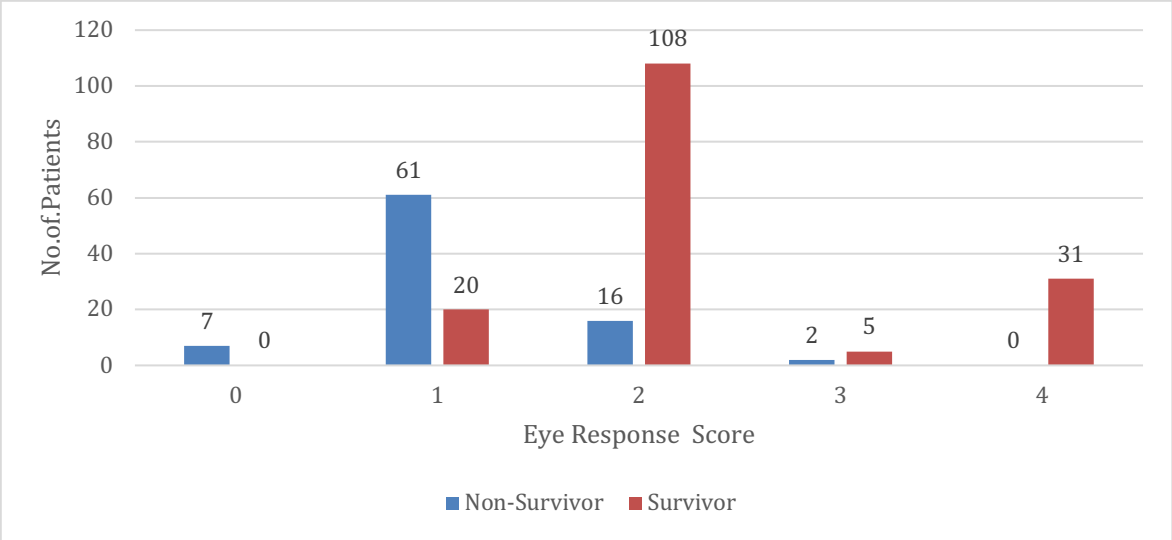


Table 14: Comparative analysis of the Motor Response of the FOUR score at 24 hours after admission and Outcome in the study population (N=250)

Motor Responses	Outcome		Total	Modified Rankin scale for Survivors At 3 months post Discharge	P-value
	Non-Survivor	Survivor			
0	15(17.44%)	4(2.44%)	19(7.60%)	4±0	**<0.001***
1	21(24.42%)	0(0%)	21(8.40%)	NIL	
2	42(48.84%)	33(20.12%)	75(30%)	1.60±1.41	
3	8(9.30%)	99(6037%)	107(42.80%)	1.69±1.67	
4	0(0%)	28(17.07%)	28(11.20%)	1.57±1.31	
Total	86(100%)	164(100%)	250(100%)		

Figure 14: Bar chart of motor response of the FOUR score vs outcome

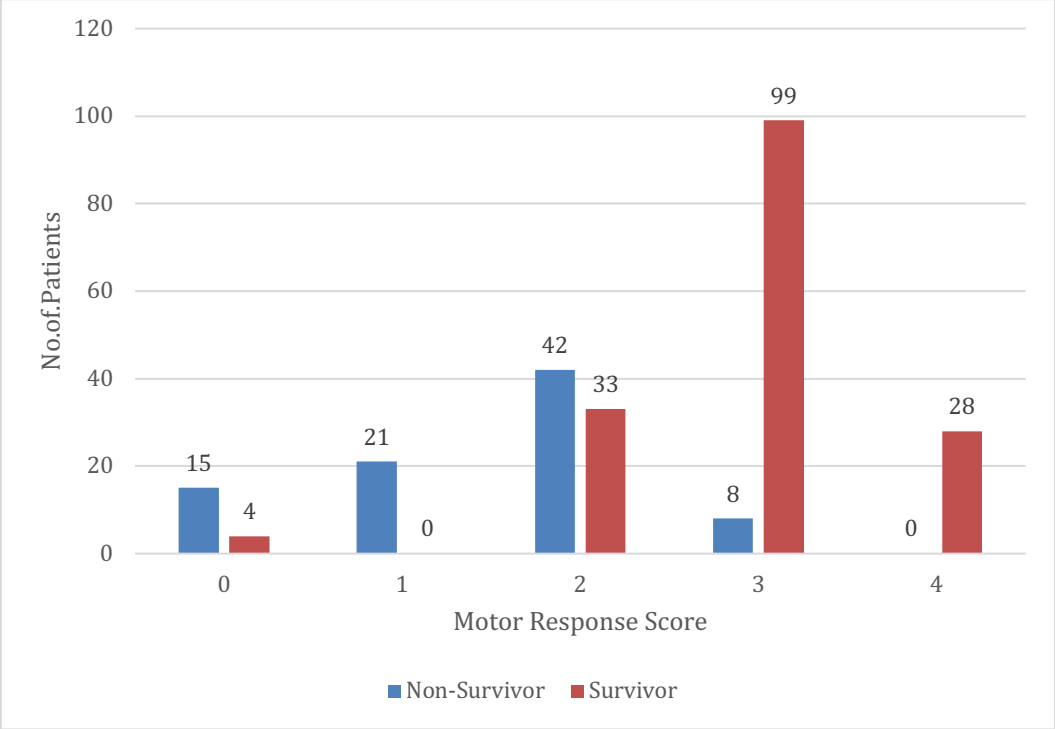


Table 15: Comparative analysis of the Brainstem Reflexes Response of the FOUR score at 24 hours after admission vs Outcome in study population (N=250)

Brainstem Reflexes	Outcome		Total	Modified Rankin scale for Survivors At 3 months post Discharge	P-value
	Non-Survivor	Survivor			
0	1(1.16%)	0(0%)	1(0.40%)	NIL	**<0.001 ***
1	13(15.12%)	0(0%)	13(5.20%)	NIL	
2	32(37.21%)	1(0.61%)	33(13.20%)	5±0	
3	24(27.91%)	0(0%)	24(9.60%)	NIL	
4	16(18.60%)	163(99.39%)	179(71.60%)	1.70±1.58	
Total	86(100%)	164(100%)	250(100%)		

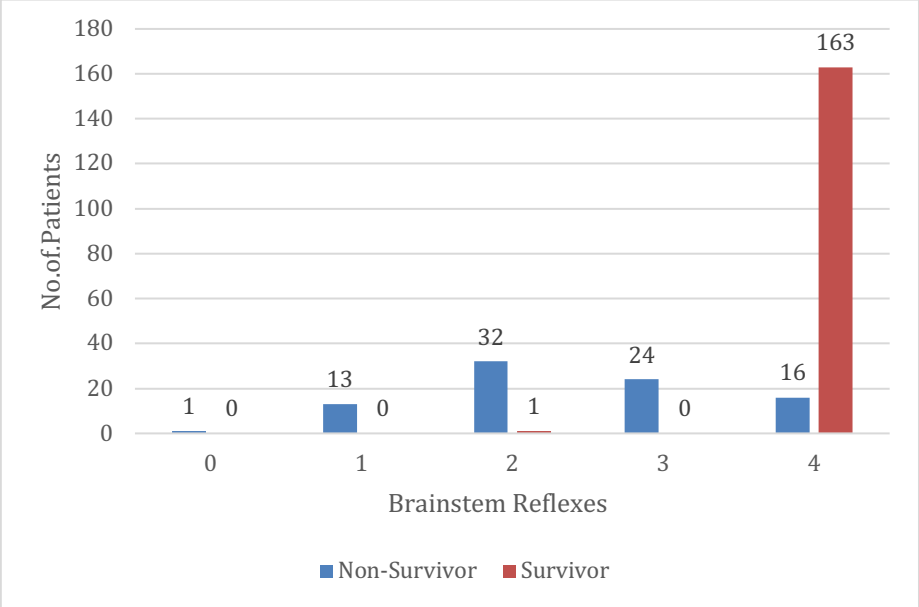


Table 16: Comparative analysis of Respiratory pattern of the FOUR score at 24hrs after admission and Outcome in study population (N=250)

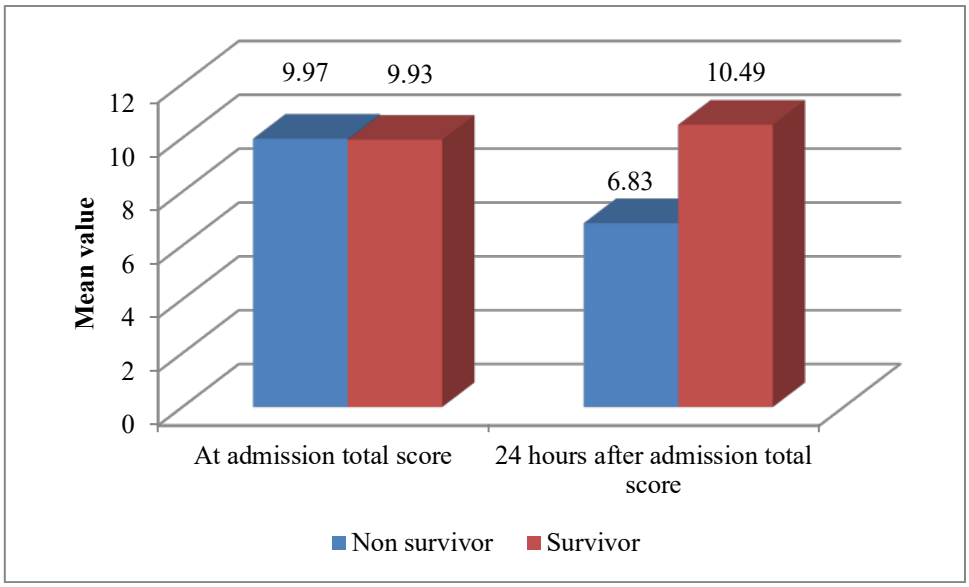
Respiration	Outcome		Total	Modified Rankin score for Survivors At 3 months post Discharge	P-value
	Non-Survivor	Survivor			
0	14(16.28%)	0(0%)	14(5.60%)	NIL	**<0.001 ***
1	14(16.28%)	13(7.93%)	27(10.80%)	1.46±1.39	
2	36(41.86%)	0(0%)	36(14.40%)	NIL	
3	14(16.28%)	0(0%)	14(5.60%)	NIL	
4	8(9.30%)	151(92.07%)	159(63.60%)	1.73±1.59	
Total	86(100%)	164(100%)	250(100%)		

From the table and Figures above, we can conclude that there is a statistically significant relationship between the various individual components of the FOUR score in predicting the outcome (lower scores are associated with increased mortality in each individual score), as given by the p value of <0.001 for each of the individual components. For predicting the neurological outcome of the survivors, we can see that the Motor component of the FOUR score and Brainstem reflexes response component of the FOUR score are better compared to the other two components. Lower scores in the motor component of the FOUR score and Brainstem reflexes responses component of the FOUR score are clearly associated with poor neurological outcome (Modified Rankin scale ≥4). Neurological outcome could not be predicted from the Eye and Respiratory components.

Table 17: Comparison of mean GLASGOW COMA SCALE vs OUTCOME

GLASGOW COMA SCALE	Outcome		Unpaired t test P value
	Non survivors (N=86)	Survivors (N=164)	
At admission total score	9.97 ± 3.08	9.93 ± 1.88	<0.001
24 hours after admission total score	6.83 ± 2.33	10.49 ± 1.91	<0.001

Figure 17: Mean GLASGOW COMA SCALE vs OUTCOME

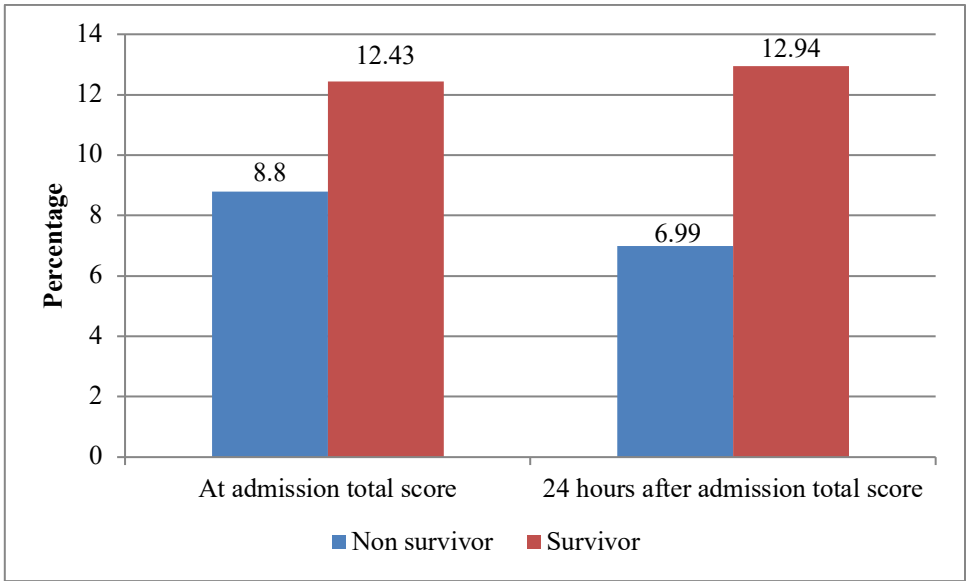


From the above table and figure, we can see that while it is still significant, as given by the p value of <0.01, the GCS done at the time of admission is not able to predict mortality accurately, while the GCS done after 24 hours is able to predict mortality better, with lower total scores associated with increased mortality compared to higher total scores. There is a statistically significant relationship between GCS and prediction of mortality, with lower total scores associated with increased mortality.

Table 18: Comparison of mean FOUR SCORE vs OUTCOME

FOUR score	Outcome		Unpaired t test P value
	Non survivor (N=86)	Survivors (N=164)	
At admission total score	8.80 ± 3.70	12.43 ± 1.69	<0.001
24 hours after admission total score	6.99 ± 3.08	12.94 ± 1.85	<0.001

Figure 18: Mean FOUR score vs OUTCOME



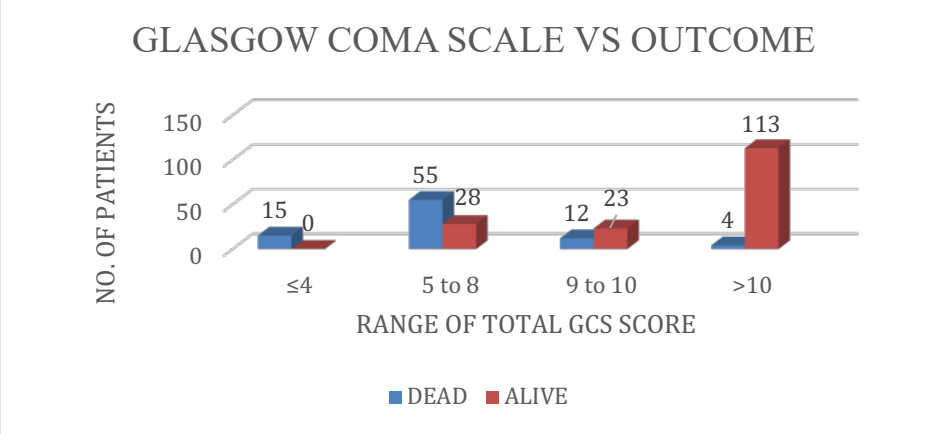
From the above table and figure, we can see that there is a statistically significant relationship between the FOUR score (done at admission as well as at 24 hours after admission) and prediction of mortality, with lower scores associated with increased mortality. FOUR score done at 24 hours after admission was able to predict mortality better compared to the one performed at the time of admission.

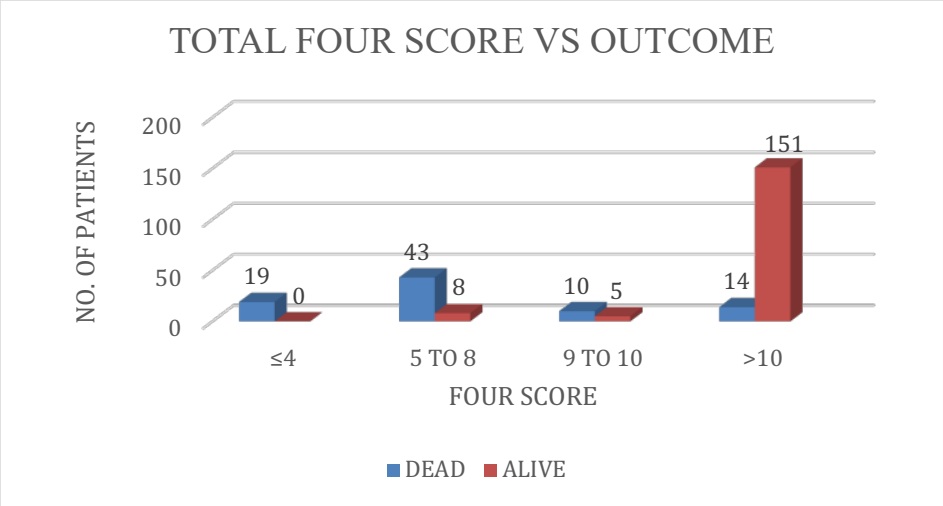
Table 19: Comparison of GCS and FOUR scores vs OUTCOME

GCS Score	Total Cases	Mortality	Mortality%	Discharged	Discharged%	Modified Rankin Scale At 3 Months for Survivors
4 or less	15	15	100%	0	0	0
5 to 8	83	55	66.27%	28	33.73%	1.92 ±1.35
9 to 10	35	12	34.29%	23	65.71%	1.78 ±2.04
>10	117	4	3.42%	113	96.58%	1.64 ±1.53

FOUR Score	Total Cases	Mortality	Mortality%	Discharged	Discharged %	Modified Rankin Scale At 3 Months for Survivors
4 or less	19	19	100%	0	0	0
5 to 8	51	43	84.31%	8	15.69	2 ±1.41
9 to 10	15	10	66.67%	5	33.33%	0.8 ±1.30
>10	165	14	8.48%	151	91.52%	1.72 ±1.59

Figure 19: Comparison of GCS and FOUR scores vs OUTCOME

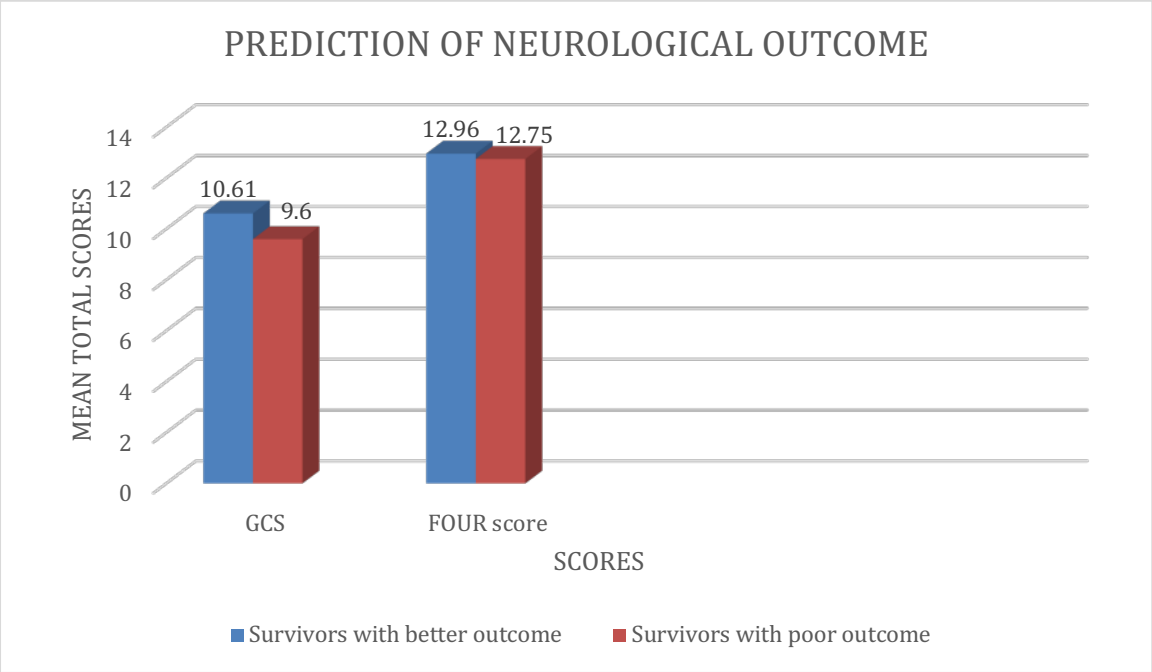




From the above table and figure, we can see that though both the GCS and FOUR score have a statistically significant relationship in predicting mortality (lower total scores are associated with higher mortality), the mortality was higher with the lowest total FOUR score compared to the lowest total GCS (FOUR score ≤4, 19 non survivors. GCS ≤4, 15 non survivors). This indicates that the probability of detecting the mortality was better with the lowest total FOUR scores than the GCS.

Figure 20: Comparison of GCS and FOUR scores in Predicting Neurological Outcome

Score	Survivors	Non-Survivors	Mean score of Survivors with better outcome (those with modified Rankin scale 0 to 3)	Mean value of Survivors with Poor outcome (those with modified Rankin scale 4 to 6)
GCS Score	10.49 ±1.91	6.82 ±2.32	10.61±1.84	9.6±2.18
Four Score	12.93 ±1.84	6.98 ±3.07	12.96±1.90	12.75±1.40



From the above figure, we can see that the mean value of the total score of GCS was better able to distinguish amongst survivors, those with better neurological outcome from those with poor neurological outcome (Better outcome – 10.61±1.84, Poor outcome- 9.6±2.18) than the mean value of the total score of the FOUR score (Better outcome - 12.96±1.90, Poor outcome - 12.75±1.40). The outcomes were determined based on the modified Rankin scale (Better outcome – score 0 to 3, Poor outcome – score 4 to 6).

Table 21: Comparative analysis of Diagnosis in the study population vs Outcome (N=250)

Diagnosis	Outcome		Intubated	Survivors after Intubation
	Non-Survivors	Survivors		
Acute Meningoencephalitis	2	10	0	0
COVID Pneumonia	10	16	2	2
Cerebral Venous Thrombosis	0	5	2	2
Hemorrhagic Stroke	23	21	11	1
Ischemic Stroke	10	49	5	4
Locked in Syndrome	0	4	4	4
Metabolic Encephalopathy	22	45	7	2
Poisoning	13	7	5	2
Seizures	0	4	0	0
Shock	3	3	4	1
Total	86	164	40	18

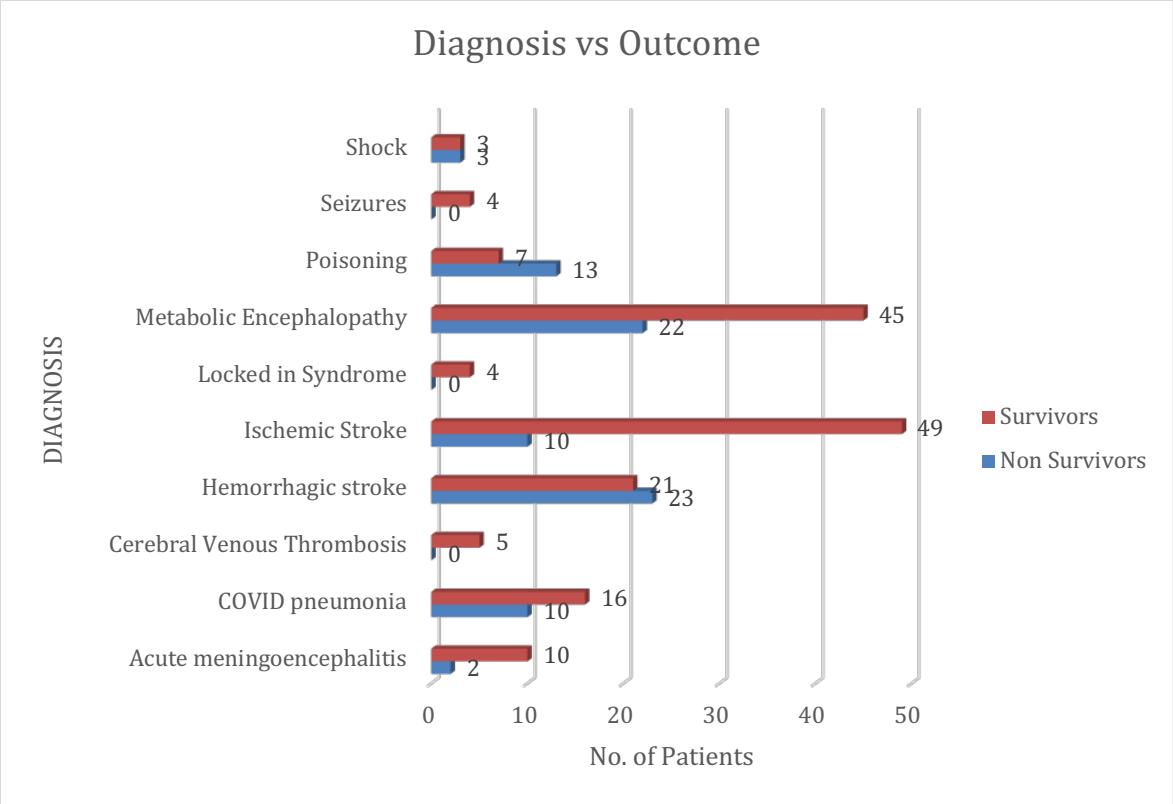


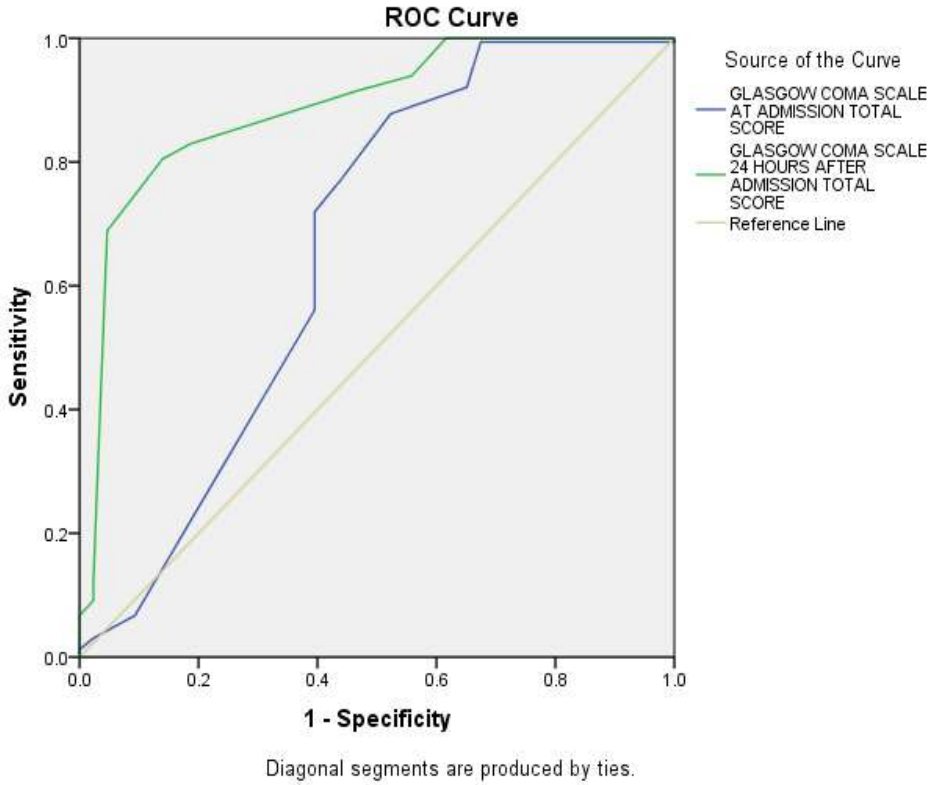
Table 22: Comparative Analysis of Diagnosis vs Mean FOUR score and GCS and Modified Rankin scale.

Diagnosis	Mean FOUR score of Survivors	Mean FOUR score of non-Survivors	Mean GCS of Survivors	Mean GCS score of non-Survivor-s	Modified Rankin Scale At 1 Month For Survivors	Modified Rankin Scale At 3 Months for Survivors
Acute Meningoencephalitiis	13.1	11	10.7	10	1.4	0
COVID Pneumonia	12.87	8.3	10.5	7.7	2.68	1.12
Cerebral Venous Thrombosis	11	0	9.2	0	2.2	0.8
Hemorrhagic Stroke	13.28	5.34	10.76	5.39	4	3.14
Ischemic Stroke	13.24	6.8	10.69	6.3	3.77	2.71
Locked in syndrome	9	0	6	0	4.5	4
Metabolic Encephalopathy	12.75	7.45	10.57	7.27	1.31	0.64
Poisoning	11.85	8.92	10.14	8.61	2.28	0.85
Seizures	14.5	0	12	0	1	0.25
Shock	13	1.33	10.33	3	1.67	2.67

From Tables 21 and 22, we can see that Metabolic encephalopathy (includes a wide range of causes like Hypoglycemia, Hyponatremia, Hepatic encephalopathy and Uremic encephalopathy) was the most common cause of altered sensorium, accounting for 26.8% of the study population. This was followed by Ischemic stroke (23.6%) and Hemorrhagic stroke (17.6%). The study was also carried out in 26 patients admitted with COVID pneumonia with altered sensorium and 20 patients with

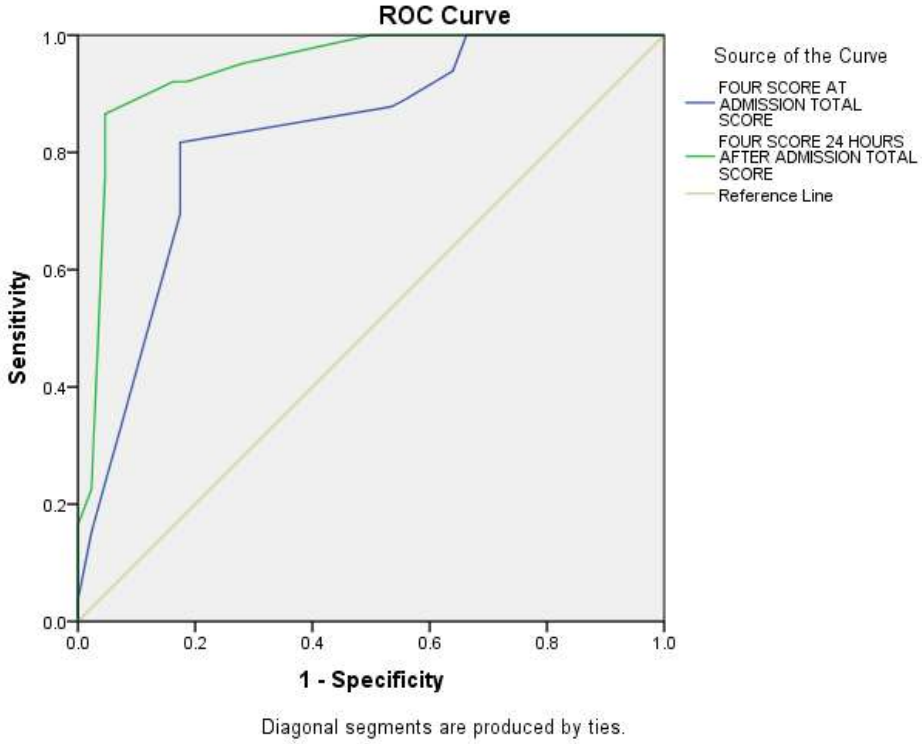
poisoning. An interesting point to note in table 22 is, the mean GCS of patients admitted with Locked in syndrome was 6, while the FOUR score was 9. All 4 patients survived. The lower mean GCS scores are because of the weightage given to the verbal component in GCS (patients with locked in syndrome cannot vocalise or move). Thus, GCS was abnormally low in these patients and was not able to predict the mortality/neurological outcome in these patients while the FOUR score was higher and thus predicted better.

Figure 23: Predictive validity of GLASGOW Coma Scale done at admission and 24 hours after admission in predicting mortality:



Test Result Variable(s)	Area Under the Curve	Std. Error	P value	95% CI	
				Lower	Upper
GLASGOW COMA scale at admission total score	0.662	0.041	<0.001	0.582	0.742
GLASGOW COMA SCALE 24 hours after admission total score	0.888	0.022	<0.001	0.844	0.932

Figure 24: Predictive validity of FOUR score done at admission and 24 hours after admission in predicting mortality:



Test Result Variable(s)	Area Under the Curve	Std. Error	P value	95% CI	
				Lower	Upper
FOUR score at admission total score	0.827	0.029	<0.001	0.770	0.883
FOUR score 24 hours after admission total score	0.944	0.017	<0.001	0.911	0.977

From figures 23 and 24, it can be inferred that both GCS and FOUR score have a statistically significant relationship in predicting mortality (lower scores are associated with higher mortality). However, the Area under the curve is significantly higher with the FOUR score (done at admission as well as 24 hours after admission) than the

GCS. Hence, we can conclude that the overall predictive accuracy of the FOUR score was better than that of GCS.

Table 24: Comparison of the sensitivity, specificity, Positive and Negative Predictive Value, Accuracy and the Area under the ROC curve of GCS and FOUR score in predicting mortality:

Variable	Cut off Score	Sensitivity %	Specificity %	PPV %	NPV %	Accuracy %	Area under ROC Curve
FOUR Score	6.5	77.36	93%	94%	53.19	80.80	0.725
GCS	7.5	78.95	76.67	91.46	44.49	74.40	0.701

From the above table, it is clear that the FOUR score, at a cut off of 6.5, has a sensitivity of 77%, specificity of 93% (significantly higher than that of GCS) and an accuracy of 80% (significantly higher than that of GCS). GCS has a marginally better sensitivity than the FOUR score in predicting mortality but falls behind the FOUR score in other parameters. Thus, we can conclude that the FOUR score has a better accuracy than the GCS in predicting mortality. With regards to the Neurological outcome amongst survivors, as has been shown in figure 20, the GCS predicts and classifies neurological outcome better than the FOUR score.

DISCUSSION

In this study, we included a sample size of 250 patients. This was much higher than the main study done by Wijdicks et al in 2005, which was done with a sample size of 120 patients (it was Wijdicks et al who introduced the FOUR score to the wider medical community in 2005). Ramazani et al in 2019 had included a sample size of 300 patients.

In our study, we found that, there was no statistical significance between the gender of the study population and the outcome, similar to other studies.

We found that there was no statistical significance between the age group of the study population and the outcome, in our study, similar to other studies.

In our study, there was no statistical significance between intubation and outcome (p value = 0.121)

In our study, we found that there is a statistically significant relationship between the various individual components of the GCS in predicting the outcome in agreement with similar studies.

In our study, we found that for predicting the neurological outcome of the survivors, the Motor component of the GCS was superior compared to the other two components, in agreement with other studies. We concluded that there is a statistically significant relationship between the various individual components of the FOUR score in predicting the outcome. For predicting the neurological outcome of the survivors, we found that the Motor component of the FOUR score and Brainstem reflexes

response component of the FOUR score are better compared to the other two components.

In our study, we found that the GCS done at the time of admission was not able to predict mortality accurately, while the GCS done after 24 hours was able to predict mortality better. This has not been assessed in previous published studies.

We found that the FOUR score done at 24 hours after admission was able to predict mortality better compared to the one performed at the time of admission. Overall, FOUR score was better in predicting mortality compared to the GCS.

In agreement with the study done by Wijdicks et al, the mortality was higher with the lowest total FOUR score compared to the lowest total GCS (FOUR score ≤4, 19 non survivors. GCS ≤4, 15 non survivors). This indicates that the probability of detecting the mortality was better with the lowest total FOUR scores than the GCS.

In our study, we found that the mean value of the total score of GCS was better able to distinguish amongst survivors, those with better neurological outcome from this with poor neurological outcome (Better outcome – 10.61±1.84, Poor outcome- 9.6±2.18) than the mean value of the total score of the FOUR score (Better outcome - 12.96±1.90, Poor outcome - 12.75±1.40). This was in contrast to the results obtained by Wijdicks et al who had concluded that the FOUR score was better in predicting neurological outcome.

In our study, we found that the GCS was low in patients with Locked in syndrome and was not able to predict the mortality/neurological outcome in these patients while the

FOUR score was higher and thus predicted better. This is in accordance with older studies.

In our study, we found that the Area under the curve was significantly higher with the FOUR score (done at admission as well as 24 hours after admission) than the GCS. Hence, we can conclude that the overall predictive accuracy of the FOUR score was better than that of GCS, similar to the results obtained by Ramazani et al.

In our study, we found that the FOUR score, had a better specificity, positive and negative predictive value and accuracy, compared to that of GCS, similar to the one obtained by Ramazani et al. GCS has a marginally better sensitivity than the FOUR score in predicting mortality but falls behind the FOUR score in other parameters. This was in agreement to the results obtained by similar studies.

CONCLUSION

- The FOUR score is better than the GCS in predicting mortality, in view of its superior specificity, positive predictive value and negative predictive value compared to the GCS.
- The neurological outcome among the survivors was better predicted by the GCS than the FOUR score as lower scores on the GCS were associated with poor neurological outcome, while higher scores were associated with a better neurological outcome.

LIMITATIONS OF THE STUDY

- The study, even though done over a sample size of 250, is relatively smaller. More number of patients have to be recruited for better results.
- The patients included in the study included a disproportionate number of patients with Metabolic encephalopathy and Ischemic stroke, compared to other diagnoses; hence the results are also likely to be skewed in their favor.
- The outcome of the patient is not dependent upon Neurological status alone; The presence of co morbidities, initiation of treatment at the right time, initiating disability limitation and rehabilitation measures like Physiotherapy also play an important role.
- The possibility of variations in inter rater reliability was not examined in this study.
- The patients were not followed up beyond 3 months post discharge.

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ANNEXURES

PROFORMA:

COMPARISON OF GLASGOW COMA SCALE AND THE FULL OUTLINE OF UNRESPONSIVENESS SCORE IN PREDICTING MORTALITY AND NEUROLOGICAL OUTCOME IN PATIENTS WITH ALTERED SENSORIUM ADMITTED IN A MEDICAL INTENSIVE CARE UNIT.

NAME:

AGE/ SEX:

IP NO:

OCCUPATION:

ADDRESS:

CONTACT NUMBER:

SYMPTOMS:

Altered sensorium/ Loss of consciousness for _____ days/hours

History of trauma present or absent:

History of difficulty in using arms and legs:

History of seizures:

History of consumption of drugs/toxins:

PAST HISTORY:

History of any co morbid illness:

PERSONAL HISTORY:

History of alcohol intake - type of alcohol, quantity and duration, history of last consumption of alcohol

GENERAL EXAMINATION:

Consciousness, orientation to time, place and person
Pallor/ Icterus / Cyanosis / Clubbing / Pedal edema / lymphadenopathy

VITALS: Blood pressure / Pulse rate / respiratory rate / Temperature

SYSTEMIC EXAMINATION:

CVS (Cardiovascular system):

RS (Respiratory system)

Abdomen:

CNS (Central Nervous system):

GCS (Glasgow Coma Scale):

FOUR score (Full Outline of Un Responsiveness score):

Modified Rankin scale (for survivors):

GLASGOW COMA SCALE

EYE OPENING:

Spontaneous – 4

To speech – 3

To pain – 2

No response – 1.

VERBAL RESPONSE:

Oriented to time, place and person – 5

Confused – 4

Words -3

Sounds – 2

No response – 1

MOTOR RESPONSE:

Obeys commands – 6

Localises pain – 5

Flexion response to pain – 4

Abnormal flexion to pain – 3

Extension response to pain – 2

No response – 1

Total – 15.

FOUR SCORE (Full Outline of Un Responsiveness Score)

EYE RESPONSE:

Opens eyes spontaneously, tracks, blinks to command - 4

Opens eyes, does not track or blink to command - 3

Eyes closed, open to loud voice - 2

Eyes closed, open to painful stimulation - 1

Eyes remain closed after painful stimulation - 0

MOTOR RESPONSE:

Obeys commands, makes sign (e.g., "Thumbs up" - 4

Localises Painful stimulus -3

Flexion response to Pain - 2

Extension response to Pain - 1

No response - 0

Status epilepticus - 0

BRAINSTEM REFLEXES:

Pupil and corneal reflexes present - 4

One pupil wide and fixed -3

Pupil or corneal reflexes absent - 2

Pupil and corneal reflexes absent - 1

Absent pupil, corneal and cough reflex - 0

RESPIRATION:

Not intubated, regular breathing pattern - 4

Not intubated, Cheyne Stokes pattern - 3

Not intubated, irregular breathing pattern - 2

Intubated, breaths above ventilator rate - 1

Breathes at ventilator rate or apnea - 0.

TOTAL – 16.

MODIFIED RANKIN SCALE FOR NEUROLOGIC DISABILITY

0 – No symptoms

1 – No significant disability, despite symptoms; able to perform all usual duties and activities.

2 – Slight disability, unable to perform all previous activities but able to look after own affairs without assistance.

3 – Moderate disability; requires some help, but able to walk without assistance.

4 – Moderately severe disability, unable to walk without assistance and unable to attend to own bodily needs without assistance.

5 – Severe disability, bedridden; incontinent and requires constant nursing care and attention.

6- Death.

Standardized Interview for the Modified Rankin Scale:
Ask these Yes/No Questions:

- Do you have any symptoms that are bothering you?
- Are you able to do the same work as before?
- Are you able to keep up with your hobbies?
- Have you maintained your ties to friends and family?
- Do you need help making a simple meal, doing household chores, or balancing a check book?
- Do you need help with shopping or traveling close to home?
- Do you need another person to help you walk?
- Do you need help with eating, going to the toilet, or bathing?
- Do you stay in bed most of the day and need constant nursing care?

INFORMATION TO PARTICIPANTS

INVESTIGATORS: Dr. M. SATHISH KUMAR

Dr. NALINI KUMARAVELU M.D.

NAME OF THE PARTICIPANT:

You are invited to take part in this study. The information in this document is meant to help you decide whether or not to take part. Please free to ask if you have any queries or concerns.

We are conducting a study titled "**COMPARISON OF GLASGOW COMA SCALE AND THE FULL OUTLINE OF UNRESPONSIVENESS SCORE IN PREDICTING MORTALITY AND NEUROLOGICAL OUTCOME IN PATIENTS WITH ALTERED SENSORIUM ADMITTED IN A MEDICAL INTENSIVE CARE UNIT**" among patients admitted in Rajiv Gandhi Government General Hospital, Chennai. Your co-operation to undergo examination is valuable to us. The purpose of this study is to examine patients admitted with altered sensorium and determine the GCS and FOUR score and calculate the accuracy of the scores in predicting mortality and neurological outcome.

We are selecting certain cases and if you are found eligible, you will be subjected to examination by the principal investigator.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

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/left thumb impression of Participant

Date:

Place:

PATIENT CONSENT FORM

Study Detail	:	**"COMPARISON OF GLASGOW COMA SCALE AND THE FULL OUTLINE OF UNRESPONSIVENESS SCORE IN PREDICTING MORTALITY AND NEUROLOGICAL OUTCOME IN PATIENTS WITH ALTERED SENSORIUM ADMITTED IN A MEDICAL INTENSIVE CARE UNIT"**
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Documentation of the informed consent

1. I _____ have read the information in this form (or it has been read for me). I was free to ask any questions and they have been answered. I am over 18 years of age and exercising my free power of choice, hereby give my consent to be included as a participant in the study.
2. I have read and understood this consent form and the information provided to me.
3. I have had the consent document explained to me.
4. I have been explained about the nature of the study.
5. I have been explained about my rights and responsibilities by the Investigator.
6. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.
7. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, govt. agencies and IEC.I understand that they are publicly published
8. I have understood that my identity will be kept confidential if my data are publicly presented.
9. I have had my questions answered to my satisfaction.
10. I have decided to be in the research study.

11. I am aware that if I have any question during this study, I should contact at one of the addresses listed above. By signing this consent form, I attest that the information given in this document has been clearly explained to me and apparently understood by me. I will be given a copy of this consent document.

Name and signature/thumb impression of the participant (or legal representative if participant incompetent):

_____ _____ _____

Name Signature Date

Name and signature of impartial witness (required for illiterate patients):

_____ _____ _____

Name Signature Date

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

_____ _____ _____

Name Signature Date

ஆய்வு தகவல் தாள்

ஆய்வு தலைப்பு :

தீவிர சிகிச்சை பிரிவில் அனுதிக்கப்பட்ட மயக்க நிலை நோயாளிகளின் கிளாஸ்கோ கோமா ஸ்கேல் மற்றும் ஃபோர் ஸ்கோர் கணித்து அதன் மூலம் இறப்பு மற்றும் பிழைப்போரின் நரம்பியல் நிலை கணிப்பு பற்றிய ஒரு ஒப்பீட்டு ஆய்வு.

ஆய்வாளர் பெயர் : மருத்துவர் ம. சதீஷ் குமார்

ஆய்வு நிலையம் : பொது மருத்துவப் பிரிவு,
சென்னை மருத்துவக் கல்லூரி, சென்னை-3.

இந்த ஆய்வில் தங்களை பங்கேற்க அழைக்கிறோம். இந்த தகவல் அறிக்கையில் கூறப்பட்டிருக்கும் தகவல்கள் தாங்கள் இந்த ஆராய்ச்சியில் பங்கேற்கலாமா வேண்டாமா என்பதை முடிவு செய்ய உதவியாக இருக்கும். இந்த படிவத்தில் உள்ள தகவல்கள் பற்றி உள்ள சந்தேகங்களை நீங்கள் தயங்காமல் கேட்கலாம்.

இந்த ஆய்வில் தீவிர சிகிச்சை பிரிவில் அனுதிக்கப்பட்ட மயக்க நிலை நோயாளிகளின் கிளாஸ்கோ கோமா ஸ்கேல் மற்றும் ஃபோர் ஸ்கோர் கணித்து அதன் மூலம் இறப்பு மற்றும் பிழைப்போரின் நரம்பியல் நிலை கணிப்பு பற்றிய ஒரு ஒப்பீட்டு ஆய்வு இங்கு நடைபெறுகிறது. அதற்குத் தங்கள் ஒத்துழைப்புத் தேவை.

நீங்கள் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியில் இருந்து பின் வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனையின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவில் தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்

தேதி :

பங்கேற்பாளர் கையொப்பம் /
இடது கட்டைவிரல் ரேகை

தேதி :

ஆய்வு ஒப்புதல் படிவம்

ஆய்வு தலைப்பு :
தீவிர சிகிச்சை பிரிவில் அனுதிக்கப்பட்ட மயக்க நிலை நோயாளிகளின் கிளாஸ்கோ கோமா ஸ்கேல் மற்றும் ஃபோர் ஸ்கோர் கணித்து அதன மூலம் இறப்பு மற்றும் பிழைப்போரின் நரம்பியல் நிலை கணிப்பு பற்றிய ஒரு ஒப்பீட்டு ஆய்வு.

பெயர் :
வயது :
பால் :

தேதி :
உள்நோயாளி எண் :
ஆராய்ச்சி சேர்க்கை எண் :

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

நான் ஆராய்ச்சியாளருடன் ஒத்துழைப்பேன் என்றும், எனக்கு ஏற்படக்கூடிய ஆசாதாரண நிகழ்வுகள் பற்றியும் உடனடியாக ஆராய்ச்சியாளரிடம் தெரிவிப்பேன் என்று உறுதி கூறுகிறேன். இந்த ஆய்விலிருந்து எப்போது வேண்டுமானாலும் எக்காரணமும் கூறாமல் என்னை விடுவித்துக்கொள்ளலாம் என்பதை அறிவேன்.

என்னிடம் இருந்து பெறப்படும் தகவல்களை அரசு, வரைமுறை அதிகாரிகள் ஆகியோர்களுடன் பகிர்ந்துகொள்ள ஆராய்ச்சியாளருக்கு அனுமதி அளிக்கிறேன். என்னுடைய சிகிச்சைக்கட்டுகளை பார்வையிட உரிமை உண்டு. என்னுடைய தகவல்களின் அடையாளம் இரகசியமாக வைக்கப்படும் என்பதை அறிவேன்.

இந்த ஆராய்ச்சியில் பங்கேற்க தன்னிச்சையாக முழு மனதுடன் சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் / ரேகை

பங்கேற்பவர் பெயர்

இடம் :

தேதி :

ஆய்வாளர் கையொப்பம்

ஆய்வாளர் பெயர்

இடம் :

தேதி :

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013/RR-16
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
DR.M.SATHISH KUMAR,
IInd Year Post Graduate, MD (General Medicine),
Institute of Internal Medicine,
Madras Medical College,
Chennai – 600003.

Dear DR. M.SATHISH KUMAR,

The Institutional Ethics Committee has considered your request and approved your study titled **"COMPARISON OF GLASGOW COMA SCALE AND THE FULL OUTLINE OF UNRESPONSIVENESS SCORE IN PREDICTING MORTALITY AND NEUROLOGICAL OUTCOME IN PATIENTS WITH ALTERED SENSORIUM ADMITTED IN A MEDICAL INTENSIVE CARE UNIT"- NO.12042021.** The following members of Ethics Committee were present in the meeting held on **07.04.2021** conducted at Madras Medical College, Chennai 3.

1. Prof.P.V.Jayashankar :Chairperson
2. Prof.N.Gopalakrishnan,MD.,DM., FRCP, Director, Inst.of Nephrology,MMC,Ch. : Member Secretary
3. Prof. K.M.Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
4. Prof. Alagarsamy Jamila ,MD, Vice Principal, Stanley Medical College, Chennai : Member
5. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
6. Prof.S.Lakshmi, Prof. of Paediatrics ICH Chennai :Member
7. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
8. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
9. Thiru K.Ranjith, Ch- 91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003.

Curiginal

Document Information

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Submitter email	sathishkumar5910@gmail.com
Similarity	1%
Analysis address	sathishkumar5910.mgrmu@analysis.urkund.com

ANTI PLAGIARISM CERTIFICATE

This is to certify that the dissertation work titled "**COMPARISON OF GLASGOW COMA SCALE AND THE FULL OUTLINE OF UNRESPONSIVENESS SCORE IN PREDICTING MORTALITY AND NEUROLOGICAL OUTCOME IN PATIENTS WITH ALTERED SENSORIUM ADMITTED IN A MEDICAL INTENSIVE CARE UNIT"** of the candidate **Dr**. **M. SATHISH KUMAR** with Registration Number **201911018** for the award of **M.D. degree** in the branch of **GENERAL MEDICINE** is original. I personally verified the urkund.com website for plagiarism Check. I found that the uploaded thesis file contained Introduction to Conclusion pages and result showed **1 percentage** of plagiarism.

Guide & Supervisor sign with Seal.

MASTER CHART OF THE STUDY

MASTER CHART - MD THESIS

Serial NO	NAME	SEX	AGE	GLASGOV COMA SCALE							
				AT ADMISSION				24 HOURS AFTER ADMISSION			
				EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE	EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE
1	PATIENT 1	MALE	46	2	NOT TESTABLE	1	4	2	NOT TESTABLE	1	4
2	PATIENT 2	MALE	35	1	NOT TESTABLE	1	3	1	NOT TESTABLE	1	3
3	PATIENT 3	FEMALI	36	4	3	6	13	4	4	6	14
4	PATIENT 4	FEMALI	38	1	NOT TESTABLE	1	3	1	NOT TESTABLE	1	3
5	PATIENT 5	MALE	63	3	NOT TESTABLE	6	10	4	NOT TESTABLE	6	11
6	PATIENT 6	MALE	40	4	3	6	13	4	3	6	13
7	PATIENT 7	MALE	55	1	NOT TESTABLE	1	3	1	NOT TESTABLE	1	3
8	PATIENT 8	MALE	75	2	NOT TESTABLE	3	6	2	NOT TESTABLE	3	6
9	PATIENT 9	MALE	67	4	1	5	10	4	1	5	10
10	PATIENT 1(	MALE	67	4	3	5	12	4	3	5	12
11	PATIENT 11	MALE	60	4	2	5	11	4	2	5	11
12	PATIENT 12	MALE	40	1	2	4	7	2	2	4	8
13	PATIENT 1(	FEMALI	67	3	2	4	9	3	2	4	9
14	PATIENT 14	MALE	55	2	2	4	8	3	3	5	11
15	PATIENT 1(	MALE	70	3	4	5	12	3	4	5	12
16	PATIENT 16	FEMALI	70	4	2	5	11	4	2	5	11
17	PATIENT 17	FEMALI	50	2	2	3	7	2	2	3	7
18	PATIENT 18	MALE	70	1	1	3	5	1	1	3	5
19	PATIENT 19	MALE	32	2	2	5	9	3	3	5	11
20	PATIENT 2(	FEMALI	55	3	3	5	11	3	3	5	11
21	PATIENT 2	FEMALI	64	3	2	5	10	3	2	5	10
22	PATIENT 22	MALE	84	3	3	4	10	3	3	4	10
23	PATIENT 23	FEMALI	34	2	2	4	8	3	3	5	11
24	PATIENT 24	FEMALI	70	2	NOT TESTABLE	2	5	2	NOT TESTABLE	2	5
25	PATIENT 25	MALE	64	2	NOT TESTABLE	4	7	2	NOT TESTABLE	4	7
26	PATIENT 26	FEMALI	38	2	NOT TESTABLE	3	6	2	NOT TESTABLE	3	6
27	PATIENT 27	FEMALI	64	2	2	4	8	2	2	4	8
28	PATIENT 28	MALE	61	3	2	3	8	3	2	3	8
29	PATIENT 29	FEMALI	65	2	2	2	6	2	2	2	6
30	PATIENT 3(	FEMALI	71	2	2	4	8	2	2	4	8
31	PATIENT 31	MALE	74	1	NOT TESTABLE	1	3	1	NOT TESTABLE	1	3
32	PATIENT 32	MALE	38	2	NOT TESTABLE	3	6	2	NOT TESTABLE	3	6
33	PATIENT 33	MALE	55	2	2	2	6	2	2	4	8

FOUR SCORE										CT BRAIN
AT ADMISSION					24 HOURS AFTER ADMISSION					
EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEXES	RESPIRATIOI	TOTAL SCOF	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEX	RESPIRATIOI	TOTAL SCORE	
1	0	0	1	2	1	0	0	1	2	NORMAL
0	0	1	0	1	0	0	1	0	1	NORMAL
4	4	4	4	16	4	4	4	4	16	NORMAL
0	0	1	1	2	0	0	1	1	2	NORMAL
4	4	4	1	13	4	4	4	1	13	NORMAL
4	4	4	4	16	4	4	4	4	16	NORMAL
0	0	1	1	2	0	0	1	0	1	INTRACRANIAL HEMORRHAGE
1	2	4	1	8	1	2	4	1	8	RIGHT GANGLIOCAPSULAR INFARCT
4	3	4	4	15	4	3	4	4	15	LEFT FRONTAL LOBE INFARCT
3	3	4	4	14	4	3	4	4	15	LEFT THALAMIC HEMORRHAGE
3	3	4	4	14	3	3	4	4	14	NORMAL
0	2	2	4	8	1	2	4	4	11	NORMAL
2	2	4	3	11	2	2	4	3	11	NORMAL
1	2	4	3	10	2	3	4	4	13	NORMAL
2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT
3	3	4	4	14	3	3	4	4	14	NORMAL
1	2	4	4	11	1	2	2	4	9	INTRACRANIAL HEMORRHAGE
0	2	2	4	8	0	2	2	4	8	RIGHT GANGLIOCAPSULAR INFARCT
1	3	4	4	12	2	3	4	4	13	SUPERIOR SAGITTAL SINUS THROME
2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
2	3	4	4	13	2	3	4	4	13	NORMAL
2	2	4	4	12	2	2	4	4	12	NORMAL
1	2	4	4	11	2	3	4	4	13	NORMAL
1	1	2	1	5	1	1	2	1	5	INTRACRANIAL HEMORRHAGE
1	2	4	1	8	1	2	4	1	8	NORMAL
1	2	4	1	8	1	2	4	1	8	TRANSVERSE-SIGMOID SINUS THRO
1	2	4	4	11	1	2	4	4	11	NORMAL
2	2	4	4	12	2	2	4	4	12	RIGHT GANGLIOCAPSULAR INFARCT
1	1	2	2	6	1	1	2	2	6	RIGHT GANGLIOCAPSULAR INFARCT
1	2	4	4	11	1	2	4	4	11	NORMAL
0	0	1	0	1	0	0	1	0	1	NORMAL
1	2	4	1	8	1	2	4	1	8	RIGHT GANGLIOCAPSULAR INFARCT
1	1	4	3	9	1	2	4	4	11	NORMAL

PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
				AT DISCHARGE	THREE MONTHS POST DISCHARGE
NO	YES	MULTIPLE TABLET **POISONING** (ANTI SEIZURE DRUGS)	NON SURVIVOR	NIL	NIL
NO	YES	ORGANOPHOSPHATE **POISONING**	NON SURVIVOR	NIL	NIL
NO	NO	ACUTE **MENINGOENCEPHALITIS**	SURVIVOR	1	0
NO	YES	**SEPTIC ENCEPHALOPATHY/POST CARDIAC ARREST**	NON SURVIVOR	NIL	NIL
NO	YES	**CARDIOGENIC SHOCK**/PULMONARY EDEMA	SURVIVOR	2	2
NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA**	SURVIVOR	2	1
NO	YES	**HEMORRHAGIC STROKE**/HYPERTENSION	NON SURVIVOR	NIL	NIL
NO	YES	**ISCHEMIC STROKE**/DIABETES MELLITUS	SURVIVOR	4	3
NO	NO	**ISCHEMIC STROKE**/DIABETES MELLITUS	SURVIVOR	4	4
NO	NO	**HEMORRHAGIC STROKE**	SURVIVOR	5	4
NO	NO	**SEIZURE** DISORDER/**ACUTE ENCEPHALOPATHY**	SURVIVOR	0	0
NO	NO	**HEPATIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY**	SURVIVOR	2	1
NO	NO	**UREMIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY**/CHRONIC KIDNEY DISEASE	NON SURVIVOR	NIL	NIL
NO	NO	**DIABETIC KETOACIDOSIS/METABOLIC ENCEPHALOPATHY**	SURVIVOR	1	0
NO	NO	**ISCHEMIC STROKE**	SURVIVOR	4	2
NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA**	SURVIVOR	1	0
NO	NO	**HEMORRHAGIC STROKE/HYPERTENSION**	NON SURVIVOR	NIL	NIL
NO	NO	**ISCHEMIC STROKE**	NON SURVIVOR	NIL	NIL
NO	NO	**CEREBRAL VENOUS THROMBOSIS/STROKE**	SURVIVOR	2	1
NO	NO	**ISCHEMIC STROKE**	SURVIVOR	4	3
NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA**	SURVIVOR	1	0
NO	NO	**METABOLIC ENCEPHALOPATHY/HYPOGLYCEMIA**	SURVIVOR	0	0
NO	NO	**DIABETIC KETOACIDOSIS/METABOLIC ENCEPHALOPATHY**	SURVIVOR	0	0
NO	YES	**HEMORRHAGIC STROKE/HYPERTENSION/CHRONIC KIDNEY DISEASE**	NON SURVIVOR	NIL	NIL
NO	YES	**DIABETIC KETOACIDOSIS/METABOLIC ENCEPHALOPATHY/SEVERE METABOLIC ACIDOSIS**	SURVIVOR	2	1
NO	YES	**CEREBRAL VENOUS THROMBOSIS/STROKE**	SURVIVOR	3	1
NO	NO	**UREMIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY**/CHRONIC KIDNEY DISEASE	SURVIVOR	2	2
NO	NO	**ISCHEMIC STROKE/DIABETES MELLITUS**/HYPERTENSION/RECURRENT CVA	SURVIVOR	4	2
NO	NO	**ISCHEMIC STROKE WITH MIDLINE SHIFT**/DIABETES MELLITUS/HYPERTENSION	NON SURVIVOR	NIL	NIL
NO	NO	**UREMIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY/CHRONIC KIDNEY DISEASE**	SURVIVOR	2	1
NO	YES	**CARDIOGENIC SHOCK**/PULMONARY EDEMA/ **POST CARDIAC ARREST**	NON SURVIVOR	NIL	NIL
NO	YES	**ISCHEMIC STROKE WITH MIDLINE SHIFT**/ YOUNG STROKE	SURVIVOR	4	3
NO	NO	**HEPATIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY**	SURVIVOR	2	1

MASTER CHART - MD THESIS

Serial NO	NAME	SEX	AGE	GLASGOW COMA SCALE							
				AT ADMISSION				24 HOURS AFTER ADMISSION			
				EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE	EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE
34	PATIENT 3	MALE	61	3	3	4	10	3	3	4	10
35	PATIENT 3	FEMALI	65	2	1	2	5	2	1	2	5
36	PATIENT 3	MALE	22	3	2	4	9	3	2	4	9
37	PATIENT 3	FEMALI	48	3	2	4	9	3	2	4	9
38	PATIENT 3	FEMALI	49	2	2	3	7	2	2	2	6
39	PATIENT 3	MALE	64	3	3	4	10	3	3	4	10
40	PATIENT 4	MALE	66	3	2	5	10	3	2	5	10
41	PATIENT 4	MALE	72	2	2	5	9	2	2	5	9
42	PATIENT 4	MALE	55	3	2	5	10	3	2	5	10
43	PATIENT 4	MALE	58	3	NOT TESTABLE	4	8	3	NOT TESTABLE	4	8
44	PATIENT 4	FEMALI	22	4	4	5	13	4	4	5	13
45	PATIENT 4	FEMALI	31	3	3	5	11	3	3	5	11
46	PATIENT 4	MALE	62	3	3	5	11	2	2	3	7
47	PATIENT 4	MALE	58	3	3	4	10	3	3	4	10
48	PATIENT 4	MALE	59	2	2	4	8	4	5	6	15
49	PATIENT 4	MALE	53	3	2	5	9	4	4	5	13
50	PATIENT 5	MALE	48	2	2	3	7	2	2	2	6
51	PATIENT 5	MALE	55	3	3	5	11	3	3	5	11
52	PATIENT 5	MALE	54	3	3	5	11	3	3	5	11
53	PATIENT 5	MALE	64	3	3	5	11	3	3	5	11
54	PATIENT 5	FEMALI	61	3	3	5	11	3	3	5	11
55	PATIENT 5	FEMALI	57	3	3	5	11	3	3	5	11
56	PATIENT 5	MALE	64	2	2	3	7	2	1	2	5
57	PATIENT 5	MALE	61	3	3	5	11	3	3	5	11
58	PATIENT 5	FEMALI	55	3	2	6	11	3	2	6	11
59	PATIENT 5	MALE	62	4	4	6	14	4	4	6	14
60	PATIENT 6	FEMALI	41	3	2	5	10	3	2	5	10
61	PATIENT 6	MALE	49	3	3	5	11	3	3	5	11
62	PATIENT 6	MALE	51	2	2	4	8	2	2	4	8
63	PATIENT 6	MALE	88	2	NOT TESTABLE	4	7	2	NOT TESTABLE	4	7
64	PATIENT 6	MALE	55	2	NOT TESTABLE	4	7	2	NOT TESTABLE	4	7
65	PATIENT 6	FEMALI	58	3	3	5	11	3	3	5	11
66	PATIENT 6	MALE	67	3	3	5	11	3	3	5	11

	FOUR SCORE										CT BRAIN
	AT ADMISSION					24 HOURS AFTER ADMISSION					
	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEXES	RESPIRATIO	TOTAL SCOF	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEX	RESPIRATIO	TOTAL SCORE	
39	2	2	4	4	12	2	2	4	4	12	NORMAL
40	1	1	2	2	6	1	1	2	2	6	INTRACRANIAL HEMORRHAGE
41	2	2	4	4	12	2	2	4	4	12	NORMAL
42	2	2	4	4	12	2	2	4	4	12	NORMAL
43	1	2	2	4	9	1	1	1	2	5	HYDROCEPHALUS
44	2	2	4	4	12	2	2	4	4	12	LEFT GANGLIOCAPSULAR INFARCT
45	2	3	4	4	13	2	3	4	4	13	LEFT FRONTAL LOBE INFARCT
46	1	3	4	4	12	1	3	4	4	12	NORMAL
47	2	3	4	4	13	2	3	4	4	13	NORMAL
48	2	2	4	1	9	2	2	4	1	9	NORMAL
49	3	3	4	4	14	3	3	4	4	14	NORMAL
50	2	3	4	4	13	2	3	4	4	13	NORMAL
51	2	3	4	2	11	1	2	2	2	7	NORMAL
52	2	2	4	4	12	2	2	4	4	12	INTRACRANIAL HEMORRHAGE
53	1	2	4	4	11	4	4	4	4	16	NORMAL
54	2	3	4	4	13	4	3	4	4	15	NORMAL
55	1	2	4	3	10	1	1	2	2	6	NORMAL
56	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
57	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
58	2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT
59	2	3	4	4	13	2	3	4	4	13	INTRACRANIAL HEMORRHAGE
60	2	3	4	4	13	2	3	4	4	13	INTRACRANIAL HEMORRHAGE
61	1	2	4	4	11	1	1	2	2	6	INTRACRANIAL HEMORRHAGE
62	2	3	4	4	13	2	3	4	4	13	NORMAL
63	2	4	4	4	14	2	4	4	4	14	LEFT FRONTAL LOBE INFARCT
64	4	4	4	4	16	4	4	4	4	16	LEFT TEMPOROPARIETAL INFARCT
65	2	3	4	4	13	2	3	4	4	13	NORMAL
66	2	3	4	4	13	2	3	4	4	13	NORMAL
67	1	2	4	4	11	1	2	4	3	10	NORMAL
68	1	2	2	1	6	1	2	1	0	4	NORMAL
69	1	2	2	1	6	1	2	1	0	4	NORMAL
70	2	3	4	4	13	2	3	4	4	13	NORMAL
71	2	3	4	4	13	2	3	4	4	13	NORMAL

	PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
					AT DISCHARGE	THREE MONTHS POST DISCHARGE
39	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/** SIADH/CONGESTIVE CARDIAC FAILURE	SURVIVOR	2	1
40	NO	NO	**HEMORRHAGIC STROKE/** ACCELERATED HYPERTENSION	NON SURVIVOR	NIL	NIL
41	NO	NO	**ACUTE MENINGOENCEPHALITIS**	SURVIVOR	1	0
42	NO	NO	**ACUTE MENINGOENCEPHALITIS/CEREBRAL MALARIA**	SURVIVOR	1	0
43	NO	NO	**TUBERCULAR MENINGITIS/ HYDROCEPHALUS/ ACUTE ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
44	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION/ DIABETES MELLITUS	SURVIVOR	4	3
45	NO	NO	**ISCHEMIC STROKE/** BROCA'S APHASIA	SURVIVOR	3	2
46	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/** ACUTE GASTROENTERITIS	SURVIVOR	2	1
47	NO	NO	**HYPOVOLEMIC SHOCK/** UGI BLEED/ DECOMPENSATED LIVER DISEASE	SURVIVOR	1	0
48	NO	YES	**NEUROTOXIC SNAKE BITE (POISONING)/** ACUTE ENCEPHALOPATHY	SURVIVOR	2	0
49	NO	NO	**PARAQUAT POISONING/** ARDS/MODS/SEPSIS/**SEPTIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
50	NO	NO	**YELLOW PHOSPHORUS POISONING/ HEPATIC ENCEPHALOPATHY**	SURVIVOR	2	1
51	NO	NO	**YELLOW PHOSPHORUS POISONING/ HEPATIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
52	NO	NO	**HEMORRHAGIC STROKE/** HYPERTENSION	SURVIVOR	4	3
53	NO	NO	**HYPOGLYCEMIA/DIABETES MELLITUS/METABOLIC ENCEPHALOPATHY**	SURVIVOR	0	0
54	NO	NO	**STATUS EPILEPTICUS/SEIZURE DISORDER**	SURVIVOR	1	0
55	NO	NO	**HEPATIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY/** CHRONIC ALCOHOLIC	NON SURVIVOR	NIL	NIL
56	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION/ DIABETES MELLITUS	SURVIVOR	4	2
57	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION/ DIABETES MELLITUS	SURVIVOR	4	3
58	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION	SURVIVOR	4	2
59	NO	NO	**HEMORRHAGIC STROKE/ACCELERATED HYPERTENSION**	SURVIVOR	4	3
60	NO	NO	**HEMORRHAGIC STROKE/** HYPERTENSION/DIABETES MELLITUS	SURVIVOR	4	3
61	NO	NO	**HEMORRHAGIC STROKE/** ACCELERATED HYPERTENSION	NON SURVIVOR	NIL	NIL
62	NO	NO	ORGANOPHOSPHATE **POISONING**	SURVIVOR	3	2
63	NO	NO	**ISCHEMIC STROKE/** BROCA'S APHASIA/DIABETES MELLITUS	SURVIVOR	3	2
64	NO	NIO	**ISCHEMIC STROKE/** WERNICKE'S APHASIA/DIABETES MELLITUS	SURVIVOR	2	2
65	NO	NO	**ACUTE MENINGOENCEPHALITIS/** CEREBRAL MALARIA	SURVIVOR	1	0
66	NO	NO	**ACUTE MENINGOENCEPHALITIS**	SURVIVOR	1	0
67	NO	NO	**METABOLIC ENCEPHALOPATHY/** UREMIC ENCEPHALOPATHY/HYPERTENSION/CKD	NON SURVIVOR	NIL	NIL
68	NO	YES	**METABOLIC ENCEPHALOPATHY/** UREMIC ENCEPHALOPATHY/HYPERTENSION/CKD	NON SURVIVOR	NIL	NIL
69	NO	YES	**METABOLIC ENCEPHALOPATHY/** HEPATIC ENCEPHALOPATHY	NON SURVIVOR	NIL	NIL
70	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/** ACUTE GASTROENTERITIS	SURVIVOR	1	0
71	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/** ACUTE GASTROENTERITIS	SURVIVOR	1	0

MASTER CHART - MD THESIS

Serial NO	NAME	SEX	AGE	GLASGOW COMA SCALE							
				AT ADMISSION				24 HOURS AFTER ADMISSION			
				EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE	EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE
67	PATIENT 6	MALE	66	3	2	4	9	3	2	4	9
68	PATIENT 6	FEMALE	88	2	2	4	8	2	2	4	8
69	PATIENT 6	FEMALE	89	2	2	4	8	2	2	4	8
70	PATIENT 7	FEMALE	59	2	2	3	7	4	5	6	15
71	PATIENT 7	MALE	56	3	3	5	11	3	3	5	11
72	PATIENT 7	MALE	61	3	1	6	10	3	1	6	10
73	PATIENT 7	MALE	64	3	4	6	13	3	4	6	13
74	PATIENT 7	MALE	72	2	1	1	4	2	1	1	4
75	PATIENT 7	FEMALE	55	4	NOT TESTABLE	1	6	4	NOT TESTABLE	1	6
76	PATIENT 7	MALE	67	4	NOT TESTABLE	1	6	4	NOT TESTABLE	1	6
77	PATIENT 7	FEMALE	55	3	1	6	10	3	1	6	10
78	PATIENT 7	FEMALE	59	3	3	5	11	3	3	5	11
79	PATIENT 7	MALE	62	3	3	5	11	3	3	5	11
80	PATIENT 8	MALE	63	3	3	5	11	3	3	5	11
81	PATIENT 8	FEMALE	56	3	3	5	11	3	3	5	11
82	PATIENT 8	FEMALE	70	3	1	6	10	4	1	6	11
83	PATIENT 8	MALE	55	3	3	5	11	3	3	5	11
84	PATIENT 8	FEMALE	65	3	3	5	11	2	2	4	8
85	PATIENT 8	MALE	66	3	3	5	11	2	2	4	8
86	PATIENT 8	MALE	67	3	4	5	12	4	4	6	14
87	PATIENT 8	FEMALE	21	3	4	5	12	3	3	4	10
88	PATIENT 8	MALE	23	3	4	5	12	3	3	4	10
89	PATIENT 8	FEMALE	22	3	4	5	12	3	3	4	10
90	PATIENT 9	MALE	58	3	3	5	11	3	3	5	11
91	PATIENT 9	FEMALE	66	2	1	2	5	2	1	2	5
92	PATIENT 9	MALE	77	2	NOT TESTABLE	2	5	2	NOT TESTABLE	2	5
93	PATIENT 9	FEMALE	66	2	NOT TESTABLE	2	5	2	NOT TESTABLE	2	5
94	PATIENT 9	MALE	76	2	NOT TESTABLE	2	5	2	NOT TESTABLE	2	5
95	PATIENT 9	MALE	95	2	NOT TESTABLE	2	5	2	NOT TESTABLE	2	5
96	PATIENT 9	MALE	66	3	3	5	11	3	3	5	11
97	PATIENT 9	FEMALE	33	3	2	4	9	3	4	5	12
98	PATIENT 9	MALE	88	3	3	5	11	3	3	5	11
99	PATIENT 9	FEMALE	87	3	3	5	11	3	3	5	11

FOUR SCORE											CT BRAIN
AT ADMISSION					24 HOURS AFTER ADMISSION						
EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEXES	RESPIRATIO	TOTAL SCOR	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEX	RESPIRATIO	TOTAL SCORE		
2	2	3	4	11	2	2	2	3	9		NORMAL
1	2	4	4	11	1	2	4	2	9		NORMAL
1	2	4	4	11	1	2	4	4	11		NORMAL
1	1	4	3	9	4	4	4	4	16		NORMAL
2	3	4	4	13	2	3	4	4	13		LEFT GANGLIOCAPSULAR INFARCT
2	4	4	4	14	2	4	4	4	14		LEFT FRONTOTEMPORAL INFARCT
2	4	4	4	14	2	4	4	4	14		LEFT TEMPOROPARIETAL INFARCT
1	0	2	4	7	1	0	1	2	4		BILATERAL PONTINE HEMORRHAGE
4	0	4	1	9	4	0	4	1	9		PONTINE INFARCT
4	0	4	1	9	4	0	4	1	9		PONTINE INFARCT
2	4	4	4	14	2	4	4	4	14		LEFT FRONTAL LOBE HEMORRHAGE
2	3	4	4	13	2	3	4	4	13		LEFT GANGLIOCAPSULAR INFARCT
2	3	4	4	13	2	3	4	4	13		FIGHT GANGLIOCAPSULARINFARCT
2	3	4	4	13	2	3	4	4	13		FIGHT GANGLIOCAPSULARINFARCT
2	3	4	4	13	2	3	4	4	13		INTRACRANIAL HEMORRHAGE
2	4	4	4	14	4	4	4	4	16		INTRACRANIAL HEMORRHAGE
2	3	4	4	13	2	3	4	4	13		FIGHT GANGLIOCAPSULARINFARCT
2	2	4	3	11	1	2	3	2	8		INFARCT WITH HEMORRHAGIC TRAI
2	2	4	3	11	1	2	3	2	8		INTRACRANIAL AND INTRAVENTRIC
2	3	4	4	13	4	4	4	4	16		LEFT TEMPOROPARIETAL INFARCT
2	3	4	4	13	2	2	4	3	11		NORMAL
2	3	4	4	13	2	2	4	3	11		NORMAL
2	3	4	4	13	2	2	4	3	11		NORMAL
2	3	4	4	13	2	3	4	4	13		LEFT FRONTAL HEMORRHAGE
1	1	2	2	6	1	1	2	2	6		INTRACRANIAL HEMORRHAGE
1	1	2	1	5	1	1	2	1	5		NORMAL
1	1	2	1	5	1	1	2	1	5		BRAINSTEM HEMORRHAGE
1	1	2	1	5	1	1	2	1	5		INFARCT WITH HEMORRHAGIC TRAI
1	1	3	1	6	1	1	3	1	6		NORMAL
2	3	4	4	13	2	3	4	4	13		LEFT GANGLIOCAPSULAR INFARCT
2	2	4	4	12	2	3	4	4	13		SIGMOID SINUS THROMBOSIS
2	3	4	4	13	2	3	4	4	13		NORMAL
2	3	4	4	13	2	3	4	4	13		NORMAL

PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
				AT DISCHARGE	THREE MONTHS POST DISCHARGE
NO	NO	METABOLIC ENCEPHALOPATHY/HYPONATREMIA/SIADH/CARCINOMA LUNG/ASPIRATION PNEUM(	NON SURVIVOR	NIL	NIL
NO	NO	METABOLIC ENCEPHALOPATHY/HYPONATREMIA/MYXEDEMA COMA	NON SURVIVOR	NIL	NIL
NO	NO	METABOLIC ENCEPHALOPATHY/HYPONATREMIA/MYXEDEMA COMA	SURVIVOR	2	2
NO	NO	METABOLIC ENCEPHALOPATHY/ HYPOGLYCEMIA	SURVIVOR	0	0
NO	NO	ISCHEMIC STROKE/ DIABETES MELLITUS	SURVIVOR	4	2
NO	NO	ISCHEMIC STROKE/ HYPERTENSION	SURVIVOR	3	2
NO	NO	ISCHEMIC STROKE	SURVIVOR	4	2
NO	NO	PONTINE HEMORRHAGIC STROKE	NON SURVIVOR	NIL	NIL
NO	YES	PONTINE INFARCT/ LOCKED IN SYNDROME/SHTN	SURVIVOR	5	4
NO	YES	PONTINE INFARCT/ LOCKED IN SYNDROME/SHTN	SURVIVOR	4	4
NO	NO	HEMORRHAGIC STROKE/ HYPERTENSION	SURVIVOR	4	2
NO	NO	ISCHEMIC STROKE	SURVIVOR	4	6
NO	NO	ISCHEMIC STROKE/ DIABETES MELLITUS	SURVIVOR	4	6
NO	NO	ISCHEMIC STROKE/ HYPERTENSION	SURVIVOR	4	3
NO	NO	HEMORRHAGIC STROKE/ DIABETES MELLITUS	SURVIVOR	4	1
NO	NO	HEMORRHAGIC STROKE/ BROCA'S APHASIA/HYPERTENSION	SURVIVOR	2	1
NO	NO	ISCHEMIC STROKE/ HYPERTENSION	SURVIVOR	3	2
NO	NO	ISCHEMIC STROKE WITH HEMORRHAGIC TRANSFORMATION/ HYPERTENSION	NON SURVIVOR	NIL	NIL
NO	NO	HEMORRHAGIC STROKE/ CHRONIC KIDNEY DISEASE/HYPERTENSION	NON SURVIVOR	NIL	NIL
NO	NO	ISCHEMIC STROKE/ VERNICKE'S APHASIA/DIABETES MELLITUS	SURVIVOR	3	2
NO	NO	PARAQUAT POISONING/ ARDS/MODS/SEPSIS/SEPTIC ENCEPHALOPATHY	NON SURVIVOR	NIL	NIL
NO	NO	PARAQUAT POISONING/ ARDS/RESPIRATORY FAILURE	NON SURVIVOR	NIL	NIL
NO	NO	PARAQUAT POISONING/ ARDS/PNEUMOMEDIASTINUM/RESPIRATORY FAILURE	NON SURVIVOR	NIL	NIL
NO	NO	HEMORRHAGIC STROKE	SURVIVOR	3	2
NO	NO	HEMORRHAGIC STROKE/ ACCELERATED HYPERTENSION	NON SURVIVOR	NIL	NIL
NO	YES	YELLOW PHOSPHORUS POISONING/ HEPATIC ENCEPHALOPATHY/POST CARDIAC ARREST	NON SURVIVOR	NIL	NIL
NO	YES	HEMORRHAGIC STROKE	NON SURVIVOR	NIL	NIL
NO	YES	ISCHEMIC STROKE WITH HEMORRHAGIC TRANSFORMATION/ HYPERTENSION	NON SURVIVOR	NIL	NIL
NO	YES	METABOLIC ENCEPHALOPATHY/UREMIC ENCEPHALOPATHY/POST CARDIAC ARREST	NON SURVIVOR	NIL	NIL
NO	NO	ISCHEMIC STROKE	SURVIVOR	4	2
NO	NO	CEREBRAL VENOUS THROMBOSIS/STROKE	SURVIVOR	2	1
NO	NO	METABOLIC ENCEPHALOPATHY/ HYPONATREMIA/ ACUTE GASTROENTERITIS	SURVIVOR	2	0
NO	NO	METABOLIC ENCEPHALOPATHY/ HYPONATREMIA/ ACUTE GASTROENTERITIS	SURVIVOR	1	0

MASTER CHART - MD THESIS

Serial NO	NAME	SEX	AGE	GLASGOW COMA SCALE							
				AT ADMISSION				24 HOURS AFTER ADMISSION			
				EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE	EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE
100	PATIENT 1(	FEMALI	77	3	3	5	11	3	3	5	11
101	PATIENT 1(	MALE	66	3	3	5	11	3	3	5	11
102	PATIENT 1(	MALE	55	3	3	5	11	3	2	5	10
103	PATIENT 1(	FEMALI	66	3	3	5	11	2	2	4	8
104	PATIENT 1(	MALE	66	3	3	5	11	2	2	4	8
105	PATIENT 1(	MALE	44	3	3	5	11	2	2	4	8
106	PATIENT 1(	FEMALI	55	3	3	5	11	2	2	4	8
107	PATIENT 1(	MALE	43	3	3	5	11	2	2	4	8
108	PATIENT 1(	FEMALI	88	2	NOT TESTABLE	1	4	2	NOT TESTABLE	1	4
109	PATIENT 1(	MALE	76	2	NOT TESTABLE	1	4	2	NOT TESTABLE	1	4
110	PATIENT 11	MALE	66	2	NOT TESTABLE	1	4	2	NOT TESTABLE	1	4
111	PATIENT 11	MALE	56	3	3	5	11	3	3	5	11
112	PATIENT 11	MALE	66	3	3	5	11	3	3	5	11
113	PATIENT 11	MALE	44	3	1	6	10	4	1	6	11
114	PATIENT 11	MALE	55	3	1	6	10	4	1	6	11
115	PATIENT 11	MALE	65	3	1	6	1	4	1	6	11
116	PATIENT 11	FEMALI	54	3	3	5	11	3	3	5	11
117	PATIENT 11	FEMALI	66	3	3	5	11	3	3	5	11
118	PATIENT 11	FEMALI	46	3	3	5	11	3	3	5	11
119	PATIENT 11	FEMALI	55	3	3	5	11	4	5	6	15
120	PATIENT 12	MALE	49	3	3	5	11	4	5	6	15
121	PATIENT 12	MALE	48	2	1	2	5	2	1	2	5
122	PATIENT 12	MALE	45	2	1	2	5	2	1	2	5
123	PATIENT 12	MALE	56	2	1	2	5	2	1	2	5
124	PATIENT 12	MALE	77	3	3	5	11	3	3	5	11
125	PATIENT 12	FEMALI	55	3	3	5	11	3	3	5	11
126	PATIENT 12	MALE	72	2	2	5	9	2	2	5	9
127	PATIENT 12	MALE	55	3	2	5	10	3	2	5	10
128	PATIENT 12	MALE	58	3	NOT TESTABLE	4	8	3	NOT TESTABLE	4	8
129	PATIENT 12	FEMALI	22	4	4	5	13	4	4	5	13
130	PATIENT 13	FEMALI	31	3	3	5	11	3	3	5	11
131	PATIENT 13	MALE	62	3	3	5	11	2	2	3	7
132	PATIENT 13	MALE	58	3	3	4	10	3	3	4	10

	FOUR SCORE										CT BRAIN
	AT ADMISSION					24 HOURS AFTER ADMISSION					
	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEXES	RESPIRATIOI	TOTAL SCOR	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEX	RESPIRATIOI	TOTAL SCORE	
105	2	3	4	4	13	2	3	4	4	13	NORMAL
106	2	3	4	4	13	2	3	4	4	13	NORMAL
107	2	3	4	4	13	2	3	3	3	11	NORMAL
108	2	2	4	3	11	1	2	3	2	8	RIGHT GANGLIOCAPSULAR INFARCT
109	2	2	4	3	11	1	2	3	2	8	INTRACRANIAL HEMORRHAGE
110	2	2	4	3	11	1	2	3	2	8	HYDROCEPHALUS
111	2	2	4	3	11	1	2	3	2	8	NORMAL
112	2	2	4	3	11	1	2	3	2	8	NORMAL
113	1	0	2	0	3	1	0	2	0	3	INTRAVENTRICULAR HEMORRHAGE
114	1	0	2	0	3	1	0	2	0	3	BRAINSTEM HEMORRHAGE
115	1	0	2	0	3	1	0	2	0	3	BRAINSTEM HEMORRHAGE
116	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR HEMORF
117	2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT
118	2	4	4	4	14	4	4	4	4	16	LEFT FRONTAL LOBE INFARCT
119	2	4	4	4	14	4	4	4	4	16	LEFT FRONTAL LOBE INFARCT
120	2	4	4	4	14	4	4	4	4	16	LEFT FRONTAL LOBE INFARCT
121	2	3	4	4	13	2	3	4	4	13	NORMAL
122	2	3	4	4	13	2	3	4	4	13	NORMAL
123	2	3	4	4	13	2	3	4	4	13	NORMAL
124	2	3	4	4	13	4	4	4	4	16	NORMAL
125	2	3	4	4	13	4	4	4	4	16	NORMAL
126	1	1	4	3	9	1	1	2	2	6	NORMAL
127	1	1	4	3	9	1	1	3	2	7	NORMAL
128	1	1	4	3	9	1	1	3	2	7	NORMAL
129	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR HEMORF
130	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR HEMORF
131	1	3	4	4	12	1	3	4	4	12	NORMAL
132	2	3	4	4	13	2	3	4	4	13	NORMAL
133	2	2	4	1	9	2	2	4	1	9	NORMAL
134	3	3	4	4	14	3	3	4	4	14	NORMAL
135	2	3	4	4	13	2	3	4	4	13	NORMAL
136	2	3	4	2	11	1	2	2	2	7	NORMAL
137	2	2	4	4	12	2	2	4	4	12	INTRACRANIAL HEMORRHAGE

	PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
					AT DISCHARGE	THREE MONTHS POST DISCHARGE
105	NO	NO	**METABOLIC ENCEPHALOPATHY/ HYPONATREMIA/** ACUTE GASTROENTERITIS	SURVIVOR	1	0
106	NO	NO	**ACUTE MENINGOENCEPHALITIS**	SURVIVOR	2	0
107	NO	NO	**ACUTE MENINGOENCEPHALITIS/** ASPIRATION PNEUMONIA	NON SURVIVOR	NIL	NIL
108	NO	NO	**ISCHEMIC STROKE/** ASPIRATION PNEUMONITIS	NON SURVIVOR	NIL	NIL
109	NO	NO	**HEMORRHAGIC STROKE/** ASPIRATION PNEUMONITIS	NON SURVIVOR	NIL	NIL
110	NO	NO	**TUBERCULAR MENINGITIS/ HYDROCEPHALUS/ ACUTE ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
111	NO	NO	**METABOLIC ENCEPHALOPATHY/ HEPATIC ENCEPHALOPATHY/** NAFLD	NON SURVIVOR	NIL	NIL
112	NO	NO	**METABOLIC ENCEPHALOPATHY/ UREMIC ENCEPHALOPATHY/** HYPERTENSION/CKD	NON SURVIVOR	NIL	NIL
113	NO	YES	**HEMORRHAGIC STROKE**	NON SURVIVOR	NIL	NIL
114	NO	YES	**HEMORRHAGIC STROKE/** HYPERTENSION	NON SURVIVOR	NIL	NIL
115	NO	YES	**HEMORRHAGIC STROKE/** HYPERTENSION	NON SURVIVOR	NIL	NIL
116	NO	NO	**HEMORRHAGIC STROKE/** HYPERTENSION	SURVIVOR	4	2
117	NO	NO	**ISCHEMIC STROKE/** DIABETES MELLITUS	SURVIVOR	4	2
118	NO	NO	**ISCHEMIC STROKE**	SURVIVOR	4	3
119	NO	NO	**ISCHEMIC STROKE/** DYSLIPIDEMIA	SURVIVOR	4	3
120	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION	SURVIVOR	4	3
121	NO	NO	**METABOLIC ENCEPHALOPATHY/ UREMIC ENCEPHALOPATHY**	SURVIVOR	2	1
122	NO	NO	**METABOLIC ENCEPHALOPATHY/ HYPONATREMIA/** SIADH/ CARCINOMA LUNG	SURVIVOR	2	6
123	NO	NO	**ACUTE MENINGOENCEPHALITIS**	SURVIVOR	2	0
124	NO	NO	**HYPOGLYCEMIA/** DIABETES MELLITUS**/METABOLIC ENCEPHALOPATHY**	SURVIVOR	0	0
125	NO	NO	**HYPOGLYCEMIA/** DIABETES MELLITUS**/METABOLIC ENCEPHALOPATHY**	SURVIVOR	0	0
126	NO	NO	**HEPATIC ENCEPHALOPATHY/ METABOLIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
127	NO	NO	**HEPATIC ENCEPHALOPATHY/** ASPIRATION PNEUMONITIS	NON SURVIVOR	NIL	NIL
128	NO	NO	**UREMIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY/** ASPIRATION PNEUMONIA	NON SURVIVOR	NIL	NIL
129	NO	NO	**HEMORRHAGIC STROKE/ HYPERTENSION**	SURVIVOR	4	3
130	NO	NO	**HEMORRHAGIC STROKE**	SURVIVOR	4	3
131	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/** ACUTE GASTROENTERITIS	SURVIVOR	2	0
132	NO	NO	**HYPOVOLEMIC SHOCK/** UGI BLEED/ DECOMPENSATED LIVER DISEASE	SURVIVOR	2	6
133	NO	YES	**NEUROTOXIC SNAKE BITE (POISONING)/** ACUTE ENCEPHALOPATHY	SURVIVOR	2	0
134	NO	NO	**PARAQUAT POISONING/** APDS/ MODS/SEPSIS**/SEPTIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
135	NO	NO	**YELLOW PHOSPHORUS POISONING/ HEPATIC ENCEPHALOPATHY**	SURVIVOR	2	0
136	NO	NO	**YELLOW PHOSPHORUS POISONING/ HEPATIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
137	NO	NO	**HEMORRHAGIC STROKE/** HYPERTENSION	SURVIVOR	4	6

MASTER CHART - MD THESIS

Serial NO	NAME	SEX	AGE	GLASGOV COMA SCALE							
				AT ADMISSION				24 HOURS AFTER ADMISSION			
				EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE	EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE
133	PATIENT 133	MALE	59	2	2	4	8	4	5	6	15
134	PATIENT 134	MALE	53	3	2	5	9	4	4	5	13
135	PATIENT 135	MALE	48	2	2	3	7	2	2	2	6
136	PATIENT 136	MALE	55	3	3	5	11	3	3	5	11
137	PATIENT 137	MALE	54	3	3	5	11	3	3	5	11
138	PATIENT 138	MALE	64	3	3	5	11	3	3	5	11
139	PATIENT 139	FEMALI	61	3	3	5	11	3	3	5	11
140	PATIENT 140	FEMALI	57	3	3	5	11	3	3	5	11
141	PATIENT 141	MALE	64	2	2	3	7	2	1	2	5
142	PATIENT 142	MALE	61	3	3	5	11	3	3	5	11
143	PATIENT 143	FEMALI	55	3	2	6	11	3	2	6	11
144	PATIENT 144	MALE	62	4	4	6	14	4	4	6	14
145	PATIENT 145	FEMALI	41	3	2	5	10	3	2	5	10
146	PATIENT 146	MALE	49	3	3	5	11	3	3	5	11
147	PATIENT 147	MALE	51	2	2	4	8	2	2	4	8
148	PATIENT 148	MALE	88	2	NOT TESTABLE	4	7	2	NOT TESTABLE	4	7
149	PATIENT 149	MALE	55	2	NOT TESTABLE	4	7	2	NOT TESTABLE	4	7
150	PATIENT 150	FEMALI	58	3	3	5	11	3	3	5	11
151	PATIENT 151	MALE	67	3	3	5	11	3	3	5	11
152	PATIENT 152	MALE	66	3	2	4	9	3	2	4	9
153	PATIENT 153	FEMALI	88	2	2	4	8	2	2	4	8
154	PATIENT 154	MALE	75	2	NOT TESTABLE	3	6	2	NOT TESTABLE	3	6
155	PATIENT 155	MALE	67	4	1	5	10	4	1	5	10
156	PATIENT 156	MALE	67	4	3	5	12	4	3	5	12
157	PATIENT 157	MALE	60	4	2	5	11	4	2	5	11
158	PATIENT 158	MALE	40	1	2	4	7	2	2	4	8
159	PATIENT 159	FEMALI	67	3	2	4	9	3	2	4	9
160	PATIENT 160	MALE	55	2	2	4	8	3	3	5	11
161	PATIENT 161	MALE	70	3	4	5	12	3	4	5	12
162	PATIENT 162	FEMALI	70	4	2	5	11	4	2	5	11
163	PATIENT 163	FEMALI	50	2	2	3	7	2	2	3	7
164	PATIENT 164	MALE	70	1	1	3	5	1	1	3	5
165	PATIENT 165	MALE	32	2	2	5	9	3	3	5	11

FOUR SCORE										CT BRAIN
AT ADMISSION					24 HOURS AFTER ADMISSION					
EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEXES	RESPIRATIOI	TOTAL SCOR	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEX	RESPIRATIOI	TOTAL SCORE	
1	2	4	4	11	4	4	4	4	16	NORMAL
2	3	4	4	13	4	3	4	4	15	NORMAL
1	2	4	3	10	1	1	2	2	6	NORMAL
2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT
2	3	4	4	13	2	3	4	4	13	INTRACRANIAL HEMORRHAGE
2	3	4	4	13	2	3	4	4	13	INTRACRANIAL HEMORRHAGE
1	2	4	4	11	1	1	2	2	6	INTRACRANIAL HEMORRHAGE
2	3	4	4	13	2	3	4	4	13	NORMAL
2	4	4	4	14	2	4	4	4	14	LEFT FRONTAL LOBE INFARCT
4	4	4	4	16	4	4	4	4	16	LEFT TEMPOROPARIETAL INFARCT
2	3	4	4	13	2	3	4	4	13	NORMAL
2	3	4	4	13	2	3	4	4	13	NORMAL
1	2	4	4	11	1	2	4	3	10	NORMAL
1	2	2	1	6	1	2	1	0	4	NORMAL
1	2	2	1	6	1	2	1	0	4	NORMAL
2	3	4	4	13	2	3	4	4	13	NORMAL
2	3	4	4	13	2	3	4	4	13	NORMAL
2	2	3	4	11	2	2	2	3	9	NORMAL
1	2	4	4	11	1	2	4	2	9	NORMAL
1	2	4	1	8	1	2	4	1	8	RIGHT GANGLIOCAPSULAR INFARCT
4	3	4	4	15	4	3	4	4	15	LEFT FRONTAL LOBE INFARCT
3	3	4	4	14	4	3	4	4	15	LEFT THALAMIC HEMORRHAGE
3	3	4	4	14	3	3	4	4	14	NORMAL
0	2	2	4	8	1	2	4	4	11	NORMAL
2	2	4	3	11	2	2	4	3	11	NORMAL
1	2	4	3	10	2	3	4	4	13	NORMAL
2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT
3	3	4	4	14	3	3	4	4	14	NORMAL
1	2	4	4	11	1	2	2	4	9	INTRACRANIAL HEMORRHAGE
0	2	2	4	8	0	2	2	4	8	RIGHT GANGLIOCAPSULAR INFARCT
1	3	4	4	12	2	3	4	4	13	SUPERIOR SAGITTAL SINUS THROME

	PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
					AT DISCHARGE	THREE MONTHS POST DISCHARGE
138	NO	NO	**HYPOGLYCEMIA/DIABETES MELLITUS/METABOLIC ENCEPHALOPATHY**	SURVIVOR	1	0
139	NO	NO	**STATUS EPILEPTICUS/SEIZURE DISORDER**	SURVIVOR	1	0
140	NO	NO	**HEPATIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY/**CHRONIC ALCOHOLIC	NON SURVIVOR	NIL	NIL
141	NO	NO	**ISCHEMIC STROKE/**HYPERTENSION/ DIABETES MELLITUS	SURVIVOR	4	6
142	NO	NO	**ISCHEMIC STROKE/**HYPERTENSION/ DIABETES MELLITUS	SURVIVOR	4	3
143	NO	NO	**ISCHEMIC STROKE/**HYPERTENSION	SURVIVOR	4	4
144	NO	NO	**HEMORRHAGIC STROKE/**ACCELERATED HYPERTENSION	SURVIVOR	5	4
145	NO	NO	**HEMORRHAGIC STROKE/**HYPERTENSION/DIABETES MELLITUS	SURVIVOR	5	4
146	NO	NO	**HEMORRHAGIC STROKE/**ACCELERATED HYPERTENSION	NON SURVIVOR	NIL	NIL
147	NO	NO	ORGANOPHOSPHATE **POISONING**	SURVIVOR	3	2
148	NO	NO	**ISCHEMIC STROKE/**BROCA'S APHASIA/DIABEETES MELLITUS	SURVIVOR	3	1
149	NO	NO	**ISCHEMIC STROKE/**VERNICKE'S APHASIA/DIABETES MELLITUS	SURVIVOR	3	2
150	NO	NO	**ACUTE MENINGOENCEPHALITIS/**CEREBRAL MALARIA	SURVIVOR	2	0
151	NO	NO	**ACUTE MENINGOENCEPHALITIS**	SURVIVOR	2	0
152	NO	NO	**METABOLIC ENCEPHALOPATHY/** UREMIC ENCEPHALOPATHY/HYPERTENSION/CKD	NON SURVIVOR	NIL	NIL
153	NO	YES	**METABOLIC ENCEPHALOPATHY/** UREMIC ENCEPHALOPATHY/HYPERTENSION/CKD	NON SURVIVOR	NIL	NIL
154	NO	YES	**METABOLIC ENCEPHALOPATHY/**HEPATIC ENCEPHALOPATHY	NON SURVIVOR	NIL	NIL
155	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/**ACUTE GASTROENTERITIS	SURVIVOR	2	0
156	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/**ACUTE GASTROENTERITIS	SURVIVOR	2	0
157	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/**SIADH/CARCINOMA LUNG/ASPIRATION PNEUMO	NON SURVIVOR	NIL	NIL
158	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/MYXEDEMA COMA**	NON SURVIVOR	NIL	NIL
159	NO	YES	**ISCHEMIC STROKE /**DIABETES MELLITUS	SURVIVOR	4	4
160	NO	NO	**ISCHEMIC STROKE /**DIABETES MELLITUS	SURVIVOR	4	2
161	NO	NO	**HEMORRHAGIC STROKE**	SURVIVOR	4	3
162	NO	NO	**SEIZURE** DISORDER/**ACUTE ENCEPHALOPATHY**	SURVIVOR	2	1
163	NO	NO	**HEPATIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY**	SURVIVOR	2	2
164	NO	NO	**UREMIC ENCEPHALOPATHY /METABOLIC ENCEPHALOPATHY/**CHRONIC KIDNEY DISEASE	NON SURVIVOR	NIL	NIL
165	NO	NO	**DIABETIC KETOACIDOSIS/METABOLIC ENCEPHALOPATHY**	SURVIVOR	2	0
166	NO	NO	**ISCHEMIC STROKE**	SURVIVOR	3	3
167	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA**	SURVIVOR	2	1
168	NO	NO	**HEMORRHAGIC STROKE/HYPERTENSION**	NON SURVIVOR	NIL	NIL
169	NO	NO	**ISCHEMIC STROKE**	NON SURVIVOR	NIL	NIL
170	NO	NO	**CEREBRAL VENOUS THROMBOSIS/STROKE**	SURVIVOR	2	1

MASTER CHART - MD THESIS

	Serial NO	NAME	SEX	AGE	GLASGOW COMA SCALE							
					AT ADMISSION				24 HOURS AFTER ADMISSION			
					EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE	EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE
171	166	PATIENT 166	FEMALE	55	3	3	5	11	3	3	5	11
172	167	PATIENT 167	FEMALE	64	3	2	5	10	3	2	5	10
173	168	PATIENT 168	MALE	84	3	3	4	10	3	3	4	10
174	169	PATIENT 169	FEMALE	34	2	2	4	8	3	3	5	11
175	170	PATIENT 170	FEMALE	70	2	NOT TESTABLE	2	5	2	NOT TESTABLE	2	5
176	171	PATIENT 171	MALE	64	2	NOT TESTABLE	4	7	2	NOT TESTABLE	4	7
177	172	PATIENT 172	FEMALE	38	2	NOT TESTABLE	3	6	2	NOT TESTABLE	3	6
178	173	PATIENT 173	FEMALE	64	2	2	4	8	2	2	4	8
179	174	PATIENT 174	MALE	61	3	2	3	8	3	2	3	8
180	175	PATIENT 175	FEMALE	65	2	2	2	6	2	2	2	6
181	176	PATIENT 176	FEMALE	71	2	2	4	8	2	2	4	8
182	177	PATIENT 177	MALE	74	1	NOT TESTABLE	1	3	1	NOT TESTABLE	1	3
183	178	PATIENT 178	MALE	38	2	NOT TESTABLE	3	6	2	NOT TESTABLE	3	6
184	179	PATIENT 179	MALE	55	2	2	2	6	2	2	4	8
185	180	PATIENT 180	MALE	61	3	3	4	10	3	3	4	10
186	181	PATIENT 181	MALE	55	3	3	5	11	3	3	5	11
187	182	PATIENT 182	FEMALE	81	3	3	5	11	3	3	5	11
188	183	PATIENT 183	MALE	72	3	3	5	11	3	3	5	11
189	184	PATIENT 184	MALE	45	3	1	6	10	4	1	6	11
190	185	PATIENT 185	FEMALE	77	2	2	4	8	2	2	4	8
191	186	PATIENT 186	FEMALE	75	2	2	4	8	2	NOT TESTABLE	4	8
192	187	PATIENT 187	FEMALE	56	2	2	4	8	4	5	6	15
193	188	PATIENT 188	FEMALE	67	3	3	5	11	3	3	5	11
194	189	PATIENT 189	MALE	70	3	3	5	11	3	3	5	11
195	190	PATIENT 190	MALE	65	2	1	1	4	2	1	1	4
196	191	PATIENT 191	FEMALE	78	2	NOT TESTABLE	1	4	2	NOT TESTABLE	1	4
197	192	PATIENT 192	FEMALE	67	3	3	5	11	3	3	5	11
198	193	PATIENT 193	FEMALE	55	3	3	5	11	3	3	5	11
199	194	PATIENT 194	MALE	59	3	3	5	11	3	3	5	11
200	195	PATIENT 195	MALE	67	2	NOT TESTABLE	5	8	2	NOT TESTABLE	5	8
201	196	PATIENT 196	FEMALE	77	3	3	5	11	3	3	5	11
202	197	PATIENT 197	FEMALE	56	3	3	5	11	4	1	6	11
203	198	PATIENT 198	FEMALE	77	3	3	5	11	3	3	5	11

	FOUR SCORE										CT BRAIN
	AT ADMISSION					24 HOURS AFTER ADMISSION					
	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEXES	RESPIRATIOI	TOTAL SCOR	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEX	RESPIRATIOI	TOTAL SCORE	
171	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
172	2	3	4	4	13	2	3	4	4	13	NORMAL
173	2	2	4	4	12	2	2	4	4	12	NORMAL
174	1	2	4	4	11	2	3	4	4	13	NORMAL
175	1	1	2	1	5	1	1	2	1	5	INTRACRANIAL HEMORRHAGE
176	1	2	4	1	8	1	2	4	1	8	NORMAL
177	1	2	4	1	8	1	2	4	1	8	TRANSVERSE+SIGMOID SINUS THRO
178	1	2	4	4	11	1	2	4	4	11	NORMAL
179	2	2	4	4	12	2	2	4	4	12	RIGHT GANGLIOCAPSULAR INFARCT
180	1	1	2	2	6	1	1	2	2	6	RIGHT GANGLIOCAPSULAR INFARCT
181	1	2	4	4	11	1	2	4	4	11	NORMAL
182	0	0	1	0	1	0	0	1	0	1	NORMAL
183	1	2	4	1	8	1	2	4	1	8	RIGHT GANGLIOCAPSULAR INFARCT
184	1	1	4	3	9	1	2	4	4	11	NORMAL
185	2	2	4	4	12	2	2	4	4	12	NORMAL
186	2	3	4	4	13	2	3	4	4	13	RIGHT FRONTAL LOBE INFARCT
187	2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT
188	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
189	2	4	4	4	14	3	4	4	4	16	LEFT FRONTAL LOBE INFARCT
190	1	2	2	4	9	1	2	2	2	7	INTRAVENTRICULAR HEMORRHAGE
191	1	2	2	4	9	1	2	2	4	9	LEFT GANGLIOCAPSULAR HEMORRH
192	1	2	4	4	11	4	4	4	4	16	NORMAL
193	2	3	4	4	13	2	3	4	4	13	NORMAL
194	2	3	4	4	13	2	3	4	4	13	NORMAL
195	1	0	1	2	4	1	0	1	2	4	INTRACRANIAL WITH INTRAVENTRIC
196	1	0	1	1	3	1	0	1	1	3	NORMAL
197	2	3	4	4	13	2	3	4	4	13	NORMAL
198	2	3	4	4	13	2	3	4	4	13	NORMAL
199	2	3	4	4	13	2	3	4	4	13	NORMAL
200	2	3	3	1	9	2	3	3	1	9	NORMAL
201	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
202	2	3	4	4	13	4	4	4	4	16	LEFT FRONTAL LOBE INFARCT
203	2	3	4	4	13	2	3	4	4	13	SUPERIOR SAGITTAL SINUS THROME

	PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
					AT DISCHARGE	THREE MONTHS POST DISCHARGE
171	NO	NO	**ISCHEMIC STROKE**	SURVIVOR	4	2
172	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA**	SURVIVOR	2	1
173	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPOGLYCEMIA**	SURVIVOR	0	0
174	NO	NO	**DIABETIC KETOACIDOSIS/METABOLIC ENCEPHALOPATHY**	SURVIVOR	0	0
175	NO	YES	**HEMORRHAGIC STROKE/HYPERTENSION/CHRONIC KIDNEY DISEASE**	NON SURVIVOR	NIL	NIL
176	NO	YES	**DIABETIC KETOACIDOSIS/METABOLIC ENCEPHALOPATHY/SEVERE METABOLIC ACIDOSIS**	SURVIVOR	2	1
177	NO	YES	**CEREBRAL VENOUS THROMBOSIS/STROKE**	SURVIVOR	2	0
178	NO	NO	**UREMIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY/**CHRONIC KIDNEY DISEASE	SURVIVOR	2	1
179	NO	NO	**ISCHEMIC STROKE/DIABETES MELLITUS/**HYPERTENSION/RECURRENT CVA	SURVIVOR	4	3
180	NO	NO	**ISCHEMIC STROKE WITH MIDLINE SHIFT/**DIABEETES MELLITUS/HYPERTENSION	NON SURVIVOR	NIL	NIL
181	NO	NO	**UREMIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY/CHRONIC KIDNEY DISEASE**	SURVIVOR	2	1
182	NO	YES	**CARDIOGENIC SHOCK/**PULMONARY EDEMA/ **POST CARDIAC ARREST**	NON SURVIVOR	NIL	NIL
183	NO	YES	**ISCHEMIC STROKE WITH MIDLINE SHIFT/** YOUNG STROKE	SURVIVOR	4	3
184	NO	NO	**HEPATIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY**	SURVIVOR	2	2
185	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/**SIADH/CONGESTIVE CARDIAC FAILURE	SURVIVOR	2	2
186	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION	SURVIVOR	4	2
187	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION	SURVIVOR	4	3
188	NO	NO	**ISCHEMIC STROKE/** DIABETES MELLITUS/ HYPERTENSION	SURVIVOR	4	4
189	NO	NO	**ISCHEMIC STROKE/** BROCA'S APHASIA	SURVIVOR	2	2
190	NO	NO	**HEMORRHAGIC STROKE/** ACCELERATED HYPERTENSION	NON SURVIVOR	NIL	NIL
191	NO	YES	**HEMORRHAGIC STROKE WITH MIDLINE SHIFT/** DIABETES MELLITUS	SURVIVOR	4	3
192	NO	NO	**HYPOGLYCEMIA/ METABOLIC ENCEPHALOPATHY**	SURVIVOR	0	0
193	NO	NO	**HYPONATREMIA/ METABOLIC ENCEPHALOPATHY/** ACUTE GASTROENTERITIS	SURVIVOR	0	0
194	NO	NO	**HYPONATREMIA/** CONGESTIVE CARDIAC FAILURE/ **METABOLIC ENCEPHALOPATHY**	SURVIVOR	1	0
195	NO	NO	**HEMORRHAGIC STROKE/ ACCELERATED HYPERTENSION/** CHRONIC KIDNEY DISEASE	NON SURVIVOR	NIL	NIL
196	NO	NO	ACUTE MYOCARDIAL INFARCTION/ **POST CARDIAC ARREST/ HYPOXIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
197	NO	NO	**HEPATIC ENCEPHALOPATHY/ RAT KILLER PASTE POISONING (YELLOW PHOSPHORUS)**	SURVIVOR	2	1
198	NO	NO	**COVID PNEUMONIA/ HYPONATREMIA/ METABOLIC ENCEPHALOPATHY/** CKD	SURVIVOR	2	1
199	NO	NO	**COVID PNEUMONIA/ UREMIC ENCEPHALOPATHY/** CHRONIC KIDNEY DISEASE/ ASPIRATION PNEUM	NON SURVIVOR	NIL	NIL
200	NO	NO	**COVID PNEUMONIA/ HYPOXIC ENCEPHALOPATHY/** HYPERTENSION	NON SURVIVOR	NIL	NIL
201	NO	NO	**COVID PNEUMONIA/ ISCHEMIC STROKE/** DIABETES MELLITUS	SURVIVOR	4	2
202	NO	NO	**COVID PNEUMONIA/ ISCHEMIC STROKE/** BROCA'S APHASIA	SURVIVOR	3	2
203	NO	NO	**COVID PNEUMONIA/ CEREBRAL VENOUS THROMBOSIS/ STROKE**	SURVIVOR	2	1

	PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
					AT DISCHARGE	THREE MONTHS POST DISCHARGE
171	NO	NO	**ISCHEMIC STROKE**	SURVIVOR	4	2
172	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA**	SURVIVOR	2	1
173	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPOGLYCEMIA**	SURVIVOR	0	0
174	NO	NO	**DIABETIC KETOACIDOSIS/METABOLIC ENCEPHALOPATHY**	SURVIVOR	0	0
175	NO	YES	**HEMORRHAGIC STROKE/HYPERTENSION/CHRONIC KIDNEY DISEASE**	NON SURVIVOR	NIL	NIL
176	NO	YES	**DIABETIC KETOACIDOSIS/METABOLIC ENCEPHALOPATHY/SEVERE METABOLIC ACIDOSIS**	SURVIVOR	2	1
177	NO	YES	**CEREBRAL VENOUS THROMBOSIS/STROKE**	SURVIVOR	2	0
178	NO	NO	**UREMIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY/**CHRONIC KIDNEY DISEASE	SURVIVOR	2	1
179	NO	NO	**ISCHEMIC STROKE/DIABETES MELLITUS/**HYPERTENSION/RECURRENT CVA	SURVIVOR	4	3
180	NO	NO	**ISCHEMIC STROKE WITH MIDLINE SHIFT/**DIABETES MELLITUS/HYPERTENSION	NON SURVIVOR	NIL	NIL
181	NO	NO	**UREMIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY/CHRONIC KIDNEY DISEASE**	SURVIVOR	2	1
182	NO	YES	**CARDIOGENIC SHOCK/**PULMONARY EDEMA/ **POST CARDIAC ARREST**	NON SURVIVOR	NIL	NIL
183	NO	YES	**ISCHEMIC STROKE WITH MIDLINE SHIFT/** YOUNG STROKE	SURVIVOR	4	3
184	NO	NO	**HEPATIC ENCEPHALOPATHY/METABOLIC ENCEPHALOPATHY**	SURVIVOR	2	2
185	NO	NO	**METABOLIC ENCEPHALOPATHY/HYPONATREMIA/**SIADH/CONGESTIVE CARDIAC FAILURE	SURVIVOR	2	2
186	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION	SURVIVOR	4	2
187	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION	SURVIVOR	4	3
188	NO	NO	**ISCHEMIC STROKE/** DIABETES MELLITUS/ HYPERTENSION	SURVIVOR	4	4
189	NO	NO	**ISCHEMIC STROKE/** BROCA'S APHASIA	SURVIVOR	2	2
190	NO	NO	**HEMORRHAGIC STROKE/** ACCELERATED HYPERTENSION	NON SURVIVOR	NIL	NIL
191	NO	YES	**HEMORRHAGIC STROKE WITH MIDLINE SHIFT/** DIABETES MELLITUS	SURVIVOR	4	3
192	NO	NO	**HYPOGLYCEMIA/ METABOLIC ENCEPHALOPATHY**	SURVIVOR	0	0
193	NO	NO	**HYPONATREMIA/ METABOLIC ENCEPHALOPATHY/** ACUTE GASTROENTERITIS	SURVIVOR	0	0
194	NO	NO	**HYPONATREMIA/** CONGESTIVE CARDIAC FAILURE/ **METABOLIC ENCEPHALOPATHY**	SURVIVOR	1	0
195	NO	NO	**HEMORRHAGIC STROKE/ ACCELERATED HYPERTENSION/** CHRONIC KIDNEY DISEASE	NON SURVIVOR	NIL	NIL
196	NO	NO	ACUTE MYOCARDIAL INFARCTION/ **POST CARDIAC ARREST/ HYPOXIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
197	NO	NO	**HEPATIC ENCEPHALOPATHY/** RAT KILLER PASTE POISONING **(YELLOW PHOSPHORUS)**	SURVIVOR	2	1
198	NO	NO	**COVID PNEUMONIA/ HYPONATREMIA/ METABOLIC ENCEPHALOPATHY/** CKD	SURVIVOR	2	1
199	NO	NO	**COVID PNEUMONIA/ UREMIC ENCEPHALOPATHY/** CHRONIC KIDNEY DISEASE/ ASPIRATION PNEUM	NON SURVIVOR	NIL	NIL
200	NO	NO	**COVID PNEUMONIA/ HYPOXIC ENCEPHALOPATHY/** HYPERTENSION	NON SURVIVOR	NIL	NIL
201	NO	NO	**COVID PNEUMONIA/ ISCHEMIC STROKE/** DIABETES MELLITUS	SURVIVOR	4	2
202	NO	NO	**COVID PNEUMONIA/ ISCHEMIC STROKE/** BROCA'S APHASIA	SURVIVOR	3	2
203	NO	NO	**COVID PNEUMONIA/ CEREBRAL VENOUS THROMBOSIS/ STROKE**	SURVIVOR	2	1

MASTER CHART - MD THESIS

	Serial NO	NAME	SEX	AGE	GLASGOW COMA SCALE							
					AT ADMISSION				24 HOURS AFTER ADMISSION			
					EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE	EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE
204	199	PATIENT 199	MALE	61	3	3	5	11	3	3	5	11
205	200	PATIENT 200	MALE	65	3	3	5	11	3	3	5	11
206	201	PATIENT 201	MALE	45	3	3	5	11	2	NOT TESTABLE	2	5
207	202	PATIENT 202	FEMALI	66	3	3	5	11	2	2	4	8
208	203	PATIENT 203	FEMALI	56	2	NOT TESTABLE	4	7	2	NOT TESTABLE	4	7
209	204	PATIENT 204	FEMALI	65	3	3	5	11	3	3	5	11
210	205	PATIENT 205	MALE	49	3	3	5	11	3	3	5	11
211	206	PATIENT 206	MALE	77	3	3	5	11	3	3	5	11
212	207	PATIENT 207	MALE	67	3	3	5	11	2	NOT TESTABLE	2	5
213	208	PATIENT 208	FEMALI	53	3	3	5	11	2	2	4	8
214	209	PATIENT 209	FEMALI	87	3	3	5	11	3	3	5	11
215	210	PATIENT 210	FEMALI	77	3	3	5	11	3	3	5	11
216	211	PATIENT 211	MALE	66	3	3	5	11	3	3	5	11
217	212	PATIENT 212	MALE	55	3	3	5	11	3	2	5	10
218	213	PATIENT 213	FEMALI	66	3	3	5	11	2	2	4	8
219	214	PATIENT 214	MALE	66	3	3	5	11	2	2	4	8
220	215	PATIENT 215	MALE	44	3	3	5	11	2	2	4	8
221	216	PATIENT 216	FEMALI	55	3	3	5	11	2	2	4	8
222	217	PATIENT 217	MALE	43	3	3	5	11	2	2	4	8
223	218	PATIENT 218	FEMALI	88	2	NOT TESTABLE	1	4	2	NOT TESTABLE	1	4
224	219	PATIENT 219	MALE	76	2	NOT TESTABLE	1	4	2	NOT TESTABLE	1	4
225	220	PATIENT 220	MALE	66	2	NOT TESTABLE	1	4	2	NOT TESTABLE	1	4
226	221	PATIENT 221	MALE	56	3	3	5	11	3	3	5	11
227	222	PATIENT 222	MALE	66	3	3	5	11	3	3	5	11
228	223	PATIENT 223	MALE	44	3	1	6	10	4	1	6	11
229	224	PATIENT 224	MALE	45	3	3	5	11	3	3	5	11
230	225	PATIENT 225	MALE	62	2	NOT TESTABLE	5	8	2	NOT TESTABLE	5	8
231	226	PATIENT 226	FEMALI	78	3	3	5	11	3	3	5	11
232	227	PATIENT 227	FEMALI	61	3	3	5	11	4	1	6	11
233	228	PATIENT 228	FEMALI	70	3	3	5	11	3	3	5	11
234	229	PATIENT 229	MALE	77	3	3	5	11	3	3	5	11
235	230	PATIENT 230	MALE	34	3	3	5	11	3	3	5	11
236	231	PATIENT 231	MALE	47	3	3	5	11	2	NOT TESTABLE	2	5

	FOUR SCORE										CT BRAIN
	AT ADMISSION					24 HOURS AFTER ADMISSION					
	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEXES	RESPIRATIOI	TOTAL SCOF	EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEX	RESPIRATIOI	TOTAL SCORE	
204	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR HEMOFF
205	2	3	4	4	13	2	3	4	4	13	NORMAL
206	2	3	4	4	11	1	1	2	1	5	NORMAL
207	2	3	4	4	13	1	2	3	2	8	NORMAL
208	2	2	4	1	9	2	2	4	1	9	SIGMOID SINUS THROMBOSIS
209	2	3	4	4	13	2	3	4	4	13	SUPERIOR SAGITTAL SINUS THROME
210	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR HEMOFF
211	2	3	4	4	13	2	3	4	4	13	NORMAL
212	2	3	4	4	11	1	1	2	1	5	NORMAL
213	2	3	4	4	13	1	2	3	2	8	NORMAL
214	2	3	4	4	13	2	3	4	4	13	NORMAL
215	2	3	4	4	13	2	3	4	4	13	NORMAL
216	2	3	4	4	13	2	3	4	4	13	NORMAL
217	2	3	4	4	13	2	3	3	3	11	NORMAL
218	2	2	4	3	11	1	2	3	2	8	RIGHT GANGLIOCAPSULAR INFARCT
219	2	2	4	3	11	1	2	3	2	8	INTRACRANIAL HEMORRHAGE
220	2	2	4	3	11	1	2	3	2	8	HYDROCEPHALUS
221	2	2	4	3	11	1	2	3	2	8	NORMAL
222	2	2	4	3	11	1	2	3	2	8	NORMAL
223	1	0	2	0	3	1	0	2	0	3	INTRAVENTRICULAR HEMORRHAGE
224	1	0	2	0	3	1	0	2	0	3	BRAINSTEM HEMORRHAGE
225	1	0	2	0	3	1	0	2	0	3	BRAINSTEM HEMORRHAGE
226	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR HEMOFF
227	2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT
228	2	4	4	4	14	4	4	4	4	16	LEFT FRONTAL LOBE INFARCT
229	2	3	4	4	13	2	3	4	4	13	NORMAL
230	2	3	3	1	9	2	3	3	1	9	NORMAL
231	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARCT
232	2	3	4	4	13	4	4	4	4	16	LEFT FRONTAL LOBE INFARCT
233	2	3	4	4	13	2	3	4	4	13	SUPERIOR SAGITTAL SINUS THROME
234	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR HEMOFF
235	2	3	4	4	13	2	3	4	4	13	NORMAL
236	2	3	4	4	11	1	1	2	1	5	NORMAL

	PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
					AT DISCHARGE	THREE MONTHS POST DISCHARGE
204	NO	NO	COVID PNEUMONIA/ HEMORRHAGIC STROKE/ HYPERTENSION	SURVIVOR	4	2
205	NO	NO	COVID PNEUMONIA/ HYPONATREMIA/ METABOLIC ENCEPHALOPATHY	SURVIVOR	2	0
206	NO	NO	COVID PNEUMONIA/ ACUTE MYOCARDIAL INFARCTION/ HYPOXIC ENCEPHALOPATHY	NON SURVIVOR	NIL	NIL
207	NO	NO	COVID PNEUMONIA/ HYPOXIC ENCEPHALOPATHY/ DIABETES MELLITUS	NON SURVIVOR	NIL	NIL
208	NO	YES	COVID PNEUMONIA/ CEREBRAL VENOUS THROMBOSIS/ STROKE	SURVIVOR	2	0
209	NO	NO	COVID PNEUMONIA/ CEREBRAL VENOUS THROMBOSIS/ STROKE	SURVIVOR	2	0
210	NO	NO	COVID PNEUMONIA/ HEMORRHAGIC STROKE/ HYPERTENSION	SURVIVOR	4	2
211	NO	NO	COVID PNEUMONIA/ HYPONATREMIA/ METABOLIC ENCEPHALOPATHY	SURVIVOR	2	0
212	NO	NO	COVID PNEUMONIA/ ACUTE MYOCARDIAL INFARCTION/ HYPOXIC ENCEPHALOPATHY	NON SURVIVOR	NIL	NIL
213	NO	NO	COVID PNEUMONIA/ HYPOXIC ENCEPHALOPATHY/ DIABETES MELLITUS	NON SURVIVOR	NIL	NIL
214	NO	NO	METABOLIC ENCEPHALOPATHY/ HYPONATREMIA/ ACUTE GASTROENTERITIS	SURVIVOR	1	0
215	NO	NO	METABOLIC ENCEPHALOPATHY/ HYPONATREMIA/ ACUTE GASTROENTERITIS	SURVIVOR	1	0
216	NO	NO	ACUTE MENINGOENCEPHALITIS	SURVIVOR	1	0
217	NO	NO	ACUTE MENINGOENCEPHALITIS/ ASPIRATION PNEUMONIA	NON SURVIVOR	NIL	NIL
218	NO	NO	ISCHEMIC STROKE/ ASPIRATION PNEUMONITIS	NON SURVIVOR	NIL	NIL
219	NO	NO	HEMORRHAGIC STROKE/ ASPIRATION PNEUMONITIS	NON SURVIVOR	NIL	NIL
220	NO	NO	TUBERCULAR MENINGITIS/ HYDROCEPHALUS/ ACUTE ENCEPHALOPATHY	NON SURVIVOR	NIL	NIL
221	NO	NO	METABOLIC ENCEPHALOPATHY/ HEPATIC ENCEPHALOPATHY/ NAFLD	NON SURVIVOR	NIL	NIL
222	NO	NO	METABOLIC ENCEPHALOPATHY/ UREMIC ENCEPHALOPATHY/ HYPERTENSIONICKD	NON SURVIVOR	NIL	NIL
223	NO	YES	HEMORRHAGIC STROKE	NON SURVIVOR	NIL	NIL
224	NO	YES	HEMORRHAGIC STROKE/ HYPERTENSION	NON SURVIVOR	NIL	NIL
225	NO	YES	HEMORRHAGIC STROKE/ HYPERTENSION	NON SURVIVOR	NIL	NIL
226	NO	NO	HEMORRHAGIC STROKE/ HYPERTENSION	SURVIVOR	4	2
227	NO	NO	ISCHEMIC STROKE/ DIABETES MELLITUS	SURVIVOR	4	2
228	NO	NO	ISCHEMIC STROKE	SURVIVOR	4	2
229	NO	NO	COVID PNEUMONIA/ UREMIC ENCEPHALOPATHY/ CHRONIC KIDNEY DISEASE/ ASPIRATION PNEUM	NON SURVIVOR	NIL	NIL
230	NO	NO	COVID PNEUMONIA/ HYPOXIC ENCEPHALOPATHY/ HYPERTENSION	NON SURVIVOR	NIL	NIL
231	NO	NO	COVID PNEUMONIA/ ISCHEMIC STROKE/ DIABETES MELLITUS	SURVIVOR	4	2
232	NO	NO	COVID PNEUMONIA/ ISCHEMIC STROKE/ BROCA'S APHASIA	SURVIVOR	2	1
233	NO	NO	COVID PNEUMONIA/ CEREBRAL VENOUS THROMBOSIS/ STROKE	SURVIVOR	2	1
234	NO	NO	COVID PNEUMONIA/ HEMORRHAGIC STROKE/ HYPERTENSION	SURVIVOR	4	2
235	NO	NO	COVID PNEUMONIA/ HYPONATREMIA/ METABOLIC ENCEPHALOPATHY	SURVIVOR	2	1
236	NO	NO	COVID PNEUMONIA/ ACUTE MYOCARDIAL INFARCTION/ HYPOXIC ENCEPHALOPATHY	NON SURVIVOR	NIL	NIL

1	MASTER CHART - MD THESIS												
2													
3	Serial NO	NAME	SEX	AGE	GLASGOV COMA SCALE								
4					AT ADMISSION				24 HOURS AFTER ADMISSION				
5					EYE RESPONSE	VERBAL RESPONSE	MOTOR RESPONSE	TOTAL SCORE	EYE RESPONSE	VERBAL RESPONSI	MOTOR RESPONSE	TOTAL SCORE	
236	231	PATIENT 231	MALE	47	3	3	5	11	2	NOT TESTABLE	2	5	
237	232	PATIENT 232	FEMAL	76	3	3	5	11	2	2	4	8	
238	233	PATIENT 233	FEMAL	42	2	NOT TESTABLE	4	7	2	NOT TESTABLE	4	7	
239	234	PATIENT 234	FEMAL	52	4	NOT TESTABLE	1	6	4	NOT TESTABLE	1	6	
240	235	PATIENT 235	MALE	67	4	NOT TESTABLE	1	6	4	NOT TESTABLE	1	6	
241	236	PATIENT 236	FEMAL	58	3	1	6	10	3	1	6	10	
242	237	PATIENT 237	FEMAL	61	3	3	5	11	3	3	5	11	
243	238	PATIENT 238	MALE	84	3	3	5	11	3	3	5	11	
244	239	PATIENT 239	MALE	67	3	3	5	11	3	3	5	11	
245	240	PATIENT 240	FEMAL	87	3	3	5	11	3	3	5	11	
246	241	PATIENT 241	FEMAL	78	3	1	6	10	4	1	6	11	
247	242	PATIENT 242	MALE	67	3	3	5	11	3	3	5	11	
248	243	PATIENT 243	FEMAL	65	3	3	5	11	2	2	4	8	
249	244	PATIENT 244	MALE	59	3	3	5	11	2	2	4	8	
250	245	PATIENT 245	MALE	80	3	4	5	12	4	4	6	14	
251	246	PATIENT 246	FEMAL	34	3	4	5	12	3	3	4	10	
252	247	PATIENT 247	MALE	31	3	4	5	12	3	3	4	10	
253	248	PATIENT 248	FEMAL	45	3	4	5	12	3	3	4	10	
254	249	PATIENT 249	MALE	58	3	3	5	11	3	3	5	11	
255	250	PATIENT 250	FEMAL	66	3	3	5	11	3	3	5	11	
256													
257													
258													

1													
2													
3	FOUR SCORE												CT BRAIN
4	AT ADMISSION					24 HOURS AFTER ADMISSION							
5	EYE RESPONSI	MOTOR RESPONSI	BRAINSTEM REFLEXES	RESPIRATIO	TOTAL SCOR	EYE RESPONSE	MOTOR RESPONSI	BRAINSTEM REFLE	RESPIRATIO	TOTAL SCORE			
206	2	3	4	4	11	1	1	2	1	5	NORMAL		
207	2	3	4	4	13	1	2	3	2	8	NORMAL		
208	2	2	4	1	9	2	2	4	1	9	SIGMOID SINUS THROMBOSIS		
209	4	0	4	1	9	4	0	4	1	9	PONTINE INFARCT		
240	4	0	4	1	9	4	0	4	1	9	PONTINE INFARCT		
241	2	4	4	4	14	2	4	4	4	14	LEFT FRONTAL LOBE HEMORRHAG		
242	2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT		
243	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARC		
244	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARC		
245	2	3	4	4	13	2	3	4	4	13	INTRACRANIAL HEMORRHAGE		
246	2	4	4	4	14	4	4	4	4	16	INTRACRANIAL HEMORRHAGE		
247	2	3	4	4	13	2	3	4	4	13	RIGHT GANGLIOCAPSULAR INFARC		
248	2	2	4	3	11	1	2	3	2	8	INFARCT WITH HEMORRHAGIC TRA		
249	2	2	4	3	11	1	2	3	2	8	INTRACRANIAL AND INTRAVENTRIC		
250	2	3	4	4	13	4	4	4	4	16	LEFT TEMPOROPARIETAL INFARCT		
251	2	3	4	4	13	2	2	4	3	11	NORMAL		
252	2	3	4	4	13	2	2	4	3	11	NORMAL		
253	2	3	4	4	13	2	2	4	3	11	NORMAL		
254	2	3	4	4	13	2	3	4	4	13	LEFT FRONTAL HEMORRHAGE		
255	2	3	4	4	13	2	3	4	4	13	LEFT GANGLIOCAPSULAR INFARCT		
256													
257													

	PRESENCE OF POLY TRAUMA	INTUBATION DONE	LIKELY DIAGNOSIS	OUTCOME	MODIFIED RANKIN SCALE - NEUROLOGICAL OUTCOME	
					AT DISCHARGE	THREE MONTHS POST DISCHARGE
236	NO	NO	**COVID PNEUMONIA/ ACUTE MYOCARDIAL INFARCTION/ HYPOXIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
237	NO	NO	**COVID PNEUMONIA/ HYPOXIC ENCEPHALOPATHY/ DIABETES MELLITUS**	NON SURVIVOR	NIL	NIL
238	NO	YES	**COVID PNEUMONIA/ CEREBRAL VENOUS THROMBOSIS/ STROKE**	SURVIVOR	2	1
239	NO	YES	**PONTINE INFARCT/** LOCKED IN SYNDROME/SHTN	SURVIVOR	4	4
240	NO	YES	**PONTINE INFARCT/** LOCKED IN SYNDROME/SHTN	SURVIVOR	5	4
241	NO	NO	**HEMORRHAGIC STROKE/** HYPERTENSION	SURVIVOR	5	6
242	NO	NO	**ISCHEMIC STROKE**	SURVIVOR	5	3
243	NO	NO	**ISCHEMIC STROKE/**DIABETES MELLITUS	SURVIVOR	5	3
244	NO	NO	**ISCHEMIC STROKE/**HYPERTENSION	SURVIVOR	4	2
245	NO	NO	**HEMORRHAGIC STROKE/**DIABETES MELLITUS	SURVIVOR	4	4
246	NO	NO	**HEMORRHAGIC STROKE/**BROCA'S APHASIA/HYPERTENSION	SURVIVOR	3	1
247	NO	NO	**ISCHEMIC STROKE/** HYPERTENSION	SURVIVOR	4	2
248	NO	NO	**ISCHEMIC STROKE WITH HEMORRHAGIC TRANSFORMATION/**HYPERTENSION	NON SURVIVOR	NIL	NIL
249	NO	NO	**HEMORRHAGIC STROKE/** CHRONIC KIDNEY DISEASE/HYPERTENSION	NON SURVIVOR	NIL	NIL
250	NO	NO	**ISCHEMIC STROKE/** VERNICKE'S APHASIA/DIABETES MELLITUS	SURVIVOR	3	2
251	NO	NO	**PARAQUAT POISONING/** ARDS/ MODS/SEPSIS/**SEPTIC ENCEPHALOPATHY**	NON SURVIVOR	NIL	NIL
252	NO	NO	**PARAQUAT POISONING/** ARDS/RESPIRATORY FAILURE	NON SURVIVOR	NIL	NIL
253	NO	NO	**PARAQUAT POISONING/** ARDS/PNEUMOMEDIASTINUM/RESPIRATORY FAILURE	NON SURVIVOR	NIL	NIL
254	NO	NO	**HEMORRHAGIC STROKE/** ACCELERATED HYPERTENSION	SURVIVOR	4	6
255	NO	NO	**ISCHEMIC STROKE/** DIABETES MELLITUS/HYPERTENSION	SURVIVOR	4	2
256						
257						